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ENVIRONMENTAL PROTECTION AGENCY

[AD-FRL-3505-6]

Assessment of Sodium Hydroxide As a Potentially Toxic Air Pollutant

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of sodium hydroxide assessment results and solicitation of information.

SUMMARY: This notice announces the results of EPA's assessment of sodium hydroxide under the Clean Air Act (CAA). The EPA initiated this assessment based on the production volume of sodium hydroxide and the potential for adverse health effects associated with exposure to sodium hydroxide. The results of EPA's preliminary analysis indicate that currently available data are insufficient to establish that emissions of sodium hydroxide to the ambient air pose a significant threat to public health. Based on this information, the EPA has determined that regulation of sodium hydroxide under the CAA is not warranted at this time.

Given the limited opportunity for prior public review of the health and exposure information incorporated in this notice, the Agency is soliciting comment on its determination. This finding has no effect on the regulation of sodium hydroxide to attain the national ambient air quality standards for particulate matter. In addition, this notice does not preclude any State or local air pollution control agency from specifically regulating emission sources of sodium hydroxide.

DATE: Written comments pertaining to this notice must be received on or before April 13, 1989.

ADDRESSES: Submit comments (duplicate copies are preferred) to: Central Docket Section (A-130), U.S. Environmental Protection Agency, Attn: Docket No. A-88-20, 401 M Street SW., Washington, DC 20460. Docket No. A-88-20, which contains information relevant to this notice, is located in the Central Docket Section of the EPA, South Conference Center, Room 4, 401 M Street SW., Washington, DC 20460. The Docket may be inspected between 8:00 a.m. and 3:30 p.m. on weekdays, and a reasonable fee may be charged for copying.

Availability of Related Information

Information on the availability of the health assessment summary document for sodium hydroxide, "Summary

Review of Health Effects Associated with Sodium Hydroxide: Health Issue Assessment" (EPA 600/8-88-081), can be obtained from ORD Publications, CERI-FR, U.S. Environmental Protection Agency, Cincinnati, Ohio 45268 (Telephone: (513) 569-7562 commercial/684-7562 FTS). The document is available through the U.S. Department of Commerce, National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, Virginia 22161. The Sodium Hydroxide Preliminary Source Assessment (EPA-450/3-88-002) is also available from NTIS. The NTIS accession number is PB88-174545. The above document and other information on the sources, emissions, and atmospheric degradation are summarized in several reports which are found in the docket.

FOR FURTHER INFORMATION CONTACT: Robert Schell, Pollutant Assessment Branch (MD-13), Emission Standards Division, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711 (Telephone: (919) 541-5519 commercial/629-5519 FTS).

SUPPLEMENTARY INFORMATION: The EPA initiated an assessment of sodium hydroxide based on the large production volume and the potential for adverse health effects associated with exposure to sodium hydroxide in the ambient air. In the course of this assessment, the Agency collected the available relevant information and today's notice provides a summary of this information on the following topics: chemical characterization, production and uses, sources and emissions, atmospheric degradation, health effects, monitored ambient air concentrations, exposure estimation, risk characterization, and existing regulations and guidelines.

Chemical Characterization

Sodium hydroxide (CAS No. 131-07-32) is a white deliquescent, crystalline solid at room temperature (U.S. EPA, 1988a). Sodium hydroxide is very soluble in water and is often used in 45 to 75 percent in acqueous solutions (U.S. EPA, 1987a). Sodium hydroxide has a melting point of 318 degrees centigrade and a boiling point of 1380 degrees centigrade (U.S. EPA, 1988a).

Production and Uses

Sodium hydroxide is produced in large quantities in the United States. There are more than 40 production facilities which in 1985 produced approximately 11 million short tons of sodium hydroxide. Most sodium hydroxide is produced as a 50 percent solution in water. Sodium hydroxide is produced by the electrolysis of sodium chloride via the diaphragm, mercury, or membrane cell process (U.S. EPA 1988a).

Sodium hydroxide is one of the most widely used chemicals in the United States. In 1982, the total United States' consumption of sodium hydroxide was slightly greater than 8 million metric tons which was 20 percent below the record consumption of 10.1 million metric tons in 1979. The largest market for sodium hydroxide is in the chemical industry (48 percent of total demand) where it is used in the production of alumina from bauxite, and also used for pH control and in the neutralization of waste acids. The next largest market is the pulp and paper industry with 26 percent of total demand (U.S. EPA, 1988a).

Sources and Emissions

Routine emissions of sodium hydroxide into the environment occur via aerosol emissions, aqueous waste streams and consumer uses. There are numerous industries which emit sodium hydroxide. The major industries that emit sodium hydroxide to the atmosphere are the kraft pulp industry, the brewing industry, auto carburetor manufacturing, plating, industrial organic chemical manufacturing, soap manufacturing and metal partitions. The largest source of sodium hydroxide emissions to the atmosphere is the kraft pulp industry. For a more complete discussion of the industrial process which emit sodium hydroxide to the atmosphere the reader is referred to the sodium hydroxide preliminary source assessment (U.S. EPA, 1988a).

Atmospheric Degradation

Sodium hydroxide is very short lived in the atmosphere. Sodium hydroxide released to the atmosphere as an aerosol in solution with water will be neutralized as a result of its reaction with carbon dioxide.

The principal reaction products are sodium carbonate and water. The atmospheric half life of sodium hydroxide has been estimated at 13 seconds (U.S. EPA, 1988c). In the short-term modeling analysis described below, this rate of degradation results in only 0.4 percent of the sodium hydroxide released remaining at a point 200 meters from the site of release. For a more complete discussion of analysis of the atmospheric fate of sodium hydroxide the reader is referred to the public docket for this action, specifically the entry entitled "Estimation of the Half-Life of Sodium Hydroxide Aerosol in the Atmosphere" (U.S. EPA, 1988c).

Health Effects

The Agency has prepared a document entitled "Summary Review of the Health Effects Associated with Sodium Hydroxide: Health Issue Assessment" (U.S. EPA, 1987a). This document discusses the relevant data available for assessing the health effects associated with exposure to sodium hydroxide in the ambient air. This information is summarized briefly below.

Carcinogenicity and Mutagenicity

No *in vivo* animal studies of sodium hydroxide carcinogenicity were found in the literature (U.S. EPA, 1988b). There are some data, however, which suggest that the incidence of carcinoma of the esophagus is greatly increased following accidental or intentional ingestion of sodium hydroxide (lye) in humans (Lansing et al., 1969; Kiviranta, 1952). The National Institute for Occupational Safety and Health (NIOSH), however, points out that these carcinomas were the result of tissue destruction and possibly scar formation and were not caused by the direct carcinogenic potential of sodium hydroxide itself. In an epidemiological study of workers chronically exposed to sodium hydroxide dust, no relationship between malignancies or mortality was observed (Ott et al., 1977).

Sodium hydroxide has not been adequately tested for mutagenic potential. Two bacterial assays have suggested sodium hydroxide is not mutagenic. However, an *in vitro* deoxyribonucleic acid (DNA) synthesis study and an *in vivo* study of insect spermatocytes suggested sodium hydroxide may be mutagenic. Based on EPA's "Guidelines for Carcinogenic Risk Assessment" (51 FR 33992), sodium hydroxide has been classified in the weight of evidence category "D," meaning it is not classifiable as to human carcinogenicity.

Developmental and Reproductive Toxicity

Only one study exists on the developmental toxicity of sodium hydroxide. This study (Dostal, 1973) indicated increased mortality in the fetuses of mice when the dams were treated intraamniotically with a weak sodium hydroxide solution. Although this study suggests an embryotoxic effect, it is difficult to envision such an exposure resulting from sodium hydroxide in the ambient air.

Other Toxic Effects

The adverse health effects associated with exposure to sodium hydroxide result from its extreme alkalinity. In

evaluating the toxicity of sodium hydroxide, therefore, it is important to consider the pH of the solution to which individuals may be exposed. Furthermore, because sodium hydroxide is a solid or liquid at room temperature, the size of the particles to which individuals may be exposed is important for evaluating toxicity resulting from inhalation exposure. Larger particles tend to settle out faster and thus have a greater effect on the upper respiratory tract, whereas smaller particles tend to affect the lower regions.

Contact with sodium hydroxide in either solid, liquid or aerosol form has resulted in severe eye injuries, damage to the skin and injury of the mucous membranes (U.S. EPA, 1988b). Most of the case histories of injuries resulting from exposure to sodium hydroxide involve the liquid or solid form. There are very few cases of health effects resulting from airborne exposure documented in the literature (NIOSH, 1975). Sax (1984) reports that inhalation of sodium hydroxide dust or concentrated mist can cause damage to the upper respiratory tract and to lung tissue, depending on the extent of the exposure. The effects associated with inhalation exposure to sodium hydroxide may thus range from mild irritation to severe pneumonitis. In a study (Lewis, 1974) of the effects of aerosol oven cleaners containing sodium hydroxide, respiratory irritation was perceived in healthy human volunteers at concentrations of 0.24 to 1.8 mg/m³. Unfortunately, this study did not take into account other potentially irritating ingredients of the aerosol.

Acute exposures of animals have produced similar results to those of humans. Frank damage of the respiratory system was observed in rats following exposures to aerosols generated from a 20 percent sodium hydroxide solution. The sodium hydroxide concentration in the exposure chambers was not given.

The only data regarding effects associated with chronic exposures come from an epidemiological study (Ott et al., 1977) of workers chronically exposed to sodium hydroxide dust ranging from 0.5 to 2.0 mg/m³. This study reported no statistically significant increased mortality in the exposed group.

Monitored Ambient Concentrations

No data on monitored ambient levels of sodium hydroxide could be found in the available literature.

Exposure Estimation

The exposure assessment for sodium hydroxide relied upon dispersion modeling to estimate ambient concentrations near emission sources. However, given the extremely short atmospheric half-life of sodium hydroxide in the atmosphere, estimation of long-term ambient concentrations are not applicable. Consequently, the focus of this assessment was on short-term exposures.

In order to assess the potential for adverse noncancer health effects from short-term exposure to sodium hydroxide, a conservative screening modeling analysis was performed (U.S. EPA, 1985). The model employs a point source Gaussian air dispersion model and applies several reasonable worst-case assumptions for source location, emissions, meteorology and terrain. Although the short-term screening model is designed to produce conservative estimates of ambient concentrations, the emission rates used in this analysis are derived from estimates of annual releases and may not be representative of short-term release rates. For this assessment, the largest emitting sources were selected and the maximum levels of sodium hydroxide that could occur near each plant were calculated. These results reflect the extremely rapid atmospheric degradation of sodium hydroxide. The maximum concentrations were estimated to be 0.02 and 0.01 mg/m³ for average times of 15 minutes and 24 hours respectively (U.S. EPA, 1988c).

Risk Characterization

Due to the paucity of data regarding health effects resulting from inhalation exposure to sodium hydroxide, it is difficult to derive a concentration level below which no significant health effects would be expected in the general population. For acute exposures, data from the study of oven cleaner aerosols containing sodium hydroxide would suggest that respiratory irritation could be perceived at levels as low as 0.24 to 1.8 mg/m³. This study, however, is flawed due to the other potentially irritating ingredients of the aerosol. The occupational exposure ceiling level developed by the American Council of Governmental Industrial Hygienists (ACGIH) is 2.0 mg/m³. A ceiling level is a level not to be exceeded at any time in work place air. The ACGIH documentation (ACGIH, 1986) referencing Patty (1949) states that 2 mg/m³ sodium hydroxide in air "represents a concentration that is noticeably, but not excessively irritant." The NIOSH also cite Patty (1949) and recommend a 2 mg/m³ limit as a 15 minute ceiling. In addition, the Occupational Safety and Health Administration (OSHA) has adopted an exposure level of 2 mg/m³ as an 8 hour time weighted average.

Although sodium hydroxide is produced in great quantities in the United States, exposure to high concentrations in the ambient air is unlikely due to its rapid atmospheric degradation (the atmospheric half life is 13 seconds). The maximum modeled ambient sodium hydroxide concentration is 0.02 mg/m³ (15 minute averaging period).

Given the paucity of data regarding systemic or acute health effects and the low potential for exposure to high concentrations of sodium hydroxide (due to its rapid atmospheric degradation), it is unlikely that routine emissions of sodium hydroxide pose a public health risk.

Existing Regulations and Guidelines

The NIOSH, OSHA, and ACGIH recommended ceiling level of 2 mg/m³ is discussed above. Six other nations have also recommended workplace standards for airborne sodium hydroxide (NIOSH 1975). All of these standards are equal to or near the NIOSH and ACGIH recommended level.

Sodium hydroxide has been removed (53 FR 49688) from the list of compounds subject to the reporting requirements of the Toxic Chemical Release Reporting, Community Right-to-Know rule (53 FR 4500), under section 313 of Title III of the Superfund Amendments and Reauthorization Act of 1986. Section 313 requires that owners and operators of certain facilities that manufacture, import, process, or otherwise use certain toxic chemicals report annually their releases of those chemicals to each environmental medium. In addition, certain suppliers of toxic chemicals must notify recipients of such chemicals in mixtures and trade name products.

Conclusions

The Agency concludes that the currently available data are insufficient to indicate health concerns that warrant specific Federal regulation of routine sodium hydroxide emissions under the CAA at this time. A number of uncertainties, however, are associated with this conclusion. With regard to the health assessment, the health data are inadequate to judge sodium hydroxide's carcinogenic potential in humans. Furthermore, there are virtually no dose-response data for noncancer health effects in humans, and the available inhalation data from animals are minimal. Regarding the exposure assessment, EPA's techniques for estimating short-term concentrations resulting from routine emissions are

based on screening models rather than on extensive site-specific modeling. The concentrations estimated from the short-term modeling exercise do not account for emissions resulting from intermittent or batch operations, thus providing a potential for underestimation of short-term concentrations. The rapid atmospheric half life of sodium hydroxide, however, has been incorporated into the exposure assessment. Ambient monitoring data for sodium hydroxide were unavailable, in part due to its rapid atmospheric degradation and the confounding effect of other sodium compounds in the atmosphere.

Although EPA considers today's action appropriate in view of the current health and exposure information, the Agency will continue to evaluate new data as they become available.

Furthermore, emissions of sodium hydroxide to the ambient air will also be evaluated in the context of multiple pollutant emissions from categories of related emission sources (source categories).

The EPA invites comments and submission of information pertinent to the determination made today. A further notice will be published if public comments or other additional information suggest a need to reevaluate today's findings and revise EPA's conclusions.

Date: January 6, 1989.

Don R. Clay,

Acting Assistant Administrator for Air and Radiation.

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