Information Profile
Sodium Azide

Prepared by:
Arthur R. Gregory Ph.D.
Pharmacologist
NIOSH/DCDSD
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Sodium Azide

In 1963, about 100,000 lbs. of sodium azide were consumed in the U.S. (Reichle, 1966). More recent production figures were not encountered; however, this compound is the largest volume azide in commercial production. In addition, the use of air bags in automobiles could greatly increase current production volumes.

In the past, the most important commercial use of sodium azide may have been in the preparation of lead azide for explosives (Rinkenbach, 1965). However, the use of a sodium azide mixture to produce nitrogen gas for air bag inflation in automobiles may soon become more important.

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.

Sodium azide is currently being used in a farm product, at a 15% concentration, for control of soil borne diseases in vegetable crops in Florida and in peanuts in Virginia, North Carolina, and Georgia (Farm Chem., 1976). The product, called Smite 15G, is marketed by PPG Industries.

Sodium azide is also used (in concentrations of 0.1%) as a preservative in diluents used in automatic blood cell counters (Chem. Eng. News, 1976).

The following companies produce sodium azide at the listed locations (Chemical Week, 1976, personal communications 1978).

Table 1. Inorganic Azides

Compound	CAS Number	RTECS Number	Molecular Formula
Sodium Azide azium	26628-22-8	VY80500	NaN_3
Lead Azide initiating explosive lead azide (DOT)	13424-46-9	OF86500	PbN_6
Hydrazoic acid azoimide diazoimide hydrogen azide hydronitric acid triazoic acid	7782-79-8	MW28000	HN_3

Atomergic Chemetals Co.	Plainview, N.Y.
Canadian International	Canada - (reportedly stopping production)
Dynamit Nobel AG	Northvale, N.J.
Guardian Chemical	Happauge, N.Y.
Fairmount Chem.	Newark, N.J.
Lonza, Inc.	Mapleton, Ill.
PPG, Inc. (Ayers & Johnson, 1976)	---
United Technology (?)	California

Sodium azide is generally not considered to be explosive under normal conditions. However, many azide salts are exceedingly explosive and extreme care must be exercised to prevent their formation. For example, lead azide (a commercial detonator) is a more sensitive primary explosive than nitroglycerin and a more effective detonating agent than mercury fulminate, in comparison with lead azide, copper azide is even more explosive and too sensitive to be used commercially. As described in the previously transmitted NIOSH Current Intelligence Bulletin; Explosive Azide Hazard, August 16, 1976, explosions have resulted from copper azide and lead azide surreptitiously formed during contact of dilute solutions of sodium azide with copper and lead plumbing lines as well as copper laboratory equipment.

NIOSH recommends that sodium azide not be permitted to come into contact with any material which may permit the formation of explosive heavy metal azide salts (e.g., copper, lead, mercury, silver).

NIOSH recommends that sodium azide not be permitted to come into contact with acids. Azides react with acids to form hydrazoic acid. Hydrazoic acid is not only an explosive and toxic gas, but it has the potential of

escaping into surrounding areas where it can react with exposed metals to form highly explosive azide salts.

Lead Azide

Production figures are not available. Lead azide is used as a detonating initiator for commercial blasting caps and military ammunition (Rinkenbach, 1965).

A listing of producers is not available; however, it is possible that lead azide is produced at government facilities operated by contractors, such as DuPont (SRC estimate).

Hydrazoic Acid

Production figures are not available.

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

There are no commercial data on production or sale of hydrazoic acid in the available literature.

1. Biological Effects of Exposure

The inorganic azides have a reputation as dangerous materials. Hydrazoic acid and its heavy metal salt, lead azide, are highly explosive which may account for the paucity of references to its pharmacological and physiological properties (Sutton, 1963). Since the percussion sensitivity of lead azide led to its important use for primers in munitions, it is not surprising that essentially no toxicological data on PbN_6 were encountered. The azide anion can be considered responsible for the similar biological effects of

hydrazoic acid and sodium azide. However, since HN_3 is a volatile liquid, the inhalation toxicity of the vapor is of additional concern.

a) Target Organs

Hydrazoic acid and sodium azide are powerful hypotensive agents (ug/kg range) which dilate blood vessels by direct action on the smooth nuscles. Azide also stimulates cardiac muscle and dilates the coronary vessels directly, thereby increasing the force of contraction and blood flow (Graham, 1949; Graham et al., 1948).

The greatest effect of azide is in the stimulation of the central nervous system (CNS), which leads to increased respiration and cardiac rate and force. With larger doses, (mg rather than ug/kg range) (Table 2), stimulation may be severe enough to cause convulsive seizures, which are characteristically followed by a period of depression leading to collapse and asphyxia from respiratory failure (Graham, 1949; Graham et al., 1948). Some survivors show injury and demyelination of nerve fibers, including the optic nerves and tracts in the CNS (Hicks, 1950). Blindness and ataxia are manifestations of the CNS damage produced by repeated doses in monkeys (Hurst, 1942; Mettler and Sax, 1972).

b) Acute Effects

The symptoms observed in animals after relatively large doses of sodium azide (Table 2) are respiratory stimulation and violent tonic convulsions leading to respiratory depression and death by asphyxiation (Graham, 1949). Mice, rats, guinea pigs, and rabbits dosed by oral, subcutaneous, intramuscular, intraperitoneal, or intravenous routes all showed similar symptoms which varied only in degree and rapidity of onset with varying dosage levels.

Hydrazoic acid has essentially the same toxicity (21.5 mg/kg) and symptomology for mice when injected intraperitoneally (Graham et al., 1948). When administered by injections and inhalation to mice, rats, guinea pigs, and rabbits, early irritation of mucous membranes with excessive salivation was noted. Inhalation of lethal doses of the vapor or spray produced, in addition, asthma and an acute bronchial inflammation in guinea pigs. Certain of the animals died of pulmonary edema, right side heart failure, and constriction of the gut which led to hepatic engorgement. When death was delayed from 3 hours to 3 days, pathological changes were observed only in the lungs. Intraperitoneal, intramuscular, intravenous, and subcutaneous injections of hydrazoic acid all gave rise to pulmonary damage.

Fairhall and coworkers, (1943) considered the acute toxicity of hydrazoic acid vapor comparable to that of hydrogen sulfide or hydrogen cyanide. Their results appear in Table 2.

Sublethal doses of sodium azide and hydrazoic acid give rise to a marked depression of blood pressure, coronary dilation, and mild respiratory stimulation with variable occurrences of convulsions (Graham el al., 1948); Graham, 1949). A long period of weakness, respiratory depression, and muscle flaccidity is followed by recovery.

Repeated intraperitoneal injections of sodium azide in rats (5 to 10 mg/kg every 15 to 30 minutes for 3 to 6 hours) resulted in severe intoxication (Hicks, 1950). At first the animals typically became stuporous, occasionally followed by brief generalized convulsions, and then comatose. This coma usually lasted 1 to 2 hours, after which the rats slowly recovered to a large extent during the next 12 hours. Hicks (1950) described nervous

Table 2. Acute Toxicity of Inorganic Azides

Organism	Route	Dose	Response	Reference
Hydrazoic Acid				
Mouse	I.P.	21.5 mg/kg	LD_{50}	Graham, 1948
Rat	Inhalation	849-967 ppm	0/14 died in 60 minutes	Fairhall et al., 1943
Rat	Inhalation	1024 ppm	3/8 died in 60 minutes	Fairhall et al., 1943
Rat	Inhalation	1081 ppm	3/4 died in 60 minutes	Fairhall et al., 1943
Rat	Inhalation	1138 ppm	17/18 died in 60 minutes	Fairhall et al., 1943
Rat	Inhalation	1162-1365 ppm	16/16 died in 60 minutes	Fairhall et al., 1943
Rat	Inhalation	1566 ppm	2/2 died in 30 minutes	Fairhall et al., 1943
Rat	Inhalation	1872 ppm	2/2 died in 19 minutes	Fairhall et al., 1943
Rat	Inhalation	2080 ppm	2/2 died in 16 minutes	Fairhall et al., 1943
Rat	Inhalation	2900 ppm	2/2 died in 10 minutes	Fairhall et al., 1943
Sodium Azide				
Rat	Oral	40 mg/kg	0/3 died in 3 hours	Fairhall et al., 1943
Rat	Oral	45 mg/kg	5/8 died in 3 hours	Fairhall et al., 1943
Rat	Oral	46 mg/kg	3/3 died in 3 hours	Fairhall et al., 1943
Rat	I.P.	25 mg/kg	0/4 died in 3 hours	Fairhall et al., 1943
Rat	I.P.	33 mg/kg	8/12 died in 3 hours	Fairhall et al., 1943
Rat	I.P.	37 mg/kg	5/5 died in 3 hours	Fairhall et al., 1943
Rat	I.P.	75 mg/kg	LD_{75} died in 3 hours	Fairhall et al., 1943
Rat	S.C.	33 mg/kg	0/5 died in 3 hours	Fairhall et al., 1943
Rat	S.C.	35 mg/kg	4/9 died in 3 hours	Fairhall et al., 1943
Rat	S.C.	38 mg/kg	8/8 died in 3 hours	Fairhall et al., 1943
Mouse	Oral	27 mg/kg	LD_{50}	Graham, 1949
Mouse	Oral	37.4 mg/kg	LD_{50}	Roth et al., 1956
Mouse	I.V.	19 mg/kg	LD_{50}	Graham, 1949
Mouse	I.P.	28-34 mg/kg	LD_{50}	Graham, 1949
Mouse	I.P.	18 mg/kg	LD_{50}	Graham et al., 1948
Mouse	I.P.	15-20 mg/kg	LD_{50}	Hicks, 1950
Mouse	I.P.	23.7 mg/kg	LD_{5}	Graham, 1949
Mouse	Unspecified	27 mg/kg	LD_{50}	Boyland & Gallico, 1952
Monkey	I.V.	12 mg/kg	LD_{Lo}	Mettler & Sax, 1972
Monkey	Intramuscular	20 mg/kg	Sick but survived	Hurst, 1942
Monkey	Intramuscular	10-12 mg/kg repeated	death after 3 to 4 doses	Hurst, 1942
Monkey	Intramuscular	5 mg/kg daily	5/6 died in 60 to 130 days	Hurst, 1942

Table 3. Summary of the Principal Lesions of the Nervous System Caused by Azide Poisoning in Eleven Rats (Hicks, 1950)

	Animals in Which Region Indicated Showed Lesions
Corpus callosum	6
Corpus striatum (gray matter)	10
Corpus striatum (white matter)	10
Optic tracts	8
Olfactory bulb	2
Thalamus	1

system lesions caused by the poisoning. Of 37 rats poisoned to the point of convulsions and coma, 15 died in the acute stages, 11 survived without brain lesions developing, and 11 survived to undergo varying degrees of destructive lesions (Table 3). They were almost exclusively limited to the corpus callosum, the corpus striatum, especially the bundles of myelinated fibers, and the optic nerve and tracts. Generally mild lesions with no particular distribution pattern were found in the hearts of most of the animals with brain lesions and in one or two instances where pathologic brain changes were absent. In one animal that showed severe lesions there was well developed necrosis of the tubular epithelium of the testis, and one other animal showed a few necrotic cells.

c) Subchronic Effects

Hurst (1942) noted as persistent symptoms in monkeys following a single large or repeated smaller doses of sodium azide temporary or permanent blindness with pupil dilatation, incoordination, cerebellar ataxia, paresis or rigidity, fibrillary muscular tremors and apathy. The animals died or were killed after 2 to 204 days, having received a total of 35 to 206 mg/kg in 2 to 165 doses. Repeated administration often produced necrosis or demyelination in the optic nerves and tracts and necrosis in the caudate nucleus and putamen of the lenticular nucleus. Lesions in the other parts of the grey and white matter were much less frequent and severe.

d) Chronic Effects

i) Carcinogenicity

Ulland and coworkers (1973) reported the results of a 2 year feeding study in which rats were fed sodium azide for 18 months and then

observed for 6 months after cessation of dosing. No carcinogenic effects could be attributed to sodium azide under the conditions of this study.

ii) Mutagenicity

Citing earlier works (Wyss et al., 1948 and Berger et al., 1953), Kleinhofs and coworkers (1975) noted that sodium azide increased the incidence of streptomycin- and penicillin-resistant mutations in Staphylococcus aureus. Later studies suggested that azide induces mutations in certain tester strains commonly used in the Ames Assay Test but not in others (Kleinhofs and Smith, 1976). Kleinhofs and Smith conclude from their study in 1976 that mutations induced by azide in susceptible tester strains of S. typhimurium and E. coli were highly specific and were not due to endogenous hydrogen peroxide accumulation. Instead, these lesions are probably either the result of direct action on DNA in the process of replication or are a result of the mutagen activated through metabolism by the cell. From these studies, they also conclude that DNA lesions induced by azide differ from those produced by U.V. (Kleinhofs and Smith, 1976). Sodium Azide inhibits the enzyme cytochrome oxidase (17). It severly compromises the ability of fibroblasts to phagocytize carmine dye particles (18), and interfers with the ability of E. coli to recover from the lethal effect of N-hydroxyurethane in the presence of chloramphenacol, puramycin, hydroxurea or uracil (19). It was inferred that sodium azide interfered with the cellular DNA repair system, and could thus act as a cocarcinogen. The mutations are intragenic and do not produce chromosomal aberrations.

Chronic treatment of rats with Sodium Azide produces an accumulation of copper, particularly in the CNS, typical of Wilson's disease (19).

iii) Teratology

No studies were encountered.

iv) Other

Mettler and Sax (1972) described a syndrome of ataxia in monkeys, developing from 1 to 55 weeks after receiving convulsant doses (8-16 mg/kg, I.V.) of sodium azide. The treatment immediately produced progressive cerebellar damage leading to states ranging from restricted Purkinje cell loss in the semilunar lubules to total decortication.

e) Human Effects

Stern (1927) described acute hydrazoic acid poisoning in a chemist who accidently inhaled fumes. The principle signs recorded were inflammation of the mucous membranes, conjunctivitis and bronchitis, swelling of both knee joints, large blue lesions on the legs, and fever lasting several days.

Kocher (1930) intentionally inhaled a 1 per cent solution of HN_3 and noted a fall of blood pressure to 70/50 before he collapsed. Recovery occurred in about 15 minutes and, apart from residual headaches for half an hour, was complete.

Graham et al., (1948) examined workers exposed to hydrazoic acid for 1-15 years in the manufacture of lead azide from lead nitrate and sodium azide from lead nitrate and sodium azide (Table 4). Detailed physical examinations revealed no evidence of any pathological condition which could be attributed to occupational exposure. Workers experienced throbbing headaches, palpitation, episodes of weakness and unsteadiness and mild eye and nose irritation. They stated that symptoms were noted only when the fume concentration was "high." The range of hydrazoic acid in the atmosphere

lay between 0.3 and 3.9 ppm. Exposure to these concentrations caused definite hypotension in the workers.

Sodium azide has been used experimentally for the therapy of hypertension. Black and coworkers (1954) found that sodium azide produces a larger fall in blood pressure in hypertensive patients than in normotensive individuals. Oral doses of 0.65 to 1.3 mg (approx. 0.01 to 0.02 mg/kg) produced a prompt fall of blood pressure which lasted 10 to 15 minutes. The administration of up to 1.3 mg three times daily for 10 days to nine normal individuals did not have a sustained effect on the pressure. The only other effect observed was an occasional transient pounding sensation in the head shortly after taking the drug. The fall in pressure was not accompanied by any significant change in pulse or respiration rate. Of 30 hypertensive individuals treated with 0.65 to 3.9 mg orally for 1 week to 2 1/2 years, 15 were found to maintain their pressure near normotensive levels. In 3 patients who took sodium azide for more than one year, there was no evidence of any organic damage on the basis of routine clinical studies. In 20 of the patients, continued treatment was followed by an increased sensitivity to the drug, necessitating a reduction in daily dosage. The administration of O_2 or Na2 S_2O_3 were without effect on the mortality rate of NaN_3 poisoned mice (20).

Table 4. Occupational History of Workers Exposed to HN_3 in the Manufacture of Lead Azide from Lead Nitrate and Sodium Azide (Graham et al., 1948).

Case Number	Years of Continuous Exposure	Age
1	15	43
2	circa 1	31
3	circa 1	31
4	16	54
5	2	36
6	circa 1	44
7	circa 1	35
8	10	44
9	1.6/12	41
10	8	52

2. TLV

A recommended ceiling TLV of 0.1 ppm for sodium azide in air has been proposed by the ACGIH (1977). It is clear from animal studies that dust concentrations should be kept at low levels if hypotension is to be avoided (Sutton, 1963).

No threshold limit values for hydrazoic acid have been proposed. The odor and irritant properties of hydrazoic acid do not present sufficient warning to prevent the occurrence of alarming symptoms. Imperial Chemicals Industries has suggested that concentrations greater than 1 ppm indicate an unsatisfactory situation for prolonged exposure (Sutton, 1963). The use of sodium azide in organic synthesis may result in significant hydrazoic acid exposure.

3. OSHA Standards

OSHA Standards have not been established for any of the inorganic azides.

4. Other Data

Table 5 lists the number of reported human exposures to inorganic azides (NIOSH, 1977).

Table 5. (NIOSH, 1977)

Compound	Number of Exposures
Sodium azide	3900
Lead azide	6780

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Sodium Azide NaN_3

Chemical Structure	Chemical Abstract Service Number
N_aN_3	026628228

Synonyms

NSC 3072, U 3886

Chemical and Physical Properties

Sodium azide is a stable, neutral, white crystaline solid. Its other properties are:

molecular weight	65.02
melting point	decomposes
specific gravity	1.846
solubility	
water	100 g @ 10°C
alcohol and benzole	slightly soluble

Consumption/Uses

Sodium azide is used as an explosive and as a source of hydrazoic acid. It is also used in the preparation of lead azide and pure sodium, and in some organic syntheses; Sodium azide is also used in in vitro diagnostic products and as a diluent in automatic blood cell counters. Consumption statistics were unavailable.

Production

There are 31 producers and distributors of sodium azide. Production statistics are unavailable.

Toxicity

Animal

Sodium azide is highly toxic to experimental animals. Signs

observed after relatively high doses include respiratory stimulation, convulsions, depression, and death. Lower doses produce variable convulsions and consistent, prompt, transient drop in blood pressure. Other effects observed in various species include hematuria, cardiac irregularities, severe intoxication, injury to and demyelination of nerve fibers, testicular damage, blindness, and rigidity with abnormal motions.

Species	Route	Dose	Effect
Rat	oral	46 mg/kg	LDLo
Rat	intraperitoneal	30 mg/kg	LDLo
Rat	subcutaneous	35 mg/kg	LDLo
Mouse	intraperitoneal	27 mg/kg	LD50
Monkey	intravenous	12 mg/kg	LDLo
Monkey	intramuscular	10-12 mg/kg	death after 3-5 treatments

Human

Sodium azide is rapidly absorbed from the gastrointestinal tract and from injection sites. It has been administered to hypertensive patients and produces drops in blood pressure which are greater in hypertensive than in normotensive individuals. Data on human toxicity is sparse, however, Patty reported a case of dizziness, weakness, blurred vision, shortness of breath, faintness, reduction in blood pressure, and bradycardia in a chemist acidifying 10 g of sodium azide in a malfunctioning hood.

Occupational Exposure

NIOSH estimates that fewer than 5,000 workers are exposed to sodium azide per year.

Recently, NIOSH has issued an alert to hospitals and clinical laboratories on the explosive hazard of sodium azide, formulated into diluents

used in conjunction with automatic blood cell counters, in plumbing systems. The alert notes that counters using these sodium azide diluents are found in over 15,000 hospitals and clinical laboratories in the U.S., and that a number of U.S. and Canadian hospitals have reported violent sodium azide-related explosions associated with automatic blood cell counters.

The NIOSH report states that sodium azide is a common preservative in many in vitro diagnostic products, and that it is found in concentrations of up to 0.1% in the diluents used with automatic blood cell counters. Sodium azide-containing waste from these machines is commonly discharged into a drain used solely for this purpose. The drain pipeline is thus bathed with solutions of sodium azide. The azide reacts with copper, lead, brass, or solder in the plumbing system forming an accumulation of lead and/or copper azide which is highly explosive. Thus, laboratory maintenance workers in general and plumbers in particular are exposed to this explosion hazard.

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11. Toxline/Chemline data bases

12. NIOSH alert on sodium azide

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15. Supplementary Notes

16. Abstract (Limit: 200 words)

Sodium-azide (26628228) (NA) was described in terms of general properties and uses, toxicity, and occupational exposures. NA has been used in preparation of explosives, herbicides, photosensitive polymers, antibacterials, antidepressants, propellants, denitrification preventors, and for various organic syntheses. Hydrazoic-acid (7782798) (HA) has been used in preparation of inorganic, organic, and organometallic azides and derivatives. Some inorganic azides and HA were highly explosive. HA and NA were found to be strong hypotensive agents which dilate blood vessels by direct smooth muscle action. Azide stimulated cardiac muscle and dilated coronary vessels directly, and stimulated the central nervous system, causing increased respiratory and cardiac rate and force. Acute effects in animals included respiratory stimulation and violent tonic convulsions, causing respiratory depression and asphyxiation. Sublethal doses of NA and HA produced marked depression of blood pressure, coronary dilation, and mild respiratory stimulation with variable convulsions. Subchronic effects included temporary or permanent blindness without dilatation, incoordination, cerebellar ataxia, paresis or rigidity, fibrillary muscular tremors, and apathy. No carcinogenic effects were found for NA. Mutation studies of NA showed interference with cellular DNA repair and possible cocarcinogenesis. Less than 5000 workers were exposed per year, but there was a hazard in hospitals and laboratories where NA could react with plumbing and be explosive.

17. Document Analysis a. Descriptors

b. Identifiers/Open-Ended Terms

NIOSH-Publication, NIOSH-Author, Laboratory-animals, Genetics, Respiratory-system-disorders, Agricultural-chemicals, Explosives-industry, Pharmaceutical-industry, Nervous-system-disorders, Cardiovascular-system, Occupational-exposure, Toxic-effects, Explosive-hazards

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