

## Blood gases in asphalt workers

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**P**olycyclic aromatic hydrocarbons (PAH) are present in asphalt structure, which also contains toxic elements such as benzene, toluene, nitric and carbonic acid, benz(a)pyrene, formaldehyde, carbon monoxide, nitrogen dioxide, sulphur dioxide and hydrogen sulphide. The ubiquitousness of asphalt in daily life results in an unavoidable health disturbance to public health.<sup>1-4</sup> Fumes and vapors, which directly affect the respiratory system, arise when asphalt is processed or used.<sup>5-8</sup> The kind of asphalt, its temperature and environmental factors affect the fume and vapor density. If protective precautions are not followed, harmful effects will be seen.<sup>9</sup> The duration of exposure and intensity of asphalt fumes and vapors affect metabolic oxidation, alveolar macrophage functions and cellular toxicity levels. It is reported that when exposure to asphalt fume is over 200mg/mL, toxic effects are seen on the respiratory system.<sup>10</sup> A tolerable inspired air particle concentration is between 0.6-1.4 mg/m<sup>3</sup>, but it is reported that some factors such as ozone, temperature, cigarette smoking and intensity of work further lower the tolerance level.<sup>3</sup> Inhaling 0.1-0.5 mg/m<sup>3</sup> of contaminated air during 3 consecutive days results in death. Asphalt can cause some disorders such as acute myocardial infarction, pulmonary fibrosis, leukemia and it may have genotoxic activity.<sup>11-14</sup> Components of asphalt fume and vapor affect not only vital functions such as the respiratory system, but it also has hematopoietic activity. Changes in the partial pressure of blood gases results in activation of compensatory mechanisms to restore the acid-base balance.<sup>15</sup> Competition of asphalt gases with O<sub>2</sub> directly affects blood PO<sub>2</sub> levels (Haldane and Bohr Effects). Asphalt fumes and vapors affect the respiratory and metabolic compensation mechanisms that control blood pH.<sup>16-20</sup>

In this study, we aimed to investigate the effects of PAH, organic solvents and other toxic elements that are aromatic products of asphalt fumes and vapors on the blood gases of asphalt workers, who were affected acutely, and office workers, who were affected chronically.

### Subjects and Methods

This study was performed between 9.00 and 11.00 hours in August and September, when asphalt paving was intensive. Sixty-eight male volunteers who worked as asphalt or office workers were enrolled. The volunteers (n=38) were 41.2±3.6 years of age (mean±SD), with a mean height of 171.0±5 cm, a mean weight of 79.3±10 kg, and had been working for 17±6 years as asphalt workers. The office workers (n=30) were all male and were 39.7±4 years old, with a mean height of 170.0±10 cm, a mean weight of 75.2±7.7 kg and had been working for 14±5 years as office workers.

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After an anamnesis, volunteers with suspicious sub-clinical and clinical symptoms were not enrolled in the study. Five mL of arterial blood was taken from the radial artery. Blood samples were transferred to the blood gases laboratory within 10 minutes. The samples were analyzed for blood partial oxygen pressure (PO<sub>2</sub>), partial carbon dioxide pressure (PCO<sub>2</sub>), oxygen saturation percentage, hydrogen concentration (pH), bicarbonate concentration (HCO<sub>3</sub>) and base excess with a CIBA-Corning 238-pH/Blood Gas Analyzer.

The data were analyzed with the unpaired Student's t-test and the chi-square test. A difference was considered significant if  $P < 0.05$ .

### Results

Based on the anamneses, the incidence of respiratory symptoms was greater in asphalt workers than in office workers (Table 1). The difference between the two groups was statistically significant for PO<sub>2</sub>, HCO<sub>3</sub>, percent oxygen saturation and base excess levels ( $P < 0.05$ ). However, differences were not statistically significant for pH, PCO<sub>2</sub> or total CO<sub>2</sub> ( $p > 0.05$ , Table 2). Percent differences in pH

**Table 1.** The incidence of respiratory symptoms in asphalt and office workers.

Symptoms	Asphalt Worker (n=38)		Office workers (n=30)		X <sup>2</sup>	P value
	n	%	n	%		
Cough	12	31.6	7	23.3	0.23	>0.05
Phlegm	12	31.6	8	26.7	0.03	>0.05
Asthmatic	13	34.2	4	13.3	3.90	<0.05

**Table 2.** Blood gas levels of asphalt and office workers.

Parameters	Asphalt workers (n=38) (mean±SD)	Office workers (n=30) (mean±SD)	Difference (%)*	t	P value
PO <sub>2</sub> (mmHg)	78.0±27.0	91.5±8.7	-14.8	2.8	<0.01
PCO <sub>2</sub> (mmHg)	33.7±4.6	33.2±3.1	+1.6	0.5	>0.05
SaO <sub>2</sub> (%)	89.0±6.1	97.0±1.1	-8.3	3.1	<0.05
pH	7.4±0.6	7.4±0.1	0.0	0.0	>0.05
HCO <sub>3</sub> (mEq/L)	21.7±1.9	22.7±1.5	-4.4	2.4	<0.05
BE (mEq/L)	-3.2±2.3	-2.1±1.9	+52.4	2.2	<0.05

\*The difference between of asphalt and office workers (+ and - represent the percentage increase and decrease, respectively.) SaO<sub>2</sub>=percent oxygen saturation; BE=base excess.

and blood gases ranged from 3.2% to 52.4%. The base excess levels of the asphalt workers were higher than the office workers and the PO<sub>2</sub>, HCO<sub>3</sub> and percent oxygen saturation levels of the office workers were higher than the asphalt workers.

### Discussion

While the volatile compounds of asphalt decreased the partial oxygen pressures by competing with oxygen, they also caused PO<sub>2</sub> levels to decrease by lowering the ventilation air oxygen concentration in the respiration zone. The PO<sub>2</sub> levels of the asphalt workers were less than those of the office workers. The O<sub>2</sub> saturation levels also declined in the asphalt workers. These results are consistent with reports in the literature, which state that exposure to asphalt reduced the PO<sub>2</sub> levels of workers.<sup>16</sup> Blood pH does not change in either asphalt or office workers due to chronic activation of intracellular and extracellular buffer systems.<sup>14,22</sup> In the literature, there are only a few studies that have reported that exposure to asphalt did not change pH levels.<sup>16,22</sup>

While long-term intracellular pH regulation is regulated by ion transportation mechanics, acute regulation is regulated by buffer systems. Therefore, there was no excess deviation in hydrogen ion concentrations. Due to the chronic response of central and peripheral chemoreceptors, the PCO<sub>2</sub> levels were similar in both groups. In the literature it is noted that exposure to asphalt fume and vapor does not alter ventilation mechanisms significantly, but it does affect the volume and capacity of respiration in a decreasing direction.<sup>5,14,20,22</sup> This state can be explained by the reduction in contractility of ventilation muscles as a result of the effect of asphalt on Ca<sup>2+</sup> metabolism.<sup>4</sup>

Also, during asphalt paving, the temperature of the asphalt is between 100 and 232°C. Increased environmental

temperature affects the respiration rate (tachypnea) by altering ventilation mechanics, and indirectly prevents hypercarbia. The explanation for the difference between asphalt and office workers' blood bicarbonate levels are: a) a decrease in respiration frequency to keep blood pH at a certain level, b) increase in blood hydrogen ion concentration, c) decrease in the amount and activity of carbonic anhydrase, and d) an increase in base losses. These are all factors that cause a decrease in bicarbonate levels, so when workers are exposed to asphalt, to compensate for the acidosis, they use more bicarbonate. By inhalation of asphalt fume and vapor, the acidic component, carbonic acid, makes the blood pH decrease, which results in bicarbonate usage, so a decrease in blood bicarbonate level is the usual response of the body.<sup>9,14</sup>

Plasma bicarbonate levels are improved by changes in cardiac performance, carbonic anhydrase activity, renal function, and respiratory and non-respiratory components. Plasma bicarbonate levels are largely controlled by kidney. Inhalation of asphalt fume and vapor inhibits the secretion of hydrogen, which decreases the bicarbonate level and causes hyperchloremic metabolic acidosis.<sup>15,19</sup> However, some authors note that long-term exposure to asphalt results in no significant change in bicarbonate levels.<sup>15</sup> As a result, chronic exposure to asphalt vapor and fumes causes a reduction in PO<sub>2</sub>, O<sub>2</sub> saturation, bicarbonate levels, and negative base excess. We think these changes affect the regulatory mechanism of respiration and maintain the steady state.<sup>15,19</sup>

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## References

1. Krasniuk EP, Chemiuk V, Rossinskaia LN. The effect of manufacturing factors in asphalt-bitumen plants on the health of workers. *Lik Sprava*. 2000; 2:106-112.
2. Brandt HC, Groot PC. A laboratory rig for studying aspects of worker exposure to bitumen fumes. *Am Ind Hyg Assoc J*. 1999; 60(2):182-190.
3. Gamble JF, Nicolich MJ, Barone NJ. Exposure-response of asphalt fumes with change in pulmonary function and symptoms. *Scand J Work Environ Health*. 1999; 25(3):186-206.
4. Romero DL, Mounho BJ, Lauer FT. Depletion of glutathione by benz(a)pyrene metabolites, ionomycin, thapsigargin, and phorbol myristate in human peripheral blood mononuclear cells. *Toxicol Appl Pharmacol*. 1997; 144(1):62-69.
5. Sobaszek A, Boulenguez C, Frimat P. Acute respiratory effects of exposure to stainless steel and mild steel welding fumes. *J Occup Environ Med*. 2000; 42(9): 923-931.
6. Mendis S, Sobotha PA, Euler DE. Expired hydrocarbons in patients with acute myocardial infarction. *Free Radic Res*. 1995; 23(2):117-122.
7. Chiaze L, Watkins DK, Amsel J. Asphalt and risk of cancer in man. *Br J In Med*. 1991; 48:538-542.
8. Kaplan SD. Update of a mortality study of workers in petroleum refineries. *J Occup Med*. 1986; 28:514-516.
9. Baruchin AM, Schraf S, Rosenberg L, Sagi AA. Hot bitumen burns. *Burns*. 1997; 23(5):438-441.
10. Carder CR, Fuerts RS. Myocardial infarction after toluene inhalation. *Pediatr Emerg Care*. 1997; 13(2): 117-119.
11. Sivak A, Niemeier R, Lynch D. Skin carcinogenicity of condensed asphalt roofing fumes and their fractions following dermal application to mice. *Cancer Lett*. 1997; 117(1):113-123.
12. Karakaya A, Yücesoy B, Turhan A. Investigation of some immunological functions in a group of asphalt workers exposed to polycyclic aromatic hydrocarbons. *Toxicology*. 1999; 135(1):43-47.
13. Burstyn I, Kromhout H, Kauppinen J. Statistical modelling of the determinants of historical exposure to bitumen and polycyclic aromatic hydrocarbons among paving workers. *Ann Occup Hyg*. 2000; 44(1):43-56.
14. Cogan MG. *Fluids and electrolytes*. San Francisco: Prentice-Hall International Inc. Appleton & Lange: 1991; 152-217.
15. Morgan WKC. Respiratory effects of particles, vapours and fumes. *Am Ind Hyg Assoc J*. 1986; 47:670-673.
16. Schnatter AR, Armstrong TW, Nicolich MC. Lymphohaematopoietic malignancies and quantitative estimates of exposure to benzene in Canadian petroleum distribution workers. *Occup Environ Med*. 1996; 53(11):773-781.
17. Armstrong TW, Pearlman ED, Schnatter AR. Retrospective benzene and total hydrocarbon exposure assessment for a petroleum marketing and distribution worker epidemiology study. *Am Ind Hyg Assoc J*. 1996; 57(4):333-343.
18. Ma JY, Barger MW, Kriech AJ, Castranova V. Effects of asphalt fume condensate exposure on acute pulmonary responses. *Arc Toxicol*. 2000; 74(8): 452-459.
19. Partanen TJ, Baffetta P, Heikkilä PR. Cancer risk for European asphalt workers. *Scand J Work Environ Health*. 1995; 21(4):252-258.
20. Hainsworth R. *Acid-base balance*. Manchester: Manchester University Press; 1986: 50-94.
21. Tolbert PE. Oils and cancer. *Cancer Causes Control*. 1997; 8(3):386-405.
22. Pleil JD, Lindstrom AB. Collection of single alveolar exhaled breath for volatile organic compounds analysis. *Am J Ind Med*. 1995; 28(1):109-121.