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Acute High Dose Exposure to Benzene in Shipyard Workers

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Fifteen degassers were acutely exposed over several days to high concentrations (>60 ppm) of benzene during removal of residual fuel (degassing) from shipboard fuel tanks. Medical surveillance evaluation mandated by the Occupational Safety and Health Administration's (OSHA) Benzene Standard initially revealed 11 workers (73%) reporting neurotoxic symptoms while degassing. Workers with more than 2 days (16 hours) of acute exposure were significantly more likely to report dizziness and nausea than those with 2 or fewer days of acute exposure. Repeated laboratory analyses performed over a 4-month period after the acute exposure revealed at least one hematologic abnormality consistent with benzene exposure in 9 (60%) of these degassers. One year later, 6 workers (40%) had persistent abnormalities; an additional worker with normal hematologic parameters at the time of our initial evaluation subsequently developed an abnormality consistent with benzene exposure. Numerous large granular lymphocytes were observed on 6 (40%) of the peripheral blood smears. Despite these laboratory findings, there were no significant associations between the presence of hematologic abnormalities and either the number of hours of acute benzene exposure or the duration of employment as a degasser. Volatilization of benzene from the residual fuel was the suspected source of benzene in the headspace of tanks. Confined space exposure to petroleum products may be exposing workers to benzene at levels above the OSHA Short-Term Exposure Limit (STEL). This situation warrants further study.

Key words: confined spaces, leukemia, aplastic anemia, aromatic hydrocarbons, solvents, degassing, fuel tanks, petroleum products, hematologic abnormalities, large granular lymphocytes, benzene exposure, shipyard work

INTRODUCTION

Benzene is an organic solvent widely used in the chemical industry and found in petroleurn products [Oak Ridge National Laboratory, 1989; Nelson, 1958; Laskin

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and Goldstein, 1979; Rappaport et al., 1987; Runion, 1975; Spear et al., 1987; Brief et al., 1980; King, 1988; Rothman and Emmett, 1988]. Some petroleum product components, specifically n-hexane. benzene, toluene, and xylenes are known to produce neurotoxic symptoms (drowsiness, headache, and dizziness) during acute high level exposures. In addition to affecting the central nervous system, acute high dose benzene exposure has been associated with hematologic abnormalities. Lymphocytopenia and an increase in the mean red cell corpuscular volume (MCV) are the earliest indicators of benzene toxicity [Goldwater, 1941; Goldstein, 19881. Other early heniatologic manifestations of benzene exposure include leukopenia, anemia, and thrombocytopenia [Goldstein, 1988; Goldwater, 1941; Bowers, 1947; Aksoy et al., 1987].

Workers subject to chronic occupational benzene exposures are at risk for developing pancytopenia, aplastic anemia, multiple myeloma, acute myelogenous leukemia, and other blood dyscrasias. These hematologic abnormalities and malignancies have been documented mainly in workers employed in shoe factories and the rubber industry [Goldstein, 1988; Aksoy, 1980; 1985a,b; Infante et al., 1977; Rinsky et al., 1987; Wong, 1987]. More recently, two cases of acute myelogenous leukemia were reported in seamen, one of whom frequently performed tank-cleaning operations [Nilsson et al., 19881.

Medical surveillance is required by the current Occupational Safety and Health Administration (OSHA) Benzene Standard after an emergency exposure, i.e., for exposures above the current permissible exposure limit (PEL) of 1 ppm as an 8-hour time-weighted average (TWA) and above the action level of 0.5 ppm averaged as an 8-hour TWA [United States Department of Labor, 19881.

This paper describes medical surveillance results for 15 degassers acutely exposed to levels of benzene ranging from 60–600 ppm.

REPORT OF INCIDENT

In October 1988, 15 degassers worked for several days without respiratory protection in fuel tanks of a ship in which the concentration of benzene exceeded the OSHA PEL. The exposure occurred while workers were performing degassing (or gas-freeing), a process involving the use of pressurized hot water to remove residual fuel from tanks. Gas-freeing must be performed before "hot work" (welding, riveting, burning), or tank repair can commence. The only protective gear usually worn at this worksite during this operation were boots and a rain suit.

Prior to tank entry, the company's safety officer checked the tanks for oxygen content and flammable atmospheres according to the OSHA Shipyard Industry Standard requirements [United States Department of Labor, 1983]. The oxygen concentration was at least 19.5% in each tank [personal communication, Health and Safety Officer]. However, the tanks were not tested for toxic atmospheres as required by this Standard, because the safety officer did not consider marine diesel fuel to be toxic.

After several days of gas-freeing, a marine chemist began inspection of tanks to certify them for "hot work." He noted the presence of benzene, in both cleaned and uncleaned tanks, using Drager equipment. At this point, these 15 men were required to stop working in the tanks.

The company's medical consultants ordered laboratory testing for the exposed

workers which included complete blood counts. Post-shift urinary phenol levels were not obtained (as required by OSHA law) on the day the benzene exposure was detected.

MATERIALS AND METHODS

Hematologic profiles for the 15 degassers were obtained and analyzed by a local commercial laboratory within 2 days of the acute exposure. Approximately 1 month after the acute exposure, the workers were referred to the Johns Hopkins Occupational Medicine Clinic for evaluation. Our initial assessment included administration of a questionnaire to obtain medical and occupational histones, a physical examination, and repeat laboratory testing which was analyzed at our hospital laboratory. The questionnaire included questions pertaining to past medical and occupational histones, smoking habits, and amount of current alcohol consumption. Each worker was asked how many days he worked in the benzene-contaminated tanks and the number of hours per day worked. In addition, the total number of years employed as a degasser was obtained for each worker. Workers were asked about symptoms they experienced while working in the contaminated tanks. Information regarding the use of personal protective gear was also obtained. Our initial blood work included a complete blood count (CBC) with white blood cell (WBC) differential count. Review of peripheral smears by a hematologist was performed only on the blood specimens obtained during our initial evaluation. Monthly hematologic profiles were obtained and analyzed by our laboratory for the subsequent 3 months.

One year after the acute exposure, we reevaluated these 15 degassers. Medical and occupational histories were obtained and physical examinations and repeat blood work were performed. Four workers with persistent hematologic abnormalities were referred to a hematologist for consultation.

Statistical Analyses

Hematologic profiles for each worker were tabulated for each of the five measurement times. Statistical analyses were performed using SAS version 6.06 [SAS Institute, Inc., 1990]. Due to the wide dispersion of hematologic exposure and alcohol data and the small sample size, nonparametric statistical methods were employed for most analyses. For analyses requiring parametric methods, log transformations were utilized. Spearman's correlation and Fisher's exact test were used to determine the association between the number of hours of acute exposure and the presence of symptoms. The Wilcoxon rank sum test was used to assess the relationship between duration of employment as a degasser, number of hours of acute benzene exposure, and the presence of at least one hematologic abnormality. This test was also used to evaluate whether workers with one hematologic abnormality (WBC count less than 4,500) were likely to have other abnormal hematologic values. Spearman's correlation was used to assess correlations between the five hematologic parameters at each time period. In addition, repeated measures analysis [Cole and Grizzle, 1966] was used to assess whether hematologic parameters changed over time, and whether benzene exposure or level of alcohol consumption influenced these changes. Statistical significance was set at the 0.05 level.

Because baseline hematologic profiles were not available, comparisons with pre-exposure values could not be performed. In addition, the daily mean laboratory

values for the laboratory performing the hematologic profiles were not available at either our hospital laboratory or the commercial laboratory. Hence, comparisons of our workers' laboratory values with laboratory means could not be performed.

RESULTS

Industrial Hygiene Sampling

The marine chemist detected benzene in the headspace of cleaned and uncleaned tanks which had contained marine diesel fuel. Testing was initially performed using a Drager pump with benzene 2/a detector tubes. Multiple samples collected in this manner revealed benzene concentrations greater than *60* ppm [personal communication with marine chemist]. These detector tubes also produce positive readings when xylene or toluene are present at concentrations above 200 ppm. To determine whether benzene was present, another sample was obtained by lowering a charcoal tube into the headspace of an uncleaned tank and taking a sample using a bellows pump. The tank sampled contained about 2,000 gallons of marine diesel fuel [personal communication with Health and Safety Officer]. A commercial laboratory performed sample analysis by gas chromatography. The total benzene concentration was found to be 653 ppm. Analysis of the back-up section of the tube revealed a benzene concentration more than 25% of that in the front section, implying that ''breakthrough'' had occurred and that the actual concentration of benzene may have been higher. Analyses for other volatile components of the fuel oil were not performed. A bulk sample of the fuel (taken from the same tank) was sent to the same laboratory for analysis; the amount of benzene present in the sample was less than the detection limit of 0.01% (100 ppm).

A site visit aboard the ship revealed that the tanks were confined spaces as defined in the Shipyard Industry Standard [United States Department of Labor, 1983]. Specifically, the tanks had: I) limited openings for entry and exit; (2) unfavorable natural ventilation; and 3) a design not compatible with continuous worker occupancy.

Demographic Information

The exposed population included 15 men, ranging in age from 29–62 years with a mean age of 42 years at the time of our initial evaluation. Fourteen subjects were black and one was white. Duration of employment as degassers ranged from 3 months to 10 years, with a mean and median of 4 years. Eight of the fifteen workers reported consumption of more than 10 alcoholic drinks per week. An alcohol score, based on reported current alcohol consumption, is presented for each worker.

Duration of Exposure

Work in the benzene-contaminated tanks spanned time periods ranging from 1 day to 3 weeks with a mean of *5* days (median 3 days). The length of time worked each day within the tanks ranged from 2.5–8 hours with a mean of 5.5 hours (median *5* hours). The total time spent degassing within the tanks ranged from 3–150 hours with a mean of 27 and median of 16.5 hours, respectively. Table I lists the duration of acute exposure and duration of employment as a degasser for each worker.

TABLE I. Duration of Acute Benzene Exposure and Length of Employment by Worker

Worker	Duration of acute exposure (hours)	Length of employment (months)
1	24	72
2	8	72
3	25	48
4	13.5	24
5	16	72
6	40	108
7	3	4
8	20	72
9	30	3
10	16	36
11	17.5	36
12	16.5	3
13	8	120
14	150	12
15	16	72
Mean (S.D.)	26.9 (35.3)	50.3 (37.5)
Median (range)	16.5 (3.0–150)	48.0 (3.0–120)

TABLE II. Symptoms Reported by 15 Degassers

Symptom	Number of workers (percent)
1. Mucous membrane irritation[a]	12 (80)
2. Peculiar or strong odor	11 (73)
3. Dyspnea	10 (67)
4. Dizziness or lightheadedness	9 (60)
5. Nausea	7 (47)
6. Chemical taste	7 (47)
7. Headache	5 (33)
8. Cough	4 (27)
9. Drowsiness	3 (20)
10. Fatigue	3 (20)
11. Skin irritation	2 (13)

[a]Includes reports of irritation of the eyes, nose, throat, and mouth.

Symptoms

The workers reported multiple symptoms while working in the benzene-contaminated tanks including symptoms of mucous membrane irritation and central nervous system depression. Table II displays the number and percent of workers reporting each symptom. Symptoms of mucous membrane irritation were reported by 80% of the workers. Eleven (73%) of the degassers stated that they noted a "peculiar" or "strong" odor in the tanks of this ship as compared to tanks in other ships. In addition, about half of the workers stated that they experienced a "chemical taste" which lasted for several days and was more noticeable with eructation.

We evaluated the relationship between duration of acute exposure and the number and types of symptoms reported. Using Spearman's correlation, no correla-

tion was found between the number of symptoms reported and duration of acute exposure. However, when workers were categorized into two exposure groups (2 days or less vs. more than 2 days [16 hours] of acute exposure), those in the higher exposure category more frequently reported dizziness ($p = 0.04$) and nausea ($p = 0.03$).

Hematologic Findings

Table III presents the hematologic profiles by date for each of the 15 degassers. Although no consistent trends are apparent during the 1 year follow-up time, 9 of the 15 workers had at least one hematologic abnormality. The peripheral smears were remarkable for the presence of numerous large granular lymphocytes (LGLs) in 6 (40%) of the workers.

No significant differences were found when comparing the duration of acute benzene exposure and the duration of employment as a degasser for workers with at least one hematologic abnormality (leukopenia, anemia, thrombocytopenia, and elevation of the MCV) vs. those without abnormalities. Neither the duration of acute benzene exposure nor the length of employment as a degasser were significantly different in workers with numerous LGLs on peripheral smear vs. those without such findings. When comparing workers with at least one hematologic abnormality with those with normal hematologic profiles, no differences in alcohol consumption were detected. Additionally, there was no difference in alcohol score for those workers with numerous LGLs as compared to those without such findings.

Hematologic parameters (WBC count, absolute lymphocyte count, MCV, hemoglobin, and platelet count) were analyzed to assess correlations at each time period using Spearman's correlation. No consistent patterns of correlation were observed.

To assess whether workers with one hematologic abnormality were likely to have statistically different levels of other blood counts, we compared workers with abnormal WBC counts ($n = 4$) to those with normal WBC counts ($n = 11$) for all other hematologic parameters at each time period. We chose an abnormality of the WBC count as a classification criterion because it was the most frequently occurring abnormality. Using the Wilcoxon rank sum test, those workers with abnormally low WBC counts had significantly lower MCVs at time 3 ($z = -2.40$; $p = 0.02$) as compared with workers with normal WBC counts. There were no significant differences between these two groups for hemoglobin or platelet count at any time period.

Using repeated measures analysis, we assessed whether hematologic parameters changed over time and whether exposure affected these changes. No significant changes in hematologic parameters over time were observed using the Greenhouse-Geyser correction [Vasey and Thayer, 1987]. Acute exposure and duration of employment were not significantly related to any of the levels of the five hematologic parameters studied, even after controlling for alcohol consumption. The repeated measures analysis excludes individuals with missing data at any one or more time periods. Hence, in studying absolute lymphocyte counts over time, the laboratory data for only seven workers were included in the analyses; for the other four hematologic parameters, the data for nine workers were used in the analyses.

DISCUSSION

We evaluated 15 degassers who were acutely exposed to high concentrations of benzene. Workers with more than 2 days of acute exposure were significantly more

likely to report dizziness and nausea than those with 2 or fewer days of acute exposure. Although sampling data were not obtained while the workers were degassing these tanks, their symptoms were consistent with exposure to benzene concentrations well above the OSHA PEL of 1 ppm. The odor threshold for benzene is 2 ppm. Symptoms of central nervous symptom depression such as dizziness, which was significantly more frequent in workers exposed for more than 2 days in this study, begin to occur at levels between 20 and 80 ppm, while fatalities occur at 10,000ppm [Oak Ridge National Laboratory, 1989].

Other components of fuel oil which could produce similar symptoms include n-hexane, xylenes, and toluene; however, analysis was performed only for benzene. Hence, it is possible that fuel components other than bezene could have contributed to the workers' symptoms.

Because benzene is highly volatile, a low concentration in the liquid phase in a confined space can result in a high ambient concentration. The concentration of benzene which could accumulate in the headspace of a fuel tank is proportional to the concentration of benzene in the liquid phase, the ambient temperature, and its vapor pressure. For example, at the limit of detection in the liquid fuel (0.01% benzene), the concentration of benzene which would be found in the headspace (at sea level and 68°F) at equilibrium is 987 ppm as shown in the following equation [Patty, 1958]:

$$\text{PPM} = 0.01 \times \frac{75\ \text{mmHg}}{760\ \text{mmHg}} \times 10^6 = 987\ \text{ppm},$$

where 75 mmHg is the vapor pressure of benzene at 68°F; 760 mmHg is the atmospheric pressure; and 10^6 is the conversion factor to ppm.

Benzene has been detected in cleaned fuel tanks in the past, both in ships at concentrations between 2 and 10 ppm [personal communication with marine chemist], and in above-ground gasoline storage tanks at levels above 1 ppm [Powers, 1990].

Following inhalation exposure to benzene, it is partially eliminated unchanged in the exhaled air [Brugnone et al., 1989; Berlin et al., 1980] and in metabolized form in the urine [Oak Ridge National Laboratory, 19891. Researchers [Berlin et al., 19801 have demonstrated that benzene is eliminated in the breath in at least two phases: a fast phase with a half-time of 2.6 hours, and a slower phase with a half-time of about 24 hours. Benzene has been detected in the breath of workers for up to 120 hours (5 days) after occupational exposure to benzene ceased [Berlin, 1985]. The dizziness reported by workers exposed for more than 16 hours may be explained by their accumulation of a body burden at which dizziness would be noted, whereas, workers exposed for less than 2 days would have been able to eliminate any accumulated dose. In the current study, 7 of the 15 degassers reported a "chemical taste" which lasted for several days after their exposure to the benzene-contaminated fuel ceased. It is possible that this description of a "chemical taste" corresponds to the elimination of benzene through exhaled air.

The increased frequency of nausea and dizziness in workers exposed more than 2 days vs. those exposed 2 days or less is biologically plausible since dose to the target organ (central nervous system) is a function of ambient concentration, and duration of exposure. The association between duration of exposure and symptoms

TABLE III. Hematologic Profiles by Date and Alcohol Scores for 15 Degassers

Worker No./ hematologic parameters	Time 1 10188	Time 2 11/88	Time 3 12/88	Time 4 1/89	Time 5 Fall 1989[b]		Peripheral smear[j]	Alcohol score[l]
1								
WBC[a]	5,000	3.800	4.400	4.700	4.700	RBC:	NC. NC; few target	0
ALC[c]	—[d]	1.311	1.628	2.068	1.457		cells: microcytosis	
HBG[f]	13.9	14.2	13.9	13.7	14.0	PLT	abundant, some large	
MCV[f]	67.6	67.5	67.9	67.5	67.3	WBC:	normal-appearing;	
PLT[g]	221	206	213	222	269		numerous LGLs	
2								
WBC	5,600	4.300	4,000	4,000	4.200	RBC	NC. NC; few	0
ALC	3,080	1.858	2,200	1,880	1,680		elliptocytes	
HBG	13.4	15.4	15.2	15.2	14.6	PLT	adequate numbers	
MCV	82.5	82.9	84.1	87.9	82.5	WBC:	normal-appearing;	
PLT	215	208	199	207	210		numerous LGLs	
3								
WBC	8.200	8,300	7,100	8.300	8.500	RBC:	NC. NC; few	2
ALC	2.624	2,515	2,560	3,240	2.720		microcytic cells	
HBG	14.8	15.2	14.9	15.8	15.0	PLT	adequate numbers	
MCV	95.9	95.3	96.5	95.3	94.0	WBC	normal-appearing;	
PLT	224	206	250	250	223		rare LGLs	
4								
WBC	3.500	5.100	N/S	4,000	4,000	RBC:	NC, NC; few	2
ALC	1,750	2.270	N/S	1,560	1.680		microspherocytes	
HBG	17.0	16.4	N/S	15.3	15.7	PLT	adequate numbers	
MCV	97.5	96.3	N/S	Y5.8	96.8	WBC:	normal-appearing;	
PLT	257	264	N/S	338	230		frequent LGLs	
5								
WBC	4,300	4.100	5,000	4.400	4,100	RBC:	NC. NC; mild anisocytosis	1
ALC	2.494	1.849	1,900	1,892	2,255		and poikilocytosis	
HBG	13.6	14.2	13.6	13.8	13.0	PLT	adequate numbers	
MCV	86.8	84.8	84.5	84.9	84.9	WBC:	normal-appearing;	
PLT	250	263	229	248	257		numerous atypical lympho-cytes; rare LGLs	
6								
WBC	7,000	6,200	6,000	6,800	6,600	RBC:	NC. NC; rare basophilic	2
ALC	—[i]	2.760	2.770	2,180	2.970		stippling	
HBG	13.8	14.2	14.1	14.2	14.9	PLT:	adequate numbers	
MCV	101.6	100.0	102.3	100.4	98.3	WBC:	normal-appearing;	
PLT	224	215	235	248	257		numerous LGLs	
7								
WBC	5,000	7,300	6.800	5,200	NIS	RBC:	NC. NC; rare	3
ALC	2.150	1,745	1.840	2.132	N/S		microspherocyte	
HBG	16.5	16.6	15.3	15.6	N/S	PLT:	adequate numbers	
MCV	94.3	94.1	93.9	94.6	N/S	WBC:	normal-appearing;	
PLT	88	100	123	95	N/S		occasional atypical lymphocytes; rare LGLs	
8								
WBC	6,600	6.800	7.300	6,500	5,900	RBC:	NC, NC	I
ALC	2,442	2,650	1,168	1,495	2,006	PLT	adequate numbers	
HRC	13.8	14.5	14.2	14.1	14.1	WBC:	normal-appearing;	
MCV	93.1	92.4	93.2	94.2	92.8		numerous LGLs	
PLT	193	198	171	212	190			

(conrinued)

TABLE III. Hematologic Profiles by Date and Alcohol Scores for 15 Degassers (Continued)

Worker No./ hematologic parameters	Time 1 10/88	Time 2 11/88	Time 3 12/88	Time 4 1/89	Time 5 Fall 1989[b]	Peripheral smear[j]	Alcohol score[l]
9							
WBC[c]	7,100	6,500	6,500	NIS	6,100	RBC:[k] NC. NC	2
ALC[c]	2,059	2,515	2,800	N/S	2.013	PLT: adequate numbers	
HBG[e]	14.2	14.9	14.3	N/S	15.1	WBC: normal-appearing;	
MCV[f]	87.4	86.7	85.5	N/S	86.3	occasional LGLs	
PLT[g]	281	278	280	N/S	314		
10							
WBC	9.400	7.300	9,200	N/S	8,400	RBC: NC, NC	0
ALC	3,854	3,300	4,420	N/S	3,108	PLT: adequate numbers	
HBG	15.6	15.3	15.0	N/S	14.7	WBC: normal-appearing;	
MCV	86.5	87.7	88.2	N/S	87.1	occasional to numerous LGLs	
PLT	399	359	358	N/S	318		
11							
WBC	7,800	6,800	N/S	6,600	8,800	RBC: NC, NC; rare fragmented cell	1
ALC	1,638	2,430	N/S	1,584	1,848	PLT: adequate numbers	
HBG	15.8	15.4	N/S	13.1	16.0	WBC: normal-appearing;	
MCV	101.5	100.5	N/S	99.2	96.5	occasional LGLs	
PLT	151	144	N/S	152	—[i]		
12							
WBC	8,100	14,100	N/S	6,700	—[h]	RBC: NC, NC; mild anisocytosis	2
ALC	2,106	2.637	N/S	1,943	—[h]	and poikilocytosis	
HBG	14.3	16.5	N/S	15.0	—[h]	PLT: adequate numbers	
MCV	93.5	91.9	NIS	90.7	—[h]	WBC: normal-appearing; numerous	
PLT	182	230	N/S	252	—[h]	atypical lymphocytes	
13							
WBC	5,500	5,500	5,900	7,500	6,800	RBC: NC, NC; slight anisocytosis	3
ALC	2,255	2,096	1,829	1.950	1,972	and poikilocytosis	
HBG	12.9	13.5	14.0	12.4	12.8	PLT: adequate numbers	
MCV	103.5	98.8	95.0	92.7	94.7	WBC: normal-appearing;	
PLT	281	281	298	412	331	occasional LGLs	
14							
WBC	6.400	5,400	6,100	5,200	5.700	RBC: NC, NC	2
ALC	2,398	2,160	1.830	1,872	2,041	PLT: adequate numbers	
HBG	12.1	13.2	13.7	12.5	13.6	WBC: normal-appearing; few atypical	
MCV	86.7	86.4	87.3	86.7	87.1	lymphocytes; rare LGLs;	
PLT	196	224	222	237	277	increased numbers of basophils	
15							
WBC	8,400	9,400	8,400	7,300	8,800	RBC: NC, NC; rare fragmented cell	0
ALC	3,528	2.341	3.110	2,140	1,584	PLT: adequate numbers	
HBG	14.3	14.0	14.0	14.1	12.7	WBC: normal-appearing;	
MCV	88.5	88.0	87.2	89.0	84.9	occasional LGLs	
PLT	343	441	453	447	426		

N/S, patient did not keep appointment; NC. NC. normocytic. normochromic; LGLs. large granular lymphocytes.
[c]White blood cell count. normal range: 4,500–11,000.
[b]Laboratory tests drawn between August a d October 1989.
[c]Absolute lymphocyte count, normal range: 1,100–4,800.
[d]Test not performed by laboratory.
[e]Hemoglobin, normal range: 13.9–16.3 grams %.
[f]Mean corpuscular volume. normal range 80–100 fl.
[g]Platelets, normal range 150,000–350,000.
[h]Laboratory accident.
Quantitative count not performed, slight decrease reported.
[j]Performed on specimens obtained 11/88.
[k]Red blood cell series.
[l]Alcohol score (current alcohol consumption): 0 = none: 1 = 1–10oz. per week; 2 = >10 oz.–20 oz. per week; 3 = >20 oz. per week.

should be interpreted with caution since workers who experienced symptoms may tend to overreport their number of exposure hours.

We observed at least one hematologic abnormality consistent with benzene exposure in 9 (60%) of the degassers in the 4-month period following their acute benzene exposure. Although the hematologic abnormalities observed in several workers were not significantly correlated with duration of acute exposure or length of employment as a degasser, it is possible that benzene exposure may have been responsible for some of the abnormalities. it is interesting to note that worker #15 who had normal hematologic profiles on the first four evaluations was anemic in the fall of 1989. In addition, his absolute lymphocyte count in the fall of 1989, while not outside the normal range. is decreased from his earlier values. This worker stated that he had gone back to work as a gas-freer (and may have been re-exposed to benzene) about 1 week before his evaluation in the fall of 1989.

Alcohol ingestion results in hematologic changes similar to those caused by benzene exposure including leukopenia, anemia. thrombocytopenia, and elevation of the MCV [Wynagaarden and Smith, 1985; Isselbacher, 1980; Chick et al., 1981; Whitehead et al., 1978; Kristensson-Aas et al., 1986]. In this study, alcohol use did not differ significantly between workers with and without hematologic abnormalities. The lack of association between duration of benzene exposure and hematologic abnormalities including the presence of numerous LGLs on peripheral smear is not surprising due to the small number of workers (n = 15) in this cohort and possible misclassification of exposure. Benzene's effect as a central nervous system depressant may have influenced a worker's ability to accurately recall the number of exposure hours. If such information bias occurred nondifferentially among workers with and without hematologic abnormalities, the bias would be toward the null, thus obscuring any potential benzene effect.

The absence of significant change in hematologic parameters over time and the lack of an exposure duration effect on these parameters, as indicated in the repeated measures analysis are not unexpected. To accurately assess the effect of exposure, baseline (pre-exposure) hematologic profiles which were unavailable to us should be used for comparison. Since baseline data were not available, the results of the first hematologic profiles were used as a basis for comparison of the hematologic parameters over time. Such an analysis is limited since the first laboratory analysis, performed by a commercial laboratory, may not produce results which are comparable to the results obtained by our laboratory. In addition, fewer workers (n = 7 or n = 9) were considered in each repeated measures analysis, reducing the power to detect a significant change over time.

in general, workers with an abnormal WBC count did not have significantly different levels of other blood counts as compared to workers with normal WBC counts, with one exception. The lower MCVs (at time 3) in workers with low WBC counts vs. those with normal WBC counts were unexpected, but could be due to chance, given the number of statistical comparisons performed (n = 20).

LGLs are identified morphologically by their abundant cytoplasm and intracytoplasmic azurophilic granules [Forbes and Leong, 1987]. While LGLs are present in the peripheral blood of normal individuals [Chan et al., 1984], the occurrence and persistence of increased numbers of LGLs in peripheral blood may indicate an LGL leukemia [Loughran and Starkebaum, 19871. Additionally, granular lymphocyte proliferative disorders (GLPDs) may be reactive [Oshimi, 19881 to underlying disorders

such as rheumatoid arthritis [Semenzato et al., 1987; Wallis et al., 1985; Saway et al., 1989], cancer [Semenzato et al., 1987; Taylor et al., 1982], and infectious diseases such as hepatitis [Chan et al., 1986; Semenzato, et al., 1987], and tuberculosis [Semenzato et al., 1987].

While the number of LGLs in each worker's blood in the current study was not quantified, 6 (40%) of these benzene-exposed workers had numerous LGLs on their peripheral smears. The relationship between benzene exposure and the presence of increased LGLs is unclear at this time but warrants further study.

In this cohort, four workers with persistent hematologic abnormalities were referred to a hematologist for evaluation. The company was advised to place these workers in jobs where there was no potential for benzene exposure. Workers with normal hematologic profiles were allowed to return to work provided they wore appropriate respiratory protection **as** specified in the **OSHA** Benzene Standard. All of these workers are required to have annual surveillance evaluations.

CONCLUSIONS AND RECOMMENDATIONS

Benzene is **a** known constituent of petroleum products which may accumulate in a confined space at concentrations well above the **OSHA** PEL of 1 ppm, even when its concentration in the liquid phase is low. Therefore, in addition to the measurement of oxygen content and testing for flammable atmospheres and residues prior to fuel tank entry, measurement of the ambient concentration of benzene in the headspace is recommended. Although not required in the current **OSHA** Shipyard Industry Standard [United States Department of Labor, 1983], sampling for benzene in the headspace of fuel tanks prior to allowing workers to enter to perform degassing may prevent potential acute fatalities, as well as decrease the risk of future hematologic malignancies.

Since no correlation was observed between the duration of acute benzene exposure and the presence of hematologic abnormalities during the year of follow-up, this acute exposure may not have caused significant hematologic changes in the short-term. The latency period for the development of hematologic malignancies is reported to range from more than 1 year to 37 years [Oak Ridge National Laboratory, 1989]. Thus, any chronic effects due to this exposure will not be apparent, perhaps for several decades. However, the finding of numerous or frequent LGLs on the peripheral smears of 40% of the exposed workers 1 month after exposure warrants further investigation, and we recommend that peripheral smears of benzene-exposed workers be examined.

This paper has identified an occupational exposure to a known human carcinogen in a work situation that has received little study or documentation. We recommend further investigation into the levels of benzene and the neurotoxic solvents present in cleaned and uncleaned fuel tanks so that appropriate precautions (ventilation, use of protective respiratory gear) can be implemented before workers enter such confined spaces.

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