Hydrogen sulfide; 7783-06-4

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR Hydrogen sulfide

File First On-Line 01/31/87

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	withdrawn; qualitative discussion	07/28/2003
Inhalation RfC (I.B.)	yes	07/28/2003
Carcinogenicity Assessment (II.)	yes	07/28/2003

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Hydrogen sulfide (H_2S)
CASRN — 7783-06-4
Last Revised — 07/28/2003

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the

noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

The RfD previously listed on IRIS for hydrogen sulfide (H_2S) was 3×10^{-3} mg/kg-day, based on an oral pig study in which the critical endpoint was gastrointestinal disturbance (Wetterau et al, 1964). That value was based on a NOAEL of 3.1 mg/kg-day and a LOAEL of 15 mg/kg-day with a composite uncertainty factor of 1,000.

In the reevaluation of the RfD, it was determined that the Wetterau et al. (1964) study is marginal in its characterization of exposure and effect on the pigs. The "diarrhetic digestive disorder" was clearly indicated in the study as not being reproducible. Therefore, the previous RfD of 3×10^{-3} mg/kg-day is withdrawn from the IRIS database based on irreproducibility of effects that may not be related to hydrogen sulfide exposure. No new RfD has been derived in this assessment due to database deficiencies.

I.A.1. Oral RfD Summary

Not applicable.

I.A.2. Principal and Supporting Studies (Oral RfD)

Not applicable.

I.A.3. Uncertainty and Modifying Factors (Oral RfD)

Not applicable.

I.A.4. Additional Studies/Comments (Oral RfD)

Not applicable.

For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).

I.A.5. Confidence in the Oral RfD

Not applicable.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

I.A.6. EPA Documentation and Review of the Oral RfD

Not applicable.

Source Document — Toxicological Review of Hydrogen Sulfide (U.S. EPA, 2003). To review the External Peer Review, ***exit to the toxicological review, Appendix A, External Peer Review -- Summary of Comments and Disposition (PDF)***

Date of Agency Consensus — 06/20/2003

I.A.7. EPA Contacts (Oral RfD)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (fax), or hotline.iris@epa.gov (Internet address).

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Hydrogen sulfide
CASRN — 7783-06-4
Last Revised — 07/28/2003

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is generally expressed in units of mg/m^3. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity

of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

The RfC for H^2S was previously listed on IRIS as 1 x 10^{-3} mg/m^3 based on a subchronic inhalation mouse study by CIIT (1983a) using inflammation of the nasal mucosa as the critical endpoint. Derivation of the RfC was based on a $NOAEL_{HEC}$ of 1 mg/m^3 and a $LOAEL_{HEC}$ of 2.6 mg/m^3, with a composite uncertainty factor of 1,000.

I.B.1. Inhalation RfC Summary

Critical Effect	Experimental Doses*	UF	MF	RfC
Nasal lesions of the olfactory mucosa **Rat Subchronic Inhalation Study** **Brenneman et al., 2000**	NOAEL: 13.9 mg/m^3 (10 ppm) NOAEL (ADJ): 3.48 mg/m^3 NOAEL (HEC): 0.64 mg/m^3 LOAEL: 41.7 mg/m^3 (30 ppm) LOAEL (ADJ): 10.4 mg/m^3 LOAEL (HEC): 1.9 mg/m^3 BMCL: not determined	300	1	2E-3 mg/m^3

*Conversion Factors and Assumptions: MW = 34.08. Assuming 25 C and 760 mmHg, NOAEL (mg/m^3) = 10 ppm x 34.08/24.45 = 13.9 mg/m^3. NOAEL(ADJ) = 13.9 mg/m^3 x 6 hours/24 hours x 7d/7d = 3.48 mg/m^3. The NOAEL(HEC) was calculated for a gas:respiratory effect in the extrathoracic region. $V_{E(rat)}$ = 0.19 liters/minute, $V_{E(human)}$ = 13.8 liters/minute, SA_{rat} = 15 cm^2, SA_{human} 200 cm^2. $RGDR_{ET} = (V_E/SA_{ET})_{rat}/(V_E/SA_{ET})_{human}$ = (0.19/15)/(13.8/200) = 0.184 = NOAEL(ADJ) x RGDR = 0.64 mg/m^3.

I.B.2. Principal and Supporting Studies (Inhalation RfC)

Brenneman, KA; James, RA; Gross, EA; Dorman, DC. (2000) Olfactory loss in adult male CD rats following inhalation exposure to hydrogen sulfide. Toxicologic Pathology 28(2): 326-333.

Brenneman et al. (2000) exposed 10-week-old male CD rats (12/exposure group) to 0, 10, 30, or 80 ppm (0, 13.9, 42, or 111 mg/m^3) H_2S for 6 hr/day, 7 days/week, for 10 weeks. At the end of the 10-week period, animals were euthanized with CO_2 while their noses were dissected

free. Nasal cavities were examined at 6 different cross-sectional levels for lesions. The lesions were graded in severity by a subjective scale: 0 = normal, 1 = mild, 2 = moderate, 3 = marked, and 4 = severe. No effects were observed in the control or 10 ppm (14 mg/m^3) exposure animals that were considered treatment-related. Nasal lesions of the olfactory mucosa were observed in the 30 and 80 ppm (42 and 111 mg/m^3) exposure animals. Lesions consisted of multifocal, bilaterally-symmetrical olfactory neuron loss and basal cell hyperplasia affecting the lining of the dorsal medial meatus and dorsal and medial region of the ethmoid recess. The severity of the observed lesions varied between mild and severe. At level 3 of the nose, the most rostral margin of the olfactory epithelium is integrated with the rostral portion of the respiratory epithelium. Olfactory neuron loss was only observed in the high-dose (80 ppm or 111 mg/m^3) group at this level of the nose. At level 4 of the nasal cavity, mild to moderate olfactory neuron loss was observed in the 30 ppm (42 mg/m^3) exposure animals, which increased in severity to moderate or severe in the 80 ppm (111 mg/m^3) exposure animals. Basal cell hyperplasia was observed in both exposure groups at this level of the nasal cavity but was more pronounced in the 30 ppm (42 mg/m^3) exposure group. At level 5 of the nasal cavity, mild to moderate olfactory neuron loss and mild basal cell hyperplasia mainly affected the nasal septum, dorsal nasal cavity and marginal ethmoturbinate was observed in both exposure groups. The nasal septum was not affected in the 30 ppm (42 mg/m^3) exposure group. The same pattern and severity of lesions were observed at level 6 in only the 80 ppm (111 mg/m^3) exposure group. These lesions were not observed in either the group exposed to 13.9 mg/m^3 or in the controls.

The Chemical Industry Institute of Toxicology (CIIT, 1983a,b,c) exposed male and female $B_6C_3F_1$/CrlBr mice, Sprague-Dawley rats, and Fischer 344 rats to 0, 10.1, 30.5, or 80 ppm (0, 14, 42, and 111 mg/m^3) H_2S for 6 hr/day, 5 days/week for 90 days (10 mice/sex/group, 15 Sprague-Dawley and rats/sex/group and 10 Fischer rats/sex/group). In mice, body weight gain depression between week 0 and 13 of exposure was 30 and 40% in high-dose males and females, respectively. Feed consumption was significantly reduced in the high-exposure animals. Gross pathology of surviving animals also revealed no gross lesions that were considered compound-related. Histological examination of surviving animals revealed only one lesion that was considered compound-related. Male (8/9) and female (7/9) mice exposed to 80 ppm (111 mg/m^3) H_2S exhibited minimal to mild inflammation of the anterior portion of the nasal mucosa. No other histological findings were considered compound-related. In Sprague-Dawley rats, body weight gain depressions of 11.7 and 20% were evident in male and females exposed to 80 ppm (111 mg/m^3) H_2S, respectively. None of the histopathologic changes were considered treatment-related by the study authors. In Fischer 344 rats, weight gain depressions of 9.8 and 10.9% were evident in male and females exposed to 80 ppm (111 mg/m^3) H_2S, respectively. Sulfhemoglobin levels were significantly increased in males exposed to 80 ppm (111 mg/m^3) H_2S. None of the histopathologic changes were considered treatment-related by the study authors.

I.B.3. Uncertainty and Modifying Factors (Inhalation RfC)

UF = A composite uncertainty factor of 300 was applied; a factor of 3 ($10^{1/2}$) was applied for interspecies extrapolation rather than 10 because of the dosimetric adjustment from rat to human, 10 for sensitive populations, and 10 for subchronic exposure.

Although a single generation reproductive study (Dorman et al., 2000) noted testicular alterations that were observed only in the high dose group, the alterations were not significantly different from the controls and had no apparent effects on reproductive performance. No other indicators of reproductive toxicity were observed in this study. No significant histopathology of reproductive organs was noted in a longer duration (subchronic) study. These results can be considered to lessen the concern for lack of a multi-generational reproductive study.

In addition, a study designed to evaluate the perinatal effects of H_2S on the developing cerebellar, Hannah and Roth (1991) reported alterations in Purkinje cells and dendritic arborization. Although the significance and relevance of the reported alterations are described in EPA's neurotoxicity risk assessment guidelines (U.S. EPA, 1998) as *possible* indicators of a neurotoxic effect, alterations in Purkinje cells are not *clearly* adverse effects. The RfC is based on an effect that could be considered neurological (e.g., olfactory neuronal loss). This effect is clearly adverse and occurs in adult animals at a lower dose than the "possible indicator of a neurotoxic effect" of altered dendritic arborization in developing animals. This dose-response relationship - between adult and neonatal effects - tends to ameliorate concerns that children are a susceptible population, at least in the situation of long-term relatively low level exposures. Certain circumstantial data suggests that children may be more susceptible than adults to the acute effects of hydrogen sulfide under high level exposures.

Although the database for H_2S currently lacks a multi-generation reproduction study, a one generation study and some existing developmental neurotoxicity data (Dorman et al., 2000) provide insight on these potential adverse effects and justification for the lack of a database uncertainty factor of 1, as there are no major omissions to the database for H_2S.

MF = None

I.B.4. Additional Studies/Comments (Inhalation RfC)

Although the effects of H_2S on humans can be from acute and/or chronic exposures, the focus of this health assessment is on long-term chronic exposures.

The exposure-response relationship for acute effects, particularly CNS and respiratory, can be very steep. Numerous case-reports that identify H_2S as a component or the chemical of exposure demonstrate that levels of 500 to 1,000 ppm (695 to 1,390 mg/m^3) for even very brief periods are life-threatening and can cause immediate unconsciousness followed by serious and debilitating neurologic and respiratory sequelae and death (Allyn, 1931; Milby 1962). Levels dangerous to human health may not be detected by odor since high levels of H_2S can paralyze the olfactory nerves making detection impossible. Exposures resulting temporary unconsciousness (e.g., 15-30 minutes) can cause profound neurophysiological, neurobehavioral, neurocognitive, neurophysical, respiratory, and ophthalmologic deficits that are persistent, with some of these deficits apparently occurring even in situations in which the exposed individual remains conscious (Wasch et al., 1989; Tvedt et al., 1991a,b; Snyder et al., 1995).

Although not clearly established due to complexities of the exposure environment, a few reports exist claiming persistent symptoms (neurobehavioral function, mood state) from intermittent or continuous exposure to "low levels" of H_2S (Kilburn and Warshaw, 1995; Kilburn, 1997, 1999). Relevancy to humans of the olfactory lesions seen in rodents to human is suggested by Hirsch and Zavala (1999) who report decreased persistent olfactory function in workers exposed chronically to hydrogen sulfide.

A study by Brenneman et al. (2000) replaces another study by CIIT (1983a) as the principal study for RfC determination. At this time, Brenneman's et al. (2000) study is considered the most applicable for deriving an inhalation RfC for the following reasons. First, the critical effect (nasal lesions of the olfactory mucosa as summarized in Table 2, Section 4.2.2 of the accompanying Toxicological Review of Hydrogen Sulfide [U.S. EPA, 2003]) has been reported by other investigators (Dorman et al., 2000; CIIT, 1983a; Lopez et al., 1988b). Second, the effect is consistent with the irritant properties of H_2S. Third, the neurological and respiratory systems have been reported as target organs of H_2S toxicity by numerous researchers. Fourth, the LOAEL (42 mg/m^3) and NOAEL (14 mg/m^3) are at lower concentrations than those in the other subchronic studies.

Both nasal tract lesions and neurologic effects occur in animals exposed to the same concentration range of H_2S. Skrajny et al. (1992) reported altered ($p<0.01$) levels of several neurotransmitters on postpartum days 7-21 in the brains of rat pups exposed to 105 mg/m^3 (75 ppm) H_2S. Norepinephrine levels of the frontal cortex were decreased compared to controls on

days 14 and 21 at 28 mg/m^3 (20 ppm). Serotonin levels in the frontal cortex were significantly increased on day 21 postpartum in rat pups exposed to 28 mg/m^3 (20 ppm) H_2S. Hannah and Roth (1991) reported changes that could be an indicator of a neurotoxic effect in the brains of rat pups that had been exposed *in utero* during development until PND 21 at 20 or 50 ppm (28 or 42 mg/m^3). The observed effect - alteration in dendritic arborization of developing cerebellar Purkinje cells - was judged by the authors to be present at both concentrations such that the 20 ppm (28 mg/m^3) level may be considered a low-effect rather than a no-effect level. The LOAEL noted for olfactory epithelium lesions in rats was 42 mg/m^3 and the NOAEL 14 mg/m^3.

The significance of these neurologic findings for humans is unclear. Since these alterations may constitute a chemically-induced change in the growth or organization of structural (or neurochemical) elements, they are to be regarded as possible rather than clear indicators of a neurotoxic effect in accordance with EPA's neurotoxicity risk assessment guidelines (U.S. EPA, 1998). Whether the alterations are adverse to humans remains unclear for several reasons. Effects reported by Hannah and Roth (1991), for example, are highly-selective and could be caused by environmental factors not directly related to exposure. The relationship of these alterations to a functional impairment is unclear, while cerebellar neurological sequelae are not the most obvious symptoms associated with H_2S exposure. These studies and issues are treated in more detail in the accompanying Toxicological Review of Hydrogen Sulfide (U.S. EPA, 2003).

Nasal tract lesions have been designated as the critical effect for this assessment, based on: the relative clarity of the mechanistic basis (most likely irritation), information available on dose-response, and the adverse nature of the endpoint for nasal tract lesions as compared to the neurologic endpoints. The levels of H_2S associated with these effects appear to be similar for either endpoint (e.g., the possible indicator of neurotoxic effects reported by Hannah and Roth [1991] to occur at 28 mg/m^3 H_2S and the no-effect level of nasal tract lesions reported by Brenneman et al. (2000) at 14 mg H_2S /m^3) is some indication that consideration for the one will also address the other. On the other hand, the same judgments made here concerning clarity of mechanism or dose-response or adversity could serve as the basis for reevaluating the critical endpoint if these matters were further resolved for neurologic effects.

Dorman et al. (2000) examined fertility and developmental effects on Sprague-Dawley rats exposed to 0, 10, 30, or 80 ppm (0, 14, 42, or 111 mg/m^3) H_2S 6 hr/day, 7 days/week for 2 weeks prior to breeding. Exposures continued during a 2-week mating period and throughout gestational days 0-19 (GD 0-19). Non-pregnant adult females were exposed for an additional 23-24 days following the 2-week breeding period. Exposure to dams and their pups (8/litter after culling) resumed between PND 5 and 18. Adult males were exposed to H_2S for 70 consecutive days. There were no statistically significant reproductive performance effects

(mating index, fertility index, postimplantation loss per litter, and number of late resorptions or stillbirths) in F_0 animals. Also, the number of live pups, litter size, average length of gestation, and average number of implants per female were not affected. There were no statistically significant effects on sperm production or morphology in F_0 males. There were no histological findings in the females which were considered treatment-related. In pups, there were no statistically significant increases in structural malformations, and there was also no significant differences in pup weight gain or development (pinnae detachment, surface righting, incisor eruption and negative geotaxis, vaginal patency, preputial separation, and eyelid separation). Pups also did not exhibit any treatment-related effects on motor activity, acoustic startle response, passive avoidance observed, FOB, or surface righting ability. Pup terminal body and organ weights in all exposure groups were comparable to controls, and no gross observations were considered treatment-related by the study authors. Microscopic examination of nervous tissues failed to demonstrate any treatment-related effects in pups.

For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).

I.B.5. Confidence in the Inhalation RfC

Study — Medium
Database — Medium - High
RfC — Medium - High

Overall confidence in this RfC assessment is medium to high. Although the principal study was well-designed and properly conducted, examining sensitive endpoints, it was subchronic in duration and only male rats were examined. For these reasons it was assigned medium confidence. On the other hand, the medium to high confidence assigned to the database for H_2S is based in large part because it addresses developmental and reproductive endpoints as well as numerous specialized studies examining, in detail, the target tissues of H_2S exposure (portal-of-entry and neurological) and mechanism.

Certain information within the database suggests that children and neonatal animals could be selectively susceptible to neurologic effects at high - much higher than the RfC - H_2S concentrations (e.g., > 0.6 mg/m^3). Effects of H_2S exposure on developing brain cells in newborn rats as well as possible effects on spermatogenesis in male rats have also been described in the database. While the data suggests that the developing human fetus could be at risk, the exposure levels producing these effects are in the same range or slightly higher than those producing the critical sentinel effect (nasal tract lesions) in adult animals. This finding ameliorates the concern that young animals (and possibly children) may be especially susceptible to the effects from relatively low-level chronic exposures to H_2S. Ascribing

relevance of this apparent susceptibility to environmental levels of H_2S such as the RfC would be conjectural.

The database for H_2S clearly demonstrates that nasal tract and neurologic effects may be elicited by this compound. Neurologic alterations in animals noted at the lowest level of H_2S exposure are regarded as possible indicators of a neurotoxic effect rather than a clearly adverse effect. These alterations, including altered morphology of cerebellar Purkinje cells (Hannah and Roth, 1991) at 28 mg/m^3 and altered neurotransmitter levels in the brains of postpartum rats pups exposed *in utero* and postpartum to 28 mg/m^3 H_2S (Skrajny et al., 1992), are in the same range as noted for the clearly adverse nasal tract lesions reported by Brenneman et al. (2000) at a NOAEL of 14 mg/m^3 H_2S. The closeness in exposure levels eliciting these effects indicate that the exposure range is in the critical transitional area in the exposure-dose-severity continuum for a variety of endpoints, with data available in some instances to discriminate the mechanistic transition from being a nonadverse to an adverse response. Therefore, the nearness in effect levels for nasal tract and neurological endpoints not only increases the confidence that a health protective RfC has been chosen, but also provides some assurance that the uncertainty factors applied for one effect will also address the other.

Confidence in the database is medium to high while the confidence in the RfC is medium to high. A chronic inhalation study in rats and mice, with additional dose groups, and further research into the effect of H_2S on the developing brain would increase the confidence in the database.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF)

I.B.6. EPA Documentation and Review of the Inhalation RfC

Source Document — Toxicological Review of Hydrogen Sulfide (U.S. EPA, 2003)

This assessment was peer reviewed by scientists external to the U.S. EPA. Their comments have been evaluated carefully and incorporated in finalization of this IRIS Summary. A record of these comments is included as an appendix to the Toxicological Review of Hydrogen Sulfide (U.S. EPA, 2003). ***To review this appendix, exit to the toxicological review, Appendix A, External Peer Review -- Summary of Comments and Disposition (PDF)***

Date of Agency Consensus — 06/20/2003

I.B.7. EPA Contacts (Inhalation RfC)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (fax), or hotline.iris@epa.gov (Internet address).

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Hydrogen sulfide (H_2S)
CASRN — 7783-06-4
Last Revised — 07/28/2003

Under the Draft Revised Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1999), data are *inadequate* for an assessment of the carcinogenic potential of hydrogen sulfide. No relevant data could be located from which to develop an assessment.

II.A. Evidence for Human Carcinogenicity

Not applicable.

II.A.1. Weight-of-Evidence Characterization

Not applicable.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).

II.A.2. Human Carcinogenicity Data

Not applicable.

II.A.3. Animal Carcinogenicity Data

Not applicable.

II.A.4. Supporting Data for Carcinogenicity

Not applicable.

II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

II.B.1. Summary of Risk Estimates

Not applicable.

II.B.2. Dose-Response Data (Carcinogenicity, Oral Exposure)

Not applicable.

II.B.3. Additional Comments (Carcinogenicity, Oral Exposure)

Not applicable.

II.B.4. Discussion of Confidence (Carcinogenicity, Oral Exposure)

Not applicable.

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

II.C.1. Summary of Risk Estimates

Not applicable.

II.C.2. Dose-Response Data for Carcinogenicity, Inhalation Exposure

Not applicable.

II.C.3. Additional Comments (Carcinogenicity, Inhalation Exposure)

Not applicable.

II.C.4. Discussion of Confidence (Carcinogenicity, Inhalation Exposure

Not applicable.

II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Not applicable.

Source Documents -- Toxicological Review of Hydrogen Sulfide (U.S. EPA, 2003). To review the External Peer Review, ***exit to the toxicological review, Appendix A, External Peer Review -- Summary of Comments and Disposition (PDF)***

II.D.2. EPA Review (Carcinogenicity Assessment)

Not applicable.

Agency Consensus Date — 06/20/2003

II.D.3. EPA Contacts (Carcinogenicity Assessment

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (fax), or hotline.iris@epa.gov (Internet address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — Hydrogen sulfide (H_2S)
CASRN — 7783-06-4

VI.A. Oral RfD References

None

VI.B. Inhalation RfC References

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VI.C. Carcinogenicity Assessment References

U.S. EPA (U.S. Environmental Protection Agency). (1999) Guidelines for carcinogen risk assessment. Review Draft, NCEA-F-0644, July 1999. Risk Assessment Forum.

VII. Revision History

Substance Name — Hydrogen sulfide (H_2S)
CASRN — 7783-06-4

Date	Section	Description
10/01/1990	I.B.	Inhalation RfC summary on-line
07/01/1995	I.B.	Inhalation RfC replaced; new RfC
07/28/2003	I., II.	Revised RfC, cancer assessment discussion added. (Previous RfD withdrawn)

VIII. Synonyms

Substance Name — Hydrogen sulfide (H_2S)
CASRN — 7783-06-4
Last Revised — 07/28/2003

- Acide sulfhydrique [French]
- Acide sulphhydrique
- Dihydrogen monosulfide
- Dihydrogen sulfide
- EINECS 231-977-3
- FEMA No. 3779
- HSDB 576
- Hydrogen sulfide
- Hydrogen sulfide (ACGIH:OSHA)
- Hydrogen sulfide (H_2S)
- Hydrogen sulfure [French]
- Hydrogen sulfuric acid
- Hydrogen sulphide
- Hydrogene sulfure [French]
- Hydrogene sulphure
- Hydrosulfuric acid

- Idrogeno solforato [Italian]
- RCRA waste number U135
- Schwefelwasserstoff [German]
- Sewer gas
- Siarkowodor [Polish]
- Stink DAMP
- Sulfur hydride
- Sulfureted hydrogen
- Zwavelwaterstof [Dutch]