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REVIEW ARTICLE

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Occupational exposure to hydrogen sulfide in the sour gas industry: some unresolved issues

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Abstract Occupational exposure to hydrogen sulfide (H_2S) and the medical management of H_2S-associated toxicity remains a problem in the sour gas industry and some other industrial settings. The acute effects of exposure to H_2S are well recognized, but accurate exposure-response data are limited to acutely lethal effects, even in animal studies. Odor followed by olfactory paralysis and keratoconjunctivitis are the characteristic effects of H_2S at lower concentrations. H_2S-induced acute central toxicity leading to reversible unconsciousness is a "knockdown"; it is controversial whether repeated or prolonged knockdowns are associated with chronic neurologic sequelae but the evidence is suggestive. Knockdowns can be acutely fatal as a consequence of respiratory paralysis and cellular anoxia. Pulmonary edema is also a well-recognized acute effect of H_2S toxicity. Human studies of sublethal exposure with satisfactory exposure assessment are almost nonexistent. There are indications, poorly documented at present, of other chronic health problems associated with H_2S exposure, including neurotoxicity, cardiac arrhythmia, and chronic eye irritation but apparently not cancer. Rigorous and comprehensive studies in the sour gas industry are difficult, in part because of confounding exposures and uncertain end points.

Key words Sour gas · Hydrogen sulfide · Health effects Oil and gas industry · Neurotoxicity · Pulmonary edema

Introduction

Sour gas is natural gas containing sulfur. Hydrogen sulfide (H_2S) is a common and potent toxic agent that is the primary chemical hazard of sour gas production [1, 3, 10, 11, 13, 26, 32, 43, 47, 48, 54]. It is also among the most disgusting common chemical hazards, typically encountered in odoriferous settings, including municipal sewers and sewage treatment plants, swine containment and manure-handling operations, pulp and paper operations (using obsolete technology), and contained spaces in which organic matieral such as fish or offal has decayed [4, 5, 20, 21, 22, 28, 42, 47, 48, 54]. However, it is most familiar to – and perhaps most respected by – that international fraternity of workers who inhabit the "oil patch," especially Texas, Oklahoma and the Gulf coast of the United States, Alberta, the North Sea, and the Middle East. It is in oil and gas production that H_2S appears most frequently as an industrial hazard.

H_2S is a variable constituent of sour gas, ranging from trace concentrations to 85% and more. The problem is uniquely severe in the Canadian province of Alberta, because of the heavy concentration of high-sulfur-content oil and gas fields in the province [1, 13, 32, 43]. Up to half a dozen deaths, on average two, have occurred annually from H_2S exposure in Alberta associated with the oil and gas industry [3, 10] and likely more from H_2S exposure from liquid manure operations on farms. Transient loss of consciousness from exposure to H_2S ("knockdowns") are common and often go unreported because until recently they were largely taken for granted.

The emphasis in this review is on unresolved issues regarding the toxicology and epidemiology of occupational and community exposure to H_2S, sour gas (sulfur-rich natural gas), and chemicals encountered in the processing of sour gas. However, many of these issues are generalizable to other exposures and other occupational health problems.

Issues in the toxicology H_2S

The toxicology of H_2S has been well reviewed recently by Reiffenstein et al. [38] and by Glass [14]. Table 1 briefly summarizes the principal effects of H_2S. This section is not intended to duplicate current comprehensive reviews

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Table 1 Health effects of H_2S at various exposure levels. (Adapted from references [2, 3, 14, 38] and the following sources: 1. Canada Safety Council data sheet "Hydrogen sulphide," No. B-3; 2. Alberta Provincial Board of Health "Guidelines for action regarding hydrogen sulphide"; 3. National Research Council of Canada, "Hydrogen sulphide in the atmospheric environment: scientific criteria for addressing its effects on environmental quality," publication # 18467)

Concentration (ppm)	Effects
0.01–0.3	Odor threshold (highly variable)
1–5	Moderate offensive odor, may be associated with nausea, tearing of the eyes, headaches or loss of sleep with prolonged exposure; healthy young male subjects experience no decline in maximal physical work capacity (Bhambhani and Sing [9])
10	8h occupational exposure limit in Alberta
15	15-min occupational exposure limit in Alberta
20	Ceiling occupational exposure limit and community evacuation level in Alberta, odor very strong
20–50	Keratoconjunctivitis (eye irritation) and lung irritation. Possible eye damage after several days of exposure; may cause digestive upset and loss of appetite
100	Eye and lung irritation; olfactory paralysis, odor disappears
150–200	Sense of smell paralyzed; severe eye and lung irritation
250–500	Pulmonary edema may occur, especially if prolonged
500	Serious damage to eyes within 30 min; severe lung irritation; unconsciousness and death within 4–8h; amnesia for period of exposure; "knockdown"
1000	Breathing may stop within one or two breaths; immediate collapse

but rather to highlight unresolved issues in the toxicology of H_2S that are significant in managing occupational and environmental exposures. Treatment, including the controversy over the efficacy of nitrite administration, will not be discussed except in passing. However, it should be noted that combined treatment with hyperbaric oxygen and nitrite appears to be emerging as the treatment of choice [16].

H_2S interacts with a number of enzymes and other macromolecules, including hemoglobin and myoglobin, because most macromolecules are held together by disulfide bonds which are prone to disruption by aqueous sulfide. Indeed, monitoring of aberrations in heme metabolism represents a promising approach to biological exposure indices for H_2S [21]. The critical target enzyme of H_2S is cytochrome oxidase, a family of related enzymes constituting the electron transport system in oxidative phosphorylation, the prinicpal energy-generating system of the cell [27]. Oxygen is the final substrate of this system and is necessary to its function. The effect of H_2S in disrupting cytochrome oxidase activity is the same as oxygen deprivation or asphyxiation except that it may act more quickly. Because the cytochrome oxidase system is common to all mammalian cells, the acute toxicity of H_2S roughly parallels the oxygen requirements of each target organ and is therefore similar to that of cyanide, although there appear to be differences at the biochemical level [6, 38]. This is one possible explanation for the prominence of the central nervous system and possibly the heart in the toxicity profile for H_2S [14, 23, 38].

The toxicologic exposure-response relationship for H_2S has been studied only for certain uncommon outcomes, such as pulmonary edema, and for lethality [45]. This is a major obstacle to deriving toxicity and risk assessment models for this agent. It forces most decisions to be made on grounds of lethality rather than sublethal health effects.

The exposure-response curve for lethality is extremely steep for hydrogen sulfide. As an inhaled toxic substance, H_2S gives little margin of safety. One can visualize an encounter with concentrations of H_2S above 500 ppm as being much like hitting a wall, with the degree of damage having much more to do with concentration, analogous to the speed with which one hits the wall, than with the duration of contact with the wall [5, 33].

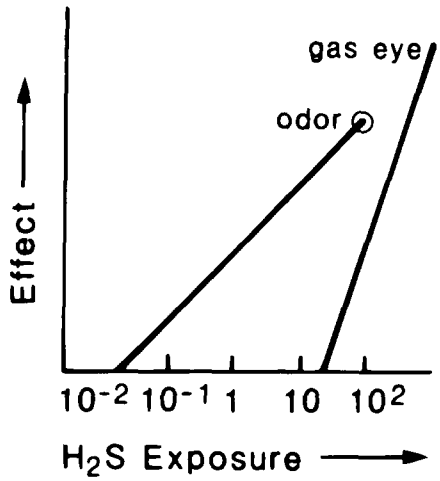

Fig. 1 Toxicological exposure-response relationship for perceived odor in response to H_2S; olfactory paralysis truncates the linear relationship [1]

One of the major problems with the toxicological data base for H_2S is the paucity of reliable information on sublethal health effects, although many have been proposed or suspected. Most standards for the protection of occupational and community populations are based on prevention of keratoconjunctivitis (eye irritation) and respiratory tract irritation [30, 45, 48]. The slope of the exposure-response relationships for these conditions is not as steep as it is for central nervous system effects. The literature on H_2S is very weak in providing the information needed to construct exposure-response relationships of this type. Two effects that have been examined in this way are odor and keratoconjunctivitis, or "gas eye" [30].

Odor has been studied on a semiquantitative basis and represents an important physiological end point, although olfactory paralysis – the loss of odor sensation – is the ac-

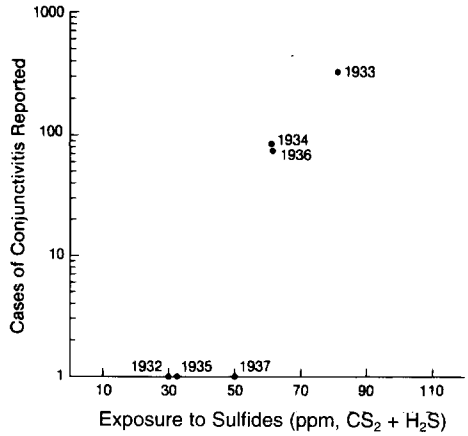

Fig. 2 Keratoconjunctivitis in a viscous rayon plant associated with exposure to sulfides; threshold for effect is apparent between 50 and 60 ppm total sulfides [47]

Table 2 Keratoconjunctivitis ("gas eye") in a viscous rayon plant associated with high levels of carbon disulfide (CS_2) and hydrogen sulfide (H_2S). (Barthelmy, 1939, cited in U.S. National Research Council [47])

Years	CS_2 (ppm)	H_2S (ppm)	Comment
1932, 1935	20, 21	9, 14	No effect
1937	32	18	No effect
1934, 1936	38	23	Same effect (71–85 cases)
1933	51	30	Maximum effect (332 cases)

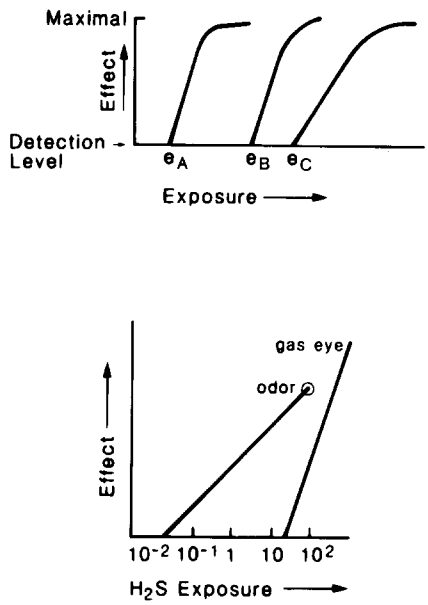

Fig. 3 H_2S exposure-response curves are very different in slope and show very different thresholds depending on the effect observed [1, 47]

tual toxic effect. Milby [33] reviewed the olfactory effects of H_2S and reported the approximate thresholds for subjective levels of perception of odor in ordinal rank. Plotted against the logarithm of concentration (Fig. 1), the response to exposure is clearly linear but truncated at the threshold for olfactory paralysis. Responses for toxic central nervous system effects may follow a similar pattern because the toxicity in both cases is probably by a common neurotoxic mechanism.

Figure 2 compares the perception of odor in response to H_2S exposure to that of gas eye, or keratoconjunctivitis, which in data from Barthelmy [47] appears to show a clear threshold for combined exposure to H_2S and carbon disulfide (Table 2). This exposure-response information, as crude as it is, is useful because it suggests a linear relationship for mucosal irritation as well as for neurotoxicity and allows comparison of the effects. In Figure 3, it is apparent that these two clinical effects of H_2S behave very differently.

Another property of H_2S is its irritant effect on mucous membranes. The respiratory tract is particularly vulnerable because of its unprotected contact with the gas in air [29, 30]. H_2S penetrates deeply into the respiratory tract because its solubility is relatively low, rendering it capable of causing alveolar injury leading to acute pulmonary edema. This injury is at least in part mitigated by host defense mechanisms that may include bronchial chemoreceptors, but little about this is known in detail [37]. H_2S may also interfere directly with host defense mechanisms in the lung [40]. However, relatively low-level exposures to H_2S do not seem to be associated with chronic respiratory abnormalities in humans [9, 22]. There do not appear to be reported cases of reactive airways dysfunction syndrome associated with H_2S exposure, for example. This is in contrast to reports of increased airways reactivity in animals exposed to 1 or 5 ppm for prolonged periods [38]. A single case report has suggested persistent dyspnea on exertion, without airflow obstruction, 5 months after recovery from inhalation of H_2S [33].

It has been known for many years that this unlovely compound has potent neurotoxic properties [2, 3, 6, 12, 14, 18, 38, 45, 47, 53, 54]. In relatively high concentrations (about 200 ppm) H_2S paralyzes the olfactory nerve, preventing perception of the otherwise strong smell. This removes a vital warning to, say, oilfield workers caught in a cloud or entering a depression in which the gas has collected (H_2S is denser than air). Respiratory paralysis may occur if exposure is prolonged. Sudden loss of consciousness following H_2S exposure, followed by equally sudden recovery, is colloquially called a "knockdown." Very high concentrations (500–1000 ppm) are associated with a knockdown. This is an abrupt loss of consciousness and collapse, described by those who witness it as appearing much like turning off the switch on a mechanical doll. A knockdown may be fatal if exposure is prolonged. If exposure is transient, as usually happens in the oil patch, the affected worker may wake up (also abruptly) feeling fine. Some veteran oilfield workers return to what they were doing without reporting the event and without treatment, considering the experience all in a day's work.

Many environmentally significant agents tend to follow "Haber's law" over a wide range of exposures: the product of the concentration and the duration of exposure

Table 3 Concentration-time products for lethal exposure to H_2S, showing deviation from "Haber's law" [33]

Concentration (ppm)	Time to death (min)	Product[a]
150	1680	0.252
300	3600	0.108
400	150	0.060
550	45	0.024
650	8.5	0.005

[a] ppm converted to 10^{-6}

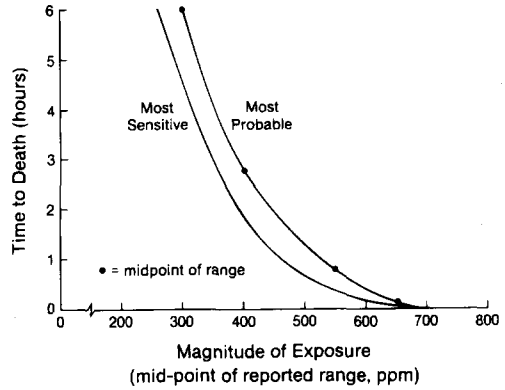

Fig. 4 Exposure-response relationship for exposure to H_2S taking lowest reported and mid-range concentrations for human fatalities [1]

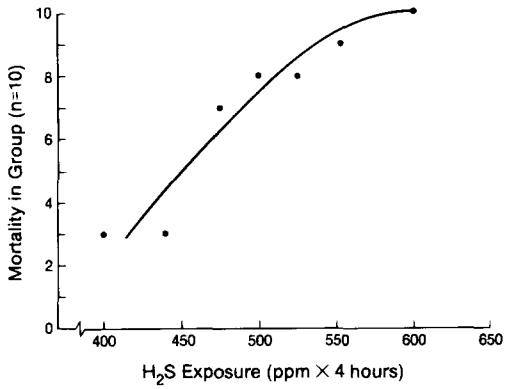

Fig. 5 Exposure-response relation for mortality among rats exposed for 4 h by inhalation to H_2S; LD_{50} was determined to be 444 ppm in this experiment [44]

needed to achieve a given effect is approximately constant. [This is the logic behind the familiar time-weighted average (TWA) in occupational hygiene.] This assumption allows us to treat variations in exposure more simply by taking the average over time. Unfortunately, this is a very crude relationship that does not hold true for many exposures. Fatal exposures to H_2S in humans, for example, may take place at 150 ppm for 6 h (concentration-time product = 0.252) or 650 ppm for 8.5 min (0.005) (Table 3). The plot of fatal exposure to H_2S shows a sharply falling product with increasing concentration (Fig. 4). This means that, for H_2S, higher concentrations are much more toxic, even with proportionally shorter exposure levels [33]. This also appears to be true for experimental pulmonary edema induced in the rat.

Figure 5 presents mortality data from an inhalation study of rats exposed to H_2S, demonstrating the curvilinear nature of the relationship [44]. This may be interpreted as representing the saturation of a detoxification mechanism or overwhelming of an adaptive mechanism. For this reason, many risk assessment models are based on toxicity relationships that weight the concentration much more heavily than the derivative of exposure, often raising it to the square.

Susceptibility states

The effects of exposure to H_2S and other chemical exposures at low levels would be much worse and longer-lasting if elaborate biochemical and physiologic mechanisms did not exist to remove, metabolize, counteract, and adapt to the H_2S exposure. For H_2S, these mechanisms include binding to metalloproteins, oxidation to sulfate, increased ventilation, and the development of limited tolerance to low levels. (Tolerance plays a much more important role in other exposures, such as oxidant gases.) These mechanisms are basic to the toxicologic response but are seldom investigated in population studies. The distribution of such host factors in a population may be as important as the level of exposure (within a given range) in determining the number of individuals affected [5, 19, 34, 47].

It is well known that in a knockdown situation, the victim ceases activity as if a light switch had been turned off. The question remains, however, whether this is fully reversible or exacts a physiologic cost each time it occurs. Recent evidence suggests that the endogenous level of sulfide in the central nervous system is rather high compared to other tissues and that it is increased in toxic exposure [15, 37, 38, 49, 52]. Recent studies have suggested that endogenous H_2S accumulation varies by anatomic region [15, 50–52] and that there may be a threshold for response: exceed a certain concentration in the brainstem and perhaps loss of consciousness occurs by inhibition of a cytochrome oxidase [27] or other mechanisms, such as sodium channel blockade [50, 51]. The sulfide load from an exposure is therefore superimposed on an existing level that may or may not be similar among normal individuals or affected by prior disease or by metabolic state [15]. It is not known what, if any, susceptibility states may predispose to central injury from H_2S exposure.

If there is much individual variability in endogenous CNS sulfide, this may be a significant factor determining susceptibility. A blood sulfide test may eventually be developed as a practical screening test for toxicity and, perhaps, susceptibility [21].

Little is known of the toxicokinetics and metabolism of H_2S. Humans are known to have enzyme systems that metabolize sulfides. These systems probably evolved to handle production of sulfides in the gut by endogenous bacteria, although it has been suggested [38] that some species

living in high-sulfide environments developed other adaptations such as mucosal defenses against irritation. It is thought, but not known, that some metabolism proceeds without enzymatic catalysis. Interaction between H_2S and macromolecules is obviously critical in the toxic response, however, so the detoxification of organosulfides is likely to be of some importance in determining susceptibility [5, 48].

There is considerable variation in the ability of normal, healthy volunteers to metabolize organosulfides. There is a subset of 10% or more of the population that shows a marked deficiency in the ability to metabolize organosulfides which appears to be inherited [19, 34]. Ordinarily this defect is of no consequence, except that such persons may excrete sulfur compounds through the skin or exhale higher concentrations in expired air after loading with sulfur-containing compounds in the diet. For these reasons they are often inconvenienced – or, more accurately, inconvenience others – for days after eating meals laden with garlic. This trait might be a marker for persons susceptible to H_2S-induced toxicity at exposure levels lower than that at which the rest of the population responds.

Exposure assessment

The relatively few human data that are the basis for occupational exposure standards are derived primarily from uncontrolled exposure incidents. In most of the these, the exposure level is only crudely estimated. This means that the human data points currently available are useful only to suggest an effect at a given level of exposure. They may also suggest comparability to animal data in which the exposure levels are known. By themselves, they are an uncertain guide to exposure-response relationships in human exposure.

Area monitors are now available that record H_2S levels in the immediate environment. However, until about 1990, most sour gas plants used alarms that triggered at a specific threshold without recording precise measurements. As a practical matter, the technology for measuring personal H_2S exposure directly is not available for most occupational applications. In the case of H_2S, blood sulfide concentration might be a way to quantify internal dosage and aberrations in heme metabolism may provide an index of biologic effect [21]. Urinary thiosulfate has also been proposed as a biologic monitoring method [24]. These biologic exposure indices are not currently available for routine use and have not been extensively validated.

Alternative outcomes of interest

Acute central neurotoxicity, pulmonary edema and the mucosal effects are well documented in their association with H_2S. The literature suggests other outcomes that should be considered in any future study of the problem. This discussion is not intended to imply that such effects are at all likely in the sour gas industry, however.

Table 4 presents chronic health outcomes plausibly associated with exposure to H_2S at levels encountered in occupational exposures but for which documentation is incomplete. The list is arranged in diminishing order of the strength of evidence for any effect involving the organ system, with that for hematologic changes judged to be

Table 4 Chronic health outcomes of concern, plausible at levels of exposure ≤ 100 ppm H_2S in humans [*A* evidence for acute effects; *?A* suggestive evidence for acute effects; *?C* suggestive evidence for chronic effects, not confirmed; *O* no evidence available; –, data available suggest no detectable effect; () data available do not directly address this outcome]

Organ system	Evidence for an effect			Chronic disease outcomes of intersest
	Animal	Human	Human-Alta.[a]	
Central nervous system	A,?C	A,C	A,?C	Dementia, cognitive dysfunction, affective disorders, cerebrovascular disease
Respiratory	A	A	A	Chronic obstructive airways disease, asthma, lung cancer
Heart	A	A	A	Coronary artery disease, sudden death, arrhythmias
Eye	A	A	A	Chronic keratoconjunctivitis, cataracts, retinal disorders
Hepatic[b]	(A)	A	(A)	Hepatic dysfunction-marginal (cirrhosis, chemical hepatitis unlikely); gallstones noted in sour oil refinery workers
Host defenses[c]	A	O	O	Infections, immune disorders, arthritis
Reproductive	A, in vitro)	O	O	Infertility, fetal loss, teratogenicity
Renal	?A	O	O	Renal insufficiency
Endocrine	A	O	O	Thyroid abnormalities
Cancer	([d])	O	O	All types
Hematologic	–	O	O	(None obvious)

[a] Human health effects reported from local experience in Alberta [3]

[b] Host defenses include reticuloendothelial function, mechanical host defenses of respiratory tract, cellular immune function, and humoral immune function

[c] A congestive hepatic lesion was described at necropsy (in an Alberta case [24]) that probably represents passive congestion due to cardiac failure. The animal studies refer to biliary function, not hepatocellular injury

[d] Sulfide salts have been reported to be mutagenic in vivo; no acceptable animal exposure study has been reported

weakest. The outcomes are tentatively described in the literature and are also suggested by local experience in Alberta [3, 10]. Clearly, central nervous system, respiratory, cardiac, eye, and host defence disorders emerge as the abnormalities of highest priority for investigation [2, 4, 5, 14, 20, 23, 26, 28, 30, 33, 35, 36, 38, 39, 42–48, 54].

Acute central nervous system toxicity has already been mentioned as a well-recognized and dramatic effect. Chronic central effects have not been extensively evaluated, however. There have been persistent questions raised over whether chronic health effects may follow knockdowns and whether neurologic toxicity may be cumulative and lead to a chronic syndrome. In the oil patch, there have long been folk tales circulating to the effect that workers who are knocked down more than a few times get a little "simple in the head" [35].

Central effects observed after a knockdown might represent primary toxicity of H_2S, secondary effects from low-level cellular anoxia, or the sequelae of acute brain injury. The long-term central effects of a knockdown in any given case may represent an episode of toxic anoxia, or even a traumatic injury, perhaps occurring if the subject struck his head in the fall [17, 46]. For this reason, subjects who have recovered from knockdowns should probably be studied separately from cases of chronic low-level exposure, even when the lifetime average exposure level such a subject has sustained might not be exceptional.

Tvedt and his colleagues at the National Institute of Occupational Health of Norway collected six cases of patients who experienced H_2S-induced knockdowns of up to 20 min, and evaluated them 5 years or more following the incident [46]. Most of these patients were exposed in settings in which products of decaying organic material accumulated in confined spaces: a sewage tank, the hold of a ship with rotting fish, a swine manure pump, inside an oil drilling platform in drydock for repair, and a tannery. None were involved in oilfield incidents. The concentrations to which these patients were exposed cannot be documented but were certainly very variable.

These patients were studied with detailed neuropsychological testing and other studies pertinent to their clinical condition. All cases showed some evidence of neurologic sequelae. They found evidence for cognitive function abnormalities after 5 years in five and anosmia in a sixth, with a suggestion of increasing severity associated with length of time unconscious. They attributed these findings to hypoxia. The patient with the shortest period of unconsciousness, a 46-year-old man out for less than a minute, had persistent anosmia for 3 years, visual accommodation changes, and subjective symptoms of fatigue and difficulty expressing himself verbally. Those with higher exposure, out for 5 min or more, showed persistent cognitive and motor impairment, EEG changes, and some localized deficits in vision and visual association. These patients also showed varying degrees of short-term memory impairment and two, unconscious for 10–15 min, showed advanced dementia [46]. All but the least-exposed case also had other factors complicating their clinical course: pulmonary edema, prolonged anoxia, and seizure disorder. It is therefore questionable to attribute these chronic effects to H_2S exposure alone. The authors did not mention whether any of the patients also sustained head trauma in their falls and this may not be known. The consistent demonstration of cognitive deficits, even in the least heavily exposed (and therefore most interesting) case, does suggest a pattern, despite these complicating factors. The Norwegian team concluded that the literature on H_2S toxicity probably underestimates the risk of neurologic sequelae.

Some oil and gas workers are exposed to levels as high as those encountered by these Norwegian patients but most are not. This one study cannot tell us whether the hundreds–perhaps thousands worldwide-who have survived relatively minor knockdowns are at risk of neurologic sequelae. Nor can we extrapolate from these cases to the hundreds of thousands of workers who are regularly exposed for prolonged periods to lower levels of H_2S although nothing obvious has presented itself.

Experimental studies help to localize the effects of H_2S in the brain and seem to suggest anoxia as the mechanism. The available animal evidence points to injury to the cerebral cortex, cerebellum, and possibly the brainstem and spinal cord at concentrations approaching those that humans might encounter [18, 28, 41]. The lesions are similar to those seen in oxygen deficiency and in poisoning by other cytochrome oxidase inhibitors [17, 41, 46].

The pattern of injury is consistent with reports of changes in the way the subject thinks and feels emotionally. Given the anatomic localization of the lesion, cognitive and affective chagnes are more likely to appear than clinical findings that can be easily detected in a physical examination.

One way to overcome this problem is to design a study using the most sensitive possible test [52]. Sophisticated tests of cortical function are difficult to perform and to standardize; tests of brain electrical activity must be done in a hospital or special laboratory and paper and pencil tests are often difficult to interpret objectively and quantitatively [52]. The tests available are not sensitive or specific. Thus, it is difficult to evaluate higher mental functions in an epidemiologic study. A negative result in such a study will not be convincing. Another approach is to use cleverer study designs, such as nested case-control or exposure-generated case-control studies to pin down the characteristics of those individuals more precisely. We have proposed to do so by establishing a registry of knockdown cases and studying these subjects more intensively, using batteries of neurobehavioral tests if preliminary inquiries point of deficits that can be evaluated by existing tests.

Other properties of H_2S are difficult to assess because mucosal irritation and cellular anoxia dominate the clinical picture. It is possible that the acute central nervous system effects of H_2S observed in a knockdown or cardiac arrhythmias observed after exposure represent a primary effect of the agent on the brain or myocardium [18, 28], but they could also be due to the anoxic effect [17]. The practical implications are very great, since a primary anesthetic action of H_2S, similar to that of many lipid-soluble

low-molecular-weight compounds [49], is likely to be reversible whereas anoxic brain injury frequently leads to long-term sequelae [17].

Additional agents encountered in the gas industry

While the single exposure of greatest interest in the sour gas industry is H_2S, numerous other exposures are involved in the gas industry. Many of these are exotic; some are unlikely to be toxic. Exposure to other agents encountered in the sour gas industry introduces potential confounders in any study of occupational health in the industry. In addition to volatile hydrocarbons, including benzene, a host of chemicals used in operations and processing should be included in any discussion of this subject (Table 5) [7, 8, 11, 13, 25, 31, 43]. Various agents may be used in the drilling, in recovery, processing, and separation of the final product. An exhaustive checklist of chemicals used in the various procedures is beyond the scope of this paper but has been presented elsewhere [11]. Every sour gas plant is slightly different. A thorough investigation requires detailed analyses of the specific procedures and plant characteristics. Evans [13] has outlined the hazard potential in a typical operating gas plant, emphasizing exposure to benzene. Dermatitis is a common response to skin contact with most of the compounds listed in Table 5 and is not noted separately [11].

Several of the chemical classes unique to the sour gas industry such as the mercaptans and carboxyl sulfide, are poorly characterized toxicologically. Mercaptans may be valuable indicators of exposure in odorized gas. Asbestos insulation is occasionally encountered on exposed pipes but there has been little indication of significantly exposure [11].

Because of this large number and wide range of chemical exposures, studies of the gas industry must at least account for and anticipate the effect of confounding exposures. It is possible that there may be significant interactive or additive effects associated with chemical mixtures in these situations but this is speculation.

A straightforward comparison with workers in the "sweet" gas industry, where low-sulfur content natural gas is processed, may not be effective for isolating H_2S effects because of the many exposures inherent in the desulfuration process. Even sweet gas may contain trace amounts of sulfur that must be removed for consumer and environmental acceptability. Workers engaged in this part of the process may be exposed to some sulfur-containing compounds, including H_2S, and to amines, although their overall exposure, and that of workers at the plant in general, would be much less than in a sour gas plant. Comparing the two industries therefore requires individual exposure assessment for workers in each.

In 1987, and again in 1991, our group at the University of Alberta proposed a comprehensive occupational health study of the sour gas industry to be carried out over 10 years, and featuring a direct approach to many of these issues. Unfortunately, funds could not be found to support this study despite independent interest in the province and support from the industry. We remain hopeful that eventually such a study can be performed. Ironically, had the study been initiated when it was first proposed, the answers to many of these unresolved issues might now be known.

Table 5 Principal exposures in the sour gas industry

Exposure	Source
Hydrogen sulfide	Raw gas
Mercaptans	Raw gas and extracted sulfur
Carboxyl sulfide	Raw gas and extracted sulfur
Hydrocarbons (C1–C5)	Raw and product gas
Solvents	Maintenance and installation of equipment
Amines[a]	Desulfuration
Elemental sulfur	Sulfur recovery
Methanol	Wax solvent, antifreeze
Demulsifiers (detergents)	Separation
Ethylene glycol	Dehydration
Corrosion agents	Pipeline and tank protection
Bactericidals	Production water, wastewater treatment
Defoamers	Separation
Asbestos	Plant insulation (significant exposure unlikely)
Noise	(Multiple)

[a] Principally monoethanolamine and diethanolamine

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