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EVIDENCE REVIEW 232

ISCRR
Institute for Safety, Compensation and Recovery Research

Bitumen contents and fumes

Evidence Review

A review of health risks associated with exposure to bitumen contents and fumes.

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A joint initiative of WorkSafe VICTORIA and MONASH University

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Disclaimer

EXECUTIVE SUMMARY

Background and scope

Bitumen is used as a binder in the production of asphalt for road surfaces and the material is heated to facilitate spreading. During this process, a complex mixture of vapours and particulate matter is emitted. Following several complaints about 'fuming' bitumen loads, WorkSafe asked ISCRR to examine the published evidence to identify risk factors associated with bitumen exposure; and to identify strategies to minimise the health risks to suppliers and roadside workers.

Method

A synthesis of the evidence of adverse health effects and risk mitigation factors associated with bitumen exposure was undertaken in October–December 2018.

Key findings

This review examined the evidence from four systematic reviews and eight primary studies to evaluate the acute or chronic health effects of bitumen exposure amongst roadside workers. Evidence from three primary studies were assessed to determine the effectiveness of strategies to mitigate the health risks associated with bitumen exposure.

Overall, evidence of an association between exposure to bitumen contents and fumes and reported adverse health outcomes was mixed.

Acute health effects

- 3/5 studies reported a statistically significant decline in lung function and increased respiratory symptoms in bitumen-exposed workers compared with non-exposed workers
- Limited evidence supported an association between bitumen exposure and eye and nose irritations

Chronic health effects

- A weak association was reported between bitumen exposure and development of some cancers
- Limited evidence showed significantly higher proportions of bitumen-exposed workers with subclinical tissue injury in liver, kidney and airways (inflammatory markers) and DNA damage compared with non-exposed workers
- Adverse health outcomes were worse for smokers and significantly higher amongst screedmen (who level the asphalt)

Protective factors

- Limited evidence supported use of personal protective equipment, air-purifying respiratory equipment, and substitution of biodiesel for cleaning equipment to reduce risk of adverse health effects due to exposure
- One evaluation study showed that a design-integrated suction slot located at the conveyor outlet in the paving machine was more efficient for capturing fumes compared with other models; thereby reducing risks of adverse health effects associated with bitumen exposure.

INTRODUCTION

Victoria is currently in the midst of long-term major road repair, construction and infrastructure works. In recent months, concerns have been raised about the composition, nature and risk profile of various bitumen loads that have been supplied to various road infrastructure worksites around Victoria.

Asphalt for road paving is manufactured by mixing heated, dry gravel with 4-5% hot bitumen, a petroleum-derived binding agent, to enable the asphalt to readily flow and spread onto the road surface.[1] While ambient temperature bitumen is non-volatile, heated bitumen produces a complex mixture of vapours, aerosols, gases and particulate matter, collectively termed 'emissions'.

WorkSafe is aware of, and has responded to, a number of complaints and service requests involving 'fuming loads'. As the temperature of the load is elevated the fuming phenomenon (which looks similar to steam) may occur on some occasions. Employees have reported suffering acute health affects (such as dizziness, nausea and respiratory discomfort), allegedly as a result of exposure to fuming loads or contact with bitumen products.

Emissions from the bitumen loads may include various levels of polycyclic aromatic hydrocarbons (PAHs), aliphatic compounds, cyclic alkanes, and heterocyclic compounds containing oxygen, nitrogen, and sulphur atoms. Some PAHs (e.g., benzo[*a*]pyrene) have been classified as *'possibly carcinogenic to humans'*.[1]

WorkSafe Inspectors have made a number of visits to various road construction workplaces and bitumen suppliers. Enquiries have been made into the nature of the bitumen including risk mitigation measures to ensure the health and safety of workers spreading the bitumen.

The Inspector's enquiries indicate that there are variations within the composition of a number of bitumen loads, including anecdotal reports of loads being supplemented with recycled materials such as printer cartridges.

One of the key objectives of WorkSafe 2030 is to take a 'prevention-led' approach. A durable and extensive research project into the bitumen fuming load issue is expected to deliver a number of prevention initiatives to the industry. This may include practical information and guidance for workers directly affected by, or required to work with, these substances; and broader industry guidance to better understand the risks associated with heated bitumen products.

In the longer term, effective implementation of the results of this research is expected to lead to a decrease in claims, injuries or illnesses arising from exposure to bitumen products.

OBJECTIVES

The primary objective of this review was to identify risk factors associated with bitumen exposure, particularly those associated with the application of heated bitumen at roadside workplaces.

A secondary objective included identification of risk mitigation measures and related controls to minimise risks to suppliers and workers, and potentially provide a foundation for an industry state of knowledge to assist duty holders to meet their obligations under the OHS legislation.

This Evidence Review is complemented by findings from an ISCRR Environmental Scan of the bitumen industry and relevant government agencies. The Environmental Scan was undertaken to identify the level of awareness of the risks of bitumen exposure and protective measures currently in place to mitigate those risks in the Australian setting. In the longer term, this research may have the potential to form the basis of industry guidance on this matter.

Research questions

1. What health risk factors have been associated with exposure to bitumen contents and fumes in the course of road construction or repairs?
2. What factors are shown to mitigate the risk of, or protect workers from, adverse health effects associated with exposure to bitumen contents and fumes during road construction and repairs?

METHODS

This Evidence Review involved a systematic search of the scientific literature to identify studies that: 1) assessed the health impacts of workers exposed to bitumen contents and fumes in the course of road construction; and/or 2) identified potential protective factors for bitumen-exposed workers. The review was undertaken between October and December 2018, according to the criteria listed in Table 1.

Table 1. Details of the literature search and sources

Population	Studies were included for review if they described health outcomes for workers exposed to bitumen contents and fumes in the course of road construction or repairs. Studies were excluded if they involved bitumen-exposed workers in other industries, such as roofing as asphalt is not used in roofing in Australia.
Intervention	Studies were included if they evaluated use of any personal protection equipment or other practices to limit or reduce exposure to bitumen. Studies that described protective strategies, without evaluating them, were excluded.
Study designs	Systematic reviews and controlled primary studies were included for review. Qualitative studies and quantitative studies without appropriate controls or non-exposed reference group were excluded.
Outcomes	Primary outcomes were any acute adverse health outcome, including headaches, eye or nasal irritation, respiratory discomfort; or chronic illness, including respiratory disorders or any cancers. Secondary outcomes included measures of exposure to bitumen contents and fumes.
Sources	Evidence sources for this review included any systematic literature reviews, meta-analyses or controlled studies identified in academic databases (PubMed, EMBASE, CINAHL, PsychInfo, Cochrane library), and relevant journals (Journal of occupational and environmental hygiene; Occupational and environmental medicine) and published between 2009 and 2018
Search terms	Combinations of the following terms and synonyms were used to search the literature databases: 1. asphalt* OR bitumen* 2. exposure OR emission* OR fume* 3. health OR risk* OR safety OR prevent* OR protect* OR mitigate

Figure 1 illustrates the search process. Citations were downloaded and combined in an Endnote library and duplicates were removed. One reviewer screened citations by title and abstract to remove those not in scope; and the full-text was obtained for remaining articles. Relevant journals and bibliographies of included articles were searched to identify articles not included in the initial searches. Two reviewers critically appraised the included articles using AMSTAR2 for systematic reviews[2] and the Effective Public Health Practice tool for primary studies.[3]

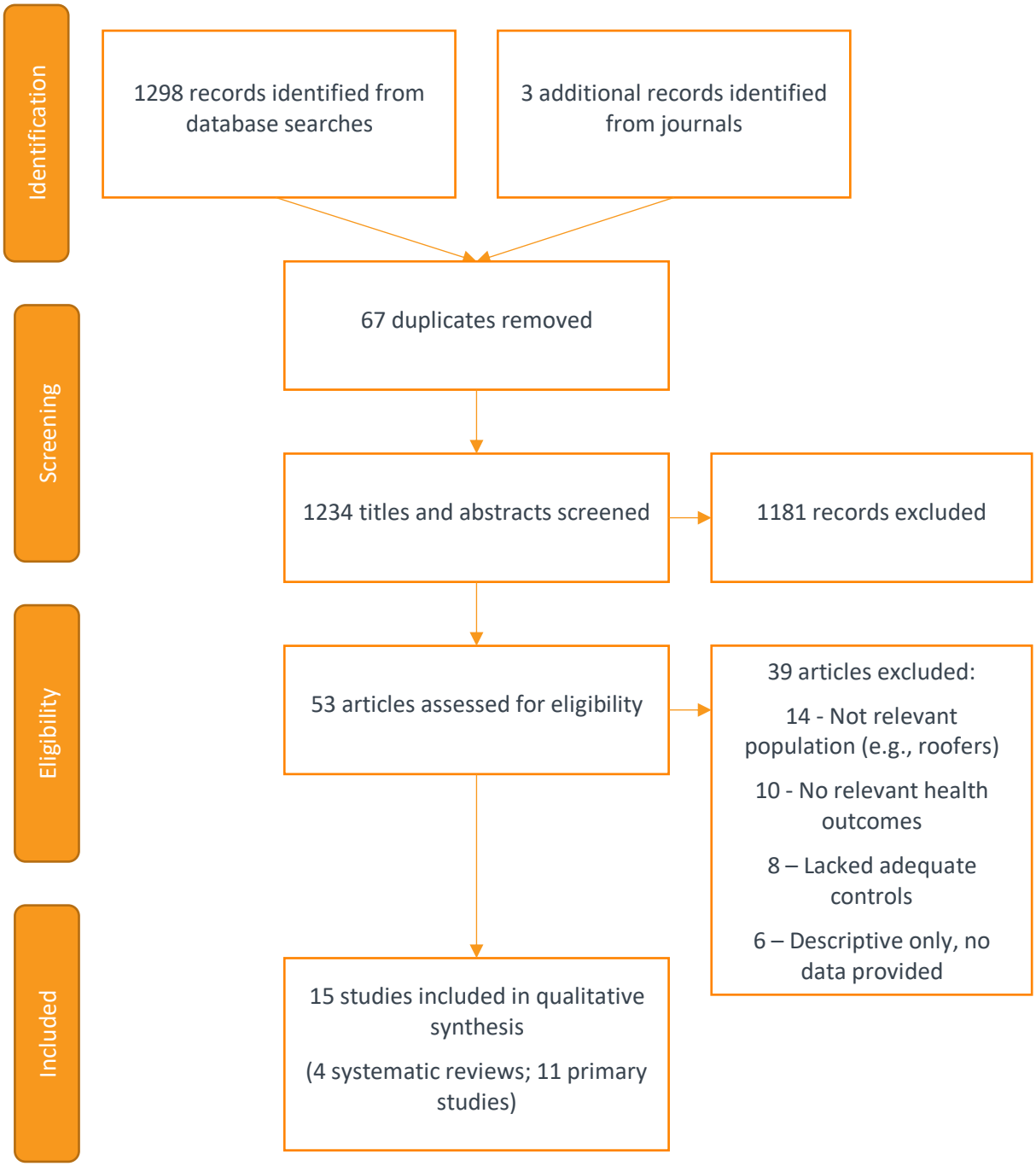

Fig 1. PRISMA diagram shows study selection process

QUESTION 1: WHAT HEALTH RISK FACTORS HAVE BEEN ASSOCIATED WITH EXPOSURE TO BITUMEN CONTENTS AND FUMES IN THE COURSE OF ROAD CONSTRUCTION AND/OR REPAIRS?

Key findings

Acute health outcomes

- **Lung function & respiratory symptoms:** Three (of 5) studies reported statistically significant reductions in lung function in bitumen-exposed workers compared with non-exposed workers, based on objective spirometry testing (controlled for smoking). The decline in function was significantly worse for smokers and higher amongst screedmen (who level the asphalt) compared with other paving workers. A significantly higher proportion of exposed workers also reported subjective respiratory symptoms (cough, wheezing, phlegm, shortness of breath and chest tightness).
- **Inflammatory response:** Levels of inflammatory markers (IL-8, IL-6, total protein and MMP-9), which represent an inflammatory response in injured tissues, were significantly higher in bitumen-exposed workers, both pre- and post-shift, compared with non-exposed workers; and the levels were higher in smokers in both groups.
- **Eye & nose irritation:** A significantly higher proportion of bitumen-exposed workers reported eye irritations, but there was no significant difference in nasal irritations between groups.

Chronic health outcomes

- **Cancers:** Overall the association between bitumen exposure and development of cancers was weak and varied across studies and with different types of cancer. Two systematic reviews reported no statistically significant excess risk of developing lung, lymphatic or haematopoietic cancers. Similarly, the evidence for laryngeal cancer was weak. One systematic review reported a significantly higher risk of developing upper aerodigestive tract (oral cavity to hypopharynx) cancers or stomach cancer in bitumen-exposed workers; however, poor methodology and failure to adjust for confounding factors limits the reliability of these findings.
- **Early genotoxic effects:** Three studies reported significantly higher levels of urinary metabolites of PAHs in bitumen-exposed workers; and significant correlations with early markers of DNA damage (DNA adducts, strand breaks).
- **Liver and kidney function:** In one study, significantly higher levels of several kidney and liver enzymes were detected in bitumen-exposed workers. Although all measures were within the normal range, elevated levels may represent early signs of organ dysfunction over the longer term.

Detailed findings – adverse health effects

Study characteristics – adverse health effects

This review identified four systematic literature reviews and nine primary studies that examined the relationship between occupational exposure to bitumen contents or emissions and acute or chronic adverse health outcomes.

All four systematic reviews evaluated the risks of developing various cancers in workers exposed to PAHs. One review specifically investigated bitumen exposure in road and roofing workers[4]. Three reviews[5-7] explored the risks of developing cancer in a broad range of workers exposed to PAHs, including asphalt workers. Table 2 shows the characteristics and key outcomes of the systematic reviews.

The nine included primary studies were undertaken in Iran,[8, 9] Germany,[10, 11] Norway,[12, 13], Turkey,[14] India[15] and Sweden.[16] The characteristics and findings of the primary studies are shown in Tables 3 and 4, respectively.

Findings are based on weak to moderate quality evidence; therefore, conclusions should be considered in the context of limited data and high variability across study settings, measures and analyses.

Table 2. Study characteristics and outcomes of systematic reviews – adverse health risks

Reference Country	N Databases searched	N primary studies on asphalt workers (total studies)	Bitumen contents and fumes exposure	Key outcomes	Quality rating**
Alicandro 2016[5] Italy	2	3 (of 41)	PAHs in asphalt paving workers	**Lymphatic & haematopoietic neoplasms** • NS excess risk compared with reference group	High
Mundt 2018[4] USA	1*	22 (of 56)	Bitumen exposure in paving workers	**Lung cancer** • NS excess risk compared with reference group (8 studies) **Upper aerodigestive tract cancers** • Significantly increased risk in bitumen-exposed workers (10 studies); MRR 1.31 [95% CI 1.07-1.67], p<0.05 **Stomach cancer** • Significant increased risk in bitumen-exposed workers (7 studies); MRR 1.29 [95% CI 1.03-1.62], p<0.05	High
Rota 2014[6] Italy	4	1 (of 13)	PAHs in asphalt roofing and paving workers	**Laryngeal cancer** • Significant increased mortality in asphalt workers; SMR 3.74 [95% CI 2.21-6.31], p<0.05	Low
Wagner 2015[7] Germany	2	1 (of 63)	PAHs in asphalt paving workers	**Laryngeal cancer** • NS excess risk compared with reference group	High

Notes: * included studies from previous meta-analyses and International Agency for Research on Cancer (IARC) multi-centre cohort studies; ** Based on AMSTAR 2 rating overall confidence in the results of the review: where high quality reviews contained no or one non-critical weakness; moderate reviews contained more than one non-critical weakness; low reviews contained one critical flaw with or without non-critical weaknesses; and critically low reviews contained more than one critical flaw with or without non-critical weaknesses; MRR = meta relative risk; NS = not significant; PAHs = polycyclic aromatic hydrocarbons; SMR = standardised mortality ratio.

Table 3. Primary study characteristics – adverse health risks

Reference Country	Study design (follow-up)	Cohort N, % male	Bitumen contents & fumes exposure assessed	Key outcomes	Quality rating*
Bal 2018[14] Turkey	Cross-sectional study (end of working week)	Asphalt paving workers N=100 (60 bitumen-exposed; 40 non-exposed controls); 100% male	• Urinary metabolites of PAHs	• Oxidative stress and DNA damage	Weak
Marczynski 2011[11] Germany	Cross-sectional study (post-shift)	Asphalt paving workers N=438 (320 bitumen-exposed; 118 non-exposed outdoor construction workers); 100% male	• Personal exposure monitoring to bitumen vapours and aerosols • Urinary metabolites of PAHs • Blood samples	• Oxidative stress and DNA damage	Moderate
Neghab 2015[9] Iran	Cross-sectional study (post-shift)	Asphalt paving workers N=184 (74 bitumen-exposed; 110 government employees); 100% male	• Atmospheric concentration of asphalt fumes (total particulate; benzene soluble fraction)	• Lung function (spirometry) • Respiratory symptoms (coughing, wheezing, shortness of breath) • Nasal symptoms • Eye symptoms	Moderate
Neghab 2017[8] Iran	Cross-sectional study	Asphalt paving workers N=210 (80 bitumen-exposed; 130 government employees); 100% male	• Atmospheric concentration of asphalt fumes (total particulate; benzene soluble fraction)	• Liver function • Kidney function • Blood cell count	Weak
Raulf-Heimsoth 2011[10] Germany	Cross-shift study	Mastic asphalt paving workers N=438 (320 bitumen-exposed; 118 non-exposed	• Atmospheric sampling of bitumen vapours and aerosols, including PAHs	• Lung function (spirometry) • Inflammatory response (nasal lavage fluids; induced sputum)	Moderate

Reference Country	Study design (follow-up)	Cohort N, % male	Bitumen contents & fumes exposure assessed	Key outcomes	Quality rating*
		outdoor construction workers); 100% male	• Personal exposure monitoring to bitumen vapours and aerosols		
Sellappa 2011[15] India	Cross-sectional study	Asphalt paving workers N=73 (36 bitumen-exposed; 37 controls); 100% male	• Urinary metabolite of PAHs (1-OHP) • Blood sample	• Oxidative stress and DNA damage	Moderate
Ulvestad 2007[12] Norway	Cross-sectional study (post-season)	Asphalt paving workers N=266 (140 bitumen-exposed; 126 non-exposed heavy construction workers); 100% male	• Personal exposure monitoring (total dust, PAHs, oil mist)	• Lung function - FVC, FEV_1, FEF_{50} • Inflammatory responses (IL-6, fibrinogen, C-reactive protein)	Moderate
Ulvestad 2017[13] Norway	Longitudinal study (annual testing 2006-2010)	Asphalt paving workers N=146 (75 bitumen-exposed; 71 non-exposed road maintenance workers); 100% male	• Personal exposure monitoring (total dust, PAHs, oil mist/vapour, PAHs) • Atmospheric sampling (ultrafine particulate matter)	• Lung function - FVC, FEV_1, FEF_{50} • Lung cancer (HRCT scan)	Moderate
Xu 2018[16] Sweden	Cross-sectional study (post-shift; post-season)	Asphalt paving workers N=267 (116 conventional asphalt; 51 crumb rubber modified asphalt; 100 non-exposed outdoors workers); 100% male	• Personal exposure monitoring (dust, PAHs, benzothiazole, nitrosamines)	• Lung function (spirometry & self-reported questionnaire) • Inflammatory response	Moderate

Notes: FEF_{50} = forced expiratory flow at 50%; FEV_1 = forced expiration volume in 1 second; FVC = forced vital capacity; HRCT = high-resolution computed tomography; IL-8 = interleukin-8; PAHs = polycyclic aromatic hydrocarbons; * quality ratings assessed using Effective Public Health Practice tool.[3]

Key outcomes – adverse health effects

Bitumen exposure health risk

Evaluating the association between occupational exposure to bitumen contents and fumes and adverse acute or chronic health outcomes is challenging. Variability in exposure risk may be related to the bitumen batch (composition, handling temperature); the process (manual or mechanised application); use of protective equipment; the job class (See Figure 2, Appendix for list of jobs); and the site conditions (weather, confinement).[17] A comprehensive international review of bitumen emissions found that the asphalt composition varied substantially depending on the asphalt supplier and the type of road surface required. *"Consequently, no two bitumen products are chemically identical" (p 40).*[1]

Two main approaches to assessing exposure were described in the literature:

1. **Atmospheric sampling**, which represents the external dose, directly measures PAHs and bitumen particulates using air sampling filters in the personal breathing zone of workers, or nearby.[18] It is difficult to compare exposure across studies as there is high variability in bitumen contents and fumes across sites and conditions, and concentration of PAHs are typically below the standard threshold limits set by the USA ($0.5mg/m^3$)[1].

2. **Biological sampling**, which represents the internal dose, indirectly measures metabolites of PAHs in urine or blood.[11] Biological sampling includes all potential routes of exposure, rather than inhalation alone; and may be a more sensitive measure of exposure compared with environmental data. However, samples included PAHs derived from other sources.

Not surprisingly, the concentrations of atmospheric bitumen fumes / particulates and urinary metabolites of PAHs were significantly higher in bitumen-exposed workers compared with non-exposed workers in all included studies. Statistical analyses were undertaken to determine the correlations between exposure and adverse health outcomes.

Cancer risk

Based on studies that examined the association between bitumen exposure and the development of cancer, two systematic reviews reported no significantly increased risk of developing lung, lymphatic or haematopoietic cancers in workers exposed to bitumen fumes compared with non-exposed workers.[4, 5]

For laryngeal cancers, the evidence was mixed. One study in the Wagner et al.[7] systematic review reported no statistically significant excess risk for asphalt pavers; whereas another study in the Rota et al.[6] systematic review reported significantly increased mortality from laryngeal cancers amongst asphalt workers. However, the study population did not distinguish between asphalt roofers and pavers; and the risks to roofers may be higher.[1]

Mundt et al.[4] reported significantly increased risk of developing stomach cancer (from 7 studies) or upper aerodigestive tract cancers (from 10 studies). However, the authors concluded that the evidence was 'low certainty' due to methodological limitations and inadequate adjustment for important confounders (smoking, coal tar, alcohol exposure) in the studies. Table 2 shows key outcomes from the systematic reviews.

In contrast, three primary studies of asphalt paving workers examined the relationship between exposure to PAHs and biomarkers of DNA damage that may indicate early genetic damage and potential long-term cumulative risk of cancer.[11, 14, 15] PAHs generate free radicals and induce oxidative stress that is known to damage proteins and DNA. DNA adducts are segments of DNA that are bound to a cancer-causing chemical, such as PAHs; and that lead to DNA strand breaks. Increases in DNA adducts and strand breaks represent biological damage and potential mutations that may

represent early carcinogenesis. Similarly, signs of oxidative stress represent an alternative pathway to the formation of DNA adducts and strand breaks.

In two studies,[14, 15] bitumen-exposed workers had significantly higher urinary metabolites of PAHs compared with control groups; and this correlated with significantly higher levels of oxidative stress and DNA damage, irrespective of smoking or alcohol status. In contrast, while Marczynski et al.[11] reported significantly higher oxidative stress in exposed workers, levels of DNA damage were within normal range for both groups; and no statistically significant associations were identified between the magnitude of exposure to PAHs and DNA damage. Table 4 shows key outcomes in the primary studies.

Lung function and respiratory symptoms

Evidence from five studies that investigated the effects of bitumen exposure on lung function in asphalt paving workers was mixed. Compared with a reference group of non-exposed workers, spirometry testing showed a statistically significant decline in lung function amongst asphalt paving workers in three studies;[9, 12, 13] and no significant difference between the groups in two studies.[10, 16] Table 6 in the Appendix provides a glossary of terms used in spirometry testing. Neghab et al.[9] also reported a significant decline in lung function measures during the shift. All studies controlled for smoking and other potential confounders.

Two studies from the same research group stratified outcomes by job tasks.[12, 13] Ulvestad et al.[12] reported significantly higher levels of exposure to PAHs in paver operators, screedmen and roller drivers compared with truck drivers or asphalt strippers ($p<0.001$). Objective measures of lung function were also significantly worse in screedmen compared with all other asphalt workers at the end of the asphalt laying season[12] and at 5 years follow-up.[13] It is possible that screedmen get more exposure to fumes and particulate matter compared with paver operators or drivers due to a higher respiration rate related to their more physically demanding role.

Other self-reported respiratory symptoms (coughing, phlegm, wheezing, shortness of breath and chest tightness) were significantly higher in exposed workers compared with non-exposed controls.[9]

In contrast, Xu et al.[16] reported no statistically significant change in lung function during the shift; and no significant difference in upper or lower airways between groups or during the shift.

Inflammatory status

Inflammatory markers, such as interleukins (IL-6, IL-8), matrix metalloproteinase-9 (MMP-9) and proteins, are produced in epithelial cells in response to tissue injury or irritation. Evidence from two studies reported significantly higher levels of inflammatory markers (IL-8, MMP-9, total protein)[10, 16] in bitumen-exposed workers compared with non-exposed controls. In addition, changes in inflammatory markers in exposed workers may be cumulative. Although Raulf-Heimsoth et al.[10] found no statistically significant change in IL-8, MMP-9 or total protein levels during shifts, Ulvestad et al.[12] reported significant increases in IL-6 levels in exposed workers over the course of the asphalt construction season. Although these biomarkers are known to indicate an inflammatory response in the lower airways, it is not clear whether the inflammation represents an early stage of respiratory disease or whether it is simply a physiological marker of exposure to an irritant, without progressing to a clinical consequence.

Other markers of organ toxicity

Inflamed or damaged liver or kidney cells leak enzymes into the bloodstream; and elevated serum liver or kidney enzymes may be an early sign of organ toxicity. One study assessed levels of blood, liver and kidney biomarkers that may indicate haematotoxic, hepatotoxic or nephrotoxic potential in bitumen-exposed compared with a non-exposed reference group.[8] Compared with non-exposed workers, bitumen-exposed workers had significantly higher levels of liver enzymes (serum albumin, bilirubin, total protein, alanine amino transferase, aspartate amino transferase); and kidney enzyme

(blood urea nitrogen). They also had significantly lower levels of alkaline phosphatase and calcium. Although all liver and kidney enzymes were within normal range, the authors suggested that the differences may indicate early, non-pathologic liver or kidney dysfunction.

Similarly, blood analyses showed significantly lower levels of white blood cells in bitumen-exposed workers compared with non-exposed reference group.

Other irritative symptoms

A significantly higher proportion of exposed workers (23%) reported eye irritations compared with non-exposed workers (10%) in one study;[16] but there was no statistically significant difference in other measures of irritation (nose, airways).

Table 4. Primary studies results – adverse health effects

Reference	Key outcomes of bitumen exposure	Bitumen contents & fumes exposure
Bal 2018[14]	**Oxidative stress markers** • 8-OH-dG: significantly higher in exposed (0.07 [95% CI 0.02-1.49] µg/g creatinine) vs non-exposed workers (0.01 [95% CI 0.0-0.16] µg/g creatinine), p<0.001 • TOS: significantly higher in exposed (1.98 [95% CI 0.2-37.4] mmol/g creatinine) vs non-exposed workers (0.2 [95% CI 0.0-1.93] mmol/g creatinine), p<0.001 • TAS: significantly higher in exposed (1.79 [95% CI 0.87-17.7] µg/g creatinine) vs non-exposed workers (0.8 [95% CI 0.2-4.34] µg/g creatinine), p<0.001 • OSI: significantly higher in exposed (104.8 [95% CI 11.7-565.2] µg/g creatinine) vs non-exposed workers (25.8 [95% CI 0-68.7] µg/g creatinine), p<0.001 • TDH: significantly higher disulphide/thiol ratio in exposed (2.44±1.14) vs non-exposed (1.86±1.48), p=0.03	Urinary analysis: • PAH exposure metabolite, 1-OHP: 10 times higher in exposed (2.27 [95% CI 0.11-122.16] µg/g creatinine) vs non-exposed workers (0.2 [95% CI 0.04-4.9] µg/g creatinine), p<0.001
Marczynski 2011[11]	**Oxidative stress markers and DNA damage** • 8-OH-dG: significantly higher in exposed (median 3.72 [IQR 2.97-4.40]) vs non-exposed (median 2.93 [IQR 2.54-3.61] pre-shift, p<0.0001 • 8-OH-dG: significantly higher in exposed (median 4.13 [IQR 3.31-5.17]) vs non-exposed (median 3.28 [IQR 2.78-4.14] post-shift, p<0.0001 • NS difference in other measures of DNA damage between groups • NS association between urinary PAH metabolites and DNA damage in blood samples	Urinary analysis: • NS difference in pre-shift urinary PAH metabolites between groups • Significantly higher concentrations of PAH metabolites (150.4 lg/l 1-OHP; 7.4 lg/l OHNA; 4.6 lg/l OHPHE) post-shift in exposed workers vs non-exposed workers (80.7 lg/l 1-OHP; 3.1 lg/l OHNA; 1.3 lg/l OHPHE), p<0001
Neghab 2015[9]	**Lung function**: • Pre- vs post-shift **mean % VC**: significant decrease in exposed workers, 91.2±5 vs 83±15.3, p<0.001 • Pre- vs post-shift **mean % FVC**: significant decrease in exposed workers, 85.9±18.8 vs 78.9±18.6, p=0.004	Mean concentration of asphalt fumes < threshold limit value (0.5mg/m^3) set by American conference of governmental industrial hygienists (AGIH)

Reference	Key outcomes of bitumen exposure	Bitumen contents & fumes exposure
	• Pre- vs post-shift **mean % FEV_1**: significant decrease in exposed workers, 89.6±18.7 vs 85.4±19.4, p=0.021 • Significant difference in **FEV_1/FVC ratio**: 87.1±8.6 in exposed workers vs 107.6±9.6 in non-exposed workers, p<0.001 **Respiratory symptoms** • **Cough**: 41% [95% CI 29-52] in exposed vs 10% [95% CI 4.3-15.7] in non-exposed workers, p<0.001 • **Phlegm**: 38% [95% CI 27-49] in exposed vs 9.1% [95% CI 3.7-4.5] in non-exposed workers, p<0.001 • **Productive cough**: 36% [95% CI 25-45] in exposed vs 6.4% [95% CI 1.7-11] in non-exposed workers, p<0.001 • **Wheezing**: 42% [95% CI 30-53] in exposed vs 3.6% [95% CI 0.1-7.2] in non-exposed workers, p<0.001 • **Shortness of breath**: 30% [95% CI 19-40] in exposed vs 5.5% [95% CI 1.2-9.8] in non-exposed workers, p<0.001 • **Chest tightness**: 18% [95% CI 9-26] in exposed vs 0% [95% CI 0] in non-exposed workers, p<0.001	
Neghab 2017[8]	**Liver function** • Liver function tests were within normal range for both groups • Significantly higher mean **serum albumin** in exposed workers (56±8 g/L^{-1}) vs non-exposed workers (48±3 g/L^{-1}), p=0.006 • Significantly higher mean **total bilirubin** in exposed workers (12±3 mg/L^{-1}) vs non-exposed workers (7±2 mg/L^{-1}), p=0.001 • Significantly higher mean **total protein** in exposed workers (87±13 g/L^{-1}) vs non-exposed workers (81±6 g/L^{-1}), p=0.006 • Significantly higher mean **alanine amino transferase** in exposed workers (27±22 μ/L^{-1}) vs non-exposed workers (20±11 μ/L^{-1}), p=0.003	Mean concentration of benzene soluble fraction and total particulate < threshold limit value of 10mg/m^{-3} and 0.5mg/m^{-3}, respectively set by American conference of governmental industrial hygienists (AGIH)

Reference	Key outcomes of bitumen exposure	Bitumen contents & fumes exposure
	• Significantly higher mean **aspartate amino transferase** in exposed workers (30±11 µg/L^{-1}) vs non-exposed workers (23±10 µg/L^{-1}), p=0.006 • Significantly lower mean **alkaline phosphatase** in exposed workers (183±51 µg/L^{-1}) vs non-exposed workers (220±64 µg/L^{-1}), p=0.001 **Kidney function** • Kidney function tests were within normal range for both groups • Significantly higher **blood urea nitrogen** in exposed workers (195±73 mg/L^{-1}) vs non-exposed workers (153±23 mg/L^{-1}), p=0.001 • Significantly lower mean **calcium** in exposed workers (89±4 mg/L^{-1}) vs non-exposed workers (97±4 mg/L^{-1}), p=0.001 • NS difference in mean **creatinine** or **phosphorous** **Blood analyses** • Blood cell counts were within normal range for both groups • Significantly lower mean number of **white blood cells** in exposed workers (6800±1700 /mm^3 blood) vs non-exposed workers (7470±1650 /mm^3 blood), p=0.007	
Raulf-Heimsoth 2011[10]	**Lung function** • NS change in **FVC** or **FEV_1** in bitumen-exposed vs non-exposed, irrespective of smoking status (spirometry) • NS difference in upper airways between groups or during shift (NALF analysis) **Inflammatory status** • **Pre-shift**: significantly higher **IL-8** concentration in bitumen-exposed (5,083 pg/ml [IQR 1,723-13,468]) vs non-exposed workers (2,315 pg/ml [IQR 915-7,420]), p<0.05 (induced sputum measures) • **Post-shift:** significantly higher **IL-8** concentration in bitumen-exposed (3,667 pg/ml [IQR 1,847-9,672]) vs non-exposed workers (1,723 pg/ml [IQR 755-5,388]), p<0.05 (induced sputum measures)	Personal air sampling of bitumen vapours and aerosols: Exposed: 3.46 mg/m^3 [IQR 1.8-5.9] Non-exposed: 0.2 mg/m^3 [IQR 0.07-0.3]

Reference	Key outcomes of bitumen exposure	Bitumen contents & fumes exposure
	• **Pre-shift**: significantly higher **total protein** concentration in bitumen-exposed (671 µg/ml [IQR 404-1,108]) vs non-exposed workers (367 µg/ml [IQR 180-711]), p<0.05 (induced sputum measures) • **Post-shift**: significantly higher **total protein** concentration in bitumen-exposed (665 µg/ml [IQR 393-1,089]) vs non-exposed workers (406 µg/ml [IQR 214-673]), p<0.05 (induced sputum measures) • **Pre-shift**: significantly higher **MMP-9** concentration in bitumen-exposed (291 ng/ml [IQR 57-646]) vs non-exposed workers (92 ng/ml [IQR 16-233]), p<0.05 (induced sputum measures) • **Post-shift**: significantly higher **MMP-9** concentration in bitumen-exposed (239 ng/ml [IQR 99-573]) vs non-exposed workers (121 ng/ml [IQR 26-273]), p<0.05 (induced sputum measures) • NS differences in inflammatory markers within groups during shift • NS differences in other inflammatory markers	
Sellappa 2011[15]	**DNA damage in blood leucocytes (post-shift)** <u>Frequency of micronuclei</u> • Smokers: significantly higher in exposed (5.7±1.08) vs non-exposed (4.31±1.29), p<0.05 • Non-smokers: significantly higher in exposed (4.06±0.93) vs non-exposed (3.10±0.76), p<0.05 • Alcohol use: significantly higher in exposed (5.26±0.04) vs non-exposed (4.15±0.88), p<0.05 • No alcohol use: significantly higher in exposed (4.94±0.83) vs non-exposed (3.05±0.05), p<0.05 <u>DNA strand breaks</u> • Smokers: significantly higher in exposed (19.4±4.99) vs non-exposed (13.3±3.74), p<0.05 • Non-smokers: significantly higher in exposed (15.5±4.94) vs non-exposed (10.9±2.85), p<0.05 • Alcohol use: significantly higher in exposed (16.2±2.03) vs non-exposed (11.1±2.92), p<0.05 • No alcohol use: significantly higher in exposed (15.1±3.12) vs non-exposed (9.9±2.83), p<0.05	Urinary analysis: PAH exposure metabolite: mean 1-OHP significantly higher in exposed (1.68±0.93) vs controls (0.55±0.42 µmol/mol-1 creatinine, p<0.05

Reference	Key outcomes of bitumen exposure	Bitumen contents & fumes exposure
Ulvestad 2007[19]	**Lung function** • **Pre-season**: significantly lower mean % **FEV_1** in bitumen-exposed workers (92.6±1.1) vs non-exposed workers (96.9±1.2, p=0.01 (adjusted for smoking and BMI) • **Pre-season**: significantly lower mean % **FEF_{50}** in bitumen-exposed workers (84.8±2.6) vs non-exposed workers (92.9±3.3), p=0.03 (adjusted for smoking and BMI) *Analysis by job category of asphalt workers* • **Post-season**: Screedmen had significantly greater change in lung function (**FVC and FEV_1**) vs all other asphalt workers (paver operator, roller driver, asphalt stripper, plant operator, lorry driver), p<0.05 **Inflammatory response** • Significant increase in mean plasma concentration of **IL-6** from 1.55pg/ml pre-season to 2.67pg/ml post-season in bitumen-exposed workers, p=0.04 (adjusted for smoking) • **Pre-season**: smokers had significantly higher **IL-6** concentrations (mean 2.11±1.89ng/L) vs non-smokers (mean 1.39±2.06ng/L), p=0.026	Personal air sampling: All measures lower than accepted safe Norwegian occupational exposure limits **Total dust:** Asphalt strippers significantly higher exposure vs other asphalt workers, p<0.001 **PAHs:** Paver operators, screedmen, roller drivers significantly higher exposure vs truck drivers, asphalt strippers, plant operators, p<0.001
Ulvestad 2017[13]	**Lung function** • At 5 yrs FU, bitumen-exposed workers lost significantly more **lung volume** vs non-exposed workers, p=<0.05 (adjusted for age, BMI and smoking) • Screedmen had significantly greater decline in **FVC** than other asphalt pavers, p=0.029 **Lung cancer scan** • 59% of bitumen-exposed workers had normal HRCT scan • 4% (3/75) had fine intralobular lung fibrosis, without evident cysts	Personal air sampling: Mean concentrations of particulate matter and total dust was < Norwegian occupational exposure limits Oil mist exposure was higher than Norwegian occupational exposure limits in 1 screedman (1/22)
Xu 2018[16]	**Lung function** • NS difference in **FVC** or **FEV_1** values between groups or during shift in any group • NS differences in self-reported respiratory symptoms (upper or lower airways) **Eye and/or nose irritation symptoms**	NS difference in dust, PAHs or nitrosamine between conventional and CRM asphalt Benzothiazole exposure was significantly higher in CRM asphalt workers (median 2.09 µg/m^3 [95% CL 1.01-3.69]) vs

Reference	Key outcomes of bitumen exposure	Bitumen contents & fumes exposure
	• Significantly higher self-reported **eye irritations** in bitumen-exposed workers (23%) vs non-exposed workers (10%), p=0.014 • NS difference in reported **eye symptoms** between conventional asphalt and CRM asphalt workers • NS difference in **nasal symptoms** between bitumen-exposed and non-exposed workers **Inflammatory response** • **Pre-shift**: **IL-8** was significantly higher in CRM asphalt workers vs non-exposed workers (β=8.80 [95% CI 4.79-12.8], p<0.001 • After 4 days work, **C-reactive protein** decreased in non-exposed workers, but not in the 2 asphalt worker groups; however, NS association between years of working with asphalt and levels of **IL-8** or **C-reactive protein**	conventional asphalt workers, (median 0.37 µg/m^3 [95% CL 0.17-2.63])p<0.001

Notes: 1-OHP = 1-hydroxypyrene; 8-OH-dG = 8-hydroxy-2-deoxyguanosine; TOS, total oxidant status; TAS, total antioxidant status; CI = confidence intervals; CRM = crumb rubber modified; FEF_{50} = forced expiratory flow at 50%; FEV_1 = forced expiration volume in 1 second; FVC = forced vital capacity; HRCT = high-resolution computed tomography; IL-8 = interleukin-8; IQR = interquartile range; MMP-9 = matrix metalloproteinase-9; NALF = nasal lavage fluid; NS = not statistically significant; OHNA = hydroxynapthalene; PHPHE = hydroxyphenanthrene; OSI = oxidative stress index; PAHs = polycyclic aromatic hydrocarbons; r_s = Spearman rank coefficient; TDH = thiol disulphide homeostasis; VC = vital capacity.

QUESTION 2: WHAT FACTORS ARE SHOWN TO MITIGATE THE RISK OF, OR PROTECT WORKERS FROM, ADVERSE HEALTH EFFECTS ASSOCIATED WITH EXPOSURE TO BITUMEN CONTENTS AND FUMES DURING ROAD CONSTRUCTION AND REPAIRS?

Key findings

Equipment design

- Integrated fume extraction system fitted to the paver machinery demonstrated 2-4 times more efficient fume capture efficiency compared with 2 other models.

Personal protective equipment

- Bitumen-exposed workers wearing protective clothing (gloves, pants, neckcloth, long-sleeved shirt) had significantly lower urinary metabolites of PAHs compared with no protective clothing
- Bitumen exposed workers wearing an air-purifying respiratory device had significantly lower urinary metabolites of PAHs compared with no respiratory device.

Alternative equipment cleaning process

- Substituting biodiesel to clean equipment led to significantly lower urinary metabolites of PAHs compared with usual diesel cleaning in bitumen-exposed workers.

Detailed findings - Protective factors

Study characteristics – protective factors

Although many studies reported lower PAHs emissions from bitumen loads manufactured in lower temperatures ('warm mix'),[20] most studies that evaluated work practices pertaining to asphalt paving construction focused on the environmental or economic impact of different mixes and processes to improve performance, but did not assess the health impact on workers.[20, 21]

Two studies were identified that assessed the effectiveness of strategies to mitigate the health risks related to bitumen by reducing exposure to bitumen fumes. One study evaluated the efficiency of fume extraction systems installed in paver machines;[22] and another assessed the effectiveness of personal protective equipment in a group of asphalt paving workers.[23] Table 5 provides details of the study characteristics and outcomes.

Table 5. Primary study characteristics and outcomes – protective factors

Reference country	Study design	Cohort	Intervention / protective factors	Key outcomes	Quality rating
Bonthoux 2013[22] France	Evaluation study of paver machines	N/A	Three fume extraction systems fitted to paver machines A. Hood located in central section B. 2 suction devices centred over each half-auger C. Design-integrated suction slot located at conveyor outlet	**Fume capture efficiency** A. 26% B. 67% C. 99%	N/A
MacLean 2012 US[23]	Crossover study Four conditions over 4 weeks: 1. Baseline (normal conditions) 2. Protective clothing 3. Powered air-purifying respirator (PAPR) 4. Biodiesel substitution (cleaning equipment)	Asphalt paving workers N=12; 100% male	• Protective clothing • Inhalation protection • Biodiesel substitution	**Urinary metabolites of PAHs** Compared with baseline measures: • Protective clothing: significant reduction in OH-Pyr, 29% [95% CI 18-38%]; and I-PAC, 15% [95% CI 2-26%], p<0.05 • PAPR: significant reduction in OH-Pyr, 24% [95% CI 12-33%]; and I-PAC, 15% [95% CI 2-26%], p<0.05 • Biodiesel substitution: significant reduction in OH-Pyr, 15% [95% CI 0.3-27%], p<0.05	Weak

Notes: N/A = not applicable; I-PAC = immunochemical quantification of polycyclic aromatic compounds; OH-Pyr = 1-hydroxypyrene; PAHs = polycyclic aromatic hydrocarbons

Key outcomes – protective factors

Equipment design

Bonthoux and Patrascu[22] examined the efficiency of three different fume extraction systems incorporated into the paver machine to protect the operator from exposure to bitumen fumes (See Figure 3, Appendix):

A) A hood was located in the central section above the spreading auger

B) Two suction devices were centred over each half-auger

C) A design-integrated suction slot was located at the conveyor outlet.

Analyses showed that Model C was superior in fume capture efficiency (99%) compared with Model A (26%) or Model B (67%) (Figure 3). Moreover, as Model C was incorporated beneath the machine engine cover, it required no additional space; there was no visual or acoustic interference; and there was a lower risk of potential blowback on the operator.[22]

Personal protection

In a cross-over study, McClean et al.[23] exposed a small sample of asphalt paving workers to a different exposure scenario each week for four weeks:

1. Normal operating conditions (baseline)
2. Protective clothing (gloves, hats with neckcloth, pants, long-sleeved shirt)
3. Inhalation protection (powered air-purifying respirators (PAPR))
4. Biodiesel substitution (B-100, containing no PAHs, to replace diesel oil normally used to clean tools and equipment.

Under normal conditions, analyses showed that urinary PAH metabolites were significantly higher post-shift and at bedtime compared with pre-shift samples. In addition, personal air sampling showed a statistically significant increase in PAH metabolites post-shift, but this decreased at bedtime and reduced further in the following morning pre-shift measures. Therefore, no cumulative effect was observed through the working week.

Protective clothing led to a 28% decrease in urinary PAH metabolite (OH-Pyr); inhalation protection led to 24% decrease in OH-Pyr; and biodiesel substitution led to 15% decrease in OH-Pyr (Table 5).

Additional measures demonstrated that increasing the temperature of the asphalt (from 121°C to 154°C) led to 72% increase in OH-Fluor and OH-Pyr; and 82% increase in OH-Phen.

SUMMARY & CONCLUSIONS

Overall, evidence of an association between exposure to bitumen contents and fumes and objective adverse health outcomes was mixed. Moderate evidence suggested that bitumen exposure had a detrimental effect on lung function in the short term. While limited evidence also showed early signs of tissue or DNA damage amongst bitumen-exposed workers, the long-term cumulative effects of exposure are unclear.

Acute health effects

Three out of five studies reported a statistically significant decline in lung function and increased respiratory symptoms in bitumen-exposed workers compared with non-exposed workers. Although eye and nose irritations have been reported in bitumen-exposed workers, there was limited evidence to support a clear association with bitumen exposure due to variability in study conditions and analyses (exposure, composition of bitumen product, measures), poor study methodology and lack of appropriate controls for confounders.

Chronic health effects

There was a weak association between bitumen exposure and development of some cancers. However, poor study methodology and high variability in exposure assessment and conditions limited interpretation of findings. It is also possible that adverse health outcomes have been underestimated in studies due to a 'healthy worker effect'. For example, the worst affected workers may have been on sick leave at the time of data collection, or left due to health reasons.

In contrast, signs of tissue injury in liver, kidney and airways (inflammatory markers) and DNA damage were significantly higher in bitumen-exposed workers; and health outcomes were worse for smokers and significantly higher amongst screedmen (who level the asphalt).

Protective factors

Of the few studies that evaluated the effectiveness of strategies to mitigate adverse health risks by reducing the level of exposure in bitumen workers, limited evidence supported use of personal protective equipment, air-purifying respiratory equipment, and substitution of biodiesel for cleaning equipment to reduce risk of exposure. Lower temperature bitumen mixes were also associated with lower PAHs emissions, thereby potentially reducing the risks of PAHs-related tissue and DNA damage.

Although statistically significant differences in outcomes were demonstrated between bitumen-exposed and non-exposed paving workers, the clinical significance of these differences is unclear, and the association between exposure to bitumen contents and fumes and adverse health effects was difficult to determine conclusively. However, the early signs of tissue and DNA damage are troubling. Despite the lack of good quality evidence, effective protective strategies and technological solutions, such as using lower temperature mixes, efficient fume capture, personal protective clothing and biodiesel for cleaning equipment, should be considered for reducing the level of exposure in bitumen workers.

REFERENCES

1. IARC. Bitumens and bitumen emissions, and some N- and S-heterocyclic polycyclic aromatic hydrocarbons. France: International Agency for Research on Cancer; 2013.

2. Shea BJ, Hamel C, Wells GA, Bouter LM, Kristjansson E, Grimshaw J, et al. AMSTAR is a reliable and valid measurement tool to assess the methodological quality of systematic reviews. J Clin Epidemiol. 2009;62(10):1013-20.

3. Effective Public Health Practice Project. Quality assessment tool for quantitative studies. Hamilton, ON: Effective Public Health Practice Project; 1998.

4. Mundt KA, Dell LD, Crawford L, Sax SN, Boffetta P. Cancer risk associated with exposure to bitumen and bitumen fumes: An updated systematic review and meta-analysis. J Occup Environ Med. 2018;60(1):e6-e54.

5. Alicandro G, Rota M, Boffetta P, La Vecchia C. Occupational exposure to polycyclic aromatic hydrocarbons and lymphatic and hematopoietic neoplasms: A systematic review and meta-analysis of cohort studies. Arch Toxicol. 2016;90(11):2643-56.

6. Rota M, Bosetti C, Boccia S, Boffetta P, La Vecchia C. Occupational exposures to polycyclic aromatic hydrocarbons and respiratory and urinary tract cancers: An updated systematic review and a meta-analysis to 2014. Arch Toxicol. 2014;88(8):1479-90.

7. Wagner M, Bolm-Audorff U, Hegewald J, Fishta A, Schlattmann P, Schmitt J, et al. Occupational polycyclic aromatic hydrocarbon exposure and risk of larynx cancer: A systematic review and meta-analysis. Occup Environ Med. 2015;72(3):226.

8. Neghab M, Derisi FZ, Hassanzadeh J, Dirin V, Heidari S. Toxic responses of different organs following occupational exposure to sub-threshold limit value levels of paving asphalt fumes. Toxicol Environ Chem. 2017;99(2):331-9.

9. Neghab M, Zare Derisi F, Hassanzadeh J. Respiratory symptoms and lung functional impairments associated with occupational exposure to asphalt fumes. Int J Occup Environ Med. 2015;6(2):113-21.

10. Raulf-Heimsoth M, Pesch B, Kendzia B, Spickenheuer A, Bramer R, Marczynski B, et al. Irritative effects of vapours and aerosols of bitumen on the airways assessed by non-invasive methods. Arch Toxicol. 2011;85(1):41-52.

11 Marczynski B, Raulf-Heimsoth M, Spickenheuer A, Pesch B, Kendzia B, Mensing T, et al. DNA adducts and strand breaks in workers exposed to vapours and aerosols of bitumen: Associations between exposure and effect. Arch Toxicol. 2011;85(1):53-64.

12. Ulvestad B, Randem BG, Hetland S, Sigurdardottir G, Johannessen E, Lyberg T. Exposure, lung function decline and systemic inflammatory response in asphalt workers. Scand J Work Environ Health. 2007;33(2):114-21.

13. Ulvestad B, Randem BG, Skare O, Aalokken TM, Myranek GK, Elihn K, et al. Lung function in asphalt pavers: a longitudinal study. Int Arch Occup Environ Health. 2017;90(1):63-71.

14. Bal C, Ağış ER, Büyükşekerci M, Gündüzöz M, Tutkun L, Yılmaz ÖH. Occupational exposure to asphalt fume can cause oxidative DNA damage among road paving workers. Am J Ind Med. 2018;61(6):471-6.

15. Sellappa S, Mani B, Keyan KS. Cytogenetic biomonitoring of road paving workers occupationally exposed to polycyclic aromatic hydrocarbons. Asian Pac J Cancer Prev. 2011;12(3):713-7.

16. Xu Y, Karedal M, Nielsen J, Adlercreutz M, Bergendorf U, Strandberg B, et al. Exposure, respiratory symptoms, lung function and inflammation response of road-paving asphalt workers. Occup Environ Med. 2018;75(7):494-500.

17. Deygout F, Auburtin G. Impact of some field factors on inhalation exposure levels to bitumen emissions during road paving operations. Ann Occup Hyg. 2015;59(2):158-72.

18. Breuer D, Hahn J-U, Höber D, Emmel C, Musanke U, Rühl R, et al. Air sampling and determination of vapours and aerosols of bitumen and polycyclic aromatic hydrocarbons in the Human Bitumen Study. Arch Toxicol. 2011;85(1):11-20.

19. Agostini M, Ferro G, Burstyn I, De Vocht F, Portengen L, Olsson A, et al. Does a more refined assessment of exposure to bitumen fume and confounders alter risk estimates from a nested case-control study of lung cancer among European asphalt workers? Occup Environ Med. 2013;70(3):195-202.

20. Santos J, Flintsch G, Ferreira A. Environmental and economic assessment of pavement construction and management practices for enhancing pavement sustainability. Resour Conserv Recycl. 2017;116:15-31.

21. Santos J, Bressi S, Cerezo V, Lo Presti D, Dauvergne M. Life cycle assessment of low temperature asphalt mixtures for road pavement surfaces: A comparative analysis. Resour Conserv Recycl. 2018;138:283-97.

22. Bonthoux F, Patrascu C. The evaluation of bitumen fume capture efficiency for road pavers. Int J Vent. 2013;12(2):135-41.

23. McClean MD, Osborn LV, Snawder JE, Olsen LD, Kriech AJ, Sjodin A, et al. Using urinary biomarkers of polycyclic aromatic compound exposure to guide exposure-reduction strategies among asphalt paving workers. Ann Occup Hyg. 2012;56(9):1013-24.

24. Elihn K, Ulvestad B, Hetland S, Wallén A, Randem BG. Exposure to ultrafine particles in asphalt work. J Occup Environ Hyg. 2008;5(12):771-9.

GLOSSARY

Table 6. Glossary of terms

FEF_{50}	Forced expiratory flow: average flow rate at 50% of the volume of exhaled air, expressed as the average rate at which air is exhaled when 50% remains in the lung
FEV_1	Forced expiratory volume during the first second: volume of air exhaled during the first second of forced exhalation, expressed as a percentage of predicted value
FVC	Forced vital capacity: volume of air that can be forcibly exhaled from the lungs after a maximum inhalation, expressed as a percentage
FEV_1/FVC	Proportion of a person's vital capacity that they can exhale in the first second of a forced expiration to the full vital capacity, expressed as a percentage (normal values are approximately 75%)
PEF	Peak expiratory flow: maximum speed of expiration, expressed as litres per minute (L/min)
VC	Vital capacity: Maximum volume of air a person can exhale after a maximum inhalation, expressed as a percentage

APPENDIX

Fig 2. Occupational job groups in asphalt production and paving

Job Group	Work Description	Main Exposures
Bitumen plant operators	Responsible for receiving the bitumen conveyed on ships and to monitor the storage.	Not exposed
Bitumen tanker drivers	Transport the bitumen from the bitumen storage plant to the asphalt plant.	Not exposed
Asphalt plant operators	Monitor the asphalt mixing from a control room. Asphalt mixing is a process whereby bitumen is combined with gravel. Operators must leave their cabins several times during the day for adjustments of the plant machinery.	Total dust, respirable dust, quartz, PAHs, oil vapor
Laboratory assistant	Check the quality and composition of the hot asphalt mix in small laboratories.	Total dust, respirable dust, quartz, VOC, PAHs, oil vapor
Excavator operator	Operator seated in ventilated cabin on an excavator that loads gravel of different sizes into the asphalt mixing system.	Total dust, respirable dust, quartz, diesel exhaust
Transport truck driver	Hot asphalt mix is transferred directly to a transport truck that takes it to the paving site, where it is emptied into the hopper of the paving machine.	Oil vapor, diesel exhaust
Paver operator	Operator seated on top of paving machine between the hopper and the screed, which discharges the hot mix onto the surface being paved.	Total dust, respirable dust, PAHs, oil mist and vapor, diesel exhaust
Screedmen	Screedmen control asphalt discharge through the screed. They also fix the edges of the asphalt on the road manually and help to spread the hot mixture discharged from the screed using a hand rake.	Total dust, respirable dust, PAHs, oil mist and vapor, diesel exhaust
Roller drivers	Drive the roller that compresses the asphalt mixture once it is applied to the surface. The roller is normally equipped with a cabin.	Total dust, respirable dust, PAHs, oil mist and vapor, diesel exhaust
Asphalt strippers	Old layer of asphalt is stripped from the road and mixed with new asphalt at the asphalt plant.	Total dust, respirable dust, quartz, PAHs, oil mist and vapor, diesel exhaust
Drivers of the binding agent truck	Binding agent is applied on the road surface to help glue the new asphalt to the old surface. Drivers also assist the screedmen.	Total dust, respirable dust, oil mist and vapor, diesel exhaust

Source: Elihn et al. (2008)[24]

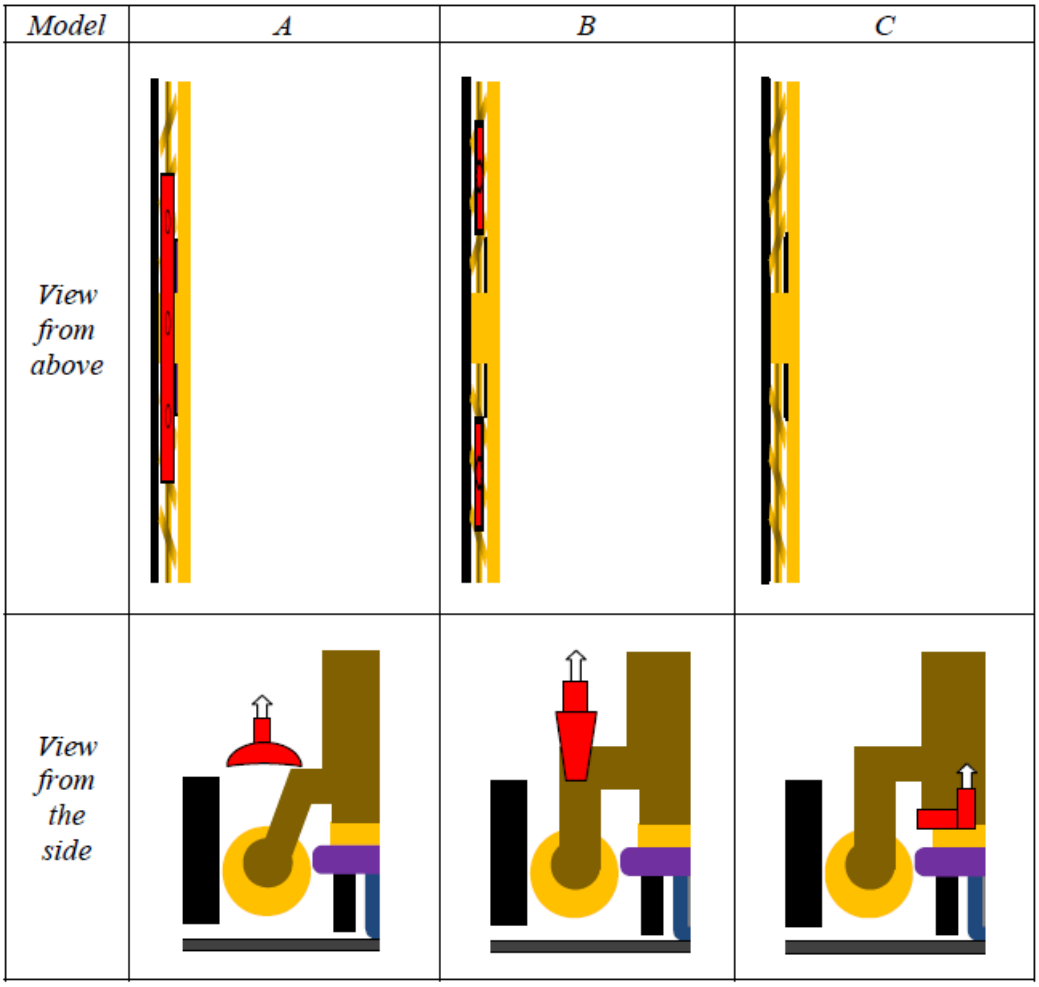

Fig 3. Layout of capture systems above the spreading auger

Source: Bonthoux[22]