March 30, 2009

Lisa Jackson, Administrator
United States Environmental Protection Agency
Ariel Rios Building
1200 Pennsylvania Avenue, N.W.
Washington, DC 20460

RE: Hydrogen sulfide needs Hazardous Air Pollutant listing under CAA Title III

Dear Administrator Jackson:

The community, environmental, and public health organizations named below request that you formally list Hydrogen Sulfide (H_2S) as a hazardous air pollutant (HAP), as defined in Title III, section 112(b) of the 1990 Clean Air Act Amendments (CAA). We assert that EPA must act to address adverse H_2S impacts based on evidence of harmful exposures in numerous communities and its toxicological effects at low concentrations such as non-cancer effects and emerging evidence that H_2S is a genotoxic agent, meaning it damages DNA. EPA has assessed the need to list H_2S as a HAP, but no formal listing action has been taken. H_2S is clearly an unlisted hazardous air pollutant.

H_2S, well known to cause death at high concentrations and respiratory-brain-nervous system effects at lower levels, escaped addition to the original list of 188 HAPs in 1990 due to opposition from the oil and gas industry, despite the EPA's attempts to include it. In January 1999, strong public support to add H_2S to Title III of federally recognized air toxics occurred when 145 public health, environmental and community groups in 32 states sent a request to EPA based on scientific studies suggesting chronic, low-level exposures cause permanent damage to the brain and central nervous system. Indeed, new toxicological evidence reveals H_2S can cause neuron death in the brain and serves as a solid regulatory basis that H_2S is far worse than just a stinky chemical triggering bad headaches, nausea and discomfort at citizens' home environment. The oil and gas industry have downplayed for decades the toxic effects of smelly H_2S emissions when citizens have to breathe horrible, nauseating rotten egg odors, while industry officials allege it's little more than a foul smelling odor with no harmful effects at sublethal low concentrations. Citizens who wrote to EPA in January 1999 are still waiting for the agency to take action on this highly toxic substance.

Today, it is unacceptable for communities to continue suffering the ill effects of toxic hydrogen sulfide gas exposure when the technology to control it is available and affordable. As EPA has learned, environmental injustice is a fact of life for thousands of communities across the nation and these residents all have a right to clean, safe air. It's past time for EPA to take action to formally acknowledge hydrogen sulfide's serious acute and chronic toxicity. As EPA Administrator, you have CAA authority to do the right thing based on a compelling body of H_2S medical evidence and air quality data indicating a need for better regulation of hydrogen sulfide. Specifically, CAA section 112(b)(2) provides "the Administrator shall periodically review the list established by this subsection and publish the results thereof and, where appropriate, revise such list by rule, adding pollutants which present, or may present, through inhalation or other routes of exposure, a threat of adverse human health effects…" Health studies support the need for EPA to list H_2S under CAA section 112(b), especially since H_2S's routine exposure effects—on a daily basis—are not addressed whatsoever under the accidental release provisions in section 112(r) of the CAA, where H_2S is currently regulated. Section 112(r) is not designed or intended to address daily toxic exposures.

The EPA began an informal review in the last ten years due to EPA's continuing health concerns and a prior request by 145 organizations sent January 25, 1999 to EPA Administrator Carol Browner. EPA's Office of Air Quality Planning & Standards has performed a new IRIS assessment and made recommendations in 2007 to the Assistant Administrator for Air and Radiation of options to address H_2S. Nonetheless, EPA has yet to make a decision to formally list H_2S as a CAA section 112(b) HAP and solicit comments in a Federal Register Notice.

Hydrogen sulfide, known as "poison gas" for its lethal properties, is probably the most common toxic air pollutant found in urban and rural communities. It is easily identifiable by its distinct rotten eggs odor. At least 73 industry categories emit H_2S in varying rates and volumes. Citizens impacted by industry H_2S have complained due to the acute and chronic effects of low level exposures. In recent years, H_2S's health concerns have occurred over frequent obnoxious odors from confined animal feeding operations (CAFOs). Data from the Poison Control Centers National Data Collection system show from 1983-1992 acute exposure to H_2S was linked to 29 deaths and 5,563 exposures, according to a 1995 article in the *American Journal of Emergency Medicine*. A U.S. multistate surveillance program found that 637 H_2S-related incidents occurred from 1993-2001, resulting in 63 public evacuations and injuring 185 people, according to a 2004 journal article written by federal health investigators and others.

Congress and the 1990 CAA study—Hydrogen Sulfide Report to Congress

The Congress considered listing hydrogen sulfide in 1990 as a hazardous air pollutant under CAA section 112(b), which regulates industrial sources with routine emissions of HAPs. Listing was negated by lobbying by the oil and gas industry, despite the health concerns of EPA. The EPA Administrator was instead directed by Congress in 1990 under section 112(n)(5) of the CAA to carry out a study "to assess the hazards to public health and the environment resulting from the emissions of H_2S associated with the extraction of oil and natural gas." However, it excluded many major point sources such as paper mills and others. The legal requirement to study H_2S associated only with oil and gas wells had been added to the CAA by the Senate Committee on Environment and Public Works, chaired by the late Quentin N. Burdick of North Dakota, due to serious concerns about lethal H_2S exposures. Witnesses testified before the Congress in 1990 that H_2S emissions related to the extraction and refining of oil and natural gas had resulted in deterioration of air quality, death and injury to livestock, and evacuation and hospitalization of untold numbers of residents located near the release point of such toxic air emissions. Similar H_2S problems continue to exist today due to a patchwork of inadequate regulations, weak H_2S ambient air standards, lack of H_2S ambient monitoring, and ineffective enforcement despite states' efforts to permit major sources for routine H_2S emissions. Loopholes remain in regulating H_2S and protecting public health as refineries receive higher sulfur crudes. The fact is H_2S is a highly neurotoxic substance deserving a HAP listing and a more protective national approach.

Conclusions of the EPA study became the *Report to Congress on Hydrogen Sulfide Air Emissions Associated with the Extraction of Oil and Natural Gas* (the Report to Congress), issued in October 1993 by the EPA Office of Air Quality Planning and Standards (EPA-453/R-93-045). The outdated status of H_2S since 1990 as a partly regulated air toxic is described in the Executive Summary of the 1993 Report to Congress where it states:

"[O]n the basis of information contained in accident records, it was determined at the time that H_2S is a chief concern from an accidental release standpoint and it would be listed under the accidental release provisions in section 112(r) of the CAA, but not under section 112(b). Substances regulated under 112(r) are known or may be anticipated to cause death, injury, or serious adverse effects to human health or the environment upon accidental release."

The executive summary of the *Hydrogen Sulfide Report to Congress* emphasizes "the [further] assessment of H_2S must include a review of existing State and industry control standards, techniques, and enforcement." However, we are not aware of ongoing efforts on H_2S by EPA since the last review in 2007 to assess it as a HAP, although an updated HAP review by EPA appears necessary based on emerging medical evidence of the human health hazards of H_2S exposure at low concentrations and its widespread occurrence in the U.S. from industrial sources.

Certainly the 112(r) approach to H_2S is necessary for handling higher lethal concentrations of H_2S. Deaths occur each year in the U.S. from accidental exposure to lethal doses of H_2S in the range of 500–900 parts per million (ppm). However, the weight of medical evidence strongly indicts H_2S as posing a serious human health hazard at low levels of exposure far below the lethality range even less than 100 ppm. In fact even levels in the parts per billion (ppb) range are associated with adverse health effects like headaches, nausea and loss of well-being.

Public concerns about routine low-level emissions of H_2S are well founded on its toxicity at unregulated low concentrations and its widespread occurrence in industries relying on processes using sulfur, sulfur compounds, or substances (i.e., oil and natural gas) containing significant sulfur as a natural contaminant. Since EPA submitted the *Hydrogen Sulfide Report to Congress* in October 1993, the agency has undertaken additional science reviews but chose not to pursue meaningful regulatory action as a national air quality priority to address widespread public health concerns about exposure to H_2S at low concentrations.

Industrial categories emitting hydrogen sulfide gas

The *Report to Congress on Hydrogen Sulfide Air Emissions Associated with the Extraction of Oil and Natural Gas* did not present a national emissions inventory of H_2S emissions in 1993 from many other industrial categories. Yet additional sources of H_2S include the following 73 industry sectors some with notorious H_2S emissions:
• Sour crude oil petroleum refineries • Pulp and paper mills • Paper production • Municipal sewage treatment plants • Large hog & livestock Confined Animal Feeding Operations (CAFOs) • Sour natural gas processing plants • Sour crude oil/sour natural gas handling stations/bulk petroleum terminals • Oil & gas production wells, flares, treating equipment at well sites & crude oil tank batteries • Sour natural gas transmission stations & compressor station sites & thereon site treating equipment • Bulk sour crude oil storage and pipelines • Carbon black mfg • Portland cement kilns • Municipal waste landfills • Coke ovens • Coal gasification plants • Tanneries of animal hides using sodium sulfide • Slaughterhouses, chicken houses with waste chicken incinerators, and rendering plants • Geothermal power plants • Sulfur products & hydrogen sulfide production plants • Animal fat and oil processing operations • Asphalt storage facilities • Blast furnaces, breweries and fermentation processes • Fertilizer production • Glue manufacturing • Metal processing (gold ore, lead ore, lead removal, copper ore sulfidizing and metallurgy) • Barium carbonate & barium salt production • Phosphoric acid production • Miscellaneous processes including the manufacture of carbon disulfide, dyes, textile printing, thiophene, sulfur, soap, phosphate purification, hydrochloric acid purification, cellophane, rubber and plastics processing, silk making, rayon making, pyrite burning, photoengraving, synthetic fibers, polysulfide caulking production, bromide-bromine, artificial flavor making, additives & sealant mfg, and refrigerant making • Fish, sugar beet and sugar cane processing, as well as other miscellaneous sources.

Many states lack effective hydrogen sulfide regulations and enforcement programs to protect public health

Every state has large sources of H_2S and many have affected communities who have complained to officials for help. States like Ohio are among 16 with no H_2S ambient air standards to enforce, and the lack of H_2S public health regulations for communities creates a nightmare that too many residents endure by suffering adverse health effects above H_2S health benchmarks. But even in the 34 states with H_2S limits citizens complain of weak ambient air standards and lack of protection. In states with larger sources of H_2S emissions and weak regulations, hydrogen sulfide is often the most common air pollution complaint filed with agencies. EPA's Regions are well aware of citizens filing thousands of complaints in Ohio, Michigan, Nebraska, Texas, Minnesota, Florida, Colorado, Alabama, Louisiana, New Mexico and others. Hundreds of H_2S complaints have been filed by several communities.

Environmental background concentrations of H_2S versus industrial concentrations

Hydrogen sulfide is primarily produced by sulfur-reducing bacteria and certain industrial processes. Generally, areas that are not exposed to industrial releases of H_2S have airborne concentrations of less than 1 ppb H_2S as an ultra-low background level. Ultra-low background H_2S levels are often below the odor threshold of 3-4 ppb and not associated with adverse health effects. Some natural areas away from industrial sites, however, can have higher ambient levels than 1 ppb, because H_2S is a natural byproduct of decomposing organic matter that contains sulfur.

Oil refineries and sour natural gas processing plants handling hydrogen sulfide can have process concentrations reaching 30-40% in certain units and pipes where small problems can allow highly obnoxious H_2S to be released. Industrial sources of H_2S can produce average concentrations in affected neighborhoods in the low parts per billion range (10-100 ppb) during low H_2S emissions, but maximum concentrations can reach 100-1,000 ppb and higher such as during industrial releases from oil pipeline breaks, leaking crude oil storage tanks, emergency flares with inefficient combustion, upset conditions in sulfur recovery units, abnormal conditions in amine treaters, fugitive leaks from process equipment, and other problems. Industrial accidents involving spills, leaks, and malfunctions of gases or liquids with H_2S can trigger higher concentrations well into the 10-1,000 ppm range. The accumulation of H_2S in low-lying areas can result in higher levels that can reach lethal concentrations.

General information about the toxicity of hydrogen sulfide gas

Inhalation of H_2S leads to adverse effects on consciousness, cardiac and pulmonary function depending on the level and length of exposure. Virtually all organ systems are affected by H_2S. The most vulnerable organs are those with exposed mucous membranes (e.g., eyes, nose and throat) and those with high oxygen demands (e.g., lungs, brain). Neurotoxicity of the central nervous system (causing nausea, dizziness, confusion, headache and sleeping problems) and pulmonary edema (build-up of fluid in the lungs) are other well-documented effects of hydrogen sulfide poisoning. Cardiovascular and gastrointestinal toxicity are associated with H_2S exposure. Hydrogen sulfide creates health effects by binding with iron in blood and cells to block the flow of oxygen to tissues and disrupting systems.

Hydrogen sulfide is similar to hydrogen cyanide (HCN) in toxicity and cellular effects. H_2S interferes with a crucial biological enzyme—cytochrome C oxidase, necessary for living cells to utilize oxygen and blockage of this vital enzyme may cause cellular death (1, 2). Cytochrome C oxidase is the last enzyme in a series in the respiratory electron transport chain of mitochondria inside the mitochondrial membrane. This key enzyme receives an electron from each of four cytochrome C molecules, and transfers them to one oxygen molecule, converting molecular oxygen (O_2) to two molecules of water (H_2O) in the cellular energy-making process. In the process, it binds four protons from the inner aqueous phase to make water, and in addition translocates four protons across the membrane, helping to establish a transmembrane difference of proton electrochemical potential that the enzyme ATP synthase then uses to make another vital cellular biochemical—ATP (adenosine triphosphate). ATP is the universal energy molecule in all living cells. ATP's cellular support function is so important that it is involved in triggering all muscle contractions including the heart and lungs. Without ATP, the heart and lungs fail to work.

Oxygen (as molecular oxygen O_2) is constantly required by living cells to run billions of complex biochemical reactions, activities and functions every single second to maintain life, including intricate cellular respiration in cascading pathways like the Krebs tricarboxylic acid cycle where the cell produces internal energy molecules in the form of ATP. Without plenty of ATP molecules being continuously supplied for cellular biochemical machinery to operate, living cells can not survive more than a few minutes before cell death occurs and tissues begin to die. Blockage of the enzyme cytochrome C oxidase by hydrogen sulfide inside red blood cells, the brain and lungs poses a serious threat to the biological integrity and well-being of the human brain and body.

How does H_2S enter the body? There are three routes: inhalation—from breathing vapors absorbed through the lungs; oral—from ingestion of contaminated substances (especially water), absorbed through the intestinal tract; and skin—from dermal contact with contaminated substances (such as air), absorbed through the skin. The main route of absorption of H_2S is through inhalation in a vast majority of communities.

Animal studies of H_2S show widespread distribution in the body after inhalation exposures (3, 4), with a selective distribution to the brain stem area compared with other areas in the brain (5). Warenycia et al. (5) found the net uptake of sulfide was greatest in the brainstem (3.02 micrograms/g) compared to other neural regions as was the selective accumulation of sulfide as calculated from normalized blood flow rates. The results of subcellular analysis showed sulfide was detectable in fractions enriched in myelin, synaptosomes and mitochondria. A major sulfide portion was found in the mitochondrial fraction, where the cell's critical energy-producing system resides. The sulfide content of these fractions increased 2- to 3-fold, the greatest increases occurring in myelin- and mitochondrial-enriched fractions (5). The brain stem is vital since it plays a primary role in the regulation of cardiac and respiratory functions. The medulla oblongata in the brain stem is the lower portion of the brainstem. It deals with autonomic physiological functions, such as breathing and blood pressure, and keeping us alive.

Research in animals has identified more than forty health effects of H_2S primarily non-cancer effects. Medical data demonstrates that numerous similar health effects occur in human exposure to H_2S. Metabolism takes place by three pathways: oxidation to sulfate, methylation, and reaction with metallo- or disulfide- containing proteins. This last appears to be the main pathway for toxicity although new evidence may offer more insights on biological oxidation and reactions (6).

Human populations most sensitive to H_2S are assumed to be the fetus (animal data only), children (7), persons with heart disease (8), individuals with asthma (9), individuals who metabolize organosulfides differently (10, 11, as reviewed in 12), and persons consuming alcohol (13, 14).

Medical information on H_2S toxicity and chronic exposure to low levels to the central nervous system (CNS)

Hydrogen sulfide's toxicity at 500-900 parts per million (and higher) is well documented as rapidly lethal to human beings by shutting down the brain's respiratory center. H_2S works by rapidly interfering with the brain's respiratory command center (sending nerve signals to the lungs) and poisoning the blood's oxygen carrying ability, but long-term, low-level or chronic exposures have been generally considered to be less toxic and less harmful.

The driving regulatory assumption has been that if an exposure to H_2S is not fatal, there are few, if any, lasting health effects. But that assumption became medically outdated in the 1990s based on numerous studies and medical conferences. Four public health scientists—including Kaye Kilburn, Ph.D., University of Southern California School of Medicine, Marvin Legator, Ph.D., toxicologist at the University of Texas Medical Branch-Galveston, and Bob Borga, Ph.D. — participated in an H_2S panel at the American Public Health Association's (APHA) annual meetings on November 11, 1997, in Indianapolis, Indiana, to present and discuss ground breaking research demonstrating the extraordinarily toxic nature of H_2S at the chronic, low levels to which communities across the nation are routinely exposed. These public health findings clearly support the thesis that exposure to hydrogen sulfide, even in extremely low concentrations, can cause lasting damage to the nervous system.

Dr. Kilburn has been conducting research on the health effects of exposure to H_2S for many years, including in communities being routinely exposed (18-20). Describing a new study, he unequivocally stated at the 1997 APHA conference that "H_2S poisons the brain, and the poisoning is irreversible" (18- 20, 24-26). Demonstrable symptoms of chronic exposure include pronounced deficits in balance and reaction time, as well as such ailments as dizziness, insomnia, and overpowering fatigue (18-20, 24-26).

In addition, Kilburn has emphasized that H_2S research since 1990 has corrected the mistaken concept that people exposed to rotten egg, chemical name hydrogen sulfide, who are not killed, recover completely (20, 23-26). The correction came from following the exposed people and doing sensitive tests of brain functions. Kilburn noted that "…their balance is abnormal, simple (one stimulus) and choice (two stimuli) reaction times are prolonged and abnormal, recall memory is impaired, as are attention and concentration measured by trail making B, that consists of connecting alternately ascending numbers and letters. Studies showed residual impairment in people rendered unconscious or knocked down by hydrogen sulfide (26)." These data indicate irreversible damage occurs at levels of hydrogen sulfide in the 1 to 5 parts per million range. A factor in such damage is probably that exposures may be for 168 hours per week – every hour of every day in contrast to workplace exposures of 40 hours or less.

Recognition of this problem is shown by 34 states that have adopted standards for hydrogen sulfide in ambient air as low as 10 parts per billion (0.010 parts per million). Thus it's obvious that the 1970's occupation standard of 10 ppm is not only obsolete but dangerous to human brains. Prudent management of cities for people means avoidance of H_2S exposure. Compromise pushes people prematurely into dementia like Alzheimer's disease.

Next, H_2S-induced impairment was found in those people without unconsciousness who were exposed at work or at home from sources such as waste sites, dumps and manure lagoons. To express diverse brain functions, Kilburn totals the numbers of abnormalities with higher numbers indicating greater severity. As a result, Kilburn concluded that H_2S damage to the brain is permanent as no treatment has ever reversed it (20, 23, 26).

Dr. Legator and research associate Chantele Singleton utilized a carefully designed health "symptom survey" to evaluate adverse health effects associated with H_2S (1, 21). In one study, they administered the survey to 97 community residents living within four miles of a large geothermal electric power plant in Hawaii, the Puna Geothermal Venture (PGV). PGV produces electricity from subsurface volcanic heat and releases hydrogen sulfide as a waste byproduct. Eighty-six percent of the subjects indicated that they had experienced central nervous system impairment similar to those described by Doctor Kilburn's research. But only 26% of those in a Puna, Hawaii control group—people who live some 20 miles away from the plant—reported such problems (1, 21, 22).

According to several studies by these researchers on chronic, low level H_2S gas exposures, one may observe abnormal neurobehavioral functioning and altered mood states (e.g., depression, fatigue, tension, vigor) (1). In addition, numerous CNS-brain effects occur including multiple effects: changes in brain density, headache, memory loss, reduced sense of smell, loss of balance, dizziness, sleep difficulties, and fatigue (1). Numerous cases reported in the literature support the CNS toxicity of H_2S (1). Many of the effects are persistent (15, 16, 17).

Dr. Bob Borda, a neuropsychologist in Texas, put neighbors of the industrial plant through a battery of tests and found that many demonstrated attention deficits and an inability to process information quickly. The condition, Borda said, is analogous to an outdated computer program: "It runs, but it is maddeningly slow and inefficient (34)."

Acute exposure to 25 ppb H_2S: Irritating to eyes of people in communities in US, Europe and New Zealand

A comprehensive literature review was conducted on the toxicology of the eye by Canadian public health scientists in 2006 (27). They reviewed ninety-six of the papers published in the last 100 years and concluded that H_2S is irritating to the eye at ultra-low concentrations below 100 ppb (27). The purpose of the historical eye toxicology review by Lambert et al. was to address an incorrect conclusion reached in an Alberta Health and Wellness review in 2002 of the H_2S literature suggesting "…that there is little evidence of eye irritation up to concentrations of 100 ppm H_2S…" and because the 2002 review incorrectly suggested that the H_2S literature on the eye is a series of unsubstantiated claims reproduced in review articles dating back to the 1930s (27b). Lambert's team sought to demonstrate the "divergence, consistency, and coherence of the perspectives and observations of H_2S eye toxicity" including a reanalysis of all the papers considered in the 2002 Alberta Health and Wellness article (27b).

Lambert et al. reviewed available clinical studies, non-clinical, and case-control studies in ninety-six papers on the PubMed and Toxline databases. They pointed out that "...almost all the scientific studies we found that discussed the eye, reported eye effects below 100 ppm H_2S in a variety of environmental contexts (Table 5)" (27). In conclusion, Lambert's team notes: "In community settings, following short-term exposure, 25 ppb H_2S appears to be the lowest concentration observed to irritate the eyes and, with chronic exposure, serious health effects on the eyes are suggested. Perhaps the most controversial question is whether H_2S can cause irreversible health effects on the eye. Blindness was suggested by Ramazzini, however, many have claimed the eye heals completely (27)."

The South Karelia, Finland air pollution studies were among the H_2S literature reviewed by Lambert et al. where effects of H_2S were observed on the eyes of children at low concentrations as part of a series of investigations conducted by Haahtela et al. in 1992 and Marttila et al. in 1994-95 (27c, d, e). Lambert et al. stated: "The South Karelia air pollution studies documented public exposures to low levels of H_2S and other reduced sulfides (methyl mercaptan, dimethyl sulfide and dimethyl disulfide) from pulp mills in Finland. Haahtela et al. (1992) presented survey results from a community that experienced low level acute H_2S exposure: the maximum 4-hr concentration 135 µg/m3 (96 ppb) H_2S and the 24-hr average of 35 and 43 µg/m3 (25 and 31 ppb H_2S). During the peak emissions, the SO_2 mean 1-hr average was only 3 µg/m3 and therefore not a significant cofounder. The authors concluded that the "observed symptoms correspond to the physiological effects of acute exposure of H_2S, suggesting direct irritative effect on mucous membranes and eye conjunctivitis but at lower concentrations than described previously" (Haahtela et al., 1992, p. 605)." (27c) Additional discussion is presented by Lambert et al. of why H_2S was considered as the most likely cause of the effects observed in South Karelia and not other sulfur compounds (27).

Additional community investigations in South Karelia by Marttila et al. in 1994-95 confirmed the presence of low concentrations of H_2S noted by Haahtela et al. in 1992 (27c, d). "Marttila et al. (1994) reported in the most polluted Karelia area that the annual mean H_2S concentration was calculated as 8 µg/m3 (5.7 ppb) H_2S, the highest 24-hr concentration was calculated as 100 µg/m3 (71.4 ppb) H_2S and maximum 4-hr average was measured as 56 µg/m3 (40 ppb) H_2S (27d)." In 1995, Marttila et al. conducted surveys of the community in a reference (non-polluted) area, medium polluted and high polluted areas evaluating daily symptom intensity in relation to exposure levels (27e). Marttila et al. found in 1995 significant differences in the eye symptoms reported between the medium and reference communities (OR 3.17, 1.21-7.47) and high vs. reference (OR 5.0, 1.66-12.65) as cited by Lambert et al. (27e). With respect to symptoms, they observed a similar increase in reporting of intensity of nasal and pharyngeal symptoms (27e). They noted that the intensity of eye symptoms was significantly higher during days of TRS > 10 µg/m3 (27e). The parents reported their children's eye symptoms over the past 12 months (OR 1.15, 95% CI 0.43-3.05) in the three communities (reference n=7/30, medium n=20/62, and high n-5/42) described in Lambert et al. (27e).

Another community health effects-eye study is also presented from Rotorua, New Zealand (natural geothermal seeps) where low concentrations of H_2S were measured ranging from 20 µg/m3 (14 ppb median concentration), 35% of the measurements >70 µg/m3 (50 ppb), 10% >400 µg/m3 (286 ppb) H_2S, and 1,000 ppb was the highest concentrations for 30-minute average (27). In summary, Lambert et al. cites the Rotorua studies of McDougal and Garland (1945), Bates (1998), Fisher (1999), and Bates (2002) in their 2006 review (27) as further evidence in support of community settings where low H_2S concentrations are associated with acute exposure to the eyes.

The Canadian public health scientists noted that in Terre Haute, Indiana, June 1964 according to an investigation by the US Department of Health, an H_2S release from a chemical lagoon resulted in recorded concentrations as 0.022 – 0.125 ppm (22 ppb – 125 ppb) for 7 hours where citizens complained about burning eyes (27). A NIOSH report (p. 44) from 1977 is quoted: "this study did suggest that hydrogen sulfide can irritate the eyes and respiratory system at concentrations below 1 ppm (27)." Lambert et al. state: "In two sour gas blow-outs in Alberta, in the early 1980s, eye injury was documented to humans and animals at 0.5 ppm [500 ppb] H_2S. Community studies in the United States, Europe and New Zealand suggest that acute exposure to 25 ppb H_2S is the lowest concentration to irritate the eyes; with chronic exposure, serious eye effects are suggested (27)."

Lambert et al. stressed that eye irritation caused by H_2S is described as the first health effect to manifest at low levels by Ramazzini in 1713 (27), which is not surprising since H_2S is also named "hydrosulfuric acid." Yet less attention has been paid to H_2S's acidic irritation to the eyes at ultra-low concentrations and the potential of damage to ultra-sensitive eye tissues. Eye irritation today is better described as occurring to the conjunctival and corneal tissues, although the mechanism remains unknown (27). One theory is that H_2S reacts with liquid water in the eye and is converted to sulfurous acid (H_2SO_3) and sulfuric acid (H_2SO_4), both acidic sulfur compounds that are known to be irritating to eye tissues. A significant community-public health point by Lambert et al. is that short-term exposure to 25 ppb H_2S irritates the eye because concentrations of 25 ppb and higher are more commonly observed in many communities close to refineries, oil & gas production wells, CAFOs, and other sources. EPA needs to more seriously assess the series of community H_2S studies in the United States, Europe and New Zealand as substantial evidence that communities are likely being adversely impacted by H_2S concentrations as low as 25 ppb. Evidence of eye irritation due to ultra-low H_2S concentrations has been disregarded by industry and officials for far too long.

Hydrogen sulfide's cytotoxicity: H_2S acts as a neuromodulator in four studies

Hydrogen sulfide is a well known cytotoxic gas recently proposed as a novel neuromodulator in four studies from 1996-2004 (28-31). A 2004 research team indicated it recently has been shown to stimulate N-methyl-d-aspartate (NMDA) receptors to enhance long-term potentiation suggesting a novel neuromodulatory role in vivo (28).

Hydrogen sulfide's cytotoxicity: H_2S induces neuron death via glutamate receptors

Evidence in a new paper indicates that H_2S is responsible for neuron death and this will significantly impact industry's view that brain damage is a secondary effect from hypoxia as opposed to direct result of H_2S exposure. A 2007 study in the *Journal of Neuropharmacology* conducted by biochemistry researchers found evidence that: "These data suggest that H_2S induced neuronal death through ionotropic glutamate receptors, which recruits apoptosis to ensure cellular demise and employs calpains and lysosomal rupture. This study provides novel insights into cell death observed in neurodegenerative diseases involving glutamate receptor activation and perturbed H_2S synthesis (32)." The new H_2S neuronal induced-death evidence provides strong impetus for the EPA to move to listing H_2S as a HAP as soon as possible.

Hydrogen sulfide's genotoxicity: new evidence H_2S damages DNA from four studies

Teams of researchers at separate institutions have discovered evidence that H_2S damages DNA in four recent studies (35-38). One team at the University of Illinois, Urbana, Illinois and a second team at the School of Medicine at the University of Singapore. Hydrogen sulfide has been shown previously to exert proapoptotic activity or cell death. However, the mechanism(s) by which H_2S affects cell growth and function have not been addressed adequately. The Singapore team of Baskar et al. concluded: "We propose that the genotoxic action of H_2S propels the cell toward apoptotic death triggered initially by stabilization of p53 and subsequently involving a cascade of downstream products. These results are of significance as they uncover a hitherto unknown and very fundamental role for H_2S in determining cell fate (37)." The University of Illinois team of Attene-Ramos et al. indicated: "In this study, we examined the chronic cytotoxicity of sulfide using a microplate assay and genotoxicity using the single-cell gel electrophoresis (SCGE; comet assay) in Chinese hamster ovary (CHO) and HT29-Cl.16E cells. … These data indicate that given a predisposing genetic background that compromises DNA repair, H_2S may lead to genomic instability or the cumulative mutations found in adenomatous polyps leading to colorectal cancer. (35, 36)." A fourth study investigated an association between effects of genetic polymorphisms of GSTT1 and GSTM1 and depression inventory scores of 124 healthy female individuals who were chronically exposed to natural sour gas containing sulfur compounds such as H_2S (38). The study was conducted in a polluted area of the Middle East.

Children and EPA's 1996 policy on setting standards designed to be protective of our youngest

President William Clinton's Executive Order of October 1995 put a new priority on the protection of children, since pollution standards have not been designed to protect our children from environmental insults such as ambient H_2S. Children are more vulnerable than adults to H_2S, first because they breathe more rapidly, inhaling more air pollution per pound of body weight than do adults. A resting infant, for example, inhales twice as much air, relative to its size, as does a resting adult. Second, national data show that children spend an average of about 50% more time outdoors than adults. Third, children are three times more active while outdoors than adults, engaged in sports and other vigorous activities; this increased activity raises breathing rates and significantly increases inhalation and in some cases swallowing of pollutants. Fourth, children are highly vulnerable to toxic substances because their bodies are immature and rapidly growing since their immune systems and developing organs are still immature. Fifth, children are in their prime learning years and H_2S exposure causes brain damage. These are just a few critical reasons why EPA needs to move on H_2S and provide better protection for the nation's children. The impairment of mental faculties in a child amounts to a lifetime of harm. Society pays an enormous cost for this harm besides individuals.

An equally persuasive argument is that exposure to toxic air contaminants like H_2S during infancy or childhood could harm the development of respiratory, nervous, endocrine and immune systems, and could increase the risk of cancer later in life. Since H_2S has been found by cancer researchers to display implications as a genotoxin, children need better protection and safeguards from this toxic air contaminant.

We are not doing enough to protect children attending the nation's Elementary Schools, according to a recent study in *USA Today*. How many children are attending Elementary Schools where exposure to hydrogen sulfide gas is a common occurrence? Unfortunately, EPA has no data on the number of affected school children and Elementary Schools in the nation, but numerous schools located near large oil refineries and oil & gas producing wells, for example, are in communities where H_2S gas exposures are almost routine daily occurrences.

Along this line of concern for children, Dr. Kaye Kilburn reported an anecdotal observation from a teacher: "Hydrogen sulfide-exposed children have trouble recalling lessons and reciting, and they lose the ability to read. They eventually drop out of school. A patient of mine in Wilmington, who is a teacher, made this observation in the months after the Texaco refinery explosion exposed Long Beach and Wilmington to levels of H2S as high as 24 parts per million in 1992." (39)

Environmental justice and hydrogen sulfide exposure: Petroleum refineries example

We want to impress upon EPA its obligation to fully comply with and enforce Title VI of the Civil Rights Act of 1964, together with President Clinton's February 11, 1994, Executive Order No. 12898 concerning the need for EPA to act on the environmental injustice in hundreds of communities impacted by H_2S emissions.

We urge EPA to seriously consider that the H_2S exposures in affected communities near major oil refineries, as one example, often create a disproportionate air toxics burden for people of color and low-income populations. Residents in the neighborhoods close to one of the largest H_2S industrial categories, the one hundred and forty oil refineries in the US, are often low income areas. A preliminary survey shows in leading petroleum refining areas in EPA regions (2, 3, 4, 5, 6, 9, 10), a majority of residents in the refinery neighborhoods are people of color.

Environmental justice is a grave need and major issue for dozens of oil refinery communities in Texas, Louisiana, California, Illinois, Pennsylvania, New Jersey, Indiana, Ohio, Kentucky, Alabama, Tennessee, Michigan, and Mississippi, which contain the bulk of the nation's refineries, and where we stress that residents in refinery communities are disproportionately poor people of color. But besides the petroleum refining sector, several other industrial source categories are located in communities where people of color disproportionately reside.

An example of repeated serious H_2S air pollution is a major Houston refinery reporting to the Texas Commission on Environmental Quality it released over 1,700 pounds of H_2S into the air during major upset incidents over several years, and the H_2S releases are a key reason why local residents often complained about noxious rotten egg odors. Yet few H_2S violations were issued by state air agency and no nuisance odor conditions were confirmed despite the large releases of rotten egg gas and many complaints. This Houston oil refinery reported H_2S releases as low as 1 pound to 235 pounds (#) including 18#, 20#, 27#, 37#, 68#, 74#, 155#, 159#, 235# and 305#. However, these H_2S

volumes were self-reported engineering estimates based on flare combustion efficiency calculations that assume 98% destruction was achieved and not based on actual H_2S monitoring. If flares were not achieving 98% destruction, H_2S releases may have been much higher than the estimated 1,700 pounds. Regulatory agencies in some cases tend to view residents living too close to industrial facilities as living in "industrial areas" where air pollution is bound to be a problem and suggest these people move away if they don't like the poor air quality.

U.S. oil refineries are importing sour crude oils with higher sulfur levels (higher H_2S) and a potential for worse H_2S community impacts along pipeline routes and around the oil refineries. U.S. refineries have been seeking permit modifications at large plants to process higher sulfur crude oils from Venezuela, Mexico and now Canada (Alberta tar sands heavy crude oil). A powerful risk in higher sulfur in the Alberta tar sands heavy crude oil is an increased lethality hazard of higher H_2S concentrations. Tar sands sulfur % is at least 4.4% and higher according to 2007 U.S. Geological Survey report (33) and it may contain 44,000 ppm of hydrogen sulfide when H_2S is deadly at 500-900 ppm. Susceptible persons with vulnerable health conditions such as heart and/or lung ailments, including thousands of senior citizens, may die at only 50 ppm H_2S levels, since lower H_2S levels can more easily poison their comprised hearts and lungs. Great concern is for infants who may succumb at even lower H_2S concentrations of 5-10 ppm exposures due to their uniquely vulnerable developing physiology, brain and nervous system. Higher sulfur crude oil is viewed by the oil industry as higher H_2S concentrations to deal with and control.

Three public health concerns result from higher H_2S content in Canadian tar sands heavy crude oil

1. Lethal hydrogen sulfide levels may reach 90 times over the minimum instant kill concentrations. H_2S is extremely dangerous being instantly lethal at 0.050% - 0.090% concentrations (500-900 ppm). The point is that pipeline breaks and leaks of crude oil with higher H_2S gas concentrations pose a significantly higher risk to public health and the environment compared to crude oil with lower sulfur and lower H_2S gas levels.
2. Increased chronic hydrogen sulfide exposure potential in affected communities. Pipelines and refineries handling higher sulfur crude with more H_2S will mean more serious community exposures to sublethal concentrations and related health effects.
3. Corrosive acid gas. H_2S is extremely corrosive and causes steel to experience sulfide stress cracking (SCC). H_2S is called an "acid gas" because it's extremely acidic and eats through solid steel leaving holes in tanks, pipes and processing equipment. The extreme corrosivity of higher H_2S in the Canadian tar sands heavy crude oil increases the potential for serious leaks and accidents from pipelines and increases the health hazards if accidental leaks occur. The presence of H_2S, a corrosive material, is of particular concern when it is also in the presence of other corrosive agents such as CO_2 +water, chlorides which cause chloride stress cracking (CSC), and oxygen (O_2) which causes rusting of steels and other materials. Such combination requires particular care in the selection of materials to resist such combined corrosion so as to adequately contain the H_2S.

EPA needs an accurate national emissions inventory (EI) for hydrogen sulfide gas

The EPA Office of Air Quality Planning & Standards has estimated the H_2S national emissions at more than 56,700 tons, but does not include sources such as CAFOs, municipal waste landfills, and sugar beet manufacturing. EPA needs a more accurate national EI for hydrogen sulfide gas from major industrial facilities, minor sources and exempted sources such as oil & gas wells. National number of sites with H_2S will be enormous since oil & gas wells number in the tens of thousands alone. The California Air Resources Board provided a 1999 EI for H_2S of 5,688,172 pounds, an indication of the large-scale H_2S volume. (http://www.oehha.org/air/chronic_rels/pdf/7783064.pdf)

Nearly 200 Texas major industrial plants estimated releases at 7,187,988.4 pounds (3,594 tons) of H_2S gas in 1997, according to EI records at the Texas Commission on Environmental Quality, not including landfills, CAFOs, oil & gas wells, and many minor facilities. That added 3,594 tons (3.3%) to the ~108,000 tons of air toxics emitted by Texas plants in 1997. Despite the need for a better national emissions inventory of H_2S from potential sources, the EPA recognizes that H_2S is a poorly regulated air pollutant with significant nationwide emissions estimated at more than 110 millions pounds annually. It is certainly ranks among the worst regulated air toxics.

California: model state hydrogen sulfide gas program to manage chronic and acute exposure risks

California has a model state program on managing H_2S emissions with an ambient air standard for both chronic and acute exposures and maintains a statewide continuous air monitoring network for H_2S. California has rules on

Construction and Demolition Debris landfills and enforces H_2S emission limits for natural energy units (geysers). The most unique public health aspect is that California requires effected sources to model non-cancer and cancer risks for all listed toxic substances even H_2S. Facilities having a non-cancer risk below a Hazard Quotient (HQ) of 1 for H_2S do not have to do any further assessments or control measures to reduce the risk to the community. But if the HQ is greater than 1, the facility is comprehensively evaluated by the air district office for the potency, toxicity, quantity of emissions released from the facility and any other factors the district considers may add to the risk. If the risk is deemed significant a public notification process is required as well as a requirement for the facility to implement a risk reduction plan.

State of California has recognized H_2S as a toxic air contaminant for years, according to the Office of Environmental Health Hazard Assessment (OEHHA). OEHHA sets reference exposure levels (RELs) for toxic air pollutants and lists H_2S as possessing an acute REL at 42 micrograms/cubic meter for one-hour triggering "headache and nausea in response to odor" effects and a chronic REL at only 10 micrograms/cubic meter (8 ppb). (http://www.oehha.org)

Diurnal variation measured in hydrogen sulfide ambient concentrations

Hydrogen sulfide is heavier than air with a molecular weight of 34.08 making it heavier than molecular oxygen at 32.00 and molecular nitrogen at 28.013. Researchers have confirmed what citizens in dozens of H_2S impacted communities have known for years: The odor of H_2S is several times worse at night versus day in most cases, except when larger releases occur during the day (34). Tarver and Dasgupta conducted field studies in West Texas oil fields with a gas chromatograph to measure the variation of ambient H_2S levels from day to night. They observed a marked variation: "At all locations, H_2S concentrations consistently exhibited a strong diurnal pattern, with nighttime maxima in the range of 1-5 ppbv followed by rapid abatement at sunrise. By 10-11 AM, H_2S levels fell below the instrument detection limit of 200 pptv" (34). Like other gases, H_2S generally does not disperse as efficiently at night, with cooler air temperatures and low wind speed conditions. For residents in impacted communities, this diurnal pattern carries the implication that by far the worst H_2S exposures are occurring when families are most likely at home, windows open (because houses in low-income communities lack air conditioning), and children are playing outside home from school. Night time is also a period when state and local regulators are the least likely to be available to verify unhealthy nuisance conditions, conduct H_2S ambient air sampling to confirm the presence of the gas, and attempt to trace the H_2S source in efforts to obtain compliance.

Control technology for Hydrogen Sulfide

Major sources such as oil refineries and sour natural gas processing plants already implement air pollution control for H_2S through a combination of processes like sulfur recovery units, amine gas treaters, hydrodesulfurization, sour gas absorbers, acid gas scrubbers, refinery fuel gas combustion, but may need to ratchet their emissions down some more to increase protection for local communities and install more ambient H_2S air monitoring systems. As EPA recognizes, H_2S pollution controls under the Clean Air Act's Prevention of Significant Deterioration (PSD) program has had a beneficial effect on a few source categories such as kraft pulp mills and petroleum refineries. Sources affected by PSD have had to install controls to reduce point source emissions, while area source emissions from lagoons and wastewater treatment units have gone unregulated. Modeling studies of H_2S at pulp and paper mills have indicated these area sources constitute the most significant risk to the public.

Voluntary efforts are being implemented in some sectors with relative success especially when encouraged by state and federal regulatory agencies. The California Air Resources Board and the air quality management districts have achieved reasonable success in reducing emissions and community impacts of H_2S in California as confirmed by the state air monitoring network. EPA needs to look at model H_2S programs in California and determine where industry may have made a more concerted effort to operate facilities in order to protect local communities from hydrogen sulfide releases. EPA Regions are working with the construction-demolition trade industry, the recycling industry, as well as several state offices to develop guidance on preventing H_2S emissions from construction debris landfills. The guidance focuses on using proven operation and maintenance guidelines which include; segregation of wastes, pH adjustment, recycling, stormwater control, leachate management practices, and applying appropriate ground cover.

Conclusion

Public health scientists have recognized for over a decade that hydrogen sulfide is a potent neurotoxin, and chronic exposure to low ambient levels causes irreversible damage to the brain and central nervous system. Ultra-low levels of H_2S down to 25 ppb have been associated with acute exposure causing eye irritation in community settings in the United States, Europe and New Zealand. Now emerging scientific evidence supports H_2S causes neuron death, confirming findings by Kilburn of irreversible brain damage. The latest scientific findings suggest H_2S causes DNA damage as a genotoxic agent, which EPA can no longer ignore. The potential carcinogenic implications of H_2S demand that EPA act to protect public health.

Children are among the most susceptible to this poison gas, and EPA needs to do a more effective job of protecting schoolchildren from H_2S impacts. Today, it is unacceptable for communities to have to continue suffering the ill effects of H_2S when the technology to monitor and control H_2S emissions exists. As EPA has learned in the last four decades, environmental injustice is a significant fact of life for thousands of communities in this nation and these residents all have a right to clean, safe air.

It's time for the EPA to take action to formally acknowledge hydrogen sulfide's clear toxicity at low concentrations. As Administrator, you have CAA authority under section 112(b)(2) to act based on a pollutant that poses or may pose "…a threat of adverse human health effects..." Health studies confirm the need for EPA to list H_2S under section 112(b) of the CAA and Title III, since routine daily exposure effects are not addressed under the accidental release provisions in section 112(r) of the CAA, where H_2S is currently regulated. However, section 112(r) is not designed or intended to address daily exposures at sublethal concentrations, but section 112(b) can bridge this gap.

EPA, in addition, needs to require annual reporting of H_2S as a toxic substance under the Toxic Release Inventory (TRI) reporting program, since H_2S is not reported due to an administrative stay issued August 22, 1994 evidently under a legal threat by the American Petroleum Institute. It's extraordinary that industry has delayed reporting of H_2S for twenty years. EPA needs a TRI reporting threshold of 1.0 pound for H_2S and not 10,000 pounds as was originally the requirement. We request that EPA immediately lift the administrative stay on H_2S and require TRI reporting in the next TRI submission cycle. The TRI data would also help EPA compile more accurate H_2S data.

Please respond to this request for EPA to take action to list H_2S under section 112(b) of the CAA. Address the EPA's response to Neil Carman at the contact information listed below.

Respectfully yours,

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