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SECOND DRAFT

Information Profiles on Potential Occupational Hazards: Inorganic Azides

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15. Supplementary Notes

16. Abstract (Limit: 200 words) Information was presented for the following inorganic azides: barium-azide (18810587), cesium-azide (22750578), hydrazoic-acid (7782798), lead-azide (13424469), lithium-azide (19597694), potassium-azide (20762601), rubidium-azide (22756361), silver-azide (13863882), and sodium-azide (26628228). The data given for each compound included chemical structure, synonyms, chemical and physical properties, production information, uses, manufacturers and distributors, manufacturing processes, impurities and additives, occupational exposures, control technology and work practices, biological effects, ongoing studies, exposure standards, and sources of additional information. While there did not appear to be a lot of research available on toxicity of inorganic azides what findings there were suggested that toxic effects are due to the azoimide radical. The major acute effects of toxic doses of either sodium-azide or hydrazoic-acid to animals included hypotension, respiratory stimulation and convulsions, followed by respiratory depression and death. Humans who have suffered accidental exposures experienced inflammation of the mucous membranes and eye irritation. Necrosis and demyelination of nerve fibers were reported among mice and monkeys following acute intraperitoneal and subchronic intramuscular exposures, respectively. Subchronic oral exposure to rats caused histologic cerebral and liver damage. Mutagenicity testing was positive for sodium-azide in a variety of assays.

17. Document Analysis a. Descriptors

b. Identifiers/Open-Ended Terms NIOSH-Publication, NIOSH-Contract, Contract-210-79-0030, Organo-metallic-compounds, Pesticides, Agricultural-chemicals, Preservatives, Occupational-exposure, Toxic-effects, Mutagens, Irritants, Brain-damage, Nervous-system-disorders

c. COSATI Field/Group

18. Availability Statement	19. Security Class (This Report)	21. No. of Pages 58
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I. SCOPE OF DOCUMENT AND SUMMARY OF MAJOR FINDINGS

A. CLASS IDENTIFICATION

The inorganic azides may be identified by the chemical sturucture $R(N_3)_x$, where R is the inorganic element such as sodium, lead, or hydrogen, and x is an integar that depends on the valence state of the inorganic element.

The azide group has a chain structure (N=N=N) rather than a ring structure.

B. CHEMICALS TO BE ADDRESSED

Individual profiles have been prepared for the following azides:

Barium azide
Cesium azide
Hydrazoic acid (hydrogen azide)
Lead azide
Lithium azide
Potassium azide
Rubidium azide
Silver azide
Sodium azide

The Appendix contains a list of all the inorganic azides considered under the class definition. The above azides were selected for individual consideration because they were either identified as having some commercial importance or had manufacturer data available from the U.S. EPA (1980). Sodium azide is the most important commercial azide followed by the military explosives lead and silver azide. The other azides profiled individually have annual production volumes no greater than several thousand pounds. Virtually all of the inorganic azides listed in the Appendix are available on a laboratory reagent basis.

C. SUMMARY OF BIOLOGICAL ACTIVITY

Most of the available literature regarding the toxicity of inorganic azides addresses sodium azide and, to a lesser extent, hydrazoic acid; very

little information is available for lead azide. No information was found on the biological effects of the other azide compounds for which profiles have been prepared. It appears that there has been little experimentation with the inorganic azides other than sodium azide because these compounds are potentially explosive.

The toxic effects of the inorganic azides appear to be due to the azoimide radical. The major acute effects resulting from administration, by various routes, of toxic doses of either sodium azide or hydrazoic acid to animals were hypotension, respiratory stimulation and convulsions, followed by respiratory depression and death. Similar effects have been observed in humans following vapor and accidental oral exposures, as well as inflammation of the mucous membranes and eye irritation. Acute intraperitoneal (mice) and subchronic intramuscular (monkey) exposure to sodium azide has resulted in necrosis and demyelination of nerve fibers (symptoms included incoordination, ataxia, paresis, tremors, and blindness), and subchronic oral exposure (rats) caused histologic cerebral and liver damage. Limited additional data indicates that sodium azide was non-carcinogenic to rats following chronic oral administration, although sodium azide has demonstrated mutagenicity in a wide variety of assays.

II. INFORMATION PROFILES

A. BARIUM AZIDE

1. Chemical Name: Barium Azide

2. Chemical Structure: $Ba(N_3)_2$

3. Synonyms: Barium trinitride

4. Chemical Abstract Service (CAS) Number: 18810-58-7

5. Registry of Toxic Effects of Chemical Substances (RTECS) Number:

   CQ8500000

6. Chemical and Physical Properties:

Description:	crystalline solid
Molecular Weight:	221.40
Boiling Point:	decomposes
Melting Point:	decomposes at 120°C
Vapor Pressure:	---
Solubility:	17.3 g/100 cc water (17°C) 0.017 g/100 cc absolute alcohol (16°C) insoluble in ether
Specific Gravity:	2.936
Stability:	explodes when shocked or heated

7. Production

Data available from the U.S. EPA (1980) regarding producers of barium azide and production volumes are presented below:

Fairmount Chemical Co.
Newark, NJ
Manufacturer
1977 Production of 0 to 1000 lb.

Atlas Powder Co.
Tamaqua, Pa
Manufactuer
1977 Production of 0 to 1000 lb.

8. Use

Barium azide is used in the preparation of cesium azide (Williams, 1979).

A substantial amount of patent literature indicates that barium azide is useful as an oxidation preventor in electron tubes, thermionic cathodes, and vacuum tubes. Patent literature also indicates that barium azide is useful as a propellant in various types of cartridges and in auto safety bag inflation.

Barium azide may also be useful in various types of pyrotechnics (Ellern, 1968).

9. Manufacturers and Distributors

The manufacturers of barium azide are presented above in the production section.

Other distributors include (Chem Sources--USA, 1980):

Alfa Products	ICN/ K and K
Apache Chem.	Noah Chem.
Atomergic Chemetals	Pfaltz and Bauer
	Reliable Chem.

10. Manufacturing Processes

The commercial production method for barium azide is not available from the literature. It is possible, however, to produce barium azide via metathesis (double decomposition) with aqueous solutions of sodium azide and barium salts. It may also be possible to make it from hydrazine hydrate, ethyl nitrate, and caustic barium or from hydrazoic acid reaction with barium metal, oxide, hydroxide, or carbonate (Smith, 1951).

11. Impurities and Additives

No information was found in the literature searched.

12. Occupational Exposure

The National Occupational Hazard Survey does not provide an estimate of the number of workers who are potentially exposed to barium azide.

13. Control Technology and Work Practices

Specific factors that may contribute to or prevent employee exposure to barium azide were not found in the literature searched.

14. Biological Effects

No information on the biological effects of barium azide was found in the literature searched.

15. Ongoing Studies

No current toxicological or environmental studies of barium azide were found.

16. Exposure Standards

No recommended or promulgated occupational exposure standards for barium azide were found.

17. Sources of Additional Relevant Information

No sources of additional relevant information were identified.

18. Other Pertinent Data

No other information that would aid in the assessment of barium azide as an occupational hazard was found in the literature searched.

B. CESIUM AZIDE

1. Chemical Name: Cesium Azide

2. Chemical Structure: C_5N_3

3. Synonyms: Cesium trinitride

4. Chemical Abstract Service (CAS) Number: 22750-57-8

5. Registry of Toxic Effects of Chemical Substances (RTECS) Number:

Not listed

6. Chemical and Physical Properties:

Description:	colorless needles
Molecular Weight:	174.93
Boiling Point:	decomposes 350°C
Melting Point:	310-326°C
Vapor Pressure:	---
Solubility:	224.2 g/100 cc water (0°C) 1.037 g/100 cc alcohol (16°C) insoluble in ether
Specific Gravity:	---
Stability:	deliquescent; potentially explosive

7. Production

Data available from the U.S. EPA (1980) regarding producers of cesium azide and production volumes are presented below:

Eastman Kodak (Rochester, NY)
Manufacturer
1977 Production of 0 to 1000 lb

Fairmount Chemical Co. (Newark, NJ)
Manufacturer
1977 Production of 0 to 1000 lb

8. Use

Cesium azide is used in the preparation of cesium metal (Williams, 1979). It may also be used in explosives.

9. Manufacturers and Distributors

The only manufacturer data available is presented above.

Distributors include (Chem Sources--USA, 1980):

Accurate Chem. and Sci.	ICN/K and K
Alfa Prod.	Noah Chem.
Atomergic Chemetals	Orion Chem.
EM Labs	Pfaltz and Bauer
Fisher Sci.	Reliable Chem.
	Var-Lac-Oid Chem.

10. Manufacturing Processes

Cesium azide is prepared by the metathetical reaction between aqueous solutions of cesium sulfate and barium azide (Williams, 1979).

11. Impurities or Additives

No information was found in the literature searched.

12. Occupational Exposure

The National Occupational Hazard Survey does not provide an estimate of the number of workers who are potentially exposed to cesium azide.

13. Control Technology and Work Practices

Specific factors that may contribute to or prevent employee exposure to cesium azide were not found in the literature searched.

14. Biological Effects

No information on the biological effects of cesium azide was found in the literature searched.

15. Ongoing Studies

No current toxicological or environmental studies of cesium azide were found.

16. Exposure Standards

No recommended or promulgated occupational exposure standards for cesium azide were found.

17. Sources of Additional Relevant Information

No sources of additional relevant information were identified.

18. Other Pertinent Data

No other information that would aid in the assessment of cesium azide as an occupational hazard was found in the literature searched.

C. HYDRAZOIC ACID

1. Chemical Name: Hydrazoic Acid

2. Chemical Structure: HN_3

3. Synonyms: Azoimide
Diazoimide
Hydroazoic acid
Hydrogen azide
Hydronitric acid
Triazoic acid

4. Chemical Abstracts Service (CAS) Number: 7782-79-8

5. Registry of Toxic Effects of Chemical Substances (RTECS) Number:

MW2800000

6. Chemical and Physical Properties (anhydrous):

Description:	mobile liquid with intolerable pungent odor
Molecular Weight:	43.03
Boiling Point:	35.7°C
Melting Point:	-80°C
Vapor Pressure:	321 mm Hg (15°C)
Solubility:	completely soluble in water; soluble in alcohol, alkali, and ether
Specific Gravity:	1.09
Stability:	pure acid is exceptionally explosive; water solutions are stable; dangerous explosion risk when stacked or heated.

7. Production

Data available from the U.S. EPA (1980) regarding producers of hydrazoic acid and production volumes are presented below:

Fairmount Chemical Co. (Newark, NJ)
Manufacturer
1977 production of 0 to 1000 pounds

8. Use

Hydrazoic acid is used in the preparation of inorganic, organic, and organometallic azides and derivatives (Reichle, 1966).

9. Manufacturers and Distributors

See Section 7 (Production) for the only available data.

10. Manufacturing Processes

Hydrazoic acid is best prepared as a dilute, aqueous, or organic solution by acidification of sodium azide using a strong, nonvolatile acid (sulfuric) and with simultaneous distillation of the acid produced. Ether extraction of the aqueous solution and subsequent drying over calcium chloride yield an anhydrous etheral solution of the acid (Reichle, 1966). It can also be prepared by the reaction of hydrazine and nitrous acid (Hawley, 1977).

11. Impurities or Additives

There is no evidence in the literature searched to indicate the presence of impurities or deliberate additives in commercially produced hydrazoic acid.

12. Occupational Exposure

The National Occupational Hazard Survey does not provide an estimate of the number of workers who are potentially exposed to hydrazoic acid.

13. Control Technology and Work Practices

Specific factors that may contribute to or prevent employee exposure to hydrazoic acid were not found in the literature searched.

14. Biological Effects

a. Animal Studies

(1) Acute Exposures

The acute toxic effects of hydrazoic acid are summarized in Table 1. The major acute effects resulting from intraperitoneal

Table 1. Acute Effects of Hyrdrazoic Acid

Route	Species	Dose	Response	Reference
i.p.[a]	mice	21.5 mg/kg	LD50.	Graham et al., 1948
inhalation	rats	849-967 ppm	0/14 deaths in 60 min.[b]	Fairhall et al., 1943
inhalation	rats	1024 ppm	3/8 deaths in 60 min.	Fairhall et al., 1943
inhalation	rats	1081 ppm	3/4 deaths in 60 min.	Fairhall et al., 1943
inhalation	rats	1138 ppm	17/18 deaths in 60 min.	Fairhall et al., 1943
inhalation	rats	1162-1365 ppm	16/16 deaths in 60 min.	Fairhall et al., 1943
inhalation	rats	1566 ppm	2/2 deaths in 30 min.	Fairhall et al., 1943
inhalation	rats	1872 ppm	2/2 deaths in 19 min.	Fairhall et al., 1943
inhalation	rats	2080 ppm	2/2 deaths in 16 min.	Fairhall et al., 1943
inhalation	rats	2900 ppm	2/2 deaths in 10 min.	Fairhall et al., 1943

[a] i.p. = intraperitoneal.

[b] min. = minute.

injections of lethal doses of hydrazoic acid into mice, rats, guinea pigs, and rabbits were hypotension, increased respiration (resulting in pulmonary damage), irritation of mucous membranes, constriction of the gut that led to hepatic engorgement, and generalized convulsions followed by depression (Graham et al., 1948; Sutton, 1963). Liver and kidney showed no damage. Inhalation of the vapors caused dyspnea, paralysis of hind legs, convulsions, acute bronchiolar inflammation, irritation of mucous membranes, and, in some instances, pulmonary edema and cardiac arrest (Graham et al., 1948; Fairhall et al. 1943). Sublethal doses caused marked stimulation of respiration with generalized convulsions, followed by a long periods of depression characterized by respiratory depression and muscle flaccidity.

(2) Subchronic Exposures

No information was found in the literature searched.

(3) Chronic Exposures

No information was found in the literature searched.

(4) Carcinogenicity

No information was found in the literature searched.

(5) Mutagenicity

No information was found in the literature searched.

(6) Teratogenicity

No information was found in the literature searched.

(7) Reproductive Effects

No information was found in the literature searched.

(8) Other Relevant Information

Based on the ferric chloride test for azides, unchanged hydrazoic acid could not be detected in the urine of rats; the test was performed within 4 hours of injection of sublethal doses (Graham et al., 1948).

b. Human Studies

(1) Pharmacokinetics

No information was found in the literature searched.

(2) Health Effects

Stern (1927) reported the case of a chemist who accidentally inhaled hydrazoic acid fumes. The victim suffered inflammation of the mucous membranes, conjunctivitis and bronchitis, swelling of both knee joints, large blue lesions on the legs, and pyrexia lasting several days.

Köcher (1930) intentionally inhaled a 1% solution of hydrazoic acid and noted a fall in blood pressure before collapse. The recovery period, which was characterized by persistent headaches, lasted 30 minutes.

The use of sodium azide in organic synthesis reactions may result in significant exposure to hydrazoic acid vapor, as observed by Sutton (1963), when a chemist was acidifying 10 g sodium azide in a malfunctioning hood. Dizziness, weakness, blurred vision, hypotension, bradycardia, and shortness of breath resulted within a few minutes. Recovery was complete within 1 hour.

(3) Target Organ Toxicity

No information was found in the literature searched.

(4) Epidemiology

Graham et al. (1948) examined 10 workmen who were exposed to hydrazoic acid fumes (0.3-3.9 ppm) for 1-15 years during the manufacture of lead azide from lead nitrate and sodium azide. Detailed clinical and laboratory examinations revealed that exposure resulted in eye and nose irritation, throbbing headaches, unsteadiness, palpitations, and severe, rapid reductions in blood pressure. No pathological conditions that could be attributed to the occupational exposure were observed.

15. Ongoing Studies

No current toxicological or environmental studies of hydrazoic acid were found.

16. Exposure Standards

No recommended or promulgated occupational exposure standards for hydrazoic acid were found.

17. Sources of Additional Relevant Information

No sources of additional relevant information were identified.

18. Other Pertinent Data

No other information that would aid in the assessment of hydrazoic acid as an occupational hazard was found in the literature searched.

D. LEAD AZIDE

1. Chemical Name: Lead Azide

2. Chemical Structure: $Pb(N_3)_2$

3. Synonyms: Dextrinated lead azide
Hydrazoic acid, lead salt
Initiating explosive lead azide
Lead trinitride

4. Chemical Abstracts Service (CAS) Number: 13424-46-9

5. Registry of Toxic Effects of Chemical Substances (RTECS) Number:

OF8650000

6. Chemical and Physical Properties:

Description:	colorless to white needles or powder
Molecular Weight:	291.25
Boiling Point:	decomposes
Melting Point:	explodes at 350°C
Vapor Pressure:	---
Solubility:	0.023 g/100 water (18°C); very soluble in acid; insoluble in NH_4OH
Specific Gravity:	--
Stability:	sensitive to impact; stable at ambient temperatures; detonates at 350°C

7. Production

Data available from the U.S. EPA (1980) regarding producers of lead azide and production volumes are presented below:

Olin Corp. - Ammunition Oper. (East Alton, IL)
Manufacturer
1977 production 0 to 1000 pounds

DuPont (Pompton Lakes, NJ)
Manufacturer
No production ranges available

Lead azide is made from sodium azide; in 1977, du Pont imported between 10-100 thousand pounds of sodium azide (U.S. EPA, 1980).

8. Use

Lead azide is the primary explosive used in military detonators in the United States (Lindner, 1980). It is also used in commercial blasting caps (Rinkenbach, 1965).

Western Electric Co. is using a high-energy bonding system for repairing circuit board contacts. This is performed by placing a gold foil replacement contact over the damaged one, followed by a covering of lead azide, that explodes when ignited by an electric spark. The miniature shock wave is effective because the charge is concentrated on a microscopically small point (Industry Week, 1978).

9. Manufacturers and Distributors

See Section 7 (Production) for the only available data.

10. Manufacturing Processes

The manufacture of lead azide is accomplished as described in the following passages from the Kirk-Othmer Encyclopedia of Chemical Technology (Lindner, 1980):

> Lead azide is made in small batches (e.g., 5 kg) buffered by the reaction solutions of lead nitrate or lead acetate with sodium azide:
>
> $$Pb(NO_3)_2 + 2NaN_3 \longrightarrow 2NaNO_3 + PbN_6$$
>
> Sodium azide is insensitive but highly toxic. Contact must be avoided with acid, with which it forms the dangerous hydrazoic acid, and with copper, lead, cadmium, silver, mercury, or their alloys, with which sensitive azides may be formed. Nucleating agents, such as poly(vinyl alcohol)(PVA), sodium carboxymethyl cellulose (CMC), or dextrin, may be added during precipitation to produce free-flowing crystals or rounded agglomerates required for the large-scale, automatic loading of detonators. The presence of hydrophilic polymeric substances also tends to eliminate the small possibility of spontaneous explosions occurring during the precipitation process. Wetting agents may also be added.
>
> All phases of the manufacturing process are conducted by remote control in stainless steel vessels using either distilled or demineralized water and filtered solutions. The overall

precipitation time is about 60 min. In the manufacture of dextrinated lead azide, lead nitrate stock solution is prepared by dissolving lead nitrate, dextrin, and sodium hydroxide in water at pH 4.6-4.8. The solution is cooled, filtered, pumped to a storage tank, and allowed to settle for 8 hours or longer. A sodium azide stock solution is similarly prepared. The precipitation vessel is a precisely made, open-topped, round-bottom, double-walled, polished stainless steel tilting pot equipped with an agitator, feed tubes, and a water-spray ring. The lead nitrate solution at 60°C is transferred to the precipitation vessel from a measuring tank and the sodium azide solution is added at a rate of about 2 L/min while maintaining a temperature of 60°C. Lead azide precipitates as free-flowing, fine white agglomerates. After settling, the mother liquor is decanted through a filter, collected, and neutralized with 30% sodium nitrite and then 30% nitric acid or with ceric ammonium nitrate to decompose the azide ion. Excess acid is neutralized with soda ash. Any soluble lead present is precipitated as the insoluble carbonate. The lead azide precipitate is washed repeatedly with water, vacuum filtered, and dried. Lead azide made without dextrin (RD 1333) usually contains more than 99% azide. When made with dextrin it contains about 92% lead azide, 4-5% lead hydroxide, 3% dextrin, and other impurities. Lead azide must be free of needle-shaped crystals longer than 0.1 mm. Dextrinated lead azide is somewhat more hygroscopic and less dense, sensitive, and efficient as an initiator than the 99% product (Lindner, 1980).

11. Impurities or Additives

See manufacturing processes above.

12. Occupational Exposure

The National Occupational Hazard Survey indicates that 7999 workers are potentially exposed to lead azide.

13. Control Technology and Work Practices

Specific factors that may contribute to or prevent employee exposure to lead azide were not found in the literature searched. Lead azide dust is kept to a minimum in industry because it is highly explosive.

14. Biological Effects

a. Animal Studies

(1) Acute Exposures

The maximum intraperitoneal dose of lead azide in rats that would not result in death was 150 mg/kg body weight (Fairhall et al., 1943).

The acute toxic effect of this substance was associated with the azoimide radical rather than with the lead.

(2) Subchronic Exposures

Fairhall et al. (1943) described experiments in which 3 groups of 10 rats each received oral doses of 20, 40, or 60 mg/rat/day of lead azide. One-hundred percent of the animals fed 60 mg died within 9 weeks; 100% of the rats given 40 mg died within 14 weeks, and 60% of the rats given 20 mg died within 44 weeks (30% of the control animals died within this total period). The quantity of lead stored in liver, kidney, and bone tissue was usually proportional to the total amount received and was not an index of toxicity.

(3) Chronic Exposures

No information was found in the literature searched.

(4) Carcinogenicity

No information was found in the literature searched.

(5) Mutagenicity

No information was found in the literature searched.

(6) Teratogenicity

No information was found in the literature searched.

(7) Reproductive Effects

No information was found in the literature searched.

(8) Other Relevant Information

No information was found in the literature searched.

b. Human Studies

(1) Pharmacokinetics

No information was found in the literature searched.

(2) Health Effects

No reactions were observed in any of 10 individuals following patch tests with lead azide, with 10 day intervals between applications (Schwartz, 1942).

(3) Target Organ Toxicity

No information was found in the literature searched.

(4) Epidemiology

No information was found in the literature searched.

15. Ongoing Studies

No current toxicological or environmental studies of lead azide were found.

16. Exposure Standards

Both NIOSH (1978) and the ACGIH (1980) currently recommend time-weighted average (TWA) concentrations for occupational exposure to inorganic lead compounds (0.10 mg Pb/m^3 and 0.15 mg $Pb/m/^3$, respectively). It appears, however, that the toxicity of lead azide is associated with the azoimide radical rather than with the lead.

17. Sources of Additional Relevant Information

No sources of additional relevant information were identified.

18. Other Pertinent Data

No other information that would aid in the assessment of lead azide as an occupational hazard was found in the literature searched.

E. LITHIUM AZIDE

1. Chemical Name: Lithium Azide

2. Chemical Structure: LiN_3

3. Synonyms: Lithium trinitride

4. Chemical Abstract Service (CAS) Number: 19597-69-4

5. Registry of Toxic Effects of Chemical Substances (RTECS) Number:

Not listed

6. Chemical and Physical Properties:

Description:	colorless crystals
Molecular Weight:	48.96
Boiling Point:	decomposes
Melting Point:	decomposes (115-298°C)
Vapor Pressure:	---
Solubility:	66.41 g/100 cc water (16°C) 20.26 g/100 cc absolute alcohol (16°C) insoluble in ether
Specific Gravity:	---
Stability:	hygroscopic; potentially explosive

7. Production

Data available from the U.S. EPA (1980) regarding producers of lithium azide and production volumes are presented below:

Eastman Kodak (Rochester, NY)
Manufacturer
1977 Production of 0 to 1000 lb

Fairmount Chemical Co. (Newark, NJ)
Manufacturer
1977 Production of 0 to 1000 lb

8. Use

According to various patent literature, lithium azide is useful in a variety of organic and inorganic syntheses, as a nitrogen generating propellant in safety bag inflation, as an emulsified agent for lithium metal in lithium chloride melts, as a catalyst for epimerizations, and as an aid for volatization of fumigants and insecticides.

9. Manufacturers and Distributors

The only manufacturer data available is listed above.

Other distributors include (Chem Sources--USA, 1980):

Apache Chem.
Fisher Sci.
ICN/K and K
Pfaltz and Bauer

10. Manufacturing Processes

The commercial production method for lithium azide is not available from the literature. It can, however, be produced by the metathesis (double decomposition) of another soluble azide and a soluble lithium salt or by the action of hydrazoic acid on lithium metal, oxide, hydroxide, or carbonate (Smith, 1951).

11. Impurities or Additives

No information was found in the literature searched.

12. Occupational Exposure

The National Occupational Hazard Survey does not provide an estimate of the number of workers who are potentially exposed to lithium azide.

13. Control Technology and Work Practices

Specific factors that may contribute to or prevent employee exposure to lithium azide were not found in the literature searched.

14. Biological Effects

No information on the biological effects of lithium azide was found in the literature searched.

15. Ongoing Studies

No current toxicological or environmental studies of lithium azide were found.

16. Exposure Standards

No recommended or promulgated occupational exposure standards for lithium azide were found.

17. Sources of Additional Relevant Information

No sources of additional relevant information were identified.

18. Other Pertinent Data

No other information that would aid in the assessment of lithium azide as an occupational hazard was found in the literature searched.

F. POTASSIUM AZIDE

1. Chemical Name: Potassium Azide

2. Chemical Structure: KN_3

3. Synonyms: Potassium trinitride
Kazoe

4. Chemical Abstract Service (CAS) Number: 20762-60-1

5. Registry of Toxic Effects of Chemical Substances (RTECS) Number:

Not listed.

6. Chemical and Physical Properties:

Description:	colorless crystals
Molecular Weight:	81.12
Boiling Point:	decomposes
Melting Point:	350°C (vacuum); decomposes 300°C
Vapor Pressure:	--
Solubility:	49.6 g/100 cc water (17°C); 54 g/100 g water (25°C) soluble in alcohol insoluble in ether
Specific Gravity:	2.04
Stability:	potentially explosive

7. Production

Data available from the U.S. EPA (1980) regarding producers of potassium azide and production volumes are presented below:

Eastman Kodak (Rochester, NY)
Manufacturer
No Manufacture or importation during 1977

PPG Industries, Inc. (Pittsburgh, PA)
Importer
No Manufacture or importation during 1977

8. Use

Potassium azide is used as a pesticide, trade named Kazoe, for control of weeds in rice and peanuts, for control of diseases in many fruits and

vegetables, as a soil fumigant, and as a turf remover (Ouellette and King, 1977). It is applied in either a 10% or 20% granular formulation.

According to various patent literature, potassium azide is also useful as a fertilizer component, in nitrification inhibition, in the whitening and cleaning of glass fabrics, and as a nitrogen generator in auto safety bag inflation.

9. Manufacturers and Distributors

The only manufacturer data available is listed above.

Distributors include (Chem Sources--USA, 1980):

Apache Chem.
Atomergic Chemetals
Chemical Procurement Lab.
EM Lab
Fisher Sci.
ICN/K and K
Noah Chem.

10. Manufacturing Processes

The commercial process for making potassium azide is not available from the literature. PPG Industries has two patents describing manufacture (Snead and McGreevy, 1974, 1970). In the first patent, sodium azide and potassium phosphate aqueous solutions are fed to a vacuum crystallizer in which KN_3 is crystallized and water is evaporated. The KN_3 is centrifuged out, washed, and dried. In the second patent, the KN_3 is precipitated from a sodium azide-potassium carbonate solution prepared by dissolving NaN_3 and KOH in water and passing CO_2 through the solution. The product KN_3 is separated by either centrifugation or filtration.

Potassium azide can also be prepared by adding potassium metal to liquid N_2O and liquid ammonia (Abe and Funaoka, 1960). The KN_3 thus obtained is 99.6% pure.

11. Impurities or Additives

As mentioned above, one method of preparing KN_3 yields a product that is 99.6% pure. No other data was available from the literature searched.

12. Occupational Exposure

The National Occupational Hazard Survey indicates that 1480 workers are potentially exposed to potassium azide.

13. Control Technology and Work Practices

Specific factors that may contribute to or prevent employee exposure to potassium azide were not found in the literature searched.

14. Biological Effects

No information on the biological effects of potassium azide was found in the literature searched.

15. Ongoing Studies

No current toxicological or environmental studies of potassium azide were found.

16. Exposure Standards

No recommended or promulgated occupational exposure standards for potassium azide were found.

17. Sources of Additional Relevant Information

No sources of additional relevant information were identified.

18. Other Pertinent Data

No other information that would aid in the assessment of potassium azide as an occupational hazard was found in the literature searched.

G. RUBIDIUM AZIDE

1. Chemical Name: Rubidium Azide

2. Chemical Structures: RbN_3

3. Synonyms: Rubidium trinitride

4. Chemical Abstract Service (CAS) Number: 22756-36-1

5. Registry of Toxic Effects of Chemical Substances (RTECS) Number:

Not listed.

6. Chemical and Physical Properties:

Description:	colorless needles or plates
Molecular Weight:	127.49
Boiling Point:	decomposes
Melting Point:	decomposes 310°C
Vapor Pressure:	---
Solubility:	107.1 g/100 cc water (16°C) 0.182 g/100 cc alcohol (16°C) insoluble in ether
Specific Gravity:	2.7876
Stability:	potentially explosive

7. Production

Data available from the U.S. EPA (1980) regarding producers of rubidium azide and production volumes are presented below:

Eastman Kodak (Rochester, NY)
Manufacturer
1977 Production: none

8. Use

Patent literature indicates that rubidium azide is useful in fumigant insecticides and in auto safety bag inflation as a propellant.

9. Manufacturers and Distributors

U.S. EPA (1980) lists Eastman Kodak in Rochester, NY as a manufacturer.

Distributors include (Chem Sources--USA, 1980):

Alfa Prod.	ICN/K and K
Chemical Procurement Lab.	Orion Chem.
Fisher Sci.	Pfaltz and Bauer
	Var-Lac-Oid Chem.

10. Manufacturing Processes

The commercial method for making rubidium azide was not available from the literature searched. It can be obtained by the metathesis (double decomposition) of a soluble azide with a soluble rubidium salt or by the action of hydrazoic acid on rubidium metal, ozide, hydroxide, or carbonate (Smith, 1951).

11. Impurities or Additives

No information was found in the literature searched.

12. Occupational Exposure

The National Occupational Hazard Survey does not provide an estimate of the number of workers who are potentially exposed to rubidium azide.

13. Control Technology and Work Practices

Specific factors that may contribute to or prevent employee exposure to rubidium azide were not found in the literature searched.

14. Biological Effects

No information on the biological effects of rubidium azide was found in the literature searched.

15. Ongoing Studies

No current toxicological or environmental studies of rubidium azide were found.

16. Exposure Standards

No recommended or promulgated occupational exposure standards for rubidium azide were found.

17. Sources of Additional Relevant Information

No sources of additional relevant information were identified.

18. Other Pertinent Data

No other information that would aid in the assessment of rubidium azide as an occupational hazard was found in the literature searched.

H. SILVER AZIDE

1. Chemical Name: Silver Azide

2. Chemical Structure: AgN_3

3. Synonyms: Silver trinitride

4. Chemical Abstracts Service (CAS) Number: 13863-88-2

5. Registry of Toxic Effects of Chemical Substances (RTECS) Number:

Not listed.

6. Chemical and Physical Properties:

Description:	white crystals consisting of fine amorphorus aggregates varying in size
Molecular Weight:	149.89
Boiling Point:	decomposes
Melting Point:	251°C
Vapor Pressure:	---
Solubility:	0.01 g/100 cc water (100°C) practically insoluble in cold water soluble in KCN and dilute HNO_3 slightly soluble in NH_4OH
Specific Gravity:	---
Stability:	detonates on sudden heating; sensitive to impact

7. Production

No data is available from the U.S. EPA (1980).

Production volumes for silver azide are judged to be much smaller than for lead azide (SRC Estimate).

8. Use

Silver azide has received attention as a potential replacement for lead azide in explosive initiators because it may be used in small quantities and thereby offers the possibility of miniaturization of fuse components. Silver azide requires somewhat less energy for initiation than lead azide and fires within a shorter amount of time (Lindner, 1980).

Silver azide is a better explosive initiator than lead azide, but the relatively high cost of silver has precluded its extensive use (Rinkenbach, 1965).

9. Manufacturers and Distributors

No data was available from the literature searched. It is likely, however, that silver azide is produced by government owned or operated munitions facilities.

10. Manufacturing Processes

Silver azide is made in the same manner as lead azide, except that silver nitrate is used in the reaction with sodium azide instead of lead nitrate (Lindner, 1980). This involves adding sodium azide solution and silver nitrate solution to a precipitation vessel and precipitating the silver azide. After settling, the mother liquor is decanted through a filter, collected, and neutralized. The silver azide precipitate is washed repeatedly with water, vacuum filtered, and dried.

11. Impurities or Additives

No information was found in the literature searched.

12. Occupational Exposure

The National Occupational Hazard Survey does not provide an estimate of the number of workers who are potentially exposed to silver azide.

13. Control Technology and Work Practices

Specific factors that may contribute to or prevent employee exposure to silver azide were not found in the literature searched.

14. Biological Effects

No information on the biological effects of silver azide was found in the literature searched.

15. Ongoing Studies

No current toxicological or environmental studies of silver azide were found.

16. Exposure Standards

No recommended or promulgated occupational exposure standards for silver azide were found.

17. Sources of Additional Relevant Information

No sources of additional relevant information were identified.

18. Other Pertinent Data

No other information that would aid in the assessment of silver azide as an occupational hazard was found in the literature searched.

I. SODIUM AZIDE

1. Chemical Name: Sodium Azide

2. Chemical Structure: NaN_3

3. Synonyms: Azide
Azium
Kazoe
NSC 3072
Smite
Sodium trinitride
U-3886

4. Chemical Abstracts Service (CAS) Number: 26628-22-8

5. Registry of Toxic Effects of Chemical Substances (RTECS) Number:

VY8050000

6. Chemical and Physical Properties:

Description:	colorless to white salt
Molecular Weight:	65.02
Boiling Point:	decomposes
Melting Point:	decomposes above 320°C
Vapor Pressure:	---
Solubility:	29.5% w/w water (21°C); 41 g/100 g water (17°C); 0.3% in alcohol (25°C) soluble in liquid ammonia; insoluble in ether
Specific Gravity:	1.846
Stability:	decomposes upon heating; is not sensitive to impact or friction; unlimited shelf life; moderately susceptible to light

7. Production

Data available from the U.S. EPA (1980) regarding producers of sodium azide and production volumes are presented in Table 2.

In 1963, about 100 thousand pounds of sodium azide were consumed in the United States (Reichle, 1966).

Future growth of the sodium azide market may depend upon the extent to which air bags are used in new automobiles, since the azide is used as a

Table 2. Producers of Sodium Azide and Production Ranges (U.S. EPA, 1980)

Producer and Location	Type of Production	1977 Production Range
Lonza Inc. Fair Lawn, NJ	Importer	not available
Fairmount Chemical Co. Newark, NJ	Manufacturer	zero
PPG Industries Pittsburgh, PA	Importer	zero
American Research Products Co. Euclid, OH	Manufacturer/ Importer/ Small Manufacturer	not available
DuPont Wilmington, DE	Importer	10-100 thousand lb
SST Corp. Clifton, NJ	Importer	10-100 thousand lb
Thiokol Corp. Newton, PA	Importer	zero
Rocket Research Co. Redmond, WA	Importer	zero

nitrogen inflator for the air bags. Figures for the quantities of sodium azide that may be required for this purpose cannot be ascertained at this time.

8. Use

Sodium azide is used commercially as a raw material in the production of the explosive detonating lead azide (Lindner, 1980); this is likely to be the largest volume use of sodium azide at the present time. Sodium azide is also used as a raw material in the production of other azides.

Large volumes of sodium azide may be consumed in automobile air-bag systems in the near future. A chemical mixture containing sodium azide is used to produce nitrogen gas, which inflates the air bags. The U.S. Congress has enacted legislation requiring all passenger cars produced after late 1983 to contain passive restraints. Two basic types of passive restraint systems have been developed by the auto industry: the air-bag system and the automatic seat-belt system. According to Dunne (1980), air bags are now out of favor in Detroit, and the auto companies are preparing to put automatic seat belts in the new cars requiring them. This is because the automatic seat belts are less expensive than air bags, and they do not have many of the liabilities of air bags. In auto models that seat three in the front, however, an air bag will still be needed for the center passenger.

In addition to numerous listings for air bag inflators, a survey of recent patent literature revealed the following uses for sodium azide: intermediate for herbicide production, production of photosensitive polymers, antibacterials, antidepressants, propellants, denitrification preventor for fertilizers, and various organic syntheses. PPG Industries markets a farm product known as Smite which is used for the control of soil-borne diseases in vegetable crops (Ouellette and King, 1977); it contains either an 8% or 16% concentration of sodium azide. Sodium azide is also used (in concentrations up to 0.1%) as a preservative in diluents used in automatic blood cell counters (NIOSH, 1978).

9. Manufacturers and Distributors

SRI International (1980) lists the following as manufacturers of sodium azide:

Lonza Inc.	Mapleton, IL
MCB Reagents	Norwood, OH

Producers cited by the U.S. EPA (1980) are presented in Table 2.

The following companies are listed as sources for purchase of sodium azide (1980-81 OPD Chemical Buyers Directory, 1980; Chemical Week: 1981 Buyers' Guide Issue, 1980); Chem Sources--USA, 1980):

Accurate Chemical and Sci.	ICN Nutritional
Aldrich Chem.	ICN/ K and K
Alfa Prod.	Kennedy and Klim
American Research Prod.	LaPine Sci.
Anachemia Chem.	Lonza Inc.
Apache Chem.	MCB Reagents
Atomergic Chemetals	Mallinchkrodt
J.T. Baker Chem.	Noah Chemical
Biochemical Labs.	Orion Chem.
Bio-Clinical Labs.	PRC Research Chem.
Chem Services	Pfaltz and Bauer
Chemical Dyanamics	Reliable Chem.
Columbia Organics	SST Corp.
Delamar Inc.	Sigma Chem.
Eastern (Guardian) Chem.	Simmler Inc.
Eastman Kodak	Spectrum Chemical Mfg.
EM Labs	Tridom Chem.
Fairmount Chem.	United States Biochem.
Fisher Sci.	Vega Biochem.
Gallard-Schlesinger Chem.	
Hach Chem.	

10. Manufacturing Processes

Sodium azide is commercially manufactured from sodamide and nitrous oxide (Reichle, 1966; Lemke, 1969). The sodamide is first prepared in this process by reacting sodium with liquid ammonia in the presence of a catalyst, such as ferric nitrate, according to the following reaction:

$$Na + NH_3 \xrightarrow{\text{catalyst}} NaNH_2 + 1/2H_2$$

The slurry of $NaNH_2$ in liquid ammonia produced by this reaction is then treated with nitrous oxide under pressure to produce the azide as follows:

$$2NaNH_2 + N_2O \longrightarrow NaNH_3 + NaOH + NH_3$$

The reaction is carried out at about 30°C.

The product of the above reactions is a slurry of sodium azide and sodium hydroxide.

The azide can be separated and purified by at least two different methods. First, the slurry is allowed to settle, the azide-rich settling layer is dissolved in water, and pure sodium azide is recovered by crystallization (Tunison, 1977). Second, the entire slurry is dissolved in water, filtered to remove solid impurities, and ethyl alcohol is added, causing a better than 99% pure sodium azide to be precipitated (Suwa, 1975). The general manufacturing operations are outlined in Figure 1.

Sodium azide has also been commercially manufactured by reacting hydrazine or its hydrate with ethyl nitrite and caustic soda (or sodium ethylate) in an alcoholic solution (Smith, 1951; Davis, [1943]). A typical reaction could be represented as follows:

$$N_2H_5OH + C_2H_5ONO + C_2H_5ONa \longrightarrow NaN_3 + 2C_2H_5OH + 2H_2O$$

The sodium azide product precipitates because it is only slightly soluble in alcohol; it is then filtered off, washed, and dried.

11. Impurities or Additives

There is no evidence in the literature searched to indicate the presence of impurities or deliberate additives in commercially produced sodium azide.

12. Occupational Exposure

The National Occupational Hazard Survey indicates that 5954 workers are potentially exposed to sodium azide.

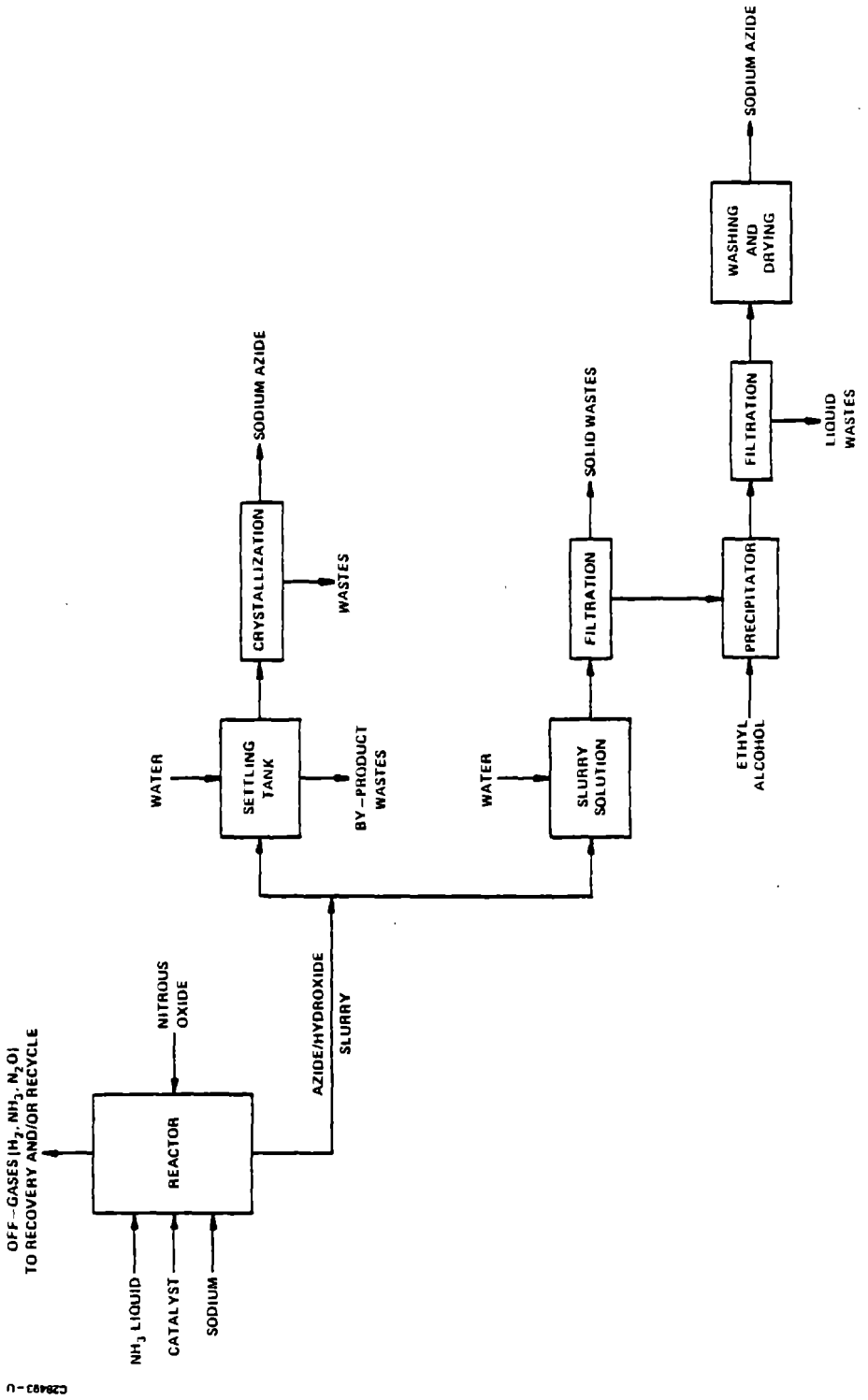

Figure 1. Sodium Azide Manufacture via Sodamide (adapted from Lemke, 1969; Tunison, 1977; Suwa, 1975)

13. Control Technology and Work Practices

Specific factors that may contribute to or prevent employee exposure to sodium azide were not found in the literature searched.

14. Biological Effects

a. Animal Studies

(1) Acute Exposures

The acute toxic effects of sodium azide are summarized in Table 3. Sodium azide is rapidly absorbed from the gastrointestinal tract. Administration of toxic doses of sodium azide by all routes resulted in respiratory stimulation and convulsions, followed by respiratory depression and death in mice, rats, guinea pigs, and rabbits (Graham, 1949; Sutton, 1963; Hicks, 1950; Mettler and Sax, 1972). Sodium azide had a direct stimulant action on cardiac muscle, due to its effect on the sympathetico-adrenal mechanism. It increased gut and bladder contractions and dilated the peripheral blood vessels directly resulting in hypotension (Graham, 1949). Sublethal doses of sodium azide stimulated the central nervous system, accelerating respiration rate, and also increased the frequency of micturition.

Black et al. (1954) reported that 0.56-0.67 mg/kg sodium azide administered intravenously to renal hypertensive rats lowered blood pressure from 190 mm Hg to 125 mm Hg for 30 to 45 minutes.

Continuous intravenous infusions of 1-10 mg/kg/minute sodium azide to dogs reduced the mean blood pressure (primarily by peripheral vascular relaxation) and maintained the reduction for several hours (Maher and Bollman, 1955). Blood pressure returned to normal 15 minutes after discontinuation of the infusion.

Mettler and Sax (1972) injected 11 monkeys with a single intravenous dose of 8-16 mg/kg sodium azide. Convulsions rapidly ensued,

Table 3. Acute Effects of Sodium Azide

Route[a]	Species	Dose[b] (mg/kg)	Response[b]	Reference
oral	rats	40	0/3 deaths in 3 h.	Fairhall et al., 1943
oral	rats	45	5/8 deaths in 3 h.	Fairhall et al., 1943
oral	rats	46	3/3 deaths in 3 h.	Fairhall et al., 1943
oral	mice	27	LD50.	Graham, 1949
oral	mice	37.4	LD50.	Roth et al., 1956
oral	rabbits	3-10	40-60% reduction in blood pressure (lasting more than 1 h), hematuria, cardiac irregularities.	Roth et al., 1956
i.p.	rats	5-10 mg/kg every 15-30 min for 3 to 6 h	Severe intoxication, stupor, convulsions, coma, 40% mortality, 30% showed brain lesions upon autopsy.	Hicks, 1950
i.p.	rats	25	0/4 deaths in 3 h.	Fairhall et al., 1943
i.p.	rats	33	8/12 deaths in 3 h.	Fairhall et al., 1943
i.p.	rats	37	5/5 deaths in 3 h.	Fairhall et al., 1943
i.p.	rats	12 mg/kg/d x 4 d	Animals became akinetic and paretic in gait and exhibited tremor of forelimbs; almost all animals survived but remained unsteady in their gait; autopsy revealed striatal lesions.	Miyoshi, 1967
i.p.	mice	28-34	LD50.	Graham, 1949
i.p.	mice	18	LD50.	Graham et al., 1948
i.p.	mice	15-20	LD50, death following coma and convulsions, histology revealed demyelination of nerve fibers in the central nervous system and testicular damage.	Hicks, 1950

Table 3. Acute Effects of Sodium Azide (Cont'd)

Route	Species	Dose (mg/kg)	Response	Reference
s.c.	rats	33	0/5 deaths in 3 h.	Fairhall et al., 1943
s.c.	rats	35	4/9 deaths in 3 h.	Fairhall et al., 1943
s.c.	rats	38	8/8 deaths in 3 h.	Fairhall et al., 1943
i.v.	mice	19	LD50.	Graham, 1949
i.v.	monkeys	12	LDLo, 18% mortality, death from respiratory arrest, survivors had ataxia due to cerebellar cortex damage.	Mettler and Sax, 1972
i.m.	monkeys	20	Unconsciousness within 6 to 8 min, recovery during next 8 h; highly susceptible to lower dose.	Hurst, 1942

[a] i.p. = intraperitoneal; s.c. = subcutaneous; i.v. = intravenous; i.m. = intramuscular.

[b] min = minute; h = hour; d= day.

followed by unconsciousness and apnea. Two animals died in respiratory failure within 5 minutes after the injection. The remaining animals recovered from the acute effects and were killed over a period of 1 to 55 weeks. Ataxia later developed in the majority of survivors due to cerebellar cortical destruction which, in turn, was probably a result of the cumulative effects of hypotension and impairment of both ventilation and oxidation enzyme activity.

(2) Subchronic Exposures

Rats fed a daily diet of 26.7 mg sodium azide showed 100% mortality in 39 days (Fairhall, 1943). A pronounced weight loss was evident during this time.

Hurst (1942) reported incoordination, cerebellar ataxia, paresis or rigidity, fibrillary muscle tremors, apathy, and temporary or permanent blindness following repeated (10-12) intramuscular injections of sodium azide in monkeys. The animals died (respiratory failure) or were killed after 2 to 204 days, having received a total of 35 to 1206 mg/kg sodium azide in 2 to 165 doses. Necrosis or demyelination in the optic nerves and necrosis in the caudate nucleus and putamen of the lenticular nucleus were evident after these repeated doses. Lesions in other parts of the grey and white matter were much less frequent and severe. Weight loss in poisoned animals was as great as 30% of the original weight.

Mortality and average weight did not differ from those of controls when rats were given a 0.2% sodium azide solution as a substitute for drinking water for 120 days (Williams, 1967). Histology of tissue samples revealed liver and cerebral damage of varying severity. Sodium azide was found to predispose the brain to increased copper deposition.

(3) Chronic Exposures

No information was found in the literature searched.

(4) Carcinogenicity

Ulland et al. (1973) reported in an abstract that sodium azide was non-carcinogenic when the maximum tolerated dose (unspecified) or half that level was added to the diet or given by gastric intubation to rats; the compound was apparently administered for 18 months followed by a 6-month observation. N-2-Fluorenylacetamide was used as a positive control in the experiment.

(5) Mutagenicity

The mutagenic properties of sodium azide have been intensely studied because this compound was originally found to be an unusually powerful and efficient mutagen that produced a remarkable lack of chromosome aberrations and a high frequency of mutations (Kleinhofs et al., 1978). The potential for sodium azide to cause mutations has also been demonstrated in bacteria, yeast, cultured mammalian cells, and Dorosophila melanogaster. The results of these assays are presented in Table 4. In the in vitro mutagenicity assay systems, sodium azide acts as a direct mutagen with the addition of metabolic activation destroying the mutagenic activity. It has been demonstrated that both barley and bacteria can metabolize sodium azide to a stable active mutagenic compound (Kleinhofs et al., 1975), however, the results from cell culture, yeast, and Drosophila suggest that at least some eucaryotic cells may also have the capability to activate this compound. Sodium azide has been tested and shown to be ineffective in the formation of chromosomal abberations in human leukocytes in culture (Sander et al., 1978).

(6) Teratogenicity

No information was found in the literature searched.

(7) Reproductive Effects

No information was found in the literature searched.

Table 4. Mutagenicity Assays of Sodium Azide

Type of Assay	Organism	Strain	With Mammalian Metabolic Activation	Dose	Results	Reference
reverse mutation, try$^-$ (plate incorporation assay)	E. coli	WP2	no	10^{-3}M	-	Kleinhofs and Smith, 1976
		WP2 (uvtA)	no	7.5 x 10^{-4}M	-	
		DM203 (uvrA, rec B_{21})	no	7.5 x 10^{-4}M	-	
		DM207 (uvrA, rec C_{22})	no	7.5 x 10^{-4}M	-	
forward mutation, 5-FU resist. (plate incorporation)	E. coli	WP2	no	10^{-3} M	-	Kleinhofs and Smith, 1976
		WP2 (uvrA)	no	7.5 x 10^{-4} M	+	
		DM203 (uvrA, rec, B_{21})	no	7.5 x 10^{-4}M	-	
		DM2107 (uvrA, rec C_{22}1)	no	7.5 x 10^{-4}M	-	
forward mutation, pencillin or streptomycin-resistant	micrococcus pyrogenes var. aureus	FDA209	no	9.23 x 10^{-3}M	+	Clark, 1953
mutation to oudbain and 8-azaguanine resistant	Chinese hamster cells	V79	no	1-10mM	+	Jones et al., 1980
forward mutation, thymidine kinase locus	mouse lymphoma cells	651784	no	500-2000 µg/ml	+	Clive et al., 1979
reversion and/or gene conversion	Saccharomyces cerevisiae	D7	no	0.5-2.0 x 10^{-5}M	+	Silhankova et al., 1979; Velemansky et al., 1979
sex-linked recessive lethal	Drosophila melanogaster (males)	NA	NA	5 x 10^{-3}M injected	-	Clark, 1958
				NR injected	-	Krama and Gollapudi, 1979
				2 x 10^{-3}M injected (0.09 µl)	-	Brink, 1963
sex-linked recessive lethal	Drosophila melanogaster (males)	NA	NA	1 x 10^{-4} M in food for 30 days	+	Krama and Gollapudi, 1979
chromosome aberrations	human keukocytes	NA	NA	10^{-2}-10^{-4} M	-	Sanders et al., 1978

[a]It was only reported that sodium azide elicited positive effects in Salmonella mutagenicity assays; results were not reported specifically by strain or by presence or absence of metabolic activation.

Table 4. Mutagenicity Assays of Sodium Azide

Type of Assay	Organism	Strain	With Mammalian Metabolic Activation	Dose	Results	Reference
reverse mutation, his$^-$ (plate incorporation assay)	S. typhimurium	G46	no	10^{-3}M	+	Kleinhofs and Smith, 1976
		TA1976	no	10^{-3}M	+	
		TA1950	no	10^{-4}M	+	
		TA1530	no	10^{-4}	+	
		TA1535	no	10^{-4}		
		C117	no	10^{-3}	-	
		C121	no	10^{-3}	-	
		C354	no	10^{-3}	-	
		C367	no	10^{-3}	-	
		D1714	no	10^{-3}	-	
reverse mutation, his$^-$ (plate incorporation assay)	S. typhimurium	TA100	no	1 µg/plate	+	McCann et al., 1975
		TA1535	no	1 µg/plate	+	
reverse mutation, his$^-$ (spot test)	S. typhimurium	TA1530	no	250 µg/well	+	Chen et al., 1975
		G46	no	250 µg/well	-	
reverse mutation, his$^-$ (plate incorporation assay)	S. typhimurium	TA100 (-nitrofurazene reductase activity)	no	25 µg/plate	+	Rosenkranz and Speck, 1975
reverse mutation, his$^-$ (plate incorporation assay)	S. typhimurium	TA1530	no	2×10^{-5}M	+	Ciesla and Filutowicz, 1979
reverse mutation, his$^-$ (plate incorporation assay)	S. typhimurium	TA100	no	1 µg/plate	+	DeFlora, 1978
		TA100	yes	1 µg/plate	-	
reverse mutation, his$^-$ (plate incorporation assay)	S. typhimurium	TA100	no	7×10^{-4}M	+	Dierickx, 1979
		TA100	yes	7×10^{-4}M	-	
reverse mutation, his$^-$	S. typhimurium	TA98, 100, 1535, 1537	no, yes	n.a.	+[a]	NTP, 1980

(8) Other Relevant Information

The activation of guanylate cyclase (GMP) by sodium azide has been investigated by several researchers. Kimura et al. (1975a, 1975b) found that maximal and half-maximal increases in cyclic GMP levels in incubated rat tissue slices of cerebellum, cerebral cortex, or liver occurred at 1 mM and 0.15 mM sodium azide, respectively. These results were confirmed by DeRubertis and Craven (1976). Katsuki et al. (1977) theorized that activation of GMP may be due to the formation of nitric oxide or another reactive material from sodium azide.

Kleinhofs et al. (1978) reviewed the abundant literature on the inhibition of various enzymes by sodium azide. Sodium azide inhibits catalase, protein synthesis, cellular respiration (but not glycolysis), the oxidation of cytochrome, and the indophenol reaction; this inhibition is due primarily to the azide's interaction with heme-containing proteins (Boyland and Gallico, 1952; Hicks, 1950; Keilin and Hartree, 1934). It also inhibits peroxidase, methemoglobin, and metmyoglobin (Hewitt and Nicholas, 1963).

Based on the ferric chloride test for azides, unchanged sodium azide could not be detected in the urine of rats; the test was performed within 4 hours of injection of sublethal doses (Graham et al., 1948).

b. Human Studies

(1) Pharmacokinetics

Sodium azide is rapidly absorbed from the gastrointestinal tract, by the lungs, and through the skin (Mettler and Sax, 1972; Köcher, 1930; Black et al., 1954).

(2) Health Effects

Ingestion of 150 mg of sodium azide by a female technician produced symptoms within 5 minutes that included a severe headache,

breathlessness, accelerated pulse, nausea, vomiting, and diarrhea (Burger and Bauer, 1965). Sweating and weakness persisted for 10 days.

Gobbi (1967) described the cases of 3 workers who handled sodium azide in bulk and developed headache, nausea, faintness, and transient hypotension. Exposure may have been dermal or via inhalation.

Richardson et al. (1975) described two cases of accidental poisoning by ingestion of Isoton, a diluting fluid for blood counters containing sodium azide (concentration unspecified). Ingestion of 50-60 mg sodium azide by a patient resulted in unconsciousness, incontinence, nausea that persisted for 1 hour, and a severe headache that subsided in 24 hours. Renal and liver function tests remained normal. The second incident involved ingestion by a female technician of 5-10 mg sodium azide that produced transient headache, sweating, and nausea that rapidly subsided.

Black et al. (1954) found sodium azide to be a potent hypotensive agent in man. Oral doses of 0.65-1.3 mg (≈0.01-0.02 mg/kg) elicited a rapid hypotensive effect in hypertensive individuals but had no effect in normotensive individuals. Chronic administration of sodium azide (0.6-1.3 mg, 3 to 4 times daily for up to 2 years) to hypertensives produced a sustained reduction in blood pressure with no damage to the kidney, heart, or liver.

(3) Target Organ Toxicity

Sodium azide is recognized as a potent respiratory poison and metabolic inhibitor (Kleinhofs et al., 1978). Cerebral and pulmonary edema and inflammation of the esophagus and gastric mucosa were revealed during autopsy of a 20-year old man who ingested an unspecified quanitity of sodium azide (Kozlicka-Gajdzinska and Brzyski, 1966).

(4) Epidemiology

No information was found in the literature searched.

15. Ongoing Studies

Carcinogenesis testing, under the auspices of the National Toxicology Program, has begun on Sodium Azide (NTP, 1981). Prechronic testing is currently in progress, and the compound is being administered via gavage to rats and mice.

16. Exposure Standards

The Threshold Limit Value (TLV) recommended by the ACGIH (1980) for exposure to sodium azide is 0.1 ppm (0.3 mg/m^3).

17. Sources of Additional Relevant Information

Sodium azide was the subject of a NIOSH Current Intelligence Bulletin (NIOSH, 1978). The purpose of the bulletin was to alert hospital and clinical laboratory personnel to the explosive hazard associated with sodium azide. The compound is used as a preservative in many *in vitro* diagnostic products and when it comes in contact with copper, lead, brass, or solder in plumbing systems, highly explosive lead and copper azides are formed.

18. Other Pertinent Data

The National Highway Traffic and Safety Administration (NHTSA) has noted that the disposal of automobiles equipped with air bags that use sodium azide to generate gas could result in the accumulation of sodium azide or its degradation products (Mechanical Engineering, 1979). It was felt that these chemicals could be hazardous to workers in the vehicle scrappage industry or to the environment.

Appendix - Inorganic Azides

The following list includes all of the inorganic azides considered under the class definition. The compounds in the list were identified primarily from the following sources: U.S. EPA TSCA list and U.S. EPA (1980), SRI International (1980), Chem Sources--USA (1980), Kirk-Othmer's Enclyclopedia of Chemical Technology, The Merck Index (1976), Hawley (1977), and other library and computer data base information pertaining to azides.

Aluminum azide
Ammonium azide
Barium azide
Beryllium azide
Bismuth azide
Boron azide
Bromine azide
Cadmium azide
Calcium azide
Carbonyl azide
Cerium azide
Cesium azide
Chlorine azide
Chromium azide
Cobalt azide
Copper azide
Cyanic azide
Fluorine azide
Gold azide
Hydrazoic acid
Iodine azide
Iron azide
Lead azide
Lithium azide
Magnesium acide
Manganese azide
Mercury azide
Molybdenum azide
Nickel azide
Scandium azide
Selenium azide
Silicon azide
Silver azide
Sodium azide
Tellurium azide
Thorium azide
Thulium azide
Tin azide
Titanium azide
Tungsten azide
Uranium azide
Vanadium azide
Ytterbium azide
Zinc azide
Zirconium azide

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