and included as part of the public comment. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

Docket: The index to the docket for this action is available electronically at *http://www.regulations.gov* and in hard copy at EPA Region IX, 75 Hawthorne Street, San Francisco, California. While all documents in the docket are listed in the index, some information may be publicly available only at the hard copy location (*e.g.,* copyrighted material), and some may not be publicly available in either location (*e.g.,* CBI). To inspect the hard copy materials, please schedule an appointment during normal business hours with the contact listed in the **FOR FURTHER INFORMATION CONTACT** section.

FOR FURTHER INFORMATION CONTACT: Mae Wang, EPA Region IX, (415) 947–4124, *wang.mae@epa.gov.*

SUPPLEMENTARY INFORMATION:

This document concerns the delegation of unchanged NESHAP to the Maricopa County Air Quality Department, the Nevada Division of Environmental Protection, and the Washoe County District Health Department, Air Quality Management Division. In the Rules and Regulations section of this **Federal Register**, EPA is amending regulations to reflect the current delegation status of NESHAP in Arizona and Nevada. EPA is taking direct final action without prior proposal because the Agency believes this action is not controversial. If we receive adverse comments, however, we will publish a timely withdrawal of the direct final rule and address the comments in subsequent action based on this proposed rule. Please note that if we receive adverse comment on an amendment, paragraph, or section of this rule and if that provision may be severed from the remainder of the rule, we may adopt as final those provisions of the rule that are not the subject of an adverse comment.

We do not plan to open a second comment period, so anyone interested in commenting should do so at this time. If we do not receive adverse comments, no further activity is planned. For further information, please see the direct final action.

Authority: This action is issued under the authority of Section 112 of the Clean Air Act, as amended, 42 U.S.C. Section 7412.

Dated: January 20, 2010.

Deborah Jordan,

Director, Air Division, Region IX.

[FR Doc. 2010–4077 Filed 2–25–10; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[EPA–HQ–TRI–2009–0844; FRL–9119–2]

RIN 2025–AA27

Hydrogen Sulfide; Community Right-to-Know Toxic Chemical Release Reporting

AGENCY: Environmental Protection Agency (EPA).

ACTION: Intent to consider lifting administrative stay; opportunity for public comment.

SUMMARY: EPA is announcing that it is considering whether to lift the Administrative Stay of the Emergency Planning and Community Right-to-Know Act (EPCRA) section 313 toxic chemical release reporting requirements for hydrogen sulfide (Chemical Abstracts Service Number (CAS No.) 7783–06–4). Hydrogen sulfide was added to the EPCRA section 313 list of toxic chemicals in a final rule published in the **Federal Register** on December 1, 1993. However, on August 22, 1994, EPA issued an Administrative Stay of the reporting requirements for hydrogen sulfide in order to evaluate issues brought to the Agency's attention after promulgation of the final rule concerning the human health effect basis for the listing and the Agency's use of exposure analysis in EPCRA section 313 listing decisions. Although the final rule listing hydrogen sulfide under section 313 of EPCRA remained in force, the stay deferred the reporting requirements for hydrogen sulfide while EPA completed this further evaluation. EPA has now completed its further evaluation, including a consideration of additional information that has become available since the stay was put in place regarding the human health and environmental effects of hydrogen sulfide. Based on this further evaluation, EPA believes that the Administrative Stay should be lifted. By this current action, EPA is not revisiting the original listing decision, which was accomplished by final rule on December 1, 1993. Rather, EPA is merely presenting its rationale for why the Administrative Stay of the reporting requirements for hydrogen sulfide should be lifted. After consideration of comments received, the Agency will issue another **Federal Register** document responding to comments and taking appropriate action.

DATES: Comments must be received on or before April 27, 2010.

ADDRESSES: Submit your comments, identified by Docket ID No. EPA–HQ–TRI–2009–0844, by one of the following methods:

- *www.regulations.gov:* Follow the on-line instructions for submitting comments.
- *E-mail: oei.docket@epa.gov.*
- *Mail:* Office of Environmental Information (OEI) Docket, Environmental Protection Agency, Mail Code: 28221T, 1200 Pennsylvania Ave., NW., Washington, DC 20460
- *Hand Delivery:* EPA Docket Center (EPA/DC), EPA West, Room 3334, 1301 Constitution Ave., NW., Washington, DC 20460. Such deliveries are only accepted during the Docket's normal hours of operation, and special arrangements should be made for deliveries of boxed information.

Instructions: Direct your comments to Docket ID No. EPA–HQ–TRI–2009–0844. EPA's policy is that all comments received will be included in the public docket without change and may be made available online at *www.regulations.gov,* including any personal information provided, unless the comment includes information claimed to be Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. Do not submit information that you consider to be CBI or otherwise protected through *www.regulations.gov* or e-mail. The *www.regulations.gov* Web site is an "anonymous access" system, which means EPA will not know your identity or contact information unless you provide it in the body of your comment. If you send an e-mail comment directly to EPA without going through *www.regulations.gov,* your e-mail address will be automatically captured and included as part of the comment that is placed in the public docket and made available on the Internet. If you submit an electronic comment, EPA recommends that you include your name and other contact information in the body of your comment and with any disk or CD–ROM you submit. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment. Electronic files should avoid the use of special characters, avoid any form of encryption, and be free of any defects or viruses.

Docket: All documents in the docket are listed in the *www.regulations.gov* index. Although listed in the index, some information is not publicly available, e.g., CBI or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, will be publicly

available only in hard copy. Publicly available docket materials are available either electronically in *www.regulations.gov* or in hard copy at the OEI Docket, EPA/DC, EPA West, Room 3334, 1301 Constitution Ave., NW., Washington, DC. This Docket Facility is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566–1744, and the telephone number for the OEI Docket is (202) 566–1752.

FOR FURTHER INFORMATION CONTACT: Daniel R. Bushman, Environmental Analysis Division, Office of Information Analysis and Access (2842T), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; *telephone number:* 202–566–0743; *fax number:* 202–566–0677; *e-mail: bushman.daniel@epa.gov,* for specific information on this document. For general information on EPCRA section 313, contact the Emergency Planning and Community Right-to-Know Hotline, toll free at (800) 424–9346 or (703) 412–9810 in Virginia and Alaska or toll free, TDD (800) 553–7672, *http://www.epa.gov/epaoswer/hotline/.*

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does This Action Apply to Me?

You may be potentially affected by this action if you manufacture, process, or otherwise use hydrogen sulfide. Potentially affected categories and entities may include, but are not limited to:

Category	Examples of potentially affected entities
Industry	Facilities included in the following NAICS manufacturing codes (corresponding to SIC codes 20 through 39): 311*, 312*, 313*, 314*, 315*, 316, 321, 322, 323*, 324, 325*, 326*, 327, 331, 332, 333, 334*, 335*, 336, 337*, 339*, 111998*, 211112*, 212324*, 212325*, 212393*, 212399*, 488390*, 511110, 511120, 511130, 511140*, 511191, 511199, 512220, 512230*, 519130*, 541712*, or 811490*. *Exceptions and/or limitations exist for these NAICS codes. Facilities included in the following NAICS codes (corresponding to SIC codes other than SIC codes 20 through 39): 212111, 212112, 212113 (correspond to SIC 12, Coal Mining (except 1241)); or 212221, 212222, 212231, 212234, 212299 (correspond to SIC 10, Metal Mining (except 1011, 1081, and 1094)); or 221111, 221112, 221113, 221119, 221121, 221122, 221330 (Limited to facilities that combust coal and/or oil for the purpose of generating power for distribution in commerce) (correspond to SIC 4911, 4931, and 4939, Electric Utilities); or 424690, 425110, 425120 (Limited to facilities previously classified in SIC 5169, Chemicals and Allied Products, Not Elsewhere Classified); or 424710 (corresponds to SIC 5171, Petroleum Bulk Terminals and Plants); or 562112 (Limited to facilities primarily engaged in solvent recovery services on a contract or fee basis (previously classified under SIC 7389, Business Services, NEC)); or 562211, 562212, 562213, 562219, 562920 (Limited to facilities regulated under the Resource Conservation and Recovery Act, subtitle C, 42 U.S.C. 6921 *et seq.*) (correspond to SIC 4953, Refuse Systems).
Federal Government	Federal facilities.

This table is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Some of the entities listed in the table have exemptions and/or limitations regarding coverage, and other types of entities not listed in the table could also be affected. To determine whether your facility would be affected by this action, you should carefully examine the applicability criteria in part 372 subpart B of Title 40 of the Code of Federal Regulations. If you have questions regarding the applicability of this action to a particular entity, consult the person listed in the preceding **FOR FURTHER INFORMATION CONTACT** section.

B. How Should I Submit CBI to the Agency?

Do not submit CBI information to EPA through *www.regulations.gov* or e-mail. Clearly mark the part or all of the information that you claim to be CBI. For CBI information in a disk or CD ROM that you mail to EPA, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is claimed as CBI. In addition to one complete version of the comment that includes information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

II. Introduction

Section 313 of EPCRA, 42 U.S.C. 11023, requires certain facilities that manufacture, process, or otherwise use listed toxic chemicals in amounts above reporting threshold levels to report their environmental releases and other waste management quantities of such chemicals annually. These facilities must also report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the Pollution Prevention Act (PPA), 42 U.S.C. 13106. EPCRA section 313 established an initial list of toxic chemicals composed of more than 300 chemicals and 20 chemical categories.

EPCRA section 313(d) authorizes EPA to add or delete chemicals from the list and sets forth criteria for these actions. Specifically, EPCRA section 313(d)(2) states that EPA may add a chemical to the list if "there is sufficient evidence to establish any one" of the listing criteria. Therefore, to add a chemical, EPA must demonstrate that at least one criterion is met, but need not determine whether any other criterion is met. Conversely, EPCRA section 313(d)(3) states that to remove a chemical from the list, EPA must determine that "there is not sufficient evidence to establish any" of the Section 313(d)(2) criteria. Therefore, to remove a chemical, EPA must demonstrate that none of the criteria are met. The EPCRA section 313(d)(2) criteria are:

(A) The chemical is known to cause or can reasonably be anticipated to cause significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases.

(B) The chemical is known to cause or can reasonably be anticipated to cause in humans—

(i) Cancer or teratogenic effects, or

(ii) Serious or irreversible—

(I) Reproductive dysfunctions,

(II) Neurological disorders,

(III) Heritable genetic mutations, or

(IV) Other chronic health effects.

(C) The chemical is known to cause or can be reasonably anticipated to cause, because of

(i) Its toxicity,

(ii) Its toxicity and persistence in the environment, or

(iii) Its toxicity and tendency to bioaccumulate in the environment, a significant adverse effect on the environment of sufficient seriousness, in the judgment of

the Administrator, to warrant reporting under this section.

EPA often refers to the section 313(d)(2)(A) criterion as the "acute human health effects criterion;" the section 313(d)(2)(B) criterion as the "chronic human health effects criterion;" and the section 313(d)(2)(C) criterion as the "environmental effects criterion."

Under EPCRA section 313(e)(1), any person may petition EPA to add chemicals to or delete chemicals from the list. EPA issued a statement of petition policy and guidance in the **Federal Register** of February 4, 1987 (52 FR 3479) to provide guidance regarding the recommended content and format for submitting petitions under EPCRA section 313(e). EPA also issued guidance in the **Federal Register** of May 23, 1991 (56 FR 23703) regarding the recommended content of petitions to delete individual members of the section 313 metal compound categories. In addition, EPA published in the **Federal Register** of November 30, 1994 (59 FR 61432) a statement clarifying its interpretation of the section 313(d)(2) and (d)(3) criteria for modifying the section 313 list of toxic chemicals.

III. Background Information

A. What is the History of the Listing of Hydrogen Sulfide Under EPCRA Section 313?

In response to a petition from the Natural Resources Defense Council and the Governor of New York, hydrogen sulfide, along with 20 other chemicals and two chemical categories, was added to the EPCRA section 313 list of toxic chemicals as part of a 1993 final rule (December 1, 1993, 58 FR 63500). Hydrogen sulfide was listed under the criteria of EPCRA section 313(d)(2)(B) (chronic human health effects) based on chronic neurotoxic effects in humans and under EPCRA section 313(d)(2)(C) (environmental effects) based on acute aquatic toxicity. However, on August 22, 1994 (59 FR 43048), EPA issued an Administrative Stay of the EPCRA section 313 reporting requirements for hydrogen sulfide. Although the final rule listing hydrogen sulfide under section 313 of EPCRA remained in force, the stay deferred the reporting requirements for hydrogen sulfide.

B. What Was the Basis for the Administrative Stay?

After the final rule was issued adding hydrogen sulfide to the EPCRA section 313 list of toxic chemicals, some members of the regulated community expressed a concern that the "chronic human health effects" basis for listing hydrogen sulfide under EPCRA section 313(d)(2)(B) changed between the proposed rule (September 8, 1992, 57 FR 41020) and the final rule (December 1, 1993, 58 FR 63500), and that commenters on the proposed rule therefore did not have an opportunity to comment on that individual basis for the listing. Specifically, although the Agency cited the same acute aquatic toxicity as an "environmental effects" basis for the listing under EPCRA section 313(d)(2)(C) in both the proposed and final rules, the Agency also cited chronic respiratory effects as a "chronic human health effects" basis under EPCRA section 313(d)(2)(B) in the proposed rule, but chronic neurotoxic effects as a "chronic human health effects" basis under that same provision in the final rule. In addition, after issuance of the final rule, some members of the regulated community expressed concern that EPA's decision not to include an exposure analysis in deciding to list hydrogen sulfide on the basis of chronic human health effects was inconsistent with past Agency practice.

Although EPA did not agree that it had been inconsistent in its use of exposure analyses, and notwithstanding the fact that the listing decision was dictated by the acute aquatic toxicity finding alone under EPCRA section 313(d)(2)(C), the Agency issued an Administrative Stay of the reporting requirements for hydrogen sulfide in order to review the concerns raised after issuance of the final rule by some members of the regulated community.

C. What Is the Purpose of This Document?

The purpose of this document is to provide the public with the opportunity to comment on EPA's review of the currently available data on the human health and environmental effects of hydrogen sulfide—specifically, chronic respiratory effects, chronic neurotoxic effects, and acute, chronic and early-life stage aquatic toxicity—and EPA's belief that the Administrative Stay should be lifted based on that data. EPA's analysis of the toxicity of hydrogen sulfide is based on the Agency's latest Toxicological Review of Hydrogen Sulfide (Ref. 1), as well as a reassessment of the environmental effects of hydrogen sulfide (Ref. 2). These assessments are discussed in detail in Unit IV. of this document. In addition, this document addresses the concerns raised regarding use of exposure analyses. After consideration of comments received, the Agency will issue another **Federal Register** document responding to comments and taking appropriate action.

IV. What Is EPA's Technical Review of Hydrogen Sulfide?

A. What Is EPA's Evaluation of the Human Health Toxicity of Hydrogen Sulfide?

The following assessment of the human health toxicity of hydrogen sulfide is based on the information contained in EPA's most recent (June 2003) Integrated Risk Information System (IRIS) Toxicological Review of Hydrogen Sulfide (Ref. 1).

Hydrogen sulfide is a colorless, acutely toxic gas at high concentrations. Hydrogen sulfide gas is absorbed rapidly through the lungs and can be absorbed through the gastrointestinal tract. Oral exposure is not likely to occur. In animals and humans, it distributes to the blood, brain, lungs, heart, liver, spleen, and kidneys. Oxidation is the primary metabolic pathway for hydrogen sulfide, with thiosulfate and sulfate as metabolites. Metabolism in laboratory animals and in humans appears to be similar. Hydrogen sulfide metabolites are excreted in the urine.

A considerable body of case studies exists on the human health impacts resulting from acute exposure to high levels of hydrogen sulfide. Levels in the range of 500 to 1,000 parts per million (ppm) (695 to 1,390 milligrams per cubic meter (mg/m^3)) are life-threatening and can cause immediate unconsciousness followed by serious and debilitating neurologic and respiratory sequelae and death (Ref. 1). While complete recovery from a high exposure episode has been reported, more often long term or even irreversible harmful neurological effects remain. Several groups of investigators (Tvedt, *et al.* (Refs. 3 and 4); Wasch, *et al.* (Ref. 5)) have reported long-term persistent adverse neurological effects from hydrogen sulfide-induced unconsciousness in humans during occupational, accidental, and chronic exposures, including neuropsychological and neurobehavorial decrements and brain damage. These irreversible effects are believed to be caused by an essentially hypoxic (low oxygen) condition existing in persons who become unconscious from a high exposure to hydrogen sulfide. Because a loss of oxygen (anoxia) utilization in tissues, particularly the brain, occurs in such poisonings, it is possible to attribute persistent neuronal damage to this effect. Permanent (chronic) damage is commonly observed clinically when

brain tissues have been deprived of oxygen due to inadequate delivery of the gas or to interrupted utilization of oxygen by cells, as is the case with hydrogen sulfide poisoning.

The observed nonirritant effects produced in mammals from exposure to hydrogen sulfide gas may primarily be attributed to the cellular anoxia produced by inhibition of cytochrome oxidase (Ref. 1). Inhibition of cytochrome oxidase reduces the oxygen dependent metabolism of the cell, reduces cell energy sources (e.g., adenosine triphosphate (ATP)), increases products of anaerobic metabolism such as lactic acid, and produces cell death. Hence, cells with a high oxygen demand such as those in brain and cardiac tissue are thought to be more sensitive to disruption of oxidative metabolism and may be considered selected targets for the toxicity of hydrogen sulfide.

1. Chronic Toxicity. EPA has reviewed the available toxicological studies on hydrogen sulfide in its most recent IRIS Toxicological Review of Hydrogen Sulfide (Ref. 1) and concluded that hydrogen sulfide can cause chronic human health toxicity. As reported in IRIS, the upper respiratory tract (neuronal and basal cells of the olfactory nasal epithelium) and neurologic tissues are both targets for hydrogen sulfide toxicity. The weight-of-evidence from the animal studies indicates that nasal tract lesions and neurological effects of hydrogen sulfide are dose-dependent, and both effects are clearly of relevance to humans. The levels of hydrogen sulfide associated with these effects appear to be similar for either endpoint (e.g., the no-effect level of nasal tract lesions reported by Brenneman, *et al.* (Ref. 6) at 10 ppm (14 mg/m^3) and the likely indicator of neurotoxic effects reported by Hannah and Roth (Ref. 7) at 20 ppm (28 mg/m^3)), which is some indication that consideration for one effect will also address the other (Ref. 1).

a. Upper Respiratory Tract Toxicity. Several subchronic exposure studies in rats and mice indicate that low concentrations of hydrogen sulfide cause nasal lesions of the olfactory mucosa (Brenneman, *et al.* (Ref. 6); Dorman, *et al.* (Ref. 8); Chemical Industry Institute of Toxicology (Ref. 9); Lopez, *et al.* (Ref. 10); Dorman, *et al.* (Ref. 11)). The nasal lesions are consistent with the neurotoxic and irritant properties of this gas. Based on the demonstrable histopathology, information available on exposure-dose-response, and the commonality of the underlying mechanism (cytochrome oxidase inhibition and irritation) between animals and humans, there is compelling indication that such effects are reasonably anticipated to occur in humans chronically exposed to hydrogen sulfide (Hirsch and Zavala (Ref. 12)).

Brenneman, *et al.* (Ref. 6) reported significant concentration-related increases in the incidence and severity of lesions to the nasal olfactory epithelium in rats exposed to 0, 10, 30, or 80 ppm of hydrogen sulfide for 10 weeks. The effects consisted of olfactory neuron loss and basal cell hyperplasia in rats exposed to 30 or 80 ppm, 6 hours/day, 7 days/week for 10 weeks; no adverse effects were observed at 10 ppm. The severity of the olfactory neuron loss was concentration-related; however, an inverse relationship between severity and concentration was observed for the basal cell hyperplasia suggesting that as the concentration increased, the ability of the olfactory epithelium to regenerate decreased. In contrast, earlier studies conducted by the Chemical Industry Institute of Toxicology (CIIT) (Refs. 13 and 14) where rats and mice were exposed to 0, 10.1, 30.5, or 80 ppm of hydrogen sulfide, 6 hours/day, 5 days/week for 13 weeks, did not find significant alterations in the nasal turbinates of Fischer-344 (F–344) or Sprague-Dawley rats exposed to 80 ppm or less of hydrogen sulfide. Inflammation of the squamous portion of the nasal mucosa was observed in mice exposed to 80 ppm hydrogen sulfide, 6 hours/day, 5 days/week for 13 weeks (CIIT (Ref. 9)); the no-observed-adverse-effect-level (NOAEL) for this effect is 30.5 ppm. However, a re-examination of the histological specimens from this study revealed a statistically significant increase in the incidence of olfactory neuron loss in Sprague-Dawley rats, F–344 rats, and B6C3F1 mice exposed to 30 or 80 ppm; no lesions were observed at 10 ppm (Dorman, *et al.* (Ref. 11)). In addition, increases in the incidence of bronchiolar epithelial hyperplasia and hypertrophy were observed in female Sprague-Dawley rats exposed to 30 or 80 ppm and male Sprague-Dawley and F–344 rats exposed to 80 ppm.

b. Neurotoxicity. The neurotoxic effects of low level hydrogen sulfide exposure have been primarily assessed from neurodevelopmental toxicity studies. Male Sprague-Dawley rats and male B6C3F1 mice exposed to 80 ppm of hydrogen sulfide (6 hours/day, 5 days/week for 13 weeks) had a statistically significant decrease in absolute (but not relative) brain weight (CIIT (Ref. 13) and Dorman, *et al.* (Ref. 11)) at 80 ppm but not 30 ppm. In an earlier study, Skrajny, *et al.* (Ref. 15) examined the effect of hydrogen sulfide exposure on serotonin and norepinephrine levels in the developing cerebellum and frontal cortex of Sprague-Dawley rats. Timed-pregnant rats were exposed to 0, 20 or 75 ppm for 7 hours/day from gestational day 5 until post natal day (PND) 21. There were statistically significant increases in serotonin levels in the frontal cortex on PND 21 in pups exposed to 20 ppm hydrogen sulfide and increases in serotonin levels in the cerebellum and frontal cortex on postpartum days 14 and 21 in pups exposed to 75 ppm hydrogen sulfide. Norepinephrine levels were increased at 75 ppm in the cerebellum on PNDs 7, 14, and 21, and in the frontal cortex on PND 21. At 20 ppm frontal cortex norepinephrine levels were decreased compared to controls on days 14 and 21.

In a similarly designed study using the same exposure protocol as the CIIT (Refs. 9, 13, and 14) and Dorman, *et al.* (Ref. 11) studies, Hannah and Roth (Ref. 7) evaluated the perinatal effect of hydrogen sulfide on developing cerebellar Purkinje cells. Sprague-Dawley dams were exposed to 0, 20 or 50 ppm hydrogen sulfide for 7 hr/day from gestational day 5 until PND 21. Exposure to both 20 and 50 ppm interrupted normal dendritic growth of Purkinje cells in the brain of offspring. In later studies using this same experimental protocol, Hannah, *et al.* (Refs. 16 and 17) also found decreases in several brain amino acid levels in Sprague-Dawley rats exposed to 75 ppm of hydrogen sulfide.

The significance of the morphological changes and alteration of brain neurotransmitters in these studies and the Skrajny, *et al.* (Ref. 15) study to humans is unclear. Since Purkinje cell alterations and changes in neurotransmitter levels may constitute a hydrogen sulfide-induced change in the growth or organization of structural (or neurochemical) elements, they are to be regarded as indicators of a neurotoxic effect in accordance with guidance in EPA's neurotoxicity risk assessment guidelines (Ref. 18). The question as to what functional impairment these alterations might lead to in humans remains unclear. Predicting particular functional impairments from decreased brain weight and specific structural alterations such as reported by the CIIT (Refs. 9, 13, and 14) and Dorman, *et al.* (Ref. 11) studies and Hannah and Roth (Ref. 7) is difficult due to the selective nature of the observed alterations and the dynamic self-organizing response of the developing brain to injury. Although behavioral testing has not indicated that alterations of brain neurotransmittters

have a functional impact (Dorman, *et al.* (Ref. 8)), further examination of the biochemical and functional aspects of the developing brain in hydrogen sulfide-exposed animals is warranted and neurotoxic effects cannot be discounted.

Available information indicates that the dose-response character of indicators of neurotoxicity (such as the alterations to cerebellar Purkinje cells reported by Hannah and Roth (Ref. 7) and nasal olfactory (neuronal cell) lesions reported by Brenneman, *et al.* (Ref. 6)) may be similar to one another, such that consideration of one may be inclusive of the other. However, more extensive and definitive information on the neurologic endpoints could reveal that these should be the most relevant endpoints, more so than nasal tract lesions. The IRIS Summary for Hydrogen Sulfide (Ref. 1) indicates that such information may provide sufficient reason to reassess hydrogen sulfide.

2. Summary. As stated in the IRIS Summary for Hydrogen Sulfide (Ref. 1) and as discussed above, both nasal tract lesions (upper respiratory effects) and neurologic effects are chronic effects of concern. These effects occur in a clear dose concentration manner with the lowest levels of hydrogen sulfide exposure associated with these effects ranging from 20 to 30 ppm (28 mg/m^3 to 41.7 mg/m^3).

B. What Is EPA's Evaluation of the Environmental Toxicity of Hydrogen Sulfide?

A number of ecotoxicity studies have been conducted on hydrogen sulfide, mainly on freshwater invertebrates and fish. Acute, chronic, and early-life stage toxicity values for hydrogen sulfide include numerous values that are well below 1 milligram per liter (mg/L), indicating that hydrogen sulfide is toxic at very low concentrations. EPA's ecological assessment (Ref. 2) includes an extensive listing of the aquatic toxicity values for hydrogen sulfide. Some examples of the values from Table 2–1 of EPA's ecological assessment (Ref. 2) are provided below.

Hydrogen sulfide acute toxicity values (96-hour LC_{50} (i.e., the concentration that is lethal to 50% of test organisms)) for freshwater fish ranged from 0.0149 mg/L (fathead minnow) to 0.0448 mg/L (bluegill). Chronic toxicity values for freshwater fish ranged from a 6-week lowest-observed-effect-concentration (LOEC) (growth rate) of 0.0005 mg/L in a tropical fish (*Mystus nemurus*) to a 430-day LOEC (final weight) of 0.009 mg/L for goldfish. Additionally, in early-life stage toxicity testing with eggs, fry, and juveniles of various freshwater fish species, endpoint values ranged from a 96-hour LC_{50} of <0.002 mg/L (yellow perch sac fry) to a 96-hour LC_{50} value of 0.0536 mg/L (fathead minnow). The hydrogen sulfide 96-hour LC_{50} values for freshwater invertebrates ranged from 0.021 mg/L (amphipod) to 1.07 mg/L (isopod), and 48- to 96-hour LC_{50} values for estuarine/marine invertebrates ranged from 0.063 mg/L (saltwater shrimp) to 0.332 mg/L (adult amphipod). The hydrogen sulfide EC_{50} (i.e., the concentration that is effective in producing a sublethal response in 50% of test organisms) values for estuarine/marine invertebrates included 0.01 mg/L (saltwater mussel) and 0.019 mg/L (sea urchin). Hydrogen sulfide chronic values for freshwater invertebrates ranged from a 65-day LOEC (reproduction) of 0.0031 mg/L to a 10-day LC_{50} value of 0.042 mg/L, both for the amphipod *Gammarus pseudolimnaeus.* For early-life stage toxicity testing, a 96-hour LC_{50} value of 0.034 mg/L was available for juvenile crayfish (freshwater species), and for estuarine/marine invertebrates, 48-hour LC_{50} values of 0.0087 mg/L and 0.0185 mg/L were available for white shrimp larvae and juveniles, respectively.

V. EPA's Use of Exposure Analyses

The Agency's position on the use of exposure analyses in listing decisions under EPCRA section 313 was presented in a proposed rule in the **Federal Register** of January 12, 1994 (59 FR 1788). The proposed rule provided the public with the opportunity to comment on the Agency's interpretation of the statutory listing criteria as it relates to the use of exposure considerations. After considering the comments received, EPA published in the **Federal Register** of November 30, 1994 (59 FR 61432) a statement clarifying its interpretation of the statutory requirements regarding how exposure will be considered in listing decisions. Subsequent to the final rule, EPA's interpretation of the statutory listing criteria as it relates to the consideration of exposure was upheld in *National Oilseed Processors Ass'n.* v. *Browner,* 924 F. Supp. 1193 (D.D.C. 1996), *aff'd in part & remanded in part, Troy Corp.* v. *Browner,* 120 F.3d 277 (D.C. Cir. 1997).

EPA has determined that hydrogen sulfide can reasonably be anticipated to cause serious or irreversible chronic human health effects at relatively low doses and thus is considered to have moderately high to high chronic toxicity. EPA does not believe that it is appropriate to consider exposure for chemicals that are moderately high to highly toxic based on a hazard assessment when determining if a chemical can be listed for chronic effects pursuant to EPCRA section 313(d)(2)(B) (see 59 FR 61432, 61433, 61440–61442). Hydrogen sulfide has also been determined to cause ecotoxicity at relatively low concentrations, and thus is considered to have high ecotoxicity. EPA believes that chemicals that induce death or serious adverse effects in aquatic organisms at relatively low concentrations (i.e., they have high ecotoxicity) have the potential to cause significant changes in the population of fish and other aquatic organisms, and can therefore reasonably be anticipated to cause a significant adverse effect on the environment of sufficient seriousness to warrant reporting. EPA does not believe that it is required to consider exposure for chemicals that have high ecotoxicity based on a hazard assessment when determining if a chemical can be listed for effects pursuant to EPCRA section 313(d)(2)(C) (*see* 59 FR 61432, 61433, 61440–61442).

VI. What Is EPA's Rationale That the Administrative Stay Should Be Lifted?

EPA's technical evaluation of hydrogen sulfide shows that it can reasonably be anticipated to cause chronic health effects in humans. The chronic health effects have been observed in laboratory animals at concentrations as low as 28 mg/m^3 (20 ppm) and 41.7 mg/m^3 (30 ppm). In addition, EPA's technical evaluation of hydrogen sulfide also shows that it can reasonably be anticipated to cause, because of its toxicity, significant adverse effects in aquatic organisms. Examples of hydrogen sulfide's ecological toxicity include acute toxicity (96-hour LC_{50}) values for freshwater fish that ranged from 0.0149 mg/L (fathead minnow) to 0.0448 mg/L (bluegill), indicating high aquatic toxicity. Examples of hydrogen sulfide's chronic ecological toxicity include freshwater fish values that ranged from a 6-week LOEC (growth rate) of 0.0005 mg/L in a tropical fish (*Mystus nemurus*) to a 430-day LOEC (final weight) of 0.009 mg/L for goldfish, also indicating high aquatic toxicity.

Based on the above findings, EPA believes that there is no basis for continuing the Administrative Stay of the reporting requirements for hydrogen sulfide, and that the Administrative Stay should therefore be lifted. As an aside, EPA notes also that it believes that the above findings clearly demonstrate the correctness of the Agency's final decision in December 1993 to list hydrogen sulfide on the EPCRA section 313 toxic chemicals list

based on the listing criteria in EPCRA sections 313(d)(2)(B) and (C).

Finally, in accordance with EPA's stated policy on the use of exposure assessments (59 FR 61432), EPA does not believe that an exposure assessment is appropriate for determining whether hydrogen sulfide meets the criteria of EPCRA section 313(d)(2)(B) or (C), and therefore the Administrative Stay should not be continued for lack of an exposure analysis.

VII. What Are the References Cited in This Document?

EPA has established an official public docket for this action under Docket ID No. EPA–HQ–TRI–2009–0844. The public docket includes information considered by EPA in developing this action, including the documents listed below, which are electronically or physically located in the docket. In addition, interested parties should consult documents that are referenced in the documents that EPA has placed in the docket, regardless of whether these referenced documents are electronically or physically located in the docket. For assistance in locating documents that are referenced in documents that EPA has placed in the docket, but that are not electronically or physically located in the docket, please consult the person listed in the above **FOR FURTHER INFORMATION CONTACT** section.

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List of Subjects in 40 CFR Part 372

Environmental protection, Community right-to-know, Reporting and recordkeeping requirements, and Toxic chemicals.

Dated: February 19, 2010.

Lisa P. Jackson,

Administrator.

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