

IS AMBIENT HYDROGEN SULFIDE A RISK TO HUMAN HEALTH?

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ABSTRACT

Hydrogen sulfide gas (H₂S) has an obnoxious odor at very low concentrations and is thus responsible for considerable annoyance and concern in communities near H₂S sources such as wastewater treatment plants. Human health data, however, do not clearly indicate that ambient concentrations (less than 1 ppm) are hazardous. Several regulatory or scientific organizations have derived standards or guidelines for H₂S exposure for different populations, and these values vary considerably. We examine here some of the reasons for the differences, and suggest that community exposures may best be evaluated in light of the acute minimal risk level of 70 ppb derived by the Agency for Toxic Substances and Disease Registry (ATSDR).

KEYWORDS

Hydrogen sulfide, H₂S, odor, air quality, public health, risk

INTRODUCTION

The noxious character of hydrogen sulfide gas, and the extremely low concentration at which it can be detected (low parts per billion), cause frequent annoyance and health concern among neighbors of facilities from which hydrogen sulfide (H₂S) is released. A natural public health response is to attempt to identify concentrations of H₂S in air that are safe if inhaled over defined periods of time. Unfortunately, the degree of health threat posed by low concentrations of H₂S in ambient air is difficult to assess for two reasons. One reason, common to many air pollutants, is a scarcity of reliable health effects data for the various health endpoints of concern, such as cancer, birth defects, and pulmonary disease. The other reason, much less common, is the difficulty in distinguishing between toxicities that are independent of olfaction and those that are. For example, the lethality of high concentrations (hundreds of ppm) H₂S does not depend on a person's ability to smell the material or on his psychological reaction to the odor. In contrast, annoyance reactions to the odor of H₂S may create or intensify other responses, such as headaches and fatigue. Presumably, persons unable to smell H₂S would not experience such responses. This phenomenon is not readily addressed by standard toxicologic and risk assessment practices, and may be viewed by public health professionals as irrelevant to the issue

of “real” harm.

In a particular situation, such as complaints due to episodic ambient concentrations of H₂S of 30 ppb in a community near to a wastewater treatment plant, one’s first step is often to look to existing standards, regulations, and guidelines for H₂S in order to gauge the severity of the problem. It is thus of interest to examine some of these values, understand their origins and limitations, and thereby be able either to use them appropriately or seek other guidance.

DISCUSSION

Some of the oldest standards or guidelines pertain to industry, where the potential for acute toxicity, including death, was greatest. Table 1 shows some current values and notes about their origins.

While these three values were developed over a period of 25 years or so, they are fairly similar. All are meant to safeguard the health of workers, and thus acknowledge that they do not protect the sick, the elderly, or the very young, which means they should not blindly be applied to the general population. Since pregnant women often work, they and their fetuses should, in theory at least, be protected. Another possible limitation of these values for safeguarding the health of the general population is that workplace exposure is clearly not continuous in the same way it can be in the general environment. That is, workers might be exposed continuously on the job, but their time away from work in the evenings and on weekends and vacations means exposure is in fact intermittent. Intermittent exposures may be less toxic than continuous exposure because damage can be repaired, the compound eliminated through excretion or metabolism, and protective mechanisms can develop.

The three organizations represented in Table 1 strive to meet different worker protection mandates that can affect the determination of a safe exposure concentration. NIOSH, part of the Centers for Disease Control and Prevention, considers only worker safety. OSHA, part of the Department of Labor, must consider the technological feasibility and expense of controlling exposure while striving to protect all workers, whatever their pre-existing health status. ACGIH, a private, independent organization, judges what exposure can be experienced by a “typical” worker without adverse effect.

One’s confidence in the values in Table 1 may also be affected by their date of issue. NIOSH’s review is clearly outdated because numerous toxicologic, clinical, and epidemiologic studies relating to H₂S have been published since 1977. If NIOSH’s value is still appropriate, that is perhaps due partly to coincidence. OSHA’s review, though more recent, depended largely on earlier work by ACGIH and was not particularly in-depth. The draft TLV from ACGIH is likely to be adopted in 2004 unless new, conflicting data are obtained.

Note, finally, that none of the values in Table 1 are derived from experimental animal toxicology studies; all are based on reports, sometimes anecdotal, of the human workplace experience. It is often unclear if the chosen value is thought to be without any adverse effect at all, or simply to pose no unreasonable or significant risk. In no case were safety or uncertainty factors explicitly used.

Other government organizations have developed H₂S exposure guidelines for the general public, as shown in Table 2. The processes by which these values were generated differ in significant ways from the occupational values given above. First, all are the product of recent reviews, particularly the new reference concentration (RfC) from U.S. EPA. Second, all are meant to protect not only the general population but also “sensitive groups” such as children and the elderly. Third, all assume continuous (*i.e.*, 24 hours/day, 7 days/week) exposure, although for varying durations. Fourth, most values are based on critical studies that used laboratory animals, rather than workers. Fifth, uncertainty and other modifying factors were applied to the critical exposure concentration to develop all four values. Similar to the occupational values, however, these ambient air guidelines were based on toxic effects not expected to be affected by annoyance.

EPA’s RfC deserves particular discussion, both since it was developed most recently and is the lowest of the ambient air guidelines. A significant factor in its development was an experimental study published in 2000 and not, or probably not, available to ATSDR or California OEHHA during their deliberations. The 2000 work, by Brenneman *et al.*, generated a no-adverse-effect level (NOAEL) of 10 ppm, one-third of the earlier NOAEL used by ATSDR and OEHHA. All else being equal, the more recent evaluation would be more reliable, scientifically. However, one can take issue with EPA’s use of the Brenneman *et al.* data, as discussed below.

Brenneman *et al.* exposed male CD rats to H₂S 6 hours/day, 7 days/week, for 10 weeks at various concentrations, then closely examined the nasal epithelium. At 10 ppm, H₂S produced no adverse responses in the olfactory or respiratory epithelia. At the next highest concentration, 30 ppm, olfactory lesions and basal cell hyperplasia (a repair response to the olfactory lesions) occurred at high incidence in most areas of the nose. At the highest concentration, 80 ppm, damage and repair in the olfactory epithelium were more frequent and extensive. Thus, 10 ppm H₂S was clearly a NOAEL for damage to the olfactory epithelium.

EPA derived the RfC from the 10-ppm NOAEL in several steps. First, the discontinuous exposure concentration was adjusted to a continuous exposure concentration by dividing by four. Second, this concentration was adjusted for differences between rats and humans in the volume of air inhaled per minute and the surface area over which the air passes; the adjustment was about a factor of 5. The “human equivalent concentration” to the 10 ppm NOAEL was thus reduced by about 20-fold. Further adjustments were made using standard uncertainty factors — totaling 300 — to account for the subchronic exposure period, interspecies differences in sensitivity to H₂S, and possible additional sensitivity among humans. Therefore, the RfC is approximately 10 ppm/6,000, or 1.5 ppb.

Each step in EPA’s derivation can be questioned. First, is loss of olfactory neurons (at 30 ppm) a clear adverse effect? Damage was estimated at mild to moderate, and repair was in process. Second, is it appropriate to adjust the discontinuous exposure concentration of 10 ppm by a factor of 4 to give a continuous exposure concentration? The unstated assumption is that the cumulative exposure, in ppm-hours, is a better indicator of toxic potential than the peak concentration (in this case a constant value). It can be argued that H₂S’s toxicity to the epithelium is concentration dependent, however. Certainly, this is the case for H₂S’s irritant properties: one’s eyes or nose may be irritated by exposure to 20 ppm for one hour, but they will

not be irritated by 1 ppm over 20 hours. Similarly, an 0.5-hour exposure to 1,000 ppm H₂S will be lethal for a person, but a 10-hour exposure to 50 ppm will not. At the cellular level, H₂S inhibits cytochrome oxidase and thus interferes with cellular respiration. If H₂S is not metabolically detoxified at a sufficient rate, cell death can result. Toxicity is thus a threshold phenomenon, occurring once the compensatory mechanisms are overwhelmed, and is related more to concentration than concentration-time. Thus, the assumption that a continuous exposure to 2.5 ppm is a better NOAEL than 10 ppm, even assuming continuous exposure, may be unnecessarily conservative.

Third, the dosimetric adjustment may be inappropriate in two respects. The specific formula used by EPA included the surface area of the “extrathoracic” section of the pulmonary tree, meaning the nose, pharynx, and mouth. Given that toxicity occurred only in the olfactory epithelium of the nose, it would perhaps be more appropriate to use the surface area for that region, specifically. Regardless, it is not clear that a dosimetric adjustment is needed at all for a chemical that is poorly absorbed in the nose and an effect likely to be concentration dependent.

Fourth, the standard uncertainty factors may be unnecessarily conservative. Damage to olfactory neurons has not been reported in humans. Loss of, or reduction in, ability to smell has been reported for a few subjects who either suffered near-lethal acute exposure to H₂S or were repeatedly exposed to concentrations of several hundred ppm. (Olfactory fatigue, meaning a temporary loss of the sense of smell, occurs at H₂S concentrations of 150 ppm or more, and recovery occurs once exposure ends.) The literature contains no reports of olfactory damage in persons exposed to lesser concentrations of H₂S, and is frankly incredible at a concentration 10- or even 100-fold higher than the RfC. Uncertainty factors for interspecies and intraspecies sensitivity may be unnecessary given the essential absence of olfactory damage in the scores of reports on the humane experience with H₂S.

Finally, one can subject the RfC to a “reality check.” EPA developed the RfC as if there were no human data regarding H₂S toxicity. The RfC of 1.5 ppb is below the H₂S odor threshold for most people (CalEPA, 2000). Given the absence of clinically significant toxicity in people exposed to H₂S well above the odor threshold (say, up to the ppm-level), there is likely to be a very large margin of exposure between the RfC and any real effect. Thus, the RfC could be significantly higher yet remain protective of public health.

Similar criticisms can be made of OEHHA’s REL and ATSDR’s MRL for intermediate duration, since these groups followed EPA’s procedure exactly, differing only in the starting exposure concentration and (in ATSDR’s case) the subchronic to chronic exposure adjustment.

Of the values presented in Tables 1 and 2, the most useful for situations of intermittent odorous exposures in neighborhoods near treatment works is the acute MRL from ATSDR, which applies for periods of up to 14 days. This guideline was developed from a human study in which asthmatics (but not severe asthmatics) were exposed to defined H₂S concentrations for half an hour. While the subjects said they became accustomed to the odor, some developed an increase in airway resistance, a possible indicator of incipient asthmatic response. ATSDR divided the LOAEL of 2 ppm by 10 to estimate the NOAEL and then by 3 to protect severe asthmatics. A concentration of 70 ppb would be considered objectionable by most people, however, and so might still create annoyance. Nonetheless, if this acute MRL is indeed protective for all people,

including severe asthmatics, then acceptable concentrations for community air will in practice be based on odor minimization, and not on avoidance of respiratory health effects. In other words, H₂S concentrations that are only minimally odorous would appear also to be safe.

CONCLUSIONS

Several H₂S exposure recommendations for workers and the general population are available and range over several orders of magnitude. Differences arise due to the literature available at the time, the populations intended to be protected, the degree of protection desired, the nature of the critical effect, the duration of anticipated exposure, use of safety and/or uncertainty factors, and other reasons. For communities exposed occasionally to odorous concentrations of H₂S, the most appropriate guidance value from those discussed here is 70 ppb for a period of up to two weeks. The reference concentration derived by U.S. EPA for continuous, lifetime exposure is considerably lower but may well be overly protective.

Table 1 - Occupational standards and guidelines for H₂S

	ACGIH	NIOSH	OSHA
type of standard or guideline	TLV-TWA (draft)	REL (recommended exposure level)	PEL (permissible exposure limit)
value	7 mg/m ³ (5 ppm)	15 mg/m ³ (10 ppm)	14 mg/m ³ (10 ppm)
exposure period	continuous, working lifetime (<i>i.e.</i> , 8/5)	ceiling (<i>i.e.</i> , 10 minutes)	continuous, working lifetime (<i>i.e.</i> , 8/5)
target population	workers	workers	workers
LOAEL, NOAEL, or other	LOAEL: 5 ppm	not clear	NOAEL: 10 ppm
critical study	acute experimental human studies by Bhambhani <i>et al.</i> , 1991-1997	various occupational studies	occupational study of a workplace adhering voluntarily to this limit, as described by ACGIH in 1986
critical effect	shift in metabolism from aerobic to anaerobic in exercising muscle	chiefly acute eye irritation	no eye irritation or conjunctivitis
adjustments	none	none	none
caveats	no information regarding cancer, sensitization, or dermal toxicity		
year developed	2003	1977	1989
comments	current value is 10 ppm		overturned in federal court; currently 20 ppm ceiling, and 50 ppm for 10 minutes once

Table 2 - H₂S exposure recommendations for the general public

	EPA	California OEHHA	ATSDR	ATSDR
type of standard or guideline	RfC (reference concentration)	REL (inhalation reference level)	MRL (minimal risk level)	MRL (minimal risk level)
value	2 µg/m ³ (1.5 ppb)	10 µg/m ³ (8 ppb)	100 µg/m ³ (70 ppb)	42 µg/m ³ (30 ppb)
exposure period	continuous, lifetime (<i>i.e.</i> , 24/7)	continuous, long term (<i>i.e.</i> , 8 years)	continuous, acute duration (up to 14 days)	continuous, intermediate duration (up to 1 year)
target population	general population, including sensitive groups	general population	general population, including sensitive groups	general population, including sensitive groups
LOAEL, NOAEL, or other	NOAEL: 10 ppm	NOAEL: 30.5 ppm	LOAEL: 2 ppm	NOAEL: 30.5 ppm
critical study	subchronic rat study by Breneman <i>et al.</i> , 2000	subchronic mouse study by CIIT, 1983	acute experimental human study by Jappinen <i>et al.</i> , 1990	subchronic mouse study by CIIT, 1983
critical effect	no destruction of olfactory neurons or inflammation/necrosis of nasal epithelium	no inflammation of nasal epithelium	increase in airway resistance	no inflammation of nasal epithelium
adjustments	①intermittent ⇒ continuous exposure ②rat ⇒ human	①intermittent ⇒ continuous exposure ②rat ⇒ human	①LOAEL ⇒ NOAEL ②interspecies sensitivity	①intermittent ⇒ continuous exposure ②rat ⇒ human

	EPA	California OEHHA	ATSDR	ATSDR
	<p>dosimetry</p> <p>③interspecies</p> <p>④sensitive humans</p> <p>⑤subchronic ⇒ chronic exposure</p>	<p>dosimetry</p> <p>③interspecies</p> <p>④sensitive humans</p> <p>⑤subchronic ⇒ chronic exposure</p>		<p>dosimetry</p> <p>③interspecies</p> <p>④sensitive humans</p>
year developed	2003	2000	1999	1999
comments		<p>same as ATSDR except for subchronic ⇒ chronic factor and rounding differences</p>		<p>used EPA procedure but different critical study and no subchronic ⇒ chronic factor since intermediate-duration exposure is assumed; same as Cal OEHHA except for subchronic ⇒ chronic factor and rounding differences</p>

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