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Fire Fighting: How Safe Are Firefighters

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Firefighting is an all-time hazardous occupation since the atmosphere of a fire is deadly to breathe. In most industrialized western countries a systematically developed legislation to protect the individual firefighter from potential health hazards exists. However, workers in this section (firefighters, police officers, and correctional facility personnel) are frequently exposed to unknown risks even in right-to-know circumstances. We report here on an incidence in which firefighters became exposed to a chemical fire without being adequately informed on the nature of the chemicals and the dangers of exposure to the toxic smoke resulting from the combustion of such substances.

A chemical fire erupted in an industrial storage complex in a densely populated suburb in Antwerp. About 400 firefighters became eventualy involved in fighting the intense fire, smoke and smell.

The following day members of the firefighting team mainly firemen and police officers - trickled to hospital with complaints of fever, fatigue and respiratory distress. A few had also developed rare symptoms, yellowish stripes on their backs, hands and feet. The admittance of more and more officers with similar complaints and symptoms led to a compulsory medical examination for all those who were involved and exposed during the fire fighting exercise.

Samples of extinguishing water were collected from the scene for analysis. The presence and identity of the various chemical substances involved was confirmed by gas chromatography (GC) and gas chromatography-mass spectrometry (GC-MS).

A clinical survey of the firefighters was conducted two days after the incidence. Blood and urine samples from exposed patients were analysed by gas chromatography (GC). Contrary to expectations, analysis only showed the presence of pentachlorophenol (PCP), a product not listed in the inventory (see Table 1). Concentrations of up to 400 μg/L of plasma in 75 % of the patients were detected. Although a number of patients remained in a prolonged

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discomfort no fatalities were reported, a fortunate escape from a catastrophe.

Table 1. An inventory of the products in store at the time of the accident.

Product	Active Component	LD_{50} (oral, rat)
Premazin	Simazin	> 5000 mg/kg
Atratex	Atrazine	> 3000 mg/kg
Herbit	Sodium chlorate	1200 mg/kg
Diuron	Diuron	3400 mg/kg
Amitrol	Amitrol	1100-24600 mg/kg
Hexazinone	Hexazinone	1690 mg/kg
Diznin	25% Premazin + 40% Diuron	-
Soria	230 g/L atrazin, 180 g/L Amitrol + 60 g/L Hexazinone	8350 mg/kg
Dalapon	Dalapon	3.86 g/kg
Guidex	Chloridazon	-
Bromone	Bromodiolone	1.75 mg/kg
Disonex V	Diazinon	-
Karathane	Dinocap	980 mg/kg
Boutanex	Diuron	437 mg/kg
Pardi	Diquat, Paraquat	231, 100 mg/kg
Cresylex	Dinitro-o-cresol (DNOC)	30 mg/kg
Lindamex	Lindane	88 mg/kg
Superdust	Malathion	1375 mg/kg
Clinex	Methoxychlor	5000 mg/kg
Premazin	Simazine	500 mg/kg

MATERIALS AND METHODS.

We randomly collected samples of extinguishing water in and around the burned out storage building complex. Samples were collected in glass stoppered Erlenmeyer flasks. Air samples were unfortunately not available. The brownish-yellow liquid had an intense sharp odour.

Pro analysi diethyl ether, hexane, methylene chloride and ethanol were used for the extraction and analysis. 500 mL aliquots of collected water were separately extracted in acid, neutral and alkali media using diethyl ether. The ether layer was dried over anhydrous sodium sulphate and evaporated to dryness. The residue was redissoved in ethanol. The resultant emulsion was centrifuged. The supernatants were qualitatively analyzed by GC-MS.

GC-MS water analysis were performed on a HP (Hewlett-Packard, Palo Alto, USA) 5890A gaschromatograph/5970B quadrupole mass spectral detector

(MSD) equipped with a 12m x 0.203 mm (internal diameter) HP cross-linked methyl silicone fused silica capillary column ana a split/splitless injection system, used in the splitless mode. The column was inserted directly into the mass spectrometer. Helium (N56 grade, Air Liquide, Liège, Belgium) was used as carrier gas at a flow rate of 2 ml/min. Gaschromatographic conditions were set as follows: Detector/injector temperature 300/230 °C; initial/final temperature 90/275 °C; initial/final time 5/20 min.; rate 10 °C/min. Data processing and acquisition was carried out on a HP 9816 workstation with a 15 Mb Winchester harddisk. Electron impact (EI) mass spectra were recorded continuosly, scanning from 50 to 500 m/z. Background was substracted manually and mass spectra were compared with the manufacturer's mass spectral library (EPA/NIH 1985).

The possible presence in blood and urine samples from the patients of products identified in the extinguishing water was also investigated. A 0.5 mL aliquot of each indivual serum was acidified using a 0.1 N solution of HCl and extracted using dichloromethane. The organic layer was dried over anhydrous sodium sulphate, evaporated to dryness and redissoved in ethanol. This was then analyzed by GC-MS.

Acetylcholinesterase activity was determined using the Merck-1-test diagnostic kit for cholinesterase activity (E. Merck, Darmstadt, Germany). Activity was only measured in 40 serum samples (20 % of the patients) randomly selected.

The possible presence of paraquat, not detected in water samples but listed in Table 1, was also included in the investigation. 10 mL of a urine sample was passed through a Bond Elut® SCX-column (Varian, Analytichem International, Inc, Harbor City, USA) preconditioned with methanol and then water (1 and 2 column volumes respectively). Paraquat is efficiently retained by the SCX benzenesulfonic acid groups. The column was then rinsed with 2 mL of methanol- water (50:50 v/v). 2 mL of sodium dithionite solution (2% $Na_2O_4S_2$ in 2 N NaOH) was then brought onto the column. A deep blue color forming along the sides at the top of the column indicates a positive test. Using this procedure, the detection limit in urine is about 50 ppb.

PCP, DNOC, lindane and dinoseb were analyzed on a HP 5890 gas chromatograph equipped with a Grob type split/splitless injection and a ^{63}Ni-electron capture detector (ECD). Peak areas were determined using a Trio integrator computer (Trivector, Bedfordshire, UK). GC separations were performed on a 30 m x 0.53 mm (i.d) fused silica capillary column Durabond 626 phase (J & W Scientific, Folsom, USA), 5 μcm film thickness. Reagent gases were helium at a flow rate of 5 ml/min and argon/methane (95:5, Air Liquide, Belgium) at a flow rate of 80 ml/min. The temperature conditions were as follows: injector/detector 250/25 °C. Oven temperature was programmed from 200 up to 285 °C at a rate of 10 °C/min. Concentrations were determined from the ratios of peak areas relative to the internal standard. Compound identity was confirmed by GC-MS. The presence of these compounds in other materials e.g. the uniforms of exposed policemen were similarly confirmed.

Extractions for the above mentioned compounds were performed as follows: 0.2 μg of 2,4,5-tribromophenol (98%, Janssen Chimica, Geel, Belgium) was added to an individual serum aliquot (0.2 - 0.5 mL) as internal standard. The samples were subsequently subjected to acid hydrolysis using a 0.1 N hydrochloric acid solution at 60 °C for 30 min. 20 mL of each urine sample was hydrolyzed using 6.0 N HCl at 100 °C in a water bath during 30 minutes following the addition of the internal standard.

Sample extraction was carried out with 3 x 25 mL batches of diethylether. The organic layers were combined, dried over anhydrous sodium sulphate and reduced in volume to about 1 mL. This was methylated with 1 mL of diazomethane in ether and then allowed to stand at room temperature for 20 minutes. The ether was removed and the residue redissolved in 2 mL hexane. This solution was washed with 0.5 mL aliquots of 0.1 N NaOH till clear, then with 0.3 mL distilled water. It was then concentrated to about 10 μl with a nitrogen stream. A 1-2 μl of treated sample was injected into the gas chromatograph.

RESULTS AND DISCUSSION.

GC-MS analysis of the water revealed the presence of the biocides pentachlorophenol, dinitro-ortho-cresol and dinoseb; the insecticides dimethoate, endosulfan, lindane and 2-imidazolinone and the herbicides simazine, burex (neburon), propachlor and propanil. Alkylated aromatic hydrocarbons, phenols and higher chlorinated (octa, hepta) dioxines were also detected.

No inhibition of acetylcholinesterase activity was observed. The presence of paraquat in the selected samples was also not detected.

Plasma and urine analysis did not reveal any of the products listed in Table 1. Only pentachlorophenol was detected. The results obtained are summarized in Fig.1. Plasma concentrations of PCP ranged from 0 to more than 300 μg/L. Table 2 shows urine PCP concentrations in function of plasma concentrations.

A comparison of results obtained for PCP analysis in this instance with about two thousand performed over the past years in this laboratory would suggest concentrations of up to 30 μg/L as "normal" background concentrations (Fig.1). Concentrations above these limits could be considered as "elevated" concentrations. The exposure profile from this diagram suggests that about 25% was not affected by PCP while the rest was exposed to varying concentrations of this product. Heavy, medium and light exposures are also reflected in this diagram.

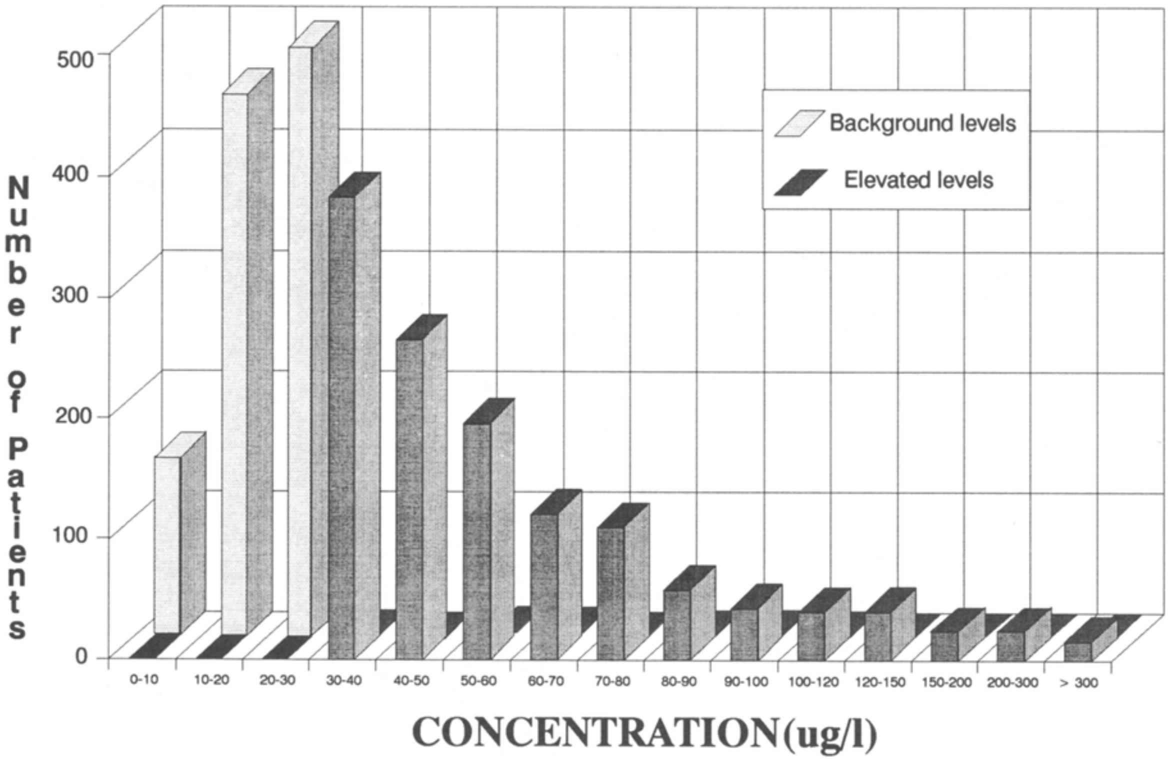

Figure 1. Results of Plasma PCP levels of exposed individuals.

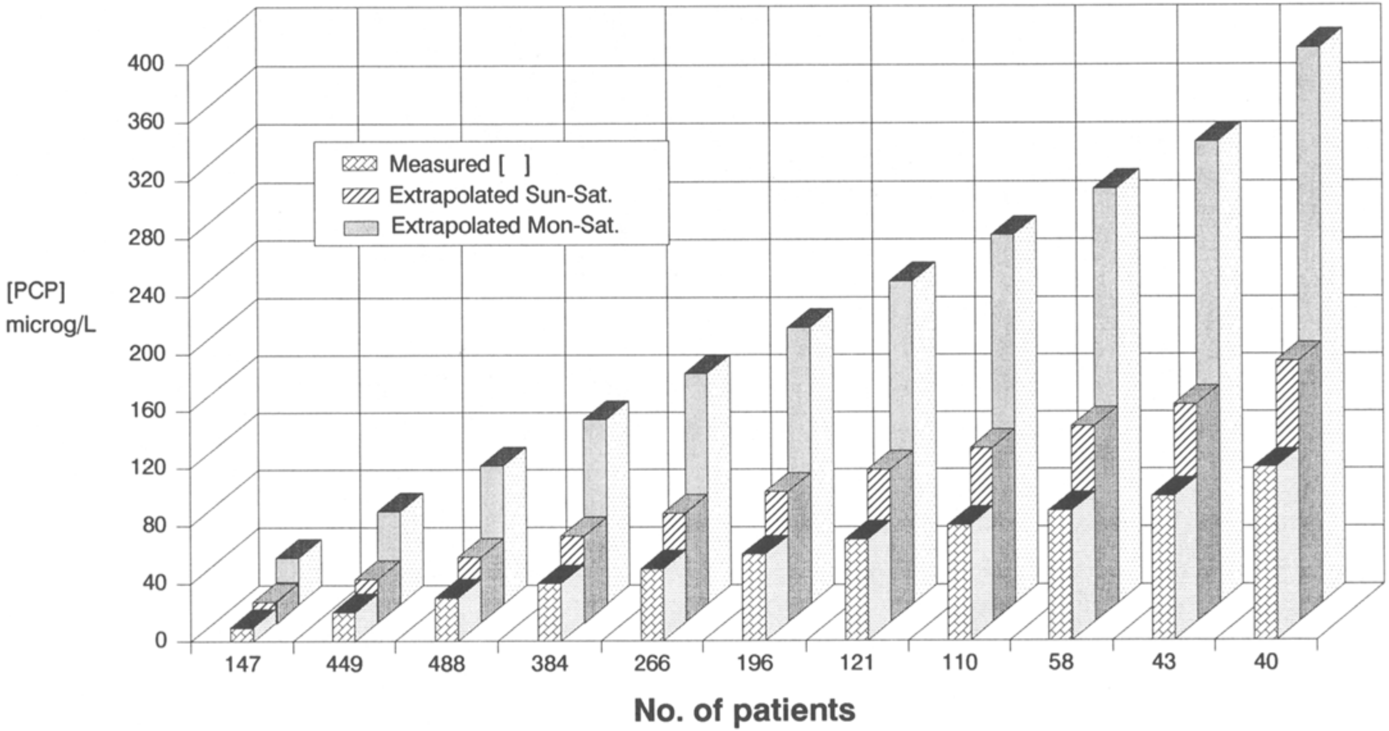

Figure 2. Measured and extrapolated PCP levels of exposed individuals.

The half-life for absorption and elimination of PCP from plasma is about 1.3 ± 0.4 h and 30.2 ± 4.0 h (Jorens and Schepens 1993). Since blood and urine samples were taken 20 to 48 hrs (Sunday and Monday) after the incident the initial exposure concentrations should have therefore been higher (Fig. 2). Individual urine analysis do not show any correlations with the plasma concentration. However, a relationship between the average urine and plasma concentrations appears to exist (Table 2).

Table 2. PCP levels in urine compared to serum concentration ranges

[PCP] μg/L Plasma Range	Urine Average	Number of Samples
200- 299	5.08	6
150-199	2.80	5
120-149	4.04	11
100-119	3.82	11
90-99	3.56	8
80-89	3.45	11
70-79	3.25	26
60-69	3.14	22
50-59	3.14	42
40-49	3.03	69
30-39	2.86	66
20-29	2.69	76
10-19	2.19	34
1-9	1.25	2

Clinical symptoms resulting from firefighting have been variously described in the literature (Cohen and Guzzardi 1983; Fitzgerald et al. 1986; Markowitz et al. 1987; Brandt-Rauf et al. 1989) are frequently a result of the irritating smoke, heat, depleted oxygen, carbon monoxide, and other toxic gases such as cyanide, hydrogen chloride and acrolein. The symptoms observed in this case were largely consistent with exposure to PCP. However, no relationship could be established between the degree of symptomatology and serum PCP levels. Unfortunately a further follow-up and evaluation of the patients was not done. Recovered patients were immediately discharged.

The submitted inventory (Table 1) conformed to existing regulations. However, the presence of a large number of unlabelled containers just outside the storage building suggested that the complex was also used for the storage of waste chemical products or solvents. Although a variety of very toxic substances was involved in the fire accident, only concentrations of pentachlorophenol gave an indication of extent of exposure to toxicity in the fire-fighting exercise. A fortunate aspect in this incident is that explosive products such as DNOC and other more toxic substances were not in the fire front. The likelyhood is that packages containing these products were destroyed by the extinguishing water. A possible source of PCP is the unlabelled containers, an indication that chemicals were stored in them without permit. This kind of practice is common in the chemical industry and presents a threat to those involved in fire fighting as well as to the public. The safety of the firefighters is, even for the legislator, difficult to guarantee.

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