AIR POLLUTION ASPECTS

OF

ARSENIC AND ITS COMPOUNDS

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FOREWORD

As the concern for air quality grows, so does the concern over the less ubiquitous but potentially harmful contaminants that are in our atmosphere. Thirty such pollutants have been identified, and available information has been summarized in a series of reports describing their sources, distribution, effects, and control technology for their abatement.

A total of 27 reports have been prepared covering the 30 pollutants. These reports were developed under contract for the National Air Pollution Control Administration (NAPCA) by Litton Systems, Inc. The complete listing is as follows:

Aeroallergens (pollens)
Aldehydes (includes acrolein
    and formaldehyde)
Ammonia
Arsenic and Its Compounds
Asbestos
Barium and Its Compounds
Beryllium and Its Compounds
Biological Aerosols
    (microorganisms)
Boron and Its Compounds
Cadmium and Its Compounds
Chlorine Gas
Chromium and Its Compounds
    (includes chromic acid)

Ethylene
Hydrochloric Acid
Hydrogen Sulfide
Iron and Its Compounds
Manganese and Its Compounds
Mercury and Its Compounds
Nickel and Its Compounds
Odorous Compounds
Organic Carcinogens
Pesticides
Phosphorus and Its Compounds
Radioactive Substances
Selenium and Its Compounds
Vanadium and Its Compounds
Zinc and Its Compounds

These reports represent current state-of-the-art literature reviews supplemented by discussions with selected knowledgeable individuals both within and outside the Federal Government. They do not however presume to be a synthesis of available information but rather a summary without an attempt to interpret or reconcile conflicting data. The reports are

necessarily limited in their discussion of health effects for some pollutants to descriptions of occupational health exposures and animal laboratory studies since only a few epidemiologic studies were available.

Initially these reports were generally intended as internal documents within NAPCA to provide a basis for sound decision-making on program guidance for future research activities and to allow ranking of future activities relating to the development of criteria and control technology documents. However, it is apparent that these reports may also be of significant value to many others in air pollution control, such as State or local air pollution control officials, as a library of information on which to base informed decisions on pollutants to be controlled in their geographic areas. Additionally, these reports may stimulate scientific investigators to pursue research in needed areas. They also provide for the interested citizen readily available information about a given pollutant. Therefore, they are being given wide distribution with the assumption that they will be used with full knowledge of their value and limitations.

This series of reports was compiled and prepared by the Litton personnel listed below:

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ABSTRACT

Arsenic and its compounds are known to be toxic to

humans, animals, and plants. Arsenical dusts may produce der-

matitis, bronchitis, and irritation to the upper respiratory

tract. Medicinal ingestion of arsenic has produced keratoses

and cancer of the skin. The relationship of arsenic to other

types of cancer, particularly lung tumors, is uncertain.

Herbivorous animals have been poisoned after eating plants con-

taminated with arsenic compounds.

Arsenic is produced by smelters processing arsenical

ores. Because there is no economic incentive to remove arsenic

from the exhaust fumes of smelting, the smelter is a potential

local pollution source.

Arsenical compounds are used as insecticides and herbi-

cides. The use of arsenic in pesticides has declined since

the appearance of organic pesticides. The largest quantity

of arsenic is used as a desiccant for cotton prior to machine

picking. As a result, arsenic air pollution occurs during

cotton ginning and the burning of cotton trash.

Coal also contains a small amount of arsenic, and for

this reason, most city air contains a small amount of arsenic

given off by combustion of coal. The 1964 average daily con-

centration was 0.02 $\mu g/m^3$.

Particulate control of emissions from smelters and cotton gins appears to be adequate to control arsenic. However, hot exhaust gases must be cooled prior to removing arsenic as particulate because arsenic trioxide sublimes at 193°C.

No information has been found on the economic costs of arsenic air pollution or on the costs of its abatement.

Methods of analysis are available for the measurement of arsenic at concentrations in ambient air.

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1. INTRODUCTION

Air pollution caused by arsenical compounds has been observed near gold[9,10] and copper[21,22,23] smelters as well as in the areas where arsenic is used for agricultural purposes.[40] A small amount of arsenic can be measured in the air in most cities.[2,3,55]

Arsenic is a common industrial nuisance wherever arsenical ores are smeltered.[28,29] Before the advent of organic insecticides (e.g., DDT) the use of arsenicals as pesticides was increasing. However, since then, their use has leveled off and perhaps declined as the organic pesticides have taken their place. As a result, the supply of arsenic is greater than the demand, and the only economic incentive to remove arsenic from the exhaust fumes of smelters is the presence of other trace metals, such as tellurium, selenium, tin, zinc, and antimony.

Arsenic trioxide (white arsenic, arsenious oxide, As_2O_3) is the common commercial form of arsenic. Most compounds of arsenic, when heated in air, are converted to this tasteless, toxic, white powder. Arsenic metal, arsenic sulfides, arsine, arsenic(V) oxides (in the presence of a reducing agent), and organic arsenates, are all converted by heat and oxygen to arsenic trioxide. Since arsenic trioxide sublimes at 193°C, it is easily suspended as small particles in the air.[28,29]

2. EFFECTS

The effect of arsenic on humans, animals, and plants depends on the level of concentration and particular chemical compound in which it is found. Arsine, AsH_3, is extremely toxic while metallic arsenic is nontoxic. While organic arsenates, such as cacodylic acid, $(CH_3)_2H\ AsO_2$, are toxic to plants, they are relatively less toxic to animals, while the reverse is true for calcium and lead arsenates. Buchanan[12] and Frost[19] have recently written excellent reviews on the toxicity and biological effects of arsenic.

2.1 Effect on Humans

When arsenical compounds are present in the air, arsenic may be absorbed by inhalation, ingestion, or absorption through the skin. The airborne arsenic dust frequently causes irritation of the skin and mucous membranes, absorption taking place most readily on moist surfaces such as folds in the skin or mucous membranes. Thus, dermatitis, mild bronchitis, and nasal irritation are common symptoms of arsenic poisoning. With more severe exposure, perforation of the nasal septum takes place. Other systemic symptoms (see Table 1) caused by ingestion are uncommon in people who are merely exposed to arsenic dust. Because of its irritant properties, inhalation of sufficient amounts of arsenic trioxide to cause systemic poisoning would be difficult.[4] It has been reported that the fatal dose by ingestion of arsenic trioxide for man is 70,000 to 180,000 μg.[4]

TABLE 1

SYMPTOMS OF ARSENIC POISONING[12,19]

Acute	Chronic
Inflammation of the stomach and intestine	Weakness
Difficulty in swallowing	Loss of appetite
Burning in the throat	Nausea and occasional vomiting
Violent cramplike pains	Pains in stomach and intestines
Nausea	Diarrhea or constipation
Vomiting	Inflammation of mucous membrane of nose and gums
Diarrhea	Sore gums
Cold, damp skin	Runny nose
Feeble, irregular heartbeat	Perforation of nasal septum
Possible death in 1-4 days or possible chronic symptoms	Sneezing
	Coughing
	Skin ulcers
	Grayish pigmentation of skin
	Dermatitis

The colorless gas, arsine, is responsible for a few deaths each year. Arsine is formed wherever hydrogen is produced in the presence of arsenic. Thus, in the pickling of metals containing arsenic, arsine can be formed.[5] An exposure of 3,000 to 30,000 $\mu g/m^3$ for one hour is probably dangerous, and 210,000 $\mu g/m^3$ is probably hazardous to life. Exposure to lower concentrations (less than 1,500 $\mu g/m^3$) may cause jaundice and hemolytic anemia, with the primary effect being the destruction of red blood cells.[5]

Any arsenic taken into the body is excreted primarily in the urine, but some also in the feces, hair, nails, and epithelium.[41] Arsenic may be found in small quantities in the blood, all the tissue, the bones, and especially the hair. The arsenic content of the hair has been used to determine the arsenical

exposure history and has served as evidence of homicidal poisoning. Since some arsenic is consumed in food, it would not be possible to determine the amount of atmospheric exposure from the quantity present in the hair. The biological half-life for the excretion of arsenic is 30-60 hours.

Arsenic compounds have been used medically for treatment of syphilis and skin disorders as well as to increase resistance to fatigue. Over a period of time, it appears that a tolerance can be developed to limited quantities of arsenic.[19] However, keratoses on the palms of the hands and soles of the feet often appear after prolonged use of arsenic for medicinal purposes.[12]

2.1.1 Carcinogenesis

Arsenic is one of the most controversial of known or suspected environmental carcinogens. As early as 1820, arsenical compounds were suspected of carcinogenic action.[12] This impression was based on the observation that skin cancer frequently occurred following therapeutic administration of arsenic for psoriasis and other disorders. According to Buchanan,[12] nearly all of these cases of skin cancer followed a prolonged period of medicinal administration (averaging 18 years) of inorganic trivalent arsenic. He states that cancer frequently (80 percent of published cases) follows the nonmalignant manifestation of keratosis, commonly on the palms of the hands or soles of the feet.

In 1963, Heuper[26] listed arsenic as one of the recognized human carcinogens. The skin, lung, and liver were listed as recognized sites of arsenic cancers, and the mouth, esophagus, larynx, and bladder as suspected sites.

The role of arsenic as a respiratory carcinogen has received some support from the finding of above-average mortality from lung cancer in South Rhodesian miners[38] of gold-arsenical ores and the frequent occurrence of lung cancer in German vineyard workers[11,48] exposed to lead arsenate dust.

In opposition, Frost[19] argued that the carcinogenic action was inappropriately attributed to arsenic because of the tendency to specify arsenic as the carcinogen even when other materials were present. Nickel in particular, appears to be a carcinogen which occurs together with arsenic in industrial dusts. (See the companion report on the air pollution aspects of nickel.) The strongest arguments against arsenic as a carcinogen are the failure to show increased prevalence of cancer among industrial workers and failure to induce cancer in experimental animals.

Snegireff and Lombard[52] examined the records of two industrial plants in relation to the number of employees who died of cancer. In a plant where the workers were exposed to arsenic, 18 of 146 deaths (12.3 percent) were caused by cancer. In the second plant, where the workers were not exposed to arsenic, 12 of 109 deaths (11.0 percent) were caused by cancer.

The authors concluded that there was no significant difference in cancer mortality between plant employees who handled arsenic and those who were not exposed.

In another study, Pinto and Bennett[43] compared the mortality of employees who handled arsenic for the American Smelting and Refining Company at Tacoma, Wash., with those whe were not exposed to arsenic. (This smelter is the only plant presently producing arsenic commercially in the U.S. See Section 3.) They observed that 6 of 38 deaths (15.8 percent) among workers who were exposed to arsenic trioxide were caused by cancer, while 37 of 191 deaths (19.4 percent) were due to cancer among workers not exposed to arsenic. The evidence that these arsenic workers were exposed to higher concentrations of arsenic was confirmed by urinalysis. This lower percentage of deaths among arsenic workers compared favorably with the male deaths due to cancer (15.9 percent) in the entire state of Washington.[43] Arsenic workers excreted an average of 820 µg/liter of arsenic in the urine compared to 130 µg/liter for unexposed workers. The authors found no evidence that arsenic trioxide caused systemic cancer or fatal cardiovascular disease in humans.

Attempts to demonstrate through animal studies that arsenic is carcinogenic have often met with failure.[7,12,27,29,50] In fact, one study showed that arsenic suppressed the appearance of spontaneous tumors of the lung.[27] However, a few cases have been reported in which arsenical cancer was induced in animals.[12]

(See Section 2.2.2)

Some investigators have mentioned that the type of arsenic compounds involved may play a role in the carcinogenesis. Cornelius and Shelley[17] suggest that arsenic trioxide to which most smelter workers are exposed is probably not as carcinogenic as other soluble arsenic compounds. The composition and effect of arsenic compounds found in the ambient air have not been determined.

This dispute regarding the relationship between arsenic and cancer is probably the most important question in relation to the air pollution aspects of arsenic.

2.1.2 Community Episodes

In June 1962, a gold mine and smelter[9,10] in one of the Western States were reopened; after they were in operation for approximately 5 months it became apparent that the emission control equipment was inadequate. Both sulfur dioxide and arsenic trioxide were emitted in visible quantities into the atmosphere. Air samples taken at the plant showed 60 to 13,000 $\mu g/m^3$ of arsenic. A yellowish-gray dust on the ground gave evidence of fallout from the plant. No air samples were taken in the small mining community adjacent to the plant. However, the results of analyses of dust, water, and grass samples in the area are shown in Table 2.

A clinical examination was made of about 40 children attending two schools. Thirty-two of the children had

TABLE 2

ARSENIC CONTAMINATION IN A WESTERN MINING COMMUNITY[9,10]

Sample	Site	Arsenic Concentration
Flue dust	Base of stack	44%
Roof dust	Shed near office	4.4%
Dust	Area near drying mill	2.7%
Dust	Roaster area	3.1%
Dust	Sulfide-ore feed	1.23%
June grass	Area near school	925 $\mu g/g$
Water	Tap water	30 $\mu g/l$

dermatosis associated with cutaneous exposure to arsenic. At
first, it was thought that this was an example of systemic
arsenic poisoning; however, it proved to be a contact
dermatitis. Local skin irritation was observed in the folds
of the skin and where the skin was moist. In a few cases
the conjunctivae and nasal mucosae were irritated. It is
interesting to note that there were no cases of dermatitis
among the older children who were bussed to a high school in
a distant town: the disease occurred only among the younger
children who spent all their time in town. Moreover, no
new cases occurred after controls were introduced at the plant.
Keratoses, epitheliomas, and melanodermas were not present.
There were, of course, typical symptoms resulting from
exposure to arsenic, such as skin disease and nasal septum
perforation, among the refinery workers. Although animals
were not systematically studied, the pet population of more
than two dozen had been reduced to one dog.

Another example of arsenical air pollution occurred in Montana[21,22,23,57] in the years 1903 to 1905. During this time, large quantities of arsenic (see section 3.2) were emitted from a copper smelter. Although large numbers of animals were killed from eating plants contaminated with arsenic trioxide, no record of human health complaints is available.

An arsenical air pollution episode occurred at a copper mine in northern Chile.[39] The concentrations of arsenic present during this incident are shown in Table 3. A survey of 124 workers showed arsenical melanosis in 7.25 percent, arsenical dermatitis in 5.65 percent, and perforation of the nasal septum in 1.6 percent. No cutaneous manifestations were encountered among a control group or among members of the mining community.

TABLE 3

THE CONCENTRATION OF ARSENIC NEAR A
COPPER MINE IN NORTHERN CHILE[39]

Sample	Arsenic Concentration
Mineral (ore)	0.054%
Concentrated ore	1.64%
Calcined ore	0.30%
Dust from electrostatic precipitator	10.36%
Dust from stack	16.64%
Soil in plant	1,000 $\mu g/g$
Soil on road to plant	650 $\mu g/g$
Soil near hospital	20 $\mu g/g$
Soil near workers' club	90 $\mu g/g$
Air at roasting plant	400-81,000 $\mu g/m^3$
Air at smelter plant	400-5,400 $\mu g/m^3$

2.2 Effects on Animals

2.2.1 Commercial and Domestic Animals

In the preceding section, the three community episodes which were cited indicate that arsenical air pollution may have deleterious effects on animals. In the Chilean[39] episode, it was noted that dogs and chickens suffered from ulcers of the feet, although it was not definite that these lesions were due to contact with arsenic. In the Western State gold-mine episode,[9,10] the pet population was reduced from over 24 to 1. The surviving collie had a large ulcer in the mouth and another on the right forepaw.

The Montana[21,22,23,57] episode caused widespread damage to herbivorous animals caused by the ingestion of arsenic trioxide, which had contaminated the forage crops. Cows, sheep, and horses suffered from symptoms similar to those often observed in humans exposed to arsenic. When a flock of 3,500 sheep was brought from an area 28 miles away to graze 15 miles from the smelter, 625 of the sheep died. Upon analysis, grass and moss from the area in which the animals had fed was found to contain 52 and 405 ppm of arsenic trioxide respectively. Moreover, horses in an area remote from the smelter died after eating hay grown in a location on which the smelter fumes could have fallen. The arsenic trioxide content of the hay was 285 ppm. In addition, large quantities of both arsenic (10-150 ppm) and copper (128-1,800 ppm) were found on the vegetation up to 15 miles from the smelter.

Both the type of symptom and data from analyses of tissues failed to implicate copper as the poisoning agent, whereas evidence concerning animals fed on graded doses of arsenic verified experimentally that arsenic was responsible.

A study of the effects of airborne arsenic from a nearby smelter on animals in Saxony Forest was reported by Prell.[44] Red deer, foxes, and horses were all affected. The deer showed signs of thickened skins and joints, malformation of the horns, and loss of hair. The arsenic content of the stomach, intestine, liver, kidneys, and hair of the different species varied from traces to 42,000 μg/kg of tissue. Bees in the area had a high mortality rate. Analysis showed as much as 1 μg of arsenic per bee and 88 μg of arsenic per g of pollen.

Arsenical compounds, especially lead and calcium arsenate, have been used as insecticides. Most arsenates are toxic to insects and have little effect on plants. If ingested in sufficiently large quantities, the other compounds of arsenic are fatal to insects.

Aquatic animals have a higher tolerance for arsenic and normally have higher arsenic contents in the tissue than other animals.[12] For this reason, arsenicals have been recommended for use in the control of aquatic weeds.[50]

2.2.2 Experimental Animals

Frost[19] has reviewed the literature on the carcinogenic effects of arsenic. He reports that more than 35 experiments

yielded negative results for carcinogenicity when mice, rats,
pigs, and dogs were tested with arsenic trioxide, potassium
arsenate, sodium arsenite, sodium arsenate, arsanilic acid,
3-nitro-4-hydroxyphenylarsonic acid, and p-dimethylaminoazo-
benzene-p[1]-arsononic acid.

Buchanan[12] points out that Leitch and Kennaway[31]
succeeded in inducing only one squamous epithelioma (after 86
applications of potassium arsenite) in 100 mice. Several
other experimenters are also cited by Buchanan as follows.
Raposo[46] developed hyperplasias and 3 papillomas in the ears
of 10 rabbits painted with arsenious oxide. Cholewa[14] painted
the ears of two rabbits with potassium arsenite and observed
a papillomatous wart and a sarcoma of the perichondrium in the
ears of one rabbit after one year. Askanazy[6], Goeckerman, and
Wilhelm[20] observed teratogenic effects when rats receiving
transplanted embryos drank water containing arsenic. Holmberg
et al.[25] injected pregnant hamsters with barely sublethal doses
(20,000 μg/kg) of sodium arsenate. Of 177 embryos, 49 percent
were malformed and 84 percent were either malformed or resorbed.
The abnormalities observed were encephalocele, exencephaly,
unilateral and bilateral eye and ear malformations, and cleft
palate or lip. Exencephalic malformations were particularly
prominent in all litters. When sodium selenite (2,000 μg/kg)
was injected simultaneously with sodium arsenate (20,000 μg/kg)
into 12 pregnant hamsters, only 19 percent of 144 embryos
showed malformations and 39 percent, malformation or resorption.

Thus, these data confirm the work of Ferm and Carpenter[18] that arsenic is teratogenic as well as the fact that selenium is antagonistic to arsenic.

Recently Milner[34] experimented with three strains of mice, CXC3H, DBA, and Balb/C. Tumors were induced with methylcholanthrene or promoted by grafting the methylcholanthrene-treated skin on the flanks of recipient animals. Approximately 20 animals were tested in each experiment. Animals tested were given arsenic trioxide in their drinking water. The only significant effect observed by Milner was that the CXC3H strain of mice showed a reduction in papillomas. Thus, arsenic appeared to have little effect on the development of tumors in mice.

The above results tend to support the work of Kanisawa and Schroeder[27] who observed that mice fed 0.46 μg/gm sodium arsenite developed fewer spontaneous tumors (11/103) than the controls (55/170). However, only the number of adenomas and carcinomas of the lung was significantly lower.

2.3 Effects on Plants

In the smelter episodes discussed in Section 2.1.2 plant damage has been observed. However, according to Birmingham et al.[10] sulfur dioxide present in the air, rather than the arsenic is more likely to have caused the damage.

Sodium arsenite is used as a soil sterilant to control vegetation around fence posts, bridge abutments, radar sites, tennis courts, roadways, and other nonagricultural areas.[50]

Sprays used to control weeds have reportedly damaged some crops in adjacent farms.

Organic arsenicals have been synthesized and are used to defoliate cotton plants prior to machine picking, to kill potato vines prior to machine picking, to control aquatic weeds, and to a limited extent to defoliate other plants.[50] Thus, arsenical compounds can be harmful to plants if their use is not properly controlled.

2.4 Effects on Materials

No information has been found on the effects of arsenic on materials. However, arsenicals are used as preservatives. For instance, arsenates are used as wood preservatives,[50] especially against termites. Arsenical paints have been used in the past, but they have been replaced by other materials.

2.5 Environmental Air Standards

No 24-hour maximum atmospheric concentration has been set in the United States for arsenic. A basic 24-hour standard of 3 $\mu g/m^3$ for arsenic and its compounds (as arsenic) has been recommended in the U.S.S.R.[47,49] and Czechoslovakia.

The threshold limit values recommended for industrial workers (8-hour/day exposures) by the American Conference of Governmental Hygienists[28] are 500 $\mu g/m^3$ for arsenic and its compounds (as arsenic) and 200 $\mu g/m^3$ for arsine. The American Industrial Hygiene Association recommends the same values.[5]

Emission standards have been summarized by Stern.[54] These values are listed in Table 4.

TABLE 4

EMISSION STANDARDS FOR ARSENIC IN EFFLUENT AIR OR GASES[54]

Location	Source of Emission	Standard Original Units	$\mu g/m^3$
Czechoslovakia		0.03 kg/hr	
Great Britain	< 5,000 cfm	0.05 grains/ft^3	115,000
Great Britain	> 5,000 cfm	0.02 grains/ft^3	46,000
New South Wales		0.01 grains/ft^3	23,000
Queensland		0.01 grains/ft^3	23,000

3. SOURCES

There are three major sources of arsenic air pollution: smelting of metals, burning of coal, and use of arsenicals as pesticides.

3.1 Natural Occurrence

Arsenic is so widely distributed that traces of it can be found almost everywhere. However, in terms of its concentration in the earth's crust (approximately 5 µg/g), it is one of the less plentiful elements. Virgin soils usually contain a few ppm of arsenic.[28]

Arsenic is present in sea water (10 to 100 ppb) and is concentrated in some aquatic creatures, such as shrimp. These supply man with an appreciable percentage of his total intake of arsenic.[28]

Arsenic is commonly found as the sulfide, arsenide, arsenite, or arsenate. Occasional deposits of elemental arsenic are found, but none are commercially important.[29]

3.2 Production Sources

Virtually all of the arsenic produced is recovered as a by-product in the smelting of lead, copper, and gold ores. The production of white arsenic as a by-product has been so great that the supply usually exceeds the demand.[30] Until this year, the United States' domestic needs have been supplied by the Anaconda Company at Anaconda, Mont. and the American Smelting and Refining Company at Tacoma, Wash.,

supplemented by some imports. However, the Bureau of Mines

reports that the Anaconda Company suspended its sale of

arsenic in 1968.[15]

In order to avoid disclosing company confidential data,

the U.S. consumption or production of white arsenic has not

been reported since 1959. Prior to that, the U.S. consumption

varied between 13,000 and 40,000 short tons per year.[35]

The price of arsenic has declined from approximately 6.5

cents per pound to about 4 cents.[15,30]

One of the problems facing these mining industries has

been the disposal of the large quantities of arsenic they

produce.[28,29] A gold smelter in a small Western town produced

14,600 tons per year, almost enough to supply all our domestic

needs. These industries are also faced with the disposal of

the very poisonous arsenic trioxide.[9,10]

The high volatility of arsenic trioxide (sublimes at

193°C) requires that most arsenic-containing ores be specially

treated to remove arsenic from the exhaust gases. Lead, copper,

and gold ores may contain up to 3 percent arsenic.[28,29]

Arsenic is also a contaminant in some nickel and cadmium ores,

and must be removed to improve the quality of the metal. In

some processes the arsenic is removed chemically, while in

others it is removed by taking advantage of the high volatility

of the arsenic trioxide. Since the latter process provides

a most important source of air pollution, it will be discussed

in detail.

In the commercial production of arsenic, arsenic trioxide
is volatilized during the smelting process and concentrated in
the flue gases. Crude flue gas dust may contain up to 30
percent arsenic trioxide, the balance being oxides of copper
or lead and perhaps of other metals, such as antimony, tin,
and zinc. To upgrade the flue dust, a small amount of pyrite
or galena is mixed with the concentrate and the mixture roasted.
The gases are finally passed through a series of brick cooling
chambers called kitchens. The temperatures of the gas and vapor
are controlled; they enter the first kitchen at approximately
220°C, and by the time the gas and vapor reach the last kitchen,
they have been cooled to 100°C or less. The condensed crude
product is 90 to 95 percent arsenic trioxide. Resublimation
at about 295°C and recondensing in kitchens at 180 to 120°C
produce 99 to 99.9 percent arsenic trioxide.

Even in the smelters where arsenic is not recovered for
commercial use, the tonnages involved are very large. A
reverberatory furnace, for example, may smelt as much as 2,100
tons of charge per day, and in doing so, burn 240 tons of coal.
The furnace would produce about 90,000,000 cubic feet of gas
per day, containing 180 tons of solids. This means that it
would be necessary to dispose of up to 60 tons of arsenic
daily.[28,29]

Two serious arsenic air pollution incidents from
smelters in the United States have been recorded in the
literature, both of which were mentioned in the preceding

section. The first incident took place in Anaconda,
Mont.,[21,22,23] where the emission rate of arsenic trioxide was
59,270 pounds per day (in 2.28 X 10^6 ft^3 of air per day) while
processing 10,000 tons of copper ore per day. This resulted
in polluting the air at the exit of the stack with approximately
450 g/m^3 of arsenic trioxide (estimated by author); the air
was then dispersed over a radius of 15 miles. Although no
atmospheric concentrations are recorded, the edible plants
were contaminated by as much as 482 µg of arsenic trioxide per
gram of plant. It is noteworthy to reiterate that while the
animals eating these plants were killed at distances up to 15
miles from the smelter, no human health complaints are recorded.

The second incident, previously mentioned, occurred in a
small Western town near a gold smelter.[9,10] (The exact location
is not mentioned.) The mine had been intermittently operated
since 1934. In 1962, the operation was resumed with a process
that required converting the sulfur and arsenic to sulfur
dioxide and arsenic trioxide to successfully accomplish
subsequent cyanidation of the gold. The smelter processed
sufficient ore to produce about 100 tons of sulfur dioxide and
40 tons of arsenic trioxide per day. The dust-collecting system
which was intended to collect approximately 90 percent of the
toxic dusts failed to operate as expected and toxic fumes
escaped into the atmosphere.

These two episodes indicate that there is an arsenical air
pollution potential at every smelter which refines arsenical

ores. An example of the arsenical pollution estimated for

Colorado[29] in 1963 is given in Table 5.

TABLE 5

ARSENICAL POLLUTION POTENTIAL FROM MILLS IN COLORADO, 1963[29]

Metal	Average Arsenic Content (Percent)	Ore (short tons)	No. of Mines	Potential Arsenic Pollution (tons)
Zinc	0.07	48,109	8	34
Lead	0.08	19,918	8	16
Copper	0.28	4,169	1	12
Total				62

Arsine is produced whenever hydrogen is emitted from the

dissolution of arsenical metals, such as in metal pickling,

soldering, etching, or in plating processes involving metals

or acids containing arsenic. This constitutes an industrial

hazard but is not an air pollution problem since the quantities

are usually very small.[28,29] Some years ago, arsine was found

to be produced by molds growing on wallpaper which had been

colored with arsenical pigments. Since this procedure is no

longer in use, a hazard no longer exists. However, based on

this evidence an arsenic cycle has been hypothesized in which

arsine is emitted into the atmosphere, oxidized in the presence

of light to form arsenic trioxide deposits on plants, eaten by

animals, and eventually returned to the earth, where it can

be reduced to arsine again.[19]

3.3 Product Sources

3.3.1 Pesticides

Arsenical pesticides constitute the primary use of
arsenic. Until 1945, when DDT made its appearance, the use
of arsenical insecticides was increasing. DDT or other
organic insecticides have almost replaced arsenical insecticides.
Arsenical herbicides were also replaced by organic herbicides
such as 2,4-D acid, which appeared on the market about the
same time as DDT. The production of arsenical pesticides is
summarized in Table 6 in the Appendix.

Several arsenical compounds are currently recommended
by the U.S. Department of Agriculture for insecticides and
herbicides. The compounds listed in Table 7 (Appendix) are
recommended.[56] In 1964, arsenic acid was the largest volume
product in the defoliant-desiccant category. About 5.0 million
pounds were used on about 1.2 million acres of cotton.[45]
Table 8 in the Appendix lists the quantities of arsenical
pesticides used by farmers.[45]

In 1968 the military used cacodylic acid extensively to
control vegetation around encampments. Another domestic
demand for cacodylic acid was for weed control around
industrial sites, rights-of-way, and fence rows. Forest
insect, forest disease, and timber management also used
cacodylic acid, in the amounts of 815 pounds in 1967 and 173
pounds in 1968.[42]

From 1937 to 1940 the U.S. Public Health Service[36] studied the effect of lead arsenate insecticides on orchardists. During this study, they measured the concentration of arsenic and lead to which the orchardists were exposed. These data are summarized in Table 9 in the Appendix. No data were given indicating the pollution area or concentration of arsenic downwind from the various operations. While the authors do not emphasize the air pollution caused by burning, it is noteworthy that the highest concentrations of arsenic were measured in the smoke from burning the pesticide containers. Moreover, the arsenic-to-lead ratios are out of proportion to the other concentrations. The high arsenic concentrations might be explained by the volatility of arsenic trioxide which is formed in the burning process.

3.3.2 Cotton Gins

Arsenicals are used for weed control and as desiccants for cotton plants prior to machine picking.[16,40,50] Thus, the dust emitted from cotton gins contains arsenic. Table 10 in the Appendix shows the concentration of dust and arsenic observed near a cotton gin in Texas and indicates that the arsenic content is approximately 0.03 percent of the particulate. Table 11 (Appendix) shows that the particulate emission concentration from the Stoneville gin may range between 11,000 and 1,258,000 $\mu g/m^3$. On the assumption that the concentration of arsenic is approximately 0.03 percent by

weight of the particulate, one can estimate that the Stoneville
plant would emit a maximum of 400 $\mu g/m^3$ of arsenic in the air
exhausted from the gin and a minimum of 3 $\mu g/m^3$. The range of
air volume exhausted from a gin is 1,410 to 2,120 per cubic
meter per minute or 9,150 to 15,900 per cubic meter per bale of
cotton. This would result in emission rates of up to 580,000
$\mu g/min$ or 6,360,000 $\mu g/bale$ as shown in Table 12 in the Appendix.

In addition to the operation of the cotton gin, the
burning of trash from a cotton gin is also a source of arsenic
pollution.[53] The fieldmen of the Cotton-Classing offices[16]
have indicated that approximately 37 percent of the gins incin-
erate the trash, 58 percent return it to the land, and 5 percent
handle it in some other manner. No estimate was made of the
arsenic emissions from incineration. However, one might expect
that all of the arsenic present in the burning trash would
be converted to volatile arsenic trioxide, which is then emitted
into the atmosphere. Arsenic has been observed in the smoke
from burning cotton burr trash.[53] Adverse effects on trees
and vegetation in areas downwind from cotton gins were observed.
Peach trees were killed by arsenic and pecan trees damaged.
These observations were confirmed by laboratory analyses. The
control of cotton gin dust is not enough; the emissions from
burning trash must also be controlled.

3.4 Other Sources

Coal contains 0.08 to 16 μg of arsenic per gram of
coal.[1] Therefore, the air of most cities contains a small

amount of arsenic. Analyses of the dusts in Hamburg, Germany[1] and Leeds, England,[1] have shown that dusts contained 30 to 230 μg of arsenic per gram. With approximately 409 million tons[51] of coal used each year in the United States, it is possible for 327 to 6,440 tons of arsenic to be emitted into the atmosphere each year. New York City used approximately 5.8 million tons[37] of coal in 1966. This could have resulted in 4.6 to 93 tons of arsenic per year being emitted into New York City air. The average particulate concentration in New York City[37] in 1966 was approximately 125 μg/m³. Assuming that the European values for arsenic in dust hold for New York City, values of 0.004 to 0.029 μg/m³ of air can be calculated. This is in agreement with the value of 0.03 μg/m³ reported in Tables 11 and 12 (Appendix) for New York City.

3.5 Environmental Air Concentrations

Air quality data taken in 1950, 1953, 1961, and 1964 of the arsenic concentration are shown in Tables 13, 14, 15, and 16 (Appendix) respectively. Of the 133 stations reporting in 1964 the year average ranged from below detection to 0.75 μg/m³ and an average for all stations of approximately 0.02 μg/m³. The highest value given in 1964 was 1.40 μg/m³ for quarterly average in El Paso, Texas. The Montana State Board of Health[55] also reported ambient air concentrations for certain cities in Montana for 1961-62 (see Table 17, Appendix). The highest con- centration was 2.5 μg/m³ in the city of Anaconda.

4. ABATEMENT

In general, the removal of particulate material will
control arsenic emissions if the control equipment operates
at a temperature low enough (~100°C) to condense the arsenic
fumes. An electrostatic precipitator has been reported to
reduce the arsenic from 5-17 ppb to 0-4 ppb.[13] Cooling flues,
bag houses, and electrostatic precipitators have been used in
the smelting industry.[19,29] No data have been reported in
the United States on their removal efficiency for arsenic.
However, at a chemical plant in the U.S.S.R., the efficiency
for arsenic removal was greatly improved by using wet vacuum
pumps instead of fabric filters. When the fabric filters
were used, the arsenic content in the air frequently reached
several hundred thousand micrograms per cubic meter. After
the wet scrubbing vacuum pumps were installed, the removal is
reportedly 100 percent effective.[32]

In the cotton industry, removal of particulate material
emitted from cotton gins should control the arsenic emissions.
However, methods need to be developed to control the arsenical
emissions produced by the burning of cotton trash.[50]

5. ECONOMICS

No information has been found on the economic costs of arsenic air pollution or on the costs of its abatement.

The production and consumption data for arsenic have been discussed in Section 3.

6. METHODS OF ANALYSIS

6.1 Sampling Methods

Dusts and fumes of arsenic compounds may be collected by any method suitable for collection of other dusts and fumes; the impinger, electrostatic precipitator, and filters are commonly used. The National Air Sampling Network uses a high-volume filtration sampler.[58]

6.2 Quantitative Methods

Several methods are available for detecting trace amounts of arsenic in dusts; however, only a few of these are quantitative. The chemical methods generally rely on the reduction of arsenical compounds to arsine. The arsine is transported as a gas from the reaction vessel to a second reaction chamber, where it reacts with copper foil (Reinsch's method) or is heated to produce metallic arsenic (Marsh's test), silver nitrate or mercuric chloride (Gutzeit's test), and silver diethyldithiocarbamate (ACGIH tests).[4,12] The National Air Pollution Control Administration uses silver diethyldithiocarbamate in the second reaction vessel. Neutron activation methods are both quantitative and extremely sensitive, but they require a neutron source. They are sensitive to approximately 0.1 µg of arsenic, corresponding to 0.24 µg/m^3 in a 30 cubic foot air sample.[12]

Thompson et al.[58] have reported that the National Air Pollution Control Administration uses atomic absorption to

supplement analyses obtained by the Gutzeit method. The method has a minimum detectable limit of 0.02 $\mu g/m^3$ based on a 2,000 cubic meter air sample.

7. SUMMARY AND CONCLUSIONS

Arsenic is toxic to some degree in most chemical forms. Arsenical compounds may be ingested, inhaled, or absorbed through the skin. Industrial exposure to arsenic has shown that it can produce dermatitis, mild bronchitis, and other upper respiratory tract irritations including perforation of the nasal septum. However, because of the irritant qualities of arsenic, it is doubtful that one could inhale sufficient amounts to produce systemic poisoning.

Skin cancer can result from prolonged therapeutic administration of arsenic. Similar cancers have not been observed among industrial workers. Moreover, lung tumors which resulted from inhaling mixed industrial dusts were often thought to be the result of inhaling arsenic. Recently, this relationship has been questioned because animal experiments have failed to demonstrate that arsenic is a carcinogen. Therefore, the causal relationship between cancer and arsenic is disputed.

Arsenic is poisonous to both animals and plants, but no damage to materials was found.

Two air pollution episodes in the United States have shown that there is an arsenical air pollution potential at every smelter which refines arsenical ores.

Arsenical compounds are used as insecticides and herbi-

cides. Although the use of arsenical pesticides declined sharply after the appearance of DDT and 2,4-D, arsenical compounds are still used as desiccants, herbicides, and sterilants Some undetermined amounts of air pollution take place during spraying and dusting operations with arsenical pesticides. Pollution from cotton gins and cotton trash burning has been cited as an important source of agricultural pollution. While the emission rates from cotton trash burning have not been determined, as much as 1,258,000 $\mu g/m^3$ of exhaust air (580,000 $\mu g/min$) may be emitted during the ginning operation. This produced concentrations of only 0.14 $\mu g/m^3$ of arsenic in the air 150 feet from the gin.

Arsenic is found to the extent of approximately 5 $\mu g/g$ in coal. Therefore, the air of cities which burn coal contains some arsenic. Air quality data from 133 sites monitored by the National Air Sampling Network showed an average daily arsenic concentration of 0.02 $\mu g/m^3$ in 1964.

Control of arsenic emissions requires special attention to the temperature of exhaust gases since arsenic trioxide sublimes at $192^{\circ}C$. For this reason exhaust fumes must be cooled to approximately $100^{\circ}C$ prior to removing them as particulates.

No information has been found on the economic costs of

arsenic pollution or on the costs of its abatement.

Analytical methods are available to determine arsenic at the concentration found in ambient air.

Based on the material presented in this report, further studies are suggested in the following areas:

(1) Determination of the carcinogenic effect of long-term exposure to low concentrations of arsenic in the atmosphere.

(2) Measurement of the concentration of arsenic near smelters, pesticide dusting and spraying operations, cotton gins, and places where cotton trash is burned.

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APPENDIX

TABLE 6

PRODUCTION OF SELECTED PESTICIDES, UNITED STATES
(In Thousands of Pounds)

Item	1939	1945	1950	1955	1960	1965	1966	1967
Calcium arsenate	41,349	25,644	45,348	3,770	6,590	4,192	2,890	2,500
Lead arsenate	59,569	70,522	39,434	14,776	10,062	7,098	7,328	6,000
White arsenic	44,686	48,698	25,546	c	c	c	c	c
Copper sulfate	134,032	251,000	174,600	156,176	116,000	47,272	41,504	33,992
Aldrin-toxaphene group[a]				77,025	90,671	118,832	130,470	120,183
Benzene hexachloride[b]	c	c	76,698	56,051	37,444	c	c	c
DDT	c	33,243	78,150	129,693	164,180	140,785	141,349	103,411
Methyl bromide				9,222	12,659	14,303	16,345	19,665
Methyl parathion					11,794	29,111	35,862	33,344
Parathion			c	5,168	7,434	16,607	19,444	11,361
Nabam					2,978	2,489	2,053	1,361
2,4-D acid	c	917	14,156	34,516	36,185	63,320	68,182	77,139

[a]Includes the chlorinated compounds, aldrin, dieldrin, endrin, chlordane, heptachlor, and toxaphene.

[b]Production of gamma isomer content in BHC was 17.1 million pounds in 1951, 10.7 million in 1955, and 6.9 million in 1960. Data in the table are on a gross basis.

[c]Not available.

TABLE 7

ARSENICAL PESTICIDES RECOMMENDED FOR USE
BY THE DEPARTMENT OF AGRICULTURE, 1968[a]

| Crop | Insecticides | | |
	Insect	Insecticide	Dosage[b]
Currant and Gooseberry	Imported currantworm	Lead arsenate	3-4
Strawberry	Slugs and snails	Calcium arsenate	0.25-0.5
Apple	Apple-and-thorn skeletonizer	Lead arsenate	20
	Apple maggot	Lead arsenate	30
	Bagworm	Lead arsenate	30
	Borer-roundheaded apple tree	Lead arsenate	30
	Cankerworms	Lead arsenate	24
	Codling moth	Lead arsenate	30
	Fall webworm	lead arsenate	20
	Fruitworms	Lead arsenate	30
	Leaf roller, red-banded	Lead arsenate	30
	Plum curculio	Lead arsenate	30
	Tent caterpillars, Eastern	Lead arsenate	30
Grape	Red-banded leaf roller	Lead arsenate	9
Pear	Borer, roundheaded apple tree	Lead arsenate	30
	Fruit-tree leaf roller	Lead arsenate	32
	Fruitworms	Lead arsenate	30
	Plum curculio	Lead arsenate	30
Plum and Prune	Eye-spotted bud moth	Lead arsenate	24
Asparagus	Cutworms	Calcium arsenate	40
Nonagricultural lands	Mosquito larvae	Paris green	0.6-0.15

| Crop | Herbicides | | |
	Purpose	Herbicide	Dosage
Cotton	Preharvest dessicant	Arsenic acid	4.4

[a]Information taken from Suggested Guide for the Use of Insecticides to Control Insects Affecting Crops, Livestock, Households, Stored Products, Forests and Forest Products—1968, Agriculture Handbook No. 331, U.S. Department of Agriculture, U.S. Govt. Printing Office (1968).

[b]Pounds of active ingredient to apply per acre.

APPENDIX

TABLE 8

QUANTITIES OF ARSENICAL PESTICIDES USED BY
FARMERS IN 48 STATES, 1964

Herbicides	Crops[a]	Other[b]	Total
	(Thousands of pounds)		
Sodium, calcium, and zinc arsenites	1,183	56	1,239
Organic arsenicals	1,006	71	1,007
Insecticides			
Lead, calcium, maganesium, and manganese arsenates	7,014	142	7,156
Defoliants and Desiccants			
Arsenic acid	4,973	136	5,109

[a]Includes all crops, pasture, and land in summer fallow.

[b]Includes fence rows, ditch banks, and other usages.

APPENDIX

TABLE 9

ARSENIC AND LEAD CONCENTRATION IN THE AIR OF ORCHARDS[a]
WHERE LEAD ARSENATE WAS USED AS AN INSECTICIDE

| Insecticide Operation | Concentration,[b] $\mu g/m^3$ | | | |
| | Arsenic | | Lead | |
	Average	Range	Average	Range
Mixing insecticide	1,850	20-11,070	5,740	90-46,730
Burning containers	16,670	4,860-26,120	3,580	1,020-7,650
Spraying orchard	140	40-480	450	130-1,430
Thinning fruit	80	10-320	300	40-1,700
Picking fruit	880	260-1,900	2,930	770-7,520
Dumping fruit				
October	60	10-190	190	40-690
December	10	2-20	30	1-110
Sorting and packing (October)	6	3-8	16	7-22

[a] Wenatchee, Wash. apple orchards (1938).

[b] Concentration to which orchardist was exposed.

APPENDIX

TABLE 10

SUSPENDED PARTICULATE AND ARSENIC CONCENTRATIONS IN
THE AIR NEAR COTTON GINS IN WEST TEXAS, 1964[16]

Distance from Gin (ft)	Range of Suspended Particulate Concentrations ($\mu g/m^3$)	Range of Arsenic Concentrations ($\mu g/m^3$)	Arsenic per μg Particulates Ratio X 10^4
150-300[a]	5,000-76,000	0.6-141	1.2-18.5
1,200-1,400[a]	385-187	.07-0.08	3.7-2.1
2,200-8,000[a]	217-42	.10-0.01	4.6-2.4
[b]	67-783		
			Ave. ~0.0003

[a]Measurement downwind from the gin.

[b]Measurement upwind from the gin.

TABLE 11

PARTICULATE EMISSIONS FROM STONEVILLE COTTON GIN[16]
(In Micrograms per Cubic Meter)

Sampling Point	Settling Chamber	Sampling Filter	Total
Unloading fan		820,000	820,000
Six-cylinder cleaner	183,000	91,000	274,000
Stick and bur machine	1,190,000	68,000	1,258,000
Seven-cylinder cleaner[a]		23,000	23,000
Seven-cylinder cleaner[b]		11,000	11,000
Condenser		46,000	46,000

[a]Standard cyclone, 84-inch diameter.

[b]High-efficiency cyclone, 34-inch diameter.

TABLE 12

ESTIMATED RATES OF EMISSION OF ARSENIC FROM COTTON GINS[16]

Emission Rate of Arsenic	Minimum*	Maximum*
μg/min	4,200-6,400	560,000-850,000
μg/bale	27,000-48,000	3,700,000-6,360,000

*Assuming efficiencies based on Stoneville Gin.

TABLE 13

AIR CONCENTRATION OF ARSENIC, 1950[8]

City	Arsenic ($\mu g/m^3$)
Cincinnati	0.06
Charleston	<0.10

APPENDIX

TABLE 14

AIR CONCENTRATION OF ARSENIC IN CITIES
OF UNITED STATES, 1953

City	Population	Average Arsenic Concentration ($\mu g/m^3$)
Los Angeles	Over 2,000,000	0.02
Detroit	" "	.04
Philadelphia	" "	.16
Chicago	" "	.04
New York	" "	.05
Cincinnati	500,000-2,000,000	.02
Kansas City	" "	.02
Portland	" "	.02
Atlanta	" "	<.01
Houston	" "	.01
San Francisco	" "	.01
Minneapolis	" "	.01
Anchorage	<500,000	<.01
Charleston	"	.09
Fort Worth	"	.01
Louisville	"	.02
Near Boonsboro	Nonurban area	.01
" Salt Lake City	"	.03
" Atlanta	"	.01
" Cincinnati	"	<.01
" Portland	"	.04
Maximum urban concentration		1.41

TABLE 15

CONCENTRATION OF ARSENIC IN AIR, FULL-YEAR DATA[2]

Location	Year	No. of Samples	Micrograms per Cubic Meter											Arith Mean	Geom Mean	Std Geom Dev
			Frequency Distribution (Percent)										Max			
			Min	10	20	30	40	50	60	70	80	90				
Arizona																
Phoenix	1961	27				.01	.01	.01	.01	.02	.03	.04	.11	.02	.01	2.63
Delaware																
Wilmington	1961	23				.01	.01	.01	.01	.01	.02	.02	.03	.01	.01	1.69
District of Columbia																
Washington	1961	23				.01	.01	.01	.01	.02	.02	.03	.04	.02	.01	1.79
Georgia																
Atlanta	1962	25						.01	.01	.01	.01	.02	.04	.01	.01	1.81
Illinois																
Chicago	1962	26				.01	.01	.01	.02	.02	.03	.03	.04	.02	.02	1.91
Indiana																
Gary	1962	21			.01	.01	.02	.02	.03	.04	.04	.06	.08	.03	.02	2.29
Parke County	1961	23						.01	.01	.01	.01	.01	.02		.01	1.59
New York																
New York	1961	23	.01	.01	.01	.02	.02	.02	.03	.03	.04	.05	.11	.03	.03	1.85
Pennsylvania																
Clarion County	1961	22					.01	.01	.01	.01	.01	.02	.03	.01	.01	1.56
Pittsburgh	1961	23	.01		.01	.01	.01	.02	.02	.03	.03	.05	.09	.03	.02	1.87
Utah																
Salt Lake City	1961	18				.01	.01	.01	.01	.02	.02	.02	.11	.02	.01	2.3

(continued)

APPENDIX

TABLE 15

CONCENTRATION OF ARSENIC IN AIR, FULL-YEAR DATA[2] (Continued)

Location	Year	No. of Samples	Microgram per Cubic Meter												Std
			Min	Frequency Distribution (Percent)									Max	Arith Mean	Geom Mean	Geom Dev
				10	20	30	40	50	60	70	80	90				
West Virginia Charleston	1962	25		.01	.03	.05	.08	.20	.22	.36	.38	.53	1.0	.24	.11	4.84
United States Urban		12*	<.01										1.0		0.02	

*Number of stations.

APPENDIX

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC IN AIR, 1964[3]

Station Location	Micrograms per Cubic Meter					Station Location	Micrograms per Cubic Meter				
	1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg		1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg
Alabama						Calif. (continued)					
Birmingham	.02	.01	.00	.02	.01	Pasadena	.01	.01	.02	.02	.02
Gadsden	.01	.01	.00	.01	.01	Sacramento	.00	.00	.01	.00	.00
Mobile	.01	.00	.01	.01	.01	San Diego	.00	.00	.00	.00	.00
						San Francisco	.00	.00	.00	.01	.00
Alaska						Santa Ana	.01	.00	.01	.01	.01
Anchorage	.00	.00	.00	.00	.00	Santa Barbara	.00	.00	.00	.01	.00
Arizona											
Grand Canyon[a]	.00	.00	.00	.01	.00	Colorado					
Paradise Valley	.01	.00	.00	.02	.01	Denver	.01	.00	.01	.01	.01
Phoenix	.01	.00	.00	.02	.01	Montezuma County[a]	.00	.00	.00	.00	.00
Tucson	.01	.01	.00	.01	.01						
						Connecticut					
Arkansas						Hartford	.01	.01	.01	.02	.01
Little Rock	.00	.00	.00	.00	.00	New Haven	.01	.01	.01	.01	.01
Montgomery County[a]		.00	.00	.00							
Texarkana	.00	.00	.00	.01	.00	Delaware					
						Kent County	.01	.01	.01	.01	.01
California						Newark			.01	.01	
Bakersfield	.01	.00	.00	.01	.01	Wilmington	.02	.02	.01	.05	.03
Burbank	.01	.01	.02	.02	.02						
Humboldt County[a]	.00	.00	.00	.00	.00	District of Columbia					
Los Angeles	.01	.00	.01	.01	.01	Washington	.01	.01	.01	.02	.01
Monterey	.00	.00	.00	.00	.00						
Oakland	.01	.00	.01	.02	.01						

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC IN AIR, 1964[3] (Continued)

Station Location	Micrograms per Cubic Meter					Station Location	Micrograms per Cubic Meter				
	1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg		1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg
Florida						Indiana (continued)					
Florida Keys[a]	.00	.00		.00		Dunes State Park	.01	.01	.00	.01	.01
Jacksonville	.00	.00	.00	.00	.00	East Chicago	.03	.05	.04	.05	.04
Orlando	.00	.00		.00		Evansville	.02	.01	.01	.01	.01
Tampa	.00	.01	.01			Fort Wayne	.03	.01	.01	.01	.02
						Gary	.04	.02	.03	.02	.03
Georgia						Hammond[b]	.03	.03	.02	.02	.03
Atlanta	.01	.01	.01	.01	.01	Indianapolis	.04	.01	.01	.02	.02
						Ogden Dunes	.01	.00	.01	.02	.01
Hawaii						Portage	.01	.01	.01	.01	.01
Honolulu	.00	.00	.00	.00	.00	Parke County[a]	.01	.00	.01	.01	.01
						Lafayette	.01	.00	.01	.01	.01
Idaho											
Boise	.00	.00	.00	.01	.00	Iowa					
Buite County[a]	.00	.00	.00	.01	.00	Davenport		.01	.01	.01	
						Delaware County[a]	.00	.00	.00	.00	.00
Illinois						Des Moines	.01	.01	.00	.01	.01
Chicago	.04	.02	.03	.02	.03	Dubuque	.01	.01	.01	.02	.01
Cicero	.03	.02	.02	.02	.02						
East St. Louis[b]	.03	.01	.04	.02	.03	Kansas					
Moline	.02	.01	.00	.01	.01	Kansas City	.01	.00	.00	.01	.01
Peoria	.02	.01	.01	.03	.02	Wichita	.01	.01	.00	.01	.01
Rock Island	.01	.01	.01	.01	.01						
Springfield			.00	.01		Kentucky					
						Ashland	.04	.03	.02	.04	.03
Indiana						Covington	.01	.01	.02	.01	.01
Beverly Shores	.01	.00	.01	.01	.01	Louisville	.01	.01	.01	.02	.01
Dunes PCL Post	.01	.01	.01	.02	.01						

(continued)

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC AIR, 1964[3] (Continued)

Station Location	Micrograms per Cubic Meter					Station Location	Micrograms per Cubic Meter				
	1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg		1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg
Louisiana						Mississippi					
Baton Rouge	.01	.00	.00	.01	.01	Jackson	.00	.00	.00		
Lake Charles	.00	.00	.00	.00	.00	Jackson County[a]		.00	.00	.00	
New Orleans	.00	.00	.00	.00	.00						
						Missouri					
Maine						Kansas City	.01	.00	.00	.01	.01
Acadia National Park[a]	.00	.00	.00	.00	.00	St. Louis	.01	.01	.03	.01	.02
Portland	.01	.00	.00	.01	.00	Shannon County[a]	.00	.00	.00	.00	.00
Maryland						Montana					
Baltimore	.02	.01	.02	.03	.02	Glacier National Park[a]	.00		.00	.00	
Calvert	.00	.00	.00	.01	.00	Helena	.02	.00	.02	.05	.02
Massachusetts						Nebraska					
Boston	.02	.01	.01	.01	.01	Omaha	.04	.02	.03	.02	.03
Springfield	.02	.01	.01	.02	.02	Thomas County[a]	.00	.00	.00	.00	.00
Michigan						Nevada					
Detroit	.03	.03	.02	.02	.03	Las Vegas	.00	.00	.00	.00	.00
Wyandotte	.03	.03	.03			White Pine County[a]	.00	.00	.00	.00	.00
Minnesota						New Hampshire					
Duluth	.01	.02	.00	.01	.01	Concord	.01	.00	.00	.00	.00
Minneapolis	.01	.01	.00	.00	.01	Coos County[a]	.00	.00	.00	.00	.00
Moorhead	.01	.00	.00	.01	.01						
St. Paul	.01	.00	.00	.01	.01						

(continued)

APPENDIX

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC IN AIR, 1964[3] (Continued)

Station Location	Micrograms per Cubic Meter					Station Location	Micrograms per Cubic Meter				
	1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg		1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg
New Jersey						North Dakota					
Bridgeton			.00	.02		Bismarck	.00	.00	.00	.01	.00
Camden	.03	.02	.02	.04	.03	Fargo	.01	.00	.00	.01	.01
Glassboro	.01	.01	.00	.02	.01	Ward County[a]	.00	.00	.00	.00	.00
Jutland	.01	.01		.01							
Marlton			.02	.02		Ohio					
Newark	.02	.01	.02	.02	.02	Akron	.04	.04	.04	.05	.04
New Brunswick	.01	.03	.04			Cincinnati	.03	.02	.02	.03	.03
Pemberton		.01	.01	.02		Cleveland	.04	.02	.02	.04	.03
Perth Amboy		.10	.03	.10		Columbus	.02	.02	.02	.03	.02
Princeton		.01	.01	.02		Dayton	.02	.04	.01	.04	.03
Trenton	.01		.01	.02		Lorain	.03	.02	.01	.02	.02
						Steubenville	.04	.07	.08	.15	.09
New Mexico						Toledo	.06	.18	.03	.10	.09
Albuquerque	.01	.00	.00	.01	.01	Youngstown	.07	.05	.02	.04	.05
Colfax County[a]	.00	.00	.00	.00	.00						
						Oklahoma					
New York						Cherokee County[a]	.00	.00	.00	.00	.00
Cape Vincent[a]	.01	.01	.01	.01	.01	Oklahoma City	.01	.00	.00	.01	.01
New York	.03	.02	.03	.05	.03	Tulsa	.01	.00	.00	.01	.01
North Carolina						Oregon					
Cape Hatteras[a]	.00	.00	.00	.00	.00	Curry County[a]		.00	.00	.00	
Charlotte	.01	.00	.00	.01	.01	Portland	.00	.02	.01	.01	.01
Fayetteville	.00	.00	.00	.01	.00						
Winston-Salem	.02	.00	.00	.02	.01						

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC IN AIR, 1964[3] (Continued)

Station Location	Micrograms per Cubic Meter					Station Location	Micrograms per Cubic Meter				
	1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg		1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg
Pennsylvania						Tennessee					
Bethlehem[b]	.02	.01	.01	.01	.01	Chattanooga	.04	.02	.01	.02	.02
Clarion County[a]		.01	.01	.02		Memphis	.02	.01	.01	.01	.01
Eagleville			.01	.02		Nashville	.01	.01	.01	.01	.01
Embreeville		.01	.00	.02							
Lancaster	.02	.01	.01	.02	.02	Texas					
Philadelphia	.04	.02	.13	.05	.06	Arkansas County[a]	.00	.00	.00	.00	.00
Pittsburgh	.03	.03	.03	.06	.04	Dallas	.02	.01	.02	.03	.02
						El Paso	.50	.60	.50	1.40	.75
Puerto Rico						Houston	.01	.00	.01	.01	.01
Guayanilla	.00	.00	.00	.00	.00	Laredo		.01	.00		.01
Ponce	.00	.00	.00	.00	.00	San Antonio	.00	.00	.00	.00	.00
San Juan	.00	.00	.00	.00	.00	Texarkana	.00	.00	.00	.00	.00
						Waco	.00	.00	.01	.01	.01
Rhode Island											
Providence	.01	.01	.01	.01	.01	Utah					
Washington County[a]	.01	.00	.00	.01	.01	Ogden	.01	.00	.01	.02	.01
						Salt Lake City	.02	.01	.00	.03	.02
South Carolina											
Columbia	.01		.01	.01		Vermont					
Richland County[a]	.00	.00	.00	.00	.00	Burlington	.01	.01	.01	.01	.01
						Orange County[a]	.00	.00	.00	.01	.00
South Dakota											
Black Hills	.00	.00	.00	.01	.00						
Sioux Falls	.01	.00	.00	.00	.00						

TABLE 16

URBAN AND NONURBAN CONCENTRATION OF ARSENIC IN AIR, 1964[3] (Continued)

Station Location	Micrograms per Cubic Meter					Station Location	Micrograms per Cubic Meter				
	1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg		1st Qtr	2nd Qtr	3rd Qtr	4th Qtr	Yrly Avg
Virginia						Wyoming					
Danville	.01	.01	.00	.01	.01	Cheyenne	.00	.00	.00	.00	.00
Norfolk	.01	.01	.01	.01	.01	Yellowstone Park[a]	.01	.00	.00	.02	.01
Shenandoah Park[a]	.00	.00	.00	.00	.00						
Washington						United States	133 Stations				.02
Seattle	.06	.08	.03	.14	.08						
Tacoma	.03	.02	.04	.18	.07						
West Virginia											
Charleston	.22	.10	.24	.36	.25						
Huntington	.03	.02	.01	.03	.02						
Parkersburg	.02	.01	.02	.02	.02						
Weirton	.01	.03	.05	.05	.04						
Wheeling[b]	.04	.02	.02	.02	.03						
Wisconsin											
Door County[a]	.00	.00	.00	.01	.00						
Eau Claire	.01	.01	.00	.01	.01						
Milwaukee	.02	.02	.01	.02	.02						
Racine	.01	.01	.01	.02	.01						
Superior	.01	.01	.00	.01	.01						

[a] Nonurban Areas

[b] 1963

APPENDIX

TABLE 17

CONCENTRATION OF ARSENIC IN THE AIR OF MONTANA CITIES, 1961-62[55]

					Arsenic, $\mu g/m^3$ (Average)								
City	Maximum	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May	June
Anaconda	2.50	0.42	0.68	0.51	0.33	0.54		0.64	0.56	0.33	0.27	0.18	0.46
Butte	0.55	.05	.05	.06	.11	.09	0.05	.09	.04	.06	.09	.04	.09
Great Falls	0.11	.01	.02	.03	.01	.02	.01	.00	.00	.01	.01	.00	.00
Helena	0.16	.00	.01	.02	.02	.03	.06	.02	.02	.03	.04	.01	.00
Missoula	0.15	.00	.00	.01	.00	.02	.01	.02	.01	.02	.00	.00	.00

TABLE 18. PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS[33]

Compound	Properties	Toxicity	Uses
Arsenic trioxide (White arsenic) As_2O_3 75.74%	m.p. 313° b.p. 465° sublimes at 193°	Most forms of arsenic are highly toxic. Acute symptoms following ingestion relate to irritation of the gastrointestinal tract: nausea, vomiting, diarrhea which can progress to shock and death. Chronic poisoning can result in exfoliation and pigmentation of skin, herpes, polyneuritis, altered hematopoiesis, degeneration of liver and kidneys LD_{50} for rats 13,000 µg/kg for man 0.0-0.5 g Chronic: Under 0.1 g Phytotoxicity: very toxic to plants.	It is the primary material for all arsenic compounds. Used in the manufacture of glass, Paris green, enamels, weed killers, textile mordants, metallic arsenic; for preserving hides, killing rodents, insects; in sheep dips and weed killers. Med. use: formerly for dermatitides, chronic bronchitis, asthma, anemia, topically for skin neoplasms. Vet. use: for pulmonary emphysema, chronic coughs, anemia, general debility, chronic nonparasitic skin disease
Arsenic disulfide As_2S_2 70.03%	m.p. 320° b.p. 565°	Most forms of arsenic are highly toxic See Arsenic trioxide	As pigment in painting, in fireworks as blue fire and to give an intense white flame; to manufacture shot; for calico printing and dyeing, tanning and depilating hides
Arsenic acid H_3AsO_4 52.78%		Poisonous. See above. LD_{50} in rabbits: 8,000 µg/kg	In the manufacture of arsenates

(continued)

TABLE 18. PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

Compound	Properties	Toxicity	Uses
Arsenic hemiselenide As_2Se 65.49%	Decomposes	Same as Arsenic trioxide	In manufacture of glass
Arsenic pentafluoride AsF_5 44.09%	m.p. -79.8° b.p. -53.2°	Same as Arsenic trioxide Also, external contact— liquid or vapor—causes severe irritation of eyes	
Arsenic pentaselenide As_2Se_5 27.51%	Decomposes	Poisonous See Arsenic trioxide	
Arsenic pentoxide As_2O_5 65.20%		Poisonous. Keep well closed. LD i.v. in rabbits 8,000 µg/kg	In manufacture of colored glass, in adhesives for metals; in wood preservatives; in weed control; as fungicide
Arsenic tribromide $AsBr$ 23.83%	m.p. 31.1°	See Arsenic trioxide Intensely poisonous Also: highly irritating to skin, eyes, mucous membranes, respiratory tract	
Arsenic trichloride $AsCl_3$ 41.32%	m.p. -16° b.p. 130.21°	Extremely toxic. Readily liberates hydrochloric acid, which is a strong irritant. Also see Arsenic trioxide	In the ceramic industry; in the synthesis of chlorine-containing arsenicals (i.e., chloro derivatives of arsenic)

(continued)

TABLE 18.   PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

Compound	Properties	Toxicity	Uses
Arsenic trifluoride AsF_3 56.79%	m.p. $-8.5°$ b.p. $63°$	Extremely toxic. See Arsenic trioxide	
Arsenic triiodide AsI_3 16.44%	m.p. $140.9°$ b.p. $\sim400°$	See Arsenic trioxide	Med. use: formerly internally for chronic dermatitides (including syphilitic), various chronic arthroses, and certain cases of lymphadenitis, and topically as stimulant in dermatitis
Arsenic triselenide As_2Se_3 38.74%	m.p. $260°$	See Arsenic pentafluoride	
Arsenic trisulfide As_2S_3 60.90%	m.p. $300-325°$	See Arsenic trioxide	In manufacture of glass, particularly infrared transmitting glass; in manufacture of oil cloth, linoleum; in electrical semiconductors, photoconductors; as pigment; for depilating hides, in pyrotechnics
Arsenious acid solution Prepared with 1 g As_2O_3 5 ml dil HCl and water to 100 ml		See Arsenic trioxide	Med. use: has been used for blood dyscrasias. Vet. use: in certain blood diseases, anemias, and skin disorders

(continued)

TABLE 18.   PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

Compound	Properties	Toxicity	Uses
Arsine AsH_3 96.12%	m.p. -117° b.p. -625°	Injurious in 1:20,000 dilution; a few inhalations may be fatal. Death from anoxia or pulmonary edema. Hemolytic anemia in non-fatal cases	For chemical analyses
Arsonoacetic acid $H_2O_3AsCH_2COOH$ 40.72%	m.p. 152°		Vet. use: disodium salt used to treat anaplasmosis (babesiasis); as general stimulant in nervous disease; for eclampsia of bitches, and with adjuncts in chronic eczema and follicular mange
Calcium arsenate $Ca_3(AsO_4)_2$ 37.64%	Powder	See Arsenic trioxide Poisonous LD_{50} 35-100 mg/kg for various animals Phytotoxicity: causes leaf and fruit damage to stone fruit trees	As insecticide, particularly against insects destructive to plants; as molluscicide
Lead arsenate $PbHAsO_4$ 21.58%	White heavy powder Decomposes above $280^{\circ}C$	Poisonous LD_{50} orally in rats 80,000 µg/kg LD_{50} animals 10,000-50,000 µg/kg man 0.1-0.5 gm	As constituent of various insecticides for larvae of gypsy moth, boll weevil, etc. Vet. use: has been reported useful for tapeworms of cattle, goats, sheep

TABLE 18.   PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

Compound	Properties	Toxicity	Uses
Cupric acetoarsenite (Paris Green) $(CuOAs_2O_3)_3 \cdot Cu(C_2H_3O_2)_2$ 44.34%	Emerald green powder Decomposes on prolonged heating in water	Poisonous. Gastric disturbances, tremors, or muscular cramps, and peripheral neuritis, local effects on the skin, mucous membranes and conjunctivae	As insecticide, wood preservative; as pigment, particularly for ships and submarines
Cupric arsenite (Scheele's green) $CuHAsO_3$ 39.96%	Yellowish-gray powder	Poisonous	As pigment, wood preservative, insecticide, fungicide, rodenticide
Potassium arsenate KH_2AsO_4 41.61%	Decomposes	Poisonous	In textile, tanning, and paper industries. In insecticidal formulations (especially fly paper)
Potassium arsenite $KAsO_2 \cdot HAsO_2$ 59.00%	Decomposes	Very poisonous. Keep well closed. LD_{50} orally in rats: 14,000 µg/kg	In manufacture of mirrors to reduce the silver salt to metallic silver
Potassium arsenite solution Made from arsenic trioxide 10 g; potassium bicarbonate 7.6 g; alcohol 30 ml; distilled water to 1 liter		Very poisonous See Arsenic trioxide	Med. use: has been used in chronic myelogenous leukemia, chronic dermatitides. Vet. use: for pulmonary emphysema, chronic coughs, anemia, general debility, chronic nonparasitic skin diseases

(continued)

TABLE 18.   PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

Compound	Properties	Toxicity	Uses
Methanearsenic acid $CH_3AsO(OH)_2$ 53.53%	m.p. 161°	See Arsenic trioxide	Disodium salt, as herbicide. Med. use: has been used in anemia, leukemia, psoriasis
Cacodylic acid $(CH_3)As(O)OH$ 54.29%	m.p. 195-196°	Poisonous LD s.c. for dogs 1.0 g/kg	As herbicide. Formerly for various skin diseases
Sodium arsenate dibasic Na_2HAsO_4 40.29%	m.p. 57°	Poisonous but less so than arsenite	The technical grade, about 98% pure, is used in dyeing with Turkey-red oil and in printing fabrics. Med. use: formerly as "alterative," anthelmintic. Has been used for chronic skin diseases. Vet. use: see Arsenic trioxide
Sodium arsenite $NaAsO_2$ 57.67%		Very poisonous. Keep well closed See Arsenic trioxide	The technical grade, 90-95% pure, is used in manufacture of arsenical soap for use on skin, for treating vines against certain scale disease, as insecticide (especially for termites). Vet. use: topically against ticks of ruminants
Chloroarsenol	m/p. 115°		Formerly as tonic

TABLE 18.   PROPERTIES, TOXICITY AND USES OF SOME ARSENIC COMPOUNDS (Continued)

Compound	Properties	Toxicity	Uses
Disodium methyl arsenate $CH_3AsO_3Na_2-$ $6H_2O$	m.p. above 300°	Oral to mammals: test animals tolerate well above 50,000 µg per kilo body weight LD_{50} to rats 20% solution of agri. grade is 600,000 µg/kg	As weedkiller (crabgrass); for some control over silver or goose grass, knotweed, and chickweed
Zinc arsenate $Zn(AsO_4)_2$ 30%		Oral to mammals See Arsenic trioxide	As insecticide