high exposure group.² An increased mortality restricted to a high exposure group might be difficult to identify when the high exposure group is diluted by large and relatively low exposed groups. In the Swedish¹ and in the recent American³ cohorts an increased cancer mortality was seen only among workers who had been exposed to high concentrations of cadmium ($>0.1 \text{ mg Cd/m}^3$) for at least two years.

Even though we agree with Lam and Kazantzis that the epidemiological data on cadmium are not conclusive, we are of the opinion that the combined epidemiological and experimental⁴ evidence speaks in favour of cadmium as a potential occupational carcinogen. History has shown that it may take many years from the first suspicion of an occupational carcinogen until it becomes a recognised occupational hazard and action is taken to decrease the industrial exposure.⁵ Until additional epidemiological studies on cadmium exposed workers have been made available, from a preventive health point of view, we think that cadmium should remain on the list of chemicals which are regarded as probably carcinogenic to man.⁶

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Are hazard warnings sufficient?

SIR,—A 50 year old man presented with blurred vision and unsteadiness. He was drowsy, hyperventilating, and had dilated pupils that reacted poorly to light. Initial investigations showed a profound metabolic acidosis pH 6.95, standard bicarbonate 5.9 mmol/l (mEq/l). Further investigations showed a significant methanol level 15.5 mmol/l (500 mg/l); ethanol was not detected. He was treated with intravenous sodium bicarbonate 1000 mmol (mEq), haemodialysis, and a nasogastric infusion of ethanol. On recovering consciousness, he developed delirium tremens that responded to diazepam.

The full story then emerged. Fifteen years previously, after the breakdown of his first marriage, he had drunk heavily. In recent years he had been drinking two bottles of spirits a week, concealing this from his wife. On occasions he had stolen and drunk methylated spirits from another department at work. Unbeknown to him, however, his employers changed from using methylated spirits to methanol, using standard hazard warnings. Two days later he took some to drink, with tragic consequences. He now has permanent optic nerve damage and is registered as blind, only being able to count fingers.

There is no legal requirement for methylated spirits or methanol to be kept under lock and key. Therefore unauthorised members of the workforce may have access. The hazard symbols and risk and safety phrases differ: for methylated spirits *highly flammable*, and for methanol *highly flammable, toxic by inhalation and if swallowed*.^{1 2} These differences, however, might not be appreciated by a worker without a scientific training who was accustomed to taking quickly a little from a bottle in a particular cupboard. They are both colourless liquids (industrial methylated spirits is not dyed with methyl violet³). The odour of methylated spirits is slightly stronger.

The ingestion of 15 ml of 40% methanol has been reported to cause death, while the highest reported dose for a survivor is 500-600 ml.⁴ In survivors the most common physical sequelae are optic nerve damage and permanent blindness.

Industrial methylated spirits is 95% ethanol and 5% methanol³; both are metabolised by hepatic alcohol dehydrogenase. Toxicity arises from the metabolic products. The relatively high concentration of ethanol in methylated spirits leads to competitive inhibition of the metabolism of methanol so any toxic effects are usually those of ethanol alone.

It is probably relatively common for employees to take methylated spirits or ethanol for their own consumption. Therefore, if a change is made to methanol I suggest that all employees who might have access should be verbally informed of the potential dangers as standard hazard warnings may not be sufficient. In this way, repetition of this sad case might be avoided.

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