

**HIGHLIGHTS**

- Life-threatening effects on CNS, blood vessels, kidney, liver

**Signs and Symptoms:**

- In acute cases, garlic odor of the breath and feces, metallic taste in mouth, adverse GI symptoms
- In chronic cases, muscle weakness, fatigue, weight loss, hyperpigmentation, hyperkeratosis, Mees lines

**Treatment:**

- GI decontamination
- Chelation therapy  
Dimercaprol (BAL) or DMPS to accelerate arsenic excretion

# Arsenical Pesticides

Many arsenic compounds have been discontinued in the United States as a result of government regulations. However, arsenical pesticides are still widely available in some countries, and many homes and farms have leftover supplies that continue to represent some residual risk.

Arsine gas is discussed separately on page 132.

## Toxicology

Arsenic is a natural element that has both metal and nonmetal physical/chemical properties. In some respects, it resembles nitrogen, phosphorus, antimony, and bismuth in its chemical behavior. In nature, it exists in elemental, trivalent (-3 or +3), and pentavalent (+5) states. It binds covalently with most nonmetals (notably oxygen and sulfur) and with metals (for example, calcium and lead). It forms stable trivalent and pentavalent organic compounds. In biochemical behavior, it resembles phosphorus, competing with phosphorus analogs for chemical binding sites.

Toxicity of the various arsenic compounds in mammals extends over a wide range, determined in part by the unique biochemical actions of each compound, but also by absorbability and efficiency of biotransformation and disposition. Overall, arsines present the greatest toxic hazard, followed closely by arsenites (inorganic trivalent compounds). Inorganic pentavalent compounds (arsenates) are somewhat less toxic than arsenites, while the organic (methylated) pentavalent compounds represent the least hazard of the arsenicals that are used as pesticides.<sup>1</sup>

The pentavalent arsenicals are relatively water soluble and absorbable across mucous membranes. Trivalent arsenicals, having greater lipid solubility, are more readily absorbed across the skin.<sup>2</sup> However, poisonings by dermal absorption of either form have been extremely rare. Ingestion has been the usual basis of poisoning; gut absorption efficiency depends on the physical form of the compound, its solubility characteristics, the gastric pH, gastrointestinal motility, and gut microbial transformation. Arsine exposure occurs primarily through inhalation, and toxic effects may also occur with other arsenicals through inhalation of aerosols.

Once absorbed, many arsenicals cause toxic injury to cells of the nervous system, blood vessels, liver, kidney, and other tissues. Two biochemical mecha-

nisms of toxicity are recognized: (1) reversible combination with thiol groups contained in tissue proteins and enzymes, and (2) substitution of arsenic anions for phosphate in many reactions, including those critical to oxidative phosphorylation. Arsenic is readily metabolized in the kidney to a methylated form, which is much less toxic and easily excreted. However, it is generally safest to manage cases of arsenical pesticide ingestion as though all forms are highly toxic.

The unique toxicology of arsine gas is described later in this chapter.

## Signs and Symptoms of Poisoning

Manifestations of acute poisoning are distinguishable from those of chronic poisoning.

**Acute arsenic poisoning:** Symptoms and signs usually appear within one hour after ingestion, but may be delayed several hours. Garlic odor of the breath and feces may help to identify the toxicant in a severely poisoned patient. There is often a metallic taste in the mouth. Adverse gastrointestinal (GI) effects predominate, with vomiting, abdominal pain, and rice-water or bloody diarrhea being the most common. Other GI effects include inflammation, vesicle formation and eventual sloughing of the mucosa in the mouth, pharynx, and esophagus.<sup>3</sup> These effects result from the action of an arsenical metabolite on blood vessels generally, and the splanchnic vasculature in particular, causing dilation and increased capillary permeability.

The central nervous system is also commonly affected during acute exposure. Symptoms may begin with headache, dizziness, drowsiness, and confusion. Symptoms may progress to include muscle weakness and spasms, hypothermia, lethargy, delirium, coma, and convulsions.<sup>1</sup> Renal injury is manifest as proteinuria, hematuria, glycosuria, oliguria, casts in the urine, and, in severe poisoning, acute tubular necrosis. Cardiovascular manifestations include shock, cyanosis, and cardiac arrhythmia,<sup>4,5</sup> which are due to direct toxic action and electrolyte disturbances. Liver damage may be manifested by elevated liver enzymes and jaundice. Injury to blood-forming tissues may cause anemia, leukopenia, and thrombocytopenia.

Death usually occurs one to three days following onset of symptoms and is often the result of circulatory failure, although renal failure also may contribute.<sup>1</sup> If the patient survives, painful paresthesias, tingling, and numbness in the hands and feet may be experienced as a delayed sequela of acute exposure. This sensorimotor peripheral neuropathy, which may include muscle weakness and spasms, typically begins 1-3 weeks after exposure.<sup>6</sup> The muscle weakness may be confused with Guillain-Barre syndrome.<sup>7</sup>

**Chronic arsenic poisoning** from repeated absorption of toxic amounts generally has an insidious onset of clinical effects and may be difficult to diagnose. Neurologic, dermal, and nonspecific manifestations are usually more prominent than the gastrointestinal effects that characterize acute poisoning. Muscle

## Commercial Products

(Many have been discontinued)

arsenic acid  
 Hi-Yield Dessicant H-10  
 Zotox  
 arsenic trioxide  
 cacodylic acid (sodium cacodylate)  
 Bolate  
 Bolls-Eye  
 Bophy  
 Dilie  
 Kack  
 Phytar 560  
 Rad-E-Cate 25  
 Salvo  
 calcium acid methane arsonate (CAMA)  
 Calar  
 Super Crab-E-Rad-Calar  
 Super Dal-E-Rad  
 calcium arsenate  
 Spra-cal  
 tricalcium arsenate  
 Turf-Cal  
 calcium arsenite  
 London purple  
 mono-calcium arsenite  
 copper acetoarsenite  
 Emerald green  
 French green  
 Mitis green  
 Paris green  
 Schweinfurt green  
 copper arsenite (acid copper arsenite)  
 disodium methane arsonate  
 Ansar 8100  
 Arrhenal  
 Arsinyl  
 Crab-E-Rad  
 Di-Tac  
 DMA  
 DSMA  
 Methar 30  
 Sodar  
 Weed-E-Rad 360  
 lead arsenate  
 Gypsine  
 Soprabel  
 methane arsonic acid (MAA)  
 monoammonium methane arsonate (MAMA)  
 monosodium methane arsonate (MSMA)  
 Ansar 170

(Continued on the next page)

## Commercial Products

(Continued)

Arsonate Liquid  
Bueno 6  
Daconate 6  
Dal-E-Rad  
Drexar 530  
Herbi-All  
Merge 823  
Mesamate  
Target MSMA  
Trans-Vert  
Weed-E-Rad  
Weed-Hoe  
sodium arsenate  
disodium arsenate  
Jones Ant Killer  
sodium arsenite  
Prodalumnol Double  
Sodanit  
zinc arsenate

weakness and fatigue can occur, as can anorexia and weight loss. Hyperpigmentation is a common sign, and tends to be accentuated in areas that are already more pigmented, such as the groin and areola. Hyperkeratosis is another very common sign, especially on the palms and soles.<sup>8,9</sup> Subcutaneous edema of the face, eyelids, and ankles, stomatitis, white striations across the nails (Mees lines), and sometimes loss of nails or hair are other signs of chronic, continuous exposure.<sup>1,9</sup> On occasion, these hyperkeratotic papules have undergone malignant transformation.<sup>8</sup> Years after exposure, dermatologic findings include squamous cell and basal cell carcinoma, often in sun-protected areas.

Neurologic symptoms are also common with chronic exposure. Peripheral neuropathy, manifested by paresthesia, pain, anesthesia, paresis, and ataxia, may be a prominent feature. It may often begin with sensory symptoms in the lower extremities and progress to muscular weakness and eventually paralysis and muscle wasting. Although less common, encephalopathy can develop with speech and mental disturbances very much like those seen in thiamine deficiency (Wernicke's syndrome).

Other organ systems are affected with arsenic toxicity. Liver injury reflected in hepatomegaly and jaundice may progress to cirrhosis, portal hypertension, and ascites. Arsenic has direct glomerular and tubular toxicity resulting in oliguria, proteinuria, and hematuria. Electrocardiographic abnormalities (prolongation of the Q-T interval) and peripheral vascular disease have been reported. The latter includes acrocyanosis, Raynaud's phenomenon, and frank gangrene.<sup>1,10</sup> Hematologic abnormalities include anemia, leukopenia, and thrombocytopenia.<sup>1</sup> Late sequelae of protracted high intakes of arsenic include skin cancer as described above and an increased risk of lung cancer.<sup>1,8</sup>

## Confirmation of Poisoning

Measurement of 24-hour urinary excretion of arsenic (micrograms per day) is the most common way to confirm excessive absorption and is the preferred method to follow serial levels and evaluate chronic exposure.<sup>1,11</sup> Spot urine arsenic analysis expressed as a ratio with urinary creatinine is the recommended method to evaluate occupational exposures.<sup>12</sup> Methods to determine blood arsenic concentration are available; however blood levels tend to poorly correlate with exposure except in the initial acute phase.<sup>11,13</sup> Special metal-free acid-washed containers should be used for sample collection. Arsenic excretion above 100 mcg per day should be viewed with suspicion and the test should be repeated.

Excretions above 200 mcg per day reflect a toxic intake, unless seafood was ingested.<sup>11,13,14,15</sup> Diets rich in seafood, primarily shellfish in the previous 48 hours, may generate 24-hour urine excretion levels as high as 200 mcg per day and sometimes more.<sup>3,14</sup> The majority of marine arsenic that is excreted is in the methylated form (arsenobetaine) and is not considered acutely toxic. How-

ever, a recent study supports that some of the arsenic released from mussels may contain higher amounts of arsenic trioxide than previously thought.<sup>14</sup> Urinary arsenic may be speciated into inorganic and organic fractions to help determine the source of the exposure and to help guide treatment.

Concentrations of arsenic in blood, urine, or other biologic materials can be measured by either wet or dry ashing, followed by colorimetric or atomic absorption spectrometric analysis. The latter method is preferred. Blood concentrations in excess of about 100 mcg per liter probably indicate excessive intake or occupational exposure, provided that seafood was not ingested before the sample was taken.<sup>3,11,13,15</sup> Blood samples tend to correlate with urine samples during the early stages of acute ingestion,<sup>11</sup> but because arsenic is rapidly cleared from the blood, the 24-hour urine sample remains the preferred method for detection and for ongoing monitoring.<sup>1,11,13</sup> Hair has been used for evaluation of chronic exposure. Levels in unexposed people are usually less than 1 mg/kg; levels in individuals with chronic poisoning range between 1 and 5 mg/kg.<sup>15</sup> Hair samples should be viewed with caution because external environmental contamination such as air pollution may artificially elevate arsenic levels.

Special tests for arsine toxicosis are described on page 132 under “Arsine Gas.”

## **Treatment**

The following discussion applies principally to poisonings by arsenicals in solid or dissolved form. Treatment of poisoning by arsine gas requires special measures described below on page 132.

**1. Skin decontamination.** Wash arsenical pesticide from skin and hair with copious amounts of soap and water. Flush contaminant from eyes with clean water. If irritation persists, specialized medical treatment should be obtained. See Chapter 2.

**2. Gastrointestinal decontamination.** If arsenical pesticide has been ingested within the first hour of treatment, consideration should be given to GI decontamination, as outlined in Chapter 2. Because poisoning by ingested arsenic almost always results in profuse diarrhea, it is generally not appropriate to administer a cathartic.

**3. Intravenous fluids.** Administer intravenous fluids to restore adequate hydration, support urine flow, and correct electrolyte imbalances. Monitor intake/output continuously to guard against fluid overload. If acute renal failure occurs, monitor blood electrolytes regularly. Blood transfusions and oxygen by mask may be needed to combat shock.

**4. Cardiopulmonary monitoring.** Monitor cardiac status by ECG to detect ventricular arrhythmias including prolonged Q-T interval and ventricular tachycardia, and toxic myocardiopathy (T wave inversion, long S-T interval).

**5. Chelation therapy.** Administration of Dimercaprol (BAL) is usually indicated in symptomatic arsenic poisonings, although DMPS, where available, may prove to be a better antidote. The following dosage schedule has proven to be effective in accelerating arsenic excretion.

Monitor urinary arsenic excretion while any chelating agent is being administered. When 24-hour excretion falls below 50 mcg per day, it usually is advisable to discontinue the chelation therapy.

### RECOMMENDED INTRAMUSCULAR DOSAGE OF BAL (DIMERCAPROL) IN ARSENIC POISONING

	Severe Poisoning	Mild Poisoning
1 <sup>st</sup> day	3.0 mg/kg q4h (6 injections)	2.5 mg/kg q6h (4 injections)
2 <sup>nd</sup> day	3.0 mg/kg q4h (6 injections)	2.5 mg/kg q6h (4 injections)
3 <sup>rd</sup> day	3.0 mg/kg q6h (4 injections)	2.5 mg/kg q12h (2 injections)
Each of the following days for 10 days, or until recovery	3.0 mg/kg q12 hr (2 injections)	2.5 mg/kg qd (1 injection)

BAL is provided as a 100 mg/mL solution in oil. Dosages in the table are in terms of BAL itself, not of the solution. Dosages for children are consistent with the "Mild Poisoning" schedule and can be between 2.5 and 3.0 mg/kg per dose.<sup>16</sup>

**Caution:** Disagreeable side effects often accompany the use of BAL: nausea, headache, burning and tingling sensations, sweating, pain in the back and abdomen, tremor, restlessness, tachycardia, hypertension, and fever. Coma and convulsions occur at very high dosage. Sterile abscesses may form at injection sites. Acute symptoms usually subside in 30-90 minutes. Antihistamine drugs or an oral dose of 25-50 mg ephedrine sulfate or pseudoephedrine provide relief. These are more effective if given a few minutes before the injection of BAL. BAL may potentially have other adverse effects. In rabbits, treatment of arsenite exposure with BAL increased brain arsenic levels.<sup>17</sup>

**6. Oral treatments.** After the gastrointestinal tract is reasonably free of arsenic, oral administration of d-penicillamine, Succimer (DMSA), or DMPS should probably replace BAL therapy. However, d-penicillamine has demonstrated limited effectiveness for arsenic exposure in experimental models.<sup>18</sup>

**Dosage of d-penicillamine:**

- *Adults and children over 12 years:* 0.5 g every 6 hours, given 30-60 minutes before meals and at bedtime for about 5 days.
- *Children under 12 years:* 0.1 g/kg body weight, every 6 hours, given 30-60 minutes before meals and at bedtime for about 5 days. Not to exceed 1.0 g per day.

**Caution:** Adverse reactions to short-term therapy are rare. However, **persons allergic to penicillin should not receive d-penicillamine** as they may suffer allergic reactions to it.

Succimer (DMSA) has been shown to be an effective chelator of arsenic, though it is not labeled for this indication.<sup>19</sup> In Europe, DMPS has been used successfully in treatment of arsenic poisoning. In light of the lack of effectiveness of d-penicillamine, coupled with the low toxicity and high therapeutic index of DMPS and DMSA, it appears that the latter two agents may be the preferred method for chronic toxicity or when oral chelation is acceptable.<sup>18,19</sup>

**Dosage of DMSA (Succimer):**

- *Adults and Children:* 10 mg/kg every 8 hours for 5 days, followed by 10 mg/kg every 12 hours for an additional 14 days. (Maximum 500 mg per dose). Should be given with food.

**Dosage of DMPS:**

- *Adults:* 100 mg every 8 hours for 3 weeks to 9 months.

**7. Hemodialysis.** Extracorporeal hemodialysis, used in combination with BAL therapy, has limited effectiveness in removing arsenic from the blood. Hemodialysis is clearly indicated to enhance arsenic elimination and maintain extracellular fluid composition if acute renal failure occurs.

**8. Renal function.** In patients with intact renal function, alkalinization of the urine by sodium bicarbonate to maintain urine pH >7.5 may help protect renal function in the face of hemolysis occurring as part of the acute poisoning.

## HIGHLIGHTS

- Powerful hemolysin

### Signs and Symptoms:

- Malaise, dizziness, nausea, abdominal pain
- Hemoglobinuria and jaundice.

### Treatment:

- Supportive
- Exchange transfusion may be considered

## ARSINE GAS

Arsine is not used as a pesticide. However, some poisonings by arsine have occurred in pesticide manufacturing plants and metal refining operations when arsenicals came into contact with mineral acids or strong reducing agents.

### Toxicology

Arsine is a powerful **hemolysin**, a toxic action not exhibited by other arsenicals. In some individuals, very little inhalation exposure is required to cause a serious hemolytic reaction. Exposure times of 30 minutes at 25-50 parts per million are considered lethal.<sup>20</sup> Symptoms of poisoning usually appear 1-24 hours after exposure: headache, malaise, weakness, dizziness, dyspnea, nausea, abdominal pain, and vomiting. Dark red urine (hemoglobinuria) is often passed 4-6 hours after exposure. Usually 1-2 days after hemoglobinuria appears, jaundice is evident. Hemolytic anemia, sometimes profound, usually provides diagnostic confirmation and can cause severe weakness. Abdominal tenderness and liver enlargement are often apparent. Basophilic stippling of red cells, red cell fragments, and ghosts are seen in the blood smear. Methemoglobinemia and methemoglobinuria are evident. Elevated concentrations of arsenic are found in the urine, but these are not nearly as high as are found in poisonings by solid arsenicals. Plasma content of unconjugated bilirubin is elevated.

Renal failure due to direct toxic action of arsine and to products of hemolysis represents the chief threat to life in arsine poisoning.<sup>21</sup>

Polyneuropathy and a mild psycho-organic syndrome are reported to have followed arsine intoxication after a latency of 1-6 months.

### Treatment

1. Remove the victim to fresh air.
2. Administer intravenous fluids to keep the urine as dilute as possible and to support excretion of arsenic and products of hemolysis. Include sufficient sodium bicarbonate to keep the urine alkaline (pH greater than 7.5).

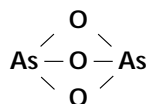
**Caution:** Monitor fluid balance carefully to avoid fluid overload if renal failure supervenes. Monitor plasma electrolytes to detect disturbances (particularly hyperkalemia) as early as possible.

3. Monitor urinary arsenic excretion to assess severity of poisoning. The amount of arsine that must be absorbed to cause poisoning is small, and therefore high levels of urinary arsenic excretion may not always occur, even in the face of significant poisoning.<sup>21,22</sup>
4. If poisoning is severe, exchange blood transfusion may be considered. It was successful in rescuing one adult victim of arsine poisoning.
5. Extracorporeal hemodialysis may be necessary to maintain normal extracellular fluid composition and to enhance arsenic elimination if renal failure occurs, but it is not very effective in removing arsine carried in the blood.

## General Chemical Structures

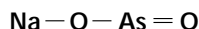
### INORGANIC TRIVALENT

Arsenic trioxide



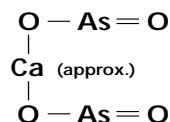
“White arsenic.” Arsenous oxide. Has been discontinued but still may be available from prior registrations.

Sodium arsenite



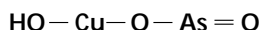
Sodanit, Prodalumol Double. All uses discontinued in the U.S.

Calcium arsenite



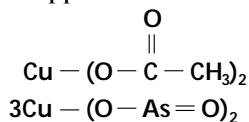
Mono-calcium arsenite, London purple. Flowable powder for insecticidal use on fruit. All uses discontinued in the U.S.

Copper arsenite  
(Acid copper arsenite)



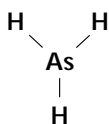
Wettable powder, for use as insecticide, wood preservative. All uses discontinued in the U.S.

Copper acetoarsenite



Insecticide. Paris green, Schweinfurt green, Emerald green, French green, Mitis green. No longer used in the U.S.; still used outside U.S.

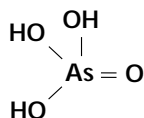
Arsine



Not a pesticide. Occasionally generated during manufacture of arsenicals.

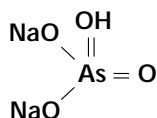
### INORGANIC PENTAVALENT

Arsenic acid

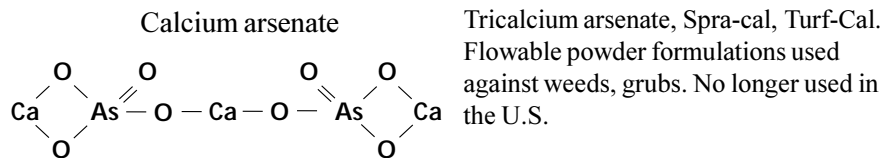


Hi-Yield Dessicant H-10, Zotox. Water solutions used as defoliants, herbicides, and wood preservatives.

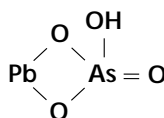
Sodium arsenate



Disodium arsenate. Jones Ant Killer. All uses discontinued, but may still be encountered from old registration.

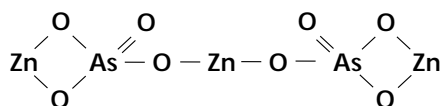


Lead arsenate



Gypsigne, Soprabel. Limited use in the U.S.; wettable powder used as insecticide outside the U.S.

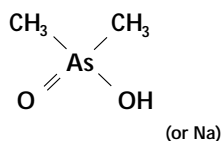
Zinc arsenate



Powder once used in U.S. as insecticide on potatoes and tomatoes.

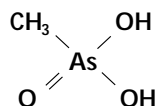
**ORGANIC (PENTAVALENT)**

Cacodylic acid (sodium cacodylate) Non-selective herbicide, defoliant, silvicide. Bolate, Bolls-Eye, Bophy, Dilic, Kack, Phytar 560, Rad-E-Cate 25, Salvo.



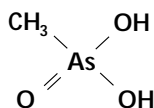
Methane arsonic acid

MAA. Non-selective herbicide.



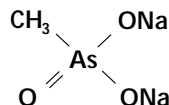
Monosodium methane arsonate

MSMA. Non-selective herbicide, defoliant, silvicide. Ansar 170, Arsonate Liquid, Bueno 6, Daconate 6, Dal-E-Rad, Drexar 530, Herbi-All, Merge 823, Mesamate, Target MSMA, Trans-Vert, Weed-E-Rad, Weed-Hoe.



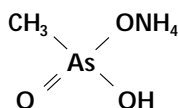
Disodium methane arsonate

DSMA. Selective post-emergence herbicide, silvicide. Ansar 8100, Arrhenal, Arsinyl, Crab-E-Rad, Di-Tac, DMA, Methar 30, Sodar, Weed-E-Rad 360.

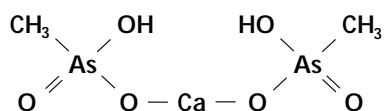


Monoammonium methane arsonate

MAMA. Selective post-emergence herbicide. No longer used in the U.S.



### Calcium acid methane arsonate



CAMA. Selective post-emergence herbicide. Calar, Super Crab-E-Rad-Calar, Super Dal-E-Rad.

## References

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