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Assessment of health effects in workers at gasoline station

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Abstract:

The aim of this study was to make assessment of health effects in 37 workers exposed to gasoline, and its constituents at gasoline stations between 1985 and 1996. Thirty-seven persons who had been exposed to gasoline for more than five years were examined. The evaluation included a medical/occupational history, haematological and biochemical examination, a physical exam, standardized psychological tests, and ultrasound examination of kidneys and liver. The groups were identical in other common parameters including age, gender (all men), and level of education ($P < 0.05$). The data were compared to two control groups: 61 healthy non-exposed controls and 25 workers at gasoline stations exposed to organic lead for only nine months. Peripheral smear revealed basophilic stippling and reticulocytosis. We found in chronic exposed gasoline workers haematological disorders: mild leukocytosis (7 of 37), lymphocytosis (20 of 37), mild lymphocytopenia (1 of 37), and decrease of red blood cells count (11 of 37). Results indicated that they have suffered from liver disorders: lipid degeneration of liver (14 of 37), chronic functional damages of liver (3 of 37), cirrhosis (1 of 37). Ultrasound examination indicated chronic kidney damages (8 of 37). These results significantly differed from those of controls ($P < 0.05$). In 13 out of 37 workers at gasoline stations exposed to gasoline for more than 5 years the symptoms of depression and decreased reaction time and motor abilities were identified. The summary of diseases of workers exposed to organic lead and gasoline are discussed.

Keywords: occupational exposure, long-term exposure effects, tetraethyl lead, gasoline constituents, oxygenates, haematological effects, physiological effects.

Introduction

Gasoline is mixture of saturated and unsaturated hydrocarbons. Gasoline fuels contain approximately 62% alkanes, 7% alkenes, and 31% aromatics and additives (1). Typically, gasoline contains more than 150 chemicals, including small amounts of benzene and tetraethyl lead. Many of these are toxic; some such as benzene are carcinogenic. Gasoline presents a serious health hazard, which is rapidly being exacerbated by the increasing number of cars on the road.

Tuzla, an industrial town of about 165 000 inhabitants, is

located in north-east Bosnia and Herzegovina. The number of cars designated to be used with the leaded petrol has doubled in our area in the last ten months from 35 000 to 70 000 cars. Every day 50 workers employed at Tuzla gasoline stations work directly with gasoline. Workers exposure occurring during the pouring of gasoline result in chronic gasoline poisoning (2).

Many the harmful effects seen after exposure to gasoline, is due to the individual chemicals in the gasoline mixture, such as benzene, lead and oxygenates. Breathing small amounts of gasoline vapours can lead nose and throat irritation, headaches, dizziness, nausea, vomiting, confusion and breathing difficulties. Some effects of skin contact with gasoline include rashes, redness, and swelling. Allergic reactions (hypersensitivity) have been reported but these are rare occurrences (3-7).

Gasoline vapours can cause central nervous system (CNS) depression (8). Prolonged and repeated exposure to n-hexane can cause irreversible damage to the peripheral nervous system. Two types of nervous system action are seen: an acute narcotizing effect induced by high concentrations and axonal neuropathy associated with repetitive or continuous exposure to n-hexane (9).

Lead can produce adverse effects, not only on mental functions, virtually on every system of the body. Ninety percent or more of the lead in the body eventually accumulates in the bones and may stay there for a lifetime (10). In the United States, the Prevention Act of the Environmental Protection Agency (US EPA) passed regulations in 1973 to rule out the use of lead in gasoline (11). Such act has not been passed in European countries. The excretion of inorganic and total lead was investigated in the urine of workers who were exposed to tetraethyl lead (11-12). Long-term exposure has been shown to lead modified number of red blood cells (13). Lead, even at low levels of exposure, is now recognized to be toxic, and it is difficult to discern a threshold for lead toxicity in a population exposed to lead at levels nearly 200 times those experienced by pre-industrial humans (14). Almost all of the blood lead is in erythrocytes, from which it is taken up by soft tissues and stored primarily in bone. This substance has demonstrated half-lives for blood and soft tissues, which can be less than a month and long-term storage in bones with half-life of more than 20 years. The toxic effects of lead on the haemopoietic system, and kidneys are well known. Less clear, however, are the

toxic effects of this metal on the liver (13-16). As of now, there are still different views on the existence of negative effects of lead on the human and animal liver exposed to this metal. Chronic, heavy exposure to volatile leaded gasoline results in encephalopathy, cerebellar and corticospinal symptoms and signs, dementia, mental status alterations, and persistent organic psychosis (17). However, heavy metal lead may cause carcinogenic changes (18-19). Some studies in gasoline-exposed workers indicated an increased mortality risk from malignant melanoma. This was result of exposure to gasoline, benzene, or sunlight, or combination of these factors (20-23). Some laboratory animals that breathed high concentrations of unleaded gasoline vapours continuously for 2 years developed liver and kidney tumours. However, there is no evidence that exposure to gasoline causes cancer in humans.

Benzene is an aromatic organic hydrocarbon present in gasoline. Benzene toxicity involves both marrow depression and leukemogenesis caused by damage to multiple classes of hematopoietic cells and variety of hematopoietic cell functions (24-26). In milder forms of benzene toxicity individual cytopenias may occur: anaemia, leucocytopenia and thrombocytopenia (26-28). The production of benzene metabolites, largely in the liver, is followed by their transport to the bone marrow and other organs. The covalent binding of hydroquinone to spindle fibre proteins could explain inhibition of cell replication by benzene (29). Benzene and their metabolites were added impacts on oxidative stress and antioxidant factors (30).

The effects of lead are always the same regardless of whether it enters the body through breathing or swallowing. The no-effect level for lead is not known (31-35). The literature on the toxicology of lead provides little evidence of the neurotoxicity of organic lead (36). It has been well known for decades that organ-lead compounds are potent neurotoxins on the central nervous system. Triethyl lead, the major metabolite of tetraethyl lead, was shown to disrupt cytoskeletal elements, particularly neurofilaments, at very low levels (nanomolar concentrations) (37-45). Occupational exposure to gasoline has been associated with numerous neurological signs including effects on intellectual capacity, modifications of psychomotor and visual-motor functions and delayed memory (45-50). After long-term gasoline lead exposure, however behavioural effects are less well recognized.

Oxygenates are used as antiknock agents in place of lead derivatives and as substitutes for high octane aromatics in fuel (51-52). Oxygenates include substances such as ethanol, methanol, methyl tertiary butyl ether (MTBE), ethyl tertiary butyl ether (ETBE), tertiary butyl alcohol

(TBA), and tertiary amyl methyl ether (TAME). Short-term effects symptoms of oxygenates such as nausea, headaches, and sensory irritation were reported. Cancer effects tumours have been observed in multiple sites in rats and mice after exposure to high levels of MTBE. After high MTBE exposure, female mice showed increased incidence of hepatocellular adenoma and male rats showed increased incidence of renal tubular cell tumours and interstitial-cell testicular tumours (53-54). Methanol is well absorbed in humans following inhalation. It produces a transient mild depression of CNS with headache, vertigo, and vomiting. There is little evidence from available information of human health effects from low-level exposure which demonstrates that methanol vapours as a gasoline additive can cause acute adverse effects to health (55-57). Few studies have been conducted on the health effects of other oxygenates, such as ETBE or TAME. They deserve substantial investigation if they are likely to be placed in widespread use (58-59).

Gasoline engines always produce carbon monoxide (CO). When CO is inhaled it binds with haemoglobin, displacing oxygen and forming carboxyhaemoglobin in a lack of oxygen in the body cells. Continued exposure can lead to vomiting, loss of consciousness, brain damage, heart irregularity, breathing difficulties, muscle weakness, and even death (60).

Exposure to gasoline compounds and lead in our country has not been controlled. We studied the relationship between gasoline exposure and the effects of inhaled gasoline in a group of workers in gasoline stations, and evaluated a spectrum of potential exposure effects.

Subjects and methods:

A prospective cohort study was conducted in the Institute of occupational medicine Tuzla. Persons who had been exposed to gasoline for more than five years between 1985 and 1996 entered the program. Exposure to gasoline occurs by breathing vapours when filling cars fuel tank.

During refuelling, total hydrocarbon concentration in the air fall within the range of 20-200p p m by volume. In Tuzla the lead content in the petrol additives is 0.6 g/L. Benzene concentration in gasoline formulation is 3% by volume in unleaded gasoline and 2.7% in leaded gasoline currently used at Tuzla stations. (2) The ratio between the different gasoline mixture in 1997 was as following: leaded gasoline: unleaded gasoline: "diesel" gasoline (1:4:3). (2) About 110 million people are exposed to gasoline constituents in the course of refuelling at self-service gasoline stations (61), an operation that requires only a few minutes per week, amounting to approximately

100 min/year. Well, the workers at gasoline stations are exposed to gasoline constituents many times a week, approximately 8 086 800 min/year.

The study comprised 129 male participants divided into four groups. All the participants worked under special occupational conditions. As such, they are obliged to undergo a systematic medical check-up every year. Clinical and laboratory evaluations included medical / occupational history, a thorough physical exam, tests of cognitive and visual motor ability (Barnardet) and psychological symptom scale (Minnesota Multiphasic Personality Inventory) and Purdue Pegboard- manual dexterity test) (62). Haematological and biochemical examination also were conducted, ultrasound examination of kidneys and liver, complete blood cell counts, serum bilirubin concentrations (to 23µmol/L, normal range), liver enzymes, and qualitative urine analysis. Complete blood cell counts included: red cell count (RBC, $4.5-5.5 \times 10^{12}$ /L, normal range), total white cell count ($5-10 \times 10^9$ /L, normal range), absolute lymphocyte count (2500-3200, normal range), reticulocyte count ($0-15 \times 10^9$ RBC, normal range).

The long-term gasoline exposed group was composed of 37 petrol-station workers of an average age 41.5 ± 6.9 years, who had a chronic exposure to petrol and tetraethyl lead for 18.3 ± 8.4 years. The nitrous vapour exposed group was composed of 31 chemical workers who worked outdoors and who had accidental nitrous vapour poisoning. They were of an average age of 40.4 ± 6.2 and length of occupational time 18.5 ± 5.9 years. The non-exposed group consisted of 30 drivers and keepers in the hospitals. They were of an average age of 38.7 ± 9.8 and length of occupational time 16.3 ± 7.3 years. These persons served as a basic control group. The short-term gasoline exposed group consisted of 22 young men, whose average time of occupational exposure was only 9 months. No effects of exposure to petrol and organic lead were expected in this group and it was introduced as a control to the long-term gasoline exposed group. All subjects were men. The workers who left Tuula during the war (1992-1996) were not included. These subjects were co-workers with similar sociological-economic and intellectual backgrounds. All groups (except for the young workers at the gasoline stations) were comparable in other common characteristics including: age, gender (all males), level of education, and occupational history (ANOVA $P < 0.05$). The summary of morbidity of workers exposed gasoline was evaluated. Microscopic observations of peripheral smear were performed for search of basophilic stippling and neutrophilic toxic granulation. There was no activity, dietary or smoking restrictions during this time. At all the workplaces (all subjects work out), the conditions were unfavourable: temperature was too low in winter and too high in summer.

All subjects brought stressful life with a lot of traumatic events and psychological consequences for majority of workers who lived in the country during the war. Smoking status was classified as current smoker, former smoker, or no smoker.

All workers were also given a comprehensive psychological symptom (self-report) scale Minnesota Multiphasic Personality Inventory (MMPI). The test measures distress in eight psychological areas (hypochondria, depression, hysteria, psychosomatics, paranoia, psychoneurosis, hypermania, schizophrenia,) by having the patient rate. The test measures distress in 201 items ranging from the true to no true (63). Psychological analyses were done consulting the psychologist.

Statistical testing included Student *t* test and the Fisher's probability exact test. As multivariate technique a one-way analysis of variance (F-test) was carried out comparing the red cell count, total white cell count, differential blood cell count, reticulocyte counts, age, occupational exposure, and neurobehavioral tests. Results were compared Multiple range test (for the effects of these factors), paired Student *t* test. A relationship was designated as statistically significant when the *P* was < 0.05 and < 0.001 .

Results:

Peripheral smear revealed basophilic stippling and reticulocytosis in 25 of 37 in long-term gasoline exposed workers, and 15 of 22 in short-term gasoline exposed workers at gasoline stations group. The mean red blood cell count (RBC) in both gasoline exposed groups was lower ($4.55 \times 10^{12} \pm 0.21 \times 10^{12}$ /L) comparing to those in controls groups ($4.75 \times 10^{12} \pm 0.23 \times 10^{12}$ /L). The lowest number of RBC were found in the workers in long-term exposed group of workers 40 to 44 years of age, and who had 19 and more years of occupational history. These findings were statistically significant according to a multivariate analysis of variance ($P < 0.05$). Eleven of 37 long-term gasoline exposed workers suffered from decrease RBC, and 3 of 22 short-term gasoline exposed workers. The analysis of mean count of RBC and the mean numbers of age indicated that the lowest counts of erythrocytes had workers from 40-44 years (duration of exposure from 14-19 years). The data showed a statistically significant relationship between decrease RBC count and mean gasoline exposure period ($F = 8.75$; $P < 0.005$). Haemoglobin was in its reference values ($7.76-10.6$ mmol / L), and only one of gasoline exposed workers had lower value of haemoglobin.

The prevalence of leukocyte in long-term exposed workers is derived from the number of greater total white

blood cell (WBC) count - (the mean of WBC $8, 16 \times 10^9$ $\pm 1, 7$ of 37. During the test years, statistically significant differences in values of leukocytes between long-term gasoline exposed and non-exposed group (from Student *t*-test) ($P < 0.05$) occurred.

In twenty of 37 long-term gasoline exposed workers lymphocytosis (mean counts of lymphocytes 4100 ± 900 , table 3) was found. Statistically significant differences between groups in absolute lymphocytes count were found for all consecutive years (from ANOVA test $F=7.41$; $P=0.05$). There were no significant differences in absolute numbers of lymphocytes between lead exposed groups ($t=0.38$). Lymphocytopenia as indicator of benzene toxicity was found in 3 of 37 workers of long-term exposed group and in 3 of 22 short-term gasoline exposed workers.

Total bilirubin has been found in the normal range ($6, 8-20, 4 \mu\text{mol} \cdot \text{L}^{-1}$), but there were statistically significant differences between long-term exposed group and non-exposed group (from paired samples *t*-test) ($t=2.36$; $P < 0.05$).

By based ultrasound examination in 18 of 37 workers of long-term lead exposed group had liver damages. In 14 of 37 was found lipid degeneration of liver, in 3 of 37 chronic inflammation changes, and in 1 of 37 cirrhosis. Proteinuria was found in six of 37 of long-term lead exposed workers. From the study data it follows that 11 of 37 long-term lead exposed workers suffered chronic kidney damages.

The most frequent central nervous symptoms in long-term lead exposed workers was headache, fatigue, suspicious, sleep changes. The psychological testing analyses using the "Purdue Pegboard test" - standard manual dexterity test, showed that there were no significant differences between the groups (from ANOVA). The scores of "Purdue Pegboard" test were in normal levels of intellectual functioning in all groups. But, the results of "Hoandel" standard motor control test (among the groups from ANOVA) ($P < 0.05$; table 2) there was significant correlation between groups. It may mean that chronic gasoline exposed workers had difficulty in concentrating on various tasks because they needed significantly more time ($172, 0 \pm 69, 16$ sec) comparing to other groups. It means that gasoline exposed workers had the cognitive disturbances: decreased reaction time and motor abilities.

Results of a Minnesota Multiphasic Personality Inventory (MMPI) (which are presented in table 3), indicated that long-term gasoline exposed workers had statistically significantly different (higher) values than other groups (ANOVA) on the following MMPI scales: scale

of odd answers, control scale, scale of hypochondria, scale of depression, and scale of hysteria ($P < 0.05$).

The summary of morbidity, as shown in table 4, illustrates that long-term gasoline exposed workers suffered gastrointestinal and liver diseases (29 of 37), diseases of genitourinary system (19 of 37), cardiovascular diseases (18 of 37) and endocrine diseases and metabolism damages in 17 of the cases.

Discussion:

Lead in gasoline remains a major problem although the lead content has decreased in many countries in the last few years (18-22). In Tuzla the amount of tetraethyl lead in polluted air in the past years from 1985 to 1996 has been increasing fifteen times from 7501 kg to 115 584 kg.

In the long-term gasoline exposed workers were found multiple haematological effects: the decreased red blood cell counts (RBC), the red blood cells are microcytic (found in 18 of 37 workers), and the increased number of reticulocytes with basophilic stippling (in 22 of 37) during the entire period of testing. Also, decreased RBC count was found in 31 of 37 of the long-term gasoline exposed workers, and in 3 of 22 short-term gasoline exposed workers working at gasoline stations. This is an agreement with the results obtained by other authors (33, 35, 40).

Amongst long-term exposed workers, the presence of neutrophilic toxic granulation was in 8 of 37. The research on the workers exposed occupationally to lead has shown significant chromosomal and chromosomal aberrations in peripheral blood lymphocytes (31,43,49).

Lymphocytosis was found in 20 of 37 long-term lead exposed workers. Lymphocytosis was not usually found in case of lead exposure. Probable, lymphocytosis has been associated by variation by a wide variety of processes including minor viral infection or from multiple chemical exposure in workers at gasoline stations exposed to different gasoline compounds too.

The phenomenon is not a single event but a complex interplay of gasoline constituent exposure (organic solvent, tetraethyl lead and other) as revealed by increased lymphocyte count. It is possible that exposure to gasoline constituents or tetraethyl lead may cause an impairment in concomitant stimulation of humoral immunity (19, 21, 42, 46.). The results have shown the presence of the lymphocytopenia (minimal count 1200) in three of 37 long-term gasoline exposed workers (in every evaluated years) and in three out of 22 short-term gasoline exposed workers. Decreased count RBC and leukopenia were found in

the same workers, and the reason is due to exposure to benzene (24,29,30,31). Haematological effects of exposure to gasoline are early sign of the gasoline constituent toxicity (seen in short-term exposed group).

So far, there are still different views on the existence of negative effects of lead on the liver of people and animals exposed to this metal (15-16). The results of the study analysis indicated liver damages in 18 of 37. Lead may play a role in the liver damages, but exposure to compounds of gasoline too.

Lead may produce a chronic interstitial nephropathy, most commonly with blood lead levels greater than 60 mg/dl (44). Restek-Samarzija and colleagues suggested that lead poisoning in the past, overall duration of lead exposure, and age as a major confounding variable related to aging process of the kidney (45). Chronic, recurrent lead poisoning with a consequently increasing lead body burden can cause impairment in renal function and a concomitant of humoral immunity (46). In long-term lead exposed workers was found chronic damages of kidneys in 11 of 37 and proteinuria in 6 of 37. This result is in accordance with the results obtained by other authors (47).

The somatic symptoms most commonly described were headache and fatigue in gasoline exposed workers. Fifty-eight workers were evaluated for potential health effects related to organic and inorganic lead exposures. Findings for which no alternative medical explanations could be found included neurobehavioral abnormalities (18 of 39 workers) and sensorimotor polyneuropathies (11 of 31 workers) (47).

The literature on the toxicology of lead provides little evidence of the neurotoxicity of organic lead compounds, but n-hexane may produce polyneuropathies too (9). Tetraethyl lead induced changes in the concentration of glial fibrillary acidic protein (31,43). Organic lead stimulates an increase of free arachidonic acid in HL-60 cells. Influence of metabolic inhibitors on metal induced arachidonic acid liberation (31). Chronic, heavy exposure to leaded gasoline results in an encephalopathy, cerebellar and corticospinal symptoms and signs, dementia, mental status alterations, and persistent organic psychosis. Lead chelation therapy is not rational and has not been shown to benefit these patients (48).

Further evaluation in this group of long-term gasoline exposed workers of the neuropsychological effects is underway, but long-term effects are evident in 11 out of 37 (depression-hypochondria symptoms). These symptoms were quantified over time with the use of the MMPI as the relating scale, the gasoline exposed workers scores markedly abnormal and remained so throughout a period of long follow-up, indicating persisting subjective dis-

tress. Other measures of neuropsychological function that assessed cognitive, visual and motor skills indicated impairment at the time of initial testing. Purdue Pegboard scores, which was not significantly different from population norms, but Bonardel scores worsened significantly in long-term exposed group. This phenomenon is very complex interplay of gasoline constituents poisoning (tetraethyl lead, n-hexane, MTHF, CO, methanol and other), smoking and alcohol consumption, as risk factors for neurological and psychological disorders (52, 53, 57, 58, 60). Cortical atrophy was more frequently seen in the gas station employees group.

These results suggest the contribution of leaded gasoline to its development (64). None of these workers visited a doctor for psychological abnormalities of any kind. Long-term lead exposed workers had difficulty in concentrating on various tasks. Schwartz and the associates had similar finding 1993 (50). Also, effects on behavioural changes, memory and psychomotor ability were seen in long-term gasoline exposed group. The significant effects were seen in workers exposed for more than 5 years.

Finally, the results of summary of diseases (table 4), showed that long-term lead exposed workers suffered from diseases that are probably caused by leaded gasoline exposure, such as gastrointestinal disorders, diseases of liver, diseases of genitourinary system, cardiovascular diseases and endocrine diseases. This phenomenon has been described by others authors (6). High prevalence of cardiovascular disease may be relatively signs of carbon monoxide toxicity. People with coronary artery disease are particularly sensitive to this effect because they have impaired ability to increase coronary blood flow. When blood flow through the heart is not sufficient to meet to oxygen demand, the heart becomes ischemic, resulting in chest pain or ECG changes. Even relatively low CO levels may bring on ischemia more quickly for some individuals with coronary artery disease (60).

Conclusions:

The health effects of being exposed to gasoline are usually exposed to many other things that also can cause health effects. This study clearly demonstrates that gasoline workers exposed workers suffer long term health problems including psychological disorders, mild haematological disorders, kidneys damage and liver damage.

Given the conclusive evidence on the negative effects of leaded gasoline that have been collected worldwide and the specific results of this study conducted in Turin, it is clear that action must be undertaken immediately to protect public health. Preventive measures should be proposed and implemented to develop adequate procedures

for reduction of lead in petrol. Such measure would protect both gasoline workers and larger public. As gasoline station workers are particularly at risk, they should be encouraged through an information campaign to pay scrupulous attention to personal hygiene especially hand washing to prevent the absorption of tetramethyl lead and other gasoline constituents through the skin. Thus, the

problem regarding the potential toxicity of gasoline is still open, even though it is clear that modern unleaded gasoline present less risk to human health due to the lower quantities of benzene and lead. Today is the insufficient data for determination of long-term effects in occupationally exposed cohorts and inadequate information on general population exposure.

Table 1 Summary of peripheral blood parameter counts detected by all evaluated workers (except younger workers at gasoline stations)

Parameters (referent range)	Long term gasoline exposed		Nitrous vapour exposed		Non exposed	
	N=27		N=31		N=50	
Leukocyte ($3-10 \times 10^9/L$)	8.12 +/-	3.54 *	6.37 +/-	1.28	6.43 +/-	1.34
Lymphocyte (2500-3500)	4100 +/-	900 *	3600 +/-	400	3600 +/-	200
Total bilirubin (0.8-20, 4 $\mu\text{mol/l}$)	11.97 +/-	3.81	9.98 +/-	3.20	9.18 +/-	3.22
Aspartate aminotransferase (< 370 nkat/l)	534.9 +/-	374.47 *	374.3 +/-	104.9	346.3 +/-	102.3
Alamine aminotransferase (< 750 nkat/l)	688.4 +/-	208.8 *	542.4 +/-	283.9	524.6 +/-	214.1
ANOVA P< 0.05*						
Results are presented as mean +/-SD						

Chronic occupational exposure to gasoline may cause bone marrow injury and haematopoietic toxicity including leukocytosis, and lymphocytosis. Results were compared to control groups from ANOVA (P<0.05).

Table 2 The mean scores (sec) of "Bonardel" for all groups:

GROUPS	"Bonardel" score (sec) (M +/- SD)	F
Long term lead exposed	172.0 +/- 69.16	F=3.43*
Short term lead exposed	118.4 +/- 43.05	F=3.45*
Nitrous vapour exposed	118.3 +/- 27.22	
Non exposed	117.7 +/- 42.4	
ANOVA *P< 0.05		

A difference in "Bonardel" scores between groups (from ANOVA) indicated that long-term lead exposed workers needed significantly more time for realization of test. It means that lead poisoning workers had difficulty in concentrating on various tasks.

Table 3 The mean Minnesota Multiphasic Personality Inventory scores for all groups (except younger workers at gasoline stations)

PERSONAL INVENTORY	Long term gasoline exposed (N=37)	Short term gasoline exposed (N=22)	Nitrous vapour exposed (N=31)	Non exposed (N=30)
Scale of sincerity	60.2±10.5	65.1±9.5	59.2±9.7	59.4±9.8
Scale of capriciousness	48.5±9.4 *	42.9±6.3	44.7±5.8	45.3±5.8
Scale of strength	52.3±7.9	59.3±8.0	54.5±7.6	54.6±7.5
Hypochondria	57.6±14.1 *	49.3±11.3	46.9±7.9	46.9±8.1
Depression	56.8±11.3*	44.0±11.2	49.4±7.6	49.7±7.4
Hysteria	57.6±10.6 *	45.9±8.8	47.8±7.5	48.0±7.5
Psychopathy	46.4±9.5	47.3±9.8	45.5±7.9	45.3±7.6
Paranoia	50.0±15.7 *	47.1±16.2	48.0±10.4	47.9±10.0
Psychastenia	52.3±11.3 *	49.2±9.9	49.1±9.8	49.2±9.9
Schizophrenia	50.8±11.1 *	48.6±8.3	47.3±9.7	47.4±10.0
Hypomania	54.3±9.2	54.9±8.6	52.4±8.9	52.3±9.0

ANOVA *P<0.05

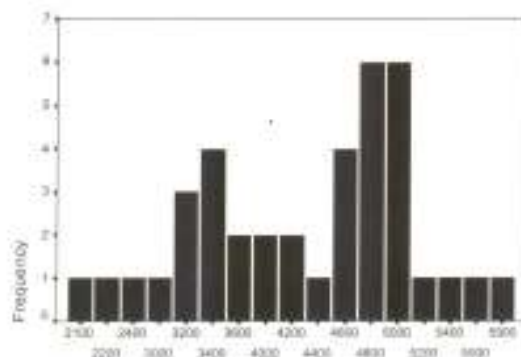
In 13 of 37 long-term gasoline exposed workers was identified high scores for following MMPI scales: depression, hypochondria, hysteria, paranoia, psychastenia, and schizophrenia (P<0.005) compared to other control groups. Results are presented as mean ±SD.

Table 4 Diseases of all evaluated workers (except younger workers at gasoline stations)

Diseases	Long term gasoline exposed (N=37)	Nitrous vapour exposed (N=31)	Non exposed (N=30)
Infective and viral diseases	2	1	1
Tumors	1	-	-
Anaemia	11*	1	*
Endocrine diseases	17*	3	3
Mental diseases	2	1	1
Neurological diseases	3	1	1
Eye diseases	10*	3	8
Ears diseases	-	7	1
Cardio-vascular diseases	18*	7	5
Respiratory diseases	6	7	4
Gastro-intestinal diseases	29*	8	4
Skin diseases	3	8	1
Bone diseases	9	7	5
Genitourinary diseases	19*	4	1
Injuries	2	3	1

ANOVA P<0.05* -Results are presented as mean ±SD.

Figure 1: Bar Chart: distribution of lymphocyte frequencies in long-term exposed gasoline group in 1997



This figure indicated that lymphocytopenia as an indicator of benzene toxicity are presented in only 3 cases, whereas lymphocytosis in 25 cases. There was no significant correlation relationship between lymphocyte count and duration exposure time (ANOVA, $F=0.205$; $P=0.653$; ns).

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Sažetak

Profesionalna izloženost benzinu i njegovim komponentama ne nadirne se u našoj zemlji. Cilj rada bilo je dugoročno otkrivanje zdravstvenih poremećaja u 37 radnika koji rade na benzinskim stanicama i koji su kronično izloženi benzinu više od pet godina. Prospektivna studija tipa kohorte provedena je u Zavodu za Medicinu rada Tuzla od 1985-1996 godine. Praćeni su slijedeći parametri: godišne starosti, laboratorijske analize krvi i urina, istorija bolesti, fizikalni pregled, radni staž (istovjetan periodu izloženosti), ultrazvučne preglede bubrega i jetre, rezultat psihološkog testiranja. Svaki parametar je bio uspoređivan sa istim u dvije kontrolne grupe: 61 benzinsko neizloženih radnika. Da bi uočili rane neželjene zdravstvene učinke izloženosti benzinu pratili smo 22 radnika zaposlena na benzinskim stanicama devet mjeseci. U Tuzlanskom području aerotagađenje olovom je porasla petnaest puta od 1985 do 1996. godine. Snižene vrijednosti eritrocita na?eme su u 11 od 37 benzinsko izloženih radnika i u 3 od 22 mlada radnika. One potiču stvaranje novih mladih stanica, po se u razmazu periferne krvi nalaze bazofilne punkcije i retikulocitoza. U radnika, dugoročno izloženih benzinu, otkrivena je: blaga leukocitoza, sniženje vrijednosti eritrocita, limfocitoza, limfocitopenia (3 od 37), kronična oštećenja bubrega (8 od 37), masna promjena jetre (14 od 37), kronična funkcionalna oštećenja jetre (3 od 37), i ciroza jetre (1 od 37). Izloženost benzinu ne izaziva karakteristične lezije na organima. Rezultati studije bili su značajno različiti u kronično izloženih benzinu i u usporednim skupinama ($P < 0.05$). U 13 od 37 radnika identificirani su simptomi somatskih poremećaja ličnosti. Zdravstvenim poremećaji u radnika kratko izloženih benzinu (devet mjeseci) bili su: limfocitoza i blaga odstupanja na psihološkom planu (2 od 22) u odnosu na kontrolne skupine. (Diskutiran je morbiditet radnika).

KLjučne riječi: profesionalna izloženost, efekat dugoročne izloženosti, tetraetilni olovo, sastavci benzina, oksigenati, hematološki efekti, psihološki efekti.