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Case studies of hydrogen sulphide occupational exposure incidents in the UK☆

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HIGHLIGHTS

- Three case studies of industrial incidents involving hydrogen sulphide are presented.
- We demonstrate the use of thiosulphate measurements in blood and urine.
- Appropriate sample collection and storage are important factors.
- The role of biological monitoring in such incidents is discussed.

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ABSTRACT

The UK Health and Safety Executive has investigated several incidents of workplace accidents involving hydrogen sulphide exposure in recent years. Biological monitoring has been used in some incidents to determine the cause of unconsciousness resulting from these incidents and as a supporting evidence in regulatory enforcement. This paper reports on three case incidents and discusses the use of biological monitoring in such cases. Biological monitoring has a role in identifying hydrogen sulphide exposure in incidents, whether these are occupational or in the wider environment. Sample type, time of collection and sample storage are important factors in the applicability of this technique. For non-fatal incidents, multiple urine samples are recommended at two or more time points between the incident and 15 h post-exposure. For routine occupational monitoring, post-shift samples should be adequate. Due to endogenous levels of urinary thiosulphate, it is likely that exposures in excess of 12 ppm for 30 min (or 360 ppm/min equivalent) would be detectable using biological monitoring. This is within the Acute Exposure Guideline Level 2 (the level of the chemical in air at or above which there may be irreversible or other serious long-lasting effects or impaired ability to escape) for hydrogen sulphide.

1. Introduction

Hydrogen sulphide is a toxic gas generated by non-specific and anaerobic bacterial reduction of sulphates and sulphur-containing organic compounds. Natural sources include crude petroleum, natural gas, volcanic gases and hot springs. It can also be found in groundwater and released from stagnant or polluted waters and manure or coal pits. The principal industrial source of hydrogen sulphide is recovery as a by-product in the purification of natural and refinery gases. It is also a by-product of pulp and paper manufacturing and carbon disulphide production. It is used as an intermediate in manufacturing processes (e.g. sulphuric acid) (WHO, 2003). In the UK, regulations are in force requiring storage of slurry (including manure) in certain areas to prevent water pollution (DEFRA, 2010). Similarly, the UK Government is committed to increasing energy production through anaerobic digestion (DEFRA, 2011). These factors have increased potential exposures to hydrogen sulphide in the UK.

Human exposure to exogenous hydrogen sulphide is principally via inhalation with rapid absorption. Hydrogen sulphide is metabolised through three pathways: oxidation, methylation, and reactions with metalloproteins or disulphide-containing proteins. Oxidation in the liver is the major detoxification pathway, forming thiosulphate, which is then converted to sulphate and excreted in the urine. The methylation pathway also serves as a detoxification route. The toxicity of hydrogen sulphide is a result of its reaction with key metabolic metalloenzymes. In the mitochondria, cytochrome oxidase (the final enzyme in the respiratory

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chain) is inhibited by hydrogen sulphide. This disrupts the electron transport chain and impairs oxidative metabolism which particularly impacts nervous and cardiac tissues (both are tissues with high oxygen demand and rely on oxidative metabolism). In the central nervous system, this effect may result in unconsciousness or even death from respiratory arrest (WHO, 2003). High flow oxygen is generally used to treat victims of hydrogen sulphide poisoning (Gresham, 2014) although other treatments such as hyperbaric oxygen and parenteral administration of a methaemoglobin inducing agent (such as sodium nitrite) have also been reported (Costigan, 2003; Belley et al., 2005).

Hydrogen sulphide is acutely toxic with fatalities associated with concentrations in excess of 500 ppm. It has a very low odour threshold (0.008 ppm) but odour perception is lost at concentrations of 150–250 ppm (WHO, 2000), adding to the danger of high level exposures as they may not be recognised, by smell, by the individual. In Europe, there is a workplace exposure limit (8 h TWA) of 5 ppm (HSE, 2011; SCOEL, 2007) with a short-term (15-min) exposure limit of 10 ppm.

Hydrogen sulphide has previously been reported as a causal agent of unconsciousness and death in a number of occupational exposure incidents (Kage et al., 2002, 2004). In the UK it has been reported (Costigan, 2003) that around 125,000 workers in the UK are potentially exposed to hydrogen sulphide in work related to the treatment of sewage, effluent waste and farm slurry. In the offshore oil and gas industries about 3000 workers are potentially exposed. The UK Health and Safety Executive has investigated several incidents of workplace accidents involving hydrogen sulphide exposure from slurry pits, animal rendering plants and biodigester facilities in recent years. The increased prevalence of biodigesters and slurry storage may indicate an increased likelihood of further incidents in the future. Here we report three case studies using biological monitoring to determine hydrogen sulphide exposure.

2. Material and methods

Blood or urine thiosulphate determination was carried out according to the method of Kage et al. (1991). Briefly, samples (200 μl) were buffered with ascorbic acid (200 mM, 50 μl) and 5% sodium chloride (50 μl) then derivatised using pentafluorobenzyl bromide (20 mM in acetone, 500 μl) and extracted into iodine ethyl acetate solution (25 mM, 2 ml) to form bis(pentafluorobenzyl) disulphide. Tribromobenzene was used as an internal standard. Analysis was by gas chromatography–mass spectrometry (positive electron ionisation) using selected ion monitoring (m/z 426 for the thiosulphate derivative). Aliquots (1 μl) were injected (220 °C, splitless) onto a BP-5 equivalent column (30 m × 0.32 mm i.d., 1 μm film) with a helium flow of 1 ml/min. The oven temperature was held at 100 °C for 2 min then ramped at 10 °C/min up to 220 °C, where it was held for 5 min. Calibration standards were prepared in blood or urine, as appropriate, and extracted as per the samples. The calibration curves were linear from 0 to 600 μmol/l (least squares regression > 0.99) and quality control samples were within the expected range showing a coefficient of variation of 12%. The detection limit was 1 μmol/l. Urine samples were also analysed for creatinine content using the alkaline picrate reaction (Cocker et al., 2011)

3. Results

3.1. Case 1

Two workers were admitted to hospital after collapsing in an enclosed waste intake area of an animal rendering plant. One was unconscious on admission. Both provided urine samples whilst at the hospital – worker 1 (male, 53 years old) approximately 9 h after the incident, worker 2 (male, 54 years old) at an unknown time (but apparently the same day) by catheter as he was still unconscious. The urine sample for worker 1 contained 326 μmol/l thiosulphate (23 mmol/mol creatinine), which is consistent with the levels seen in other survivors of reported incidents of hydrogen sulphide exposure where samples have been taken between 2 and 15 h of the incident (Kage et al., 1997, 2002). Worker 2's result (10 μmol/l, 2 mmol/mol creatinine) was within previously reported background levels (Kangas and Savolainen 1987; Chwatko and Bald, 2009) however it is not clear when the sample was collected in relation to the incident. It is possible that, if he was exposed, it might take a couple of hours for his thiosulphate level to exceed background levels (as demonstrated by a volunteer study (Kangas and Savolainen, 1987)); so if the sample was taken shortly after the incident, the sample may not reflect the extent of his exposure to hydrogen sulphide. Equally, if the sample had been taken later, the level of thiosulphate may already have reduced to background levels. There is previously reported, (Kage et al., 1997) a case (in which a man lost consciousness due to hydrogen sulphide exposure and subsequently recovered) where the urinary thiosulphate level was less than 3 μmol/l when the sample was taken 15 h after the incident.

There was evidence that worker 1 had been exposed to hydrogen sulphide in sufficient amounts to cause a feeling of unwellness or even unconsciousness. The sample of worker 2 did not demonstrate evidence of hydrogen sulphide exposure but this does not exclude the possibility of exposure due to the unknown timing of sample collection.

3.2. Case 2

A chicken waste rendering plant had a blocked condenser connected to a storage vessel. On releasing the blockage, an emission of gas (suspected to contain hydrogen sulphide) was released knocking three workers unconscious. All three workers were taken to hospital, two were subsequently released and one spent time in intensive care before being released. Blood samples were obtained from two of the workers (both male, ages unknown) but were not detectable for thiosulphate. This is in agreement with previous reports where blood thiosulphate is not detected in survivors of hydrogen sulphide incidents. Unfortunately, in this case, it was not possible to obtain urine samples. Samples of the chicken waste showed considerable potential for hydrogen sulphide generation at the sterilising temperature used (~120 °C).

3.3. Case 3

One urine sample and one blood sample were received from a fatality (male, age unknown) involving a biodigester, where hydrogen sulphide was a suspected toxic agent. The urine sample was below the detection limit for thiosulphate. The blood sample had a detectable thiosulphate level of 22 μmol/l.

The blood level reported is at the lower end of the scale of previously reported fatalities (25–230 μmol/l) but definitely indicates significant hydrogen sulphide exposure – sufficient to cause unconsciousness, and possibly fatal poisoning. No thiosulphate was detected in urine, which is consistent with literature reports of sudden death caused by hydrogen sulphide (Kage et al., 2002) whereas survivors of hydrogen sulphide poisoning incidents tend to have raised urinary thiosulphate levels in the hours following the incident as thiosulphate is excreted.

It can therefore be concluded that the results of the thiosulphate analysis from blood and urine samples are consistent with acute hydrogen sulphide poisoning causing death rapidly. However, it should be noted that these analyses were conducted some nine months after the incident occurred. The samples were