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Arsenic – Pesticides with an Ambivalent Character

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1. Introduction

Arsenic (As) is a naturally occurring ubiquitous element. It is found in the environment in the earth crust and quantities in media such as soil, water, rock and air. It is present in the environment naturally and due to human activities and other industrial processes such as mining and coal-fired power plants. Arsenic has also been used as a pesticide to protect animals, wood, fruit and vegetables from insects. Because of its therapeutic properties, arsenic has also been used as a medicinal agent. The dark side of the medicine was the reputation as an attractive poison

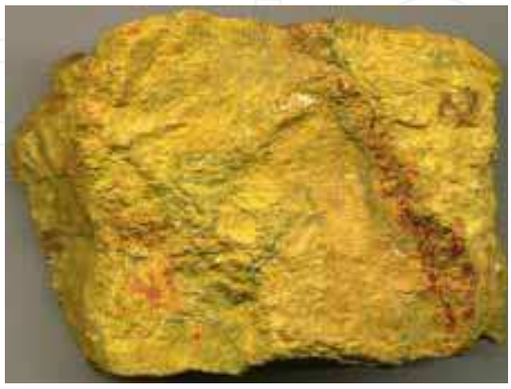
Arsenic is mainly transported in the environment by food, except in areas with high levels of arsenic in the drinking water e.g. India, Taiwan and Bangladesh (2004; Singh, Kumar, and Sahu 2007). The outbreak of As was triggered by deep drilled wells and the desire to obtain microorganism-free safety drinking water. Arsenic contamination of drinking-water is a hazard to human health. Because of the toxicities and side effects of arsenic compounds it is known as a major environmental pollutant. The IARC classified arsenic and arsenic compounds as a human carcinogen (Group 1) (2004). But although arsenic compounds have been known and used for centuries, their mechanisms of interaction in humans are not fully elucidated.

The paradox of arsenic compounds is that, on the one hand, they are considered extremely dangerous for human health with acute and chronic adverse health effects. Long-term arsenic exposure can lead to several types of cancer. The exposure to As has been associated also with non-carcinogenic effects e.g. such diabetes and cardiovascular diseases. On the other hand arsenic compounds are regarded as potential drugs against cancer and ranging from the use as poisons to applications in semiconductors and pesticides. Especially the discovery of organoarsenicals for the treatment of hematological malignancies and solid tumor has awakened interest.

2. Background and basics

Arsenic is a chemical element in the period table that has the symbol "As", the atomic number 33 and an atomic mass of 74,92159 g/ mol. Arsenic exhibits both metallic and non-metallic properties. Arsenic exists as unstable oxides and sulfides or as arsenites or arsenates of sodium, calcium and potassium. Arsenic has two biologically important oxidation states: arsenite (the trivalent form, As III) and arsenate (the pentavalent form, As V). As III is 60 times

more toxic than As V (Yousef, El-Demerdash, and Radwan 2008). From biological and toxicological view, arsenic compounds can be classified into three major groups: Inorganic arsenic compounds, organic arsenic compounds and arsine gas (Hardman et al. 1996). The metalloid is found mostly as yellow complex sulfides. Organic arsenic is non-toxic whereas inorganic arsenic is toxic. The inorganic forms of arsenic are yellow (AS_2S_3, orpiment), red (AS_2S_2 realgar) and grey to silver white (FeAsS, arsenopyrite) (Waxman and Anderson 2001).



a) Orpiment



b) Realgar



c) Arsenopyrite

Fig. 1. Orpiment, realgar and arsenopyrite - from left to right- (Photographs from the Ohio State University Newark)

Arsenic compounds have no smell or taste, but heat can cause As to sublimate to gas with a distinctive garlic odor (Jones 2007).
Arsenic naturally occures in the earth´s surface, mostly in inorganic form (Hine, Pinto, and Nelson 1977). It exists in low concentrations in many rock types but is frequently associated with metal ore deposits e.g. Au (gold) , Ag (silver), Cu (copper) and Fe (iron) (Gochfeld 1997). The most important natural sources of arsenic in the environment are volcanoes. The organic form result when arsenic combines with carbon and hydrogen.

Compound	**CAS No.**	**Molecular formula**
Arsenic	7440-38-2	As
Arsenic trioxide	1327-53-3	As_2O_3
Arsenic pentoxide	1303-28-2	As_2O_5
Arsenic sulphide	1303-33-9	As_2S_3
Dimethylarsinic acid (DMA)	75-60-5	$(CH_3)_2AsO(OH)$
Potassium arsenate	7784-41-0	KH_2AsO_4
Potassium arsenite	10124-50-2	$KasO_2HAsO_2$

Table 1. Physiochemical properties (1980)

3. History

Arsenic and arsenic compounds are known since the ancient times. As early as 500 B.C. the ancients knew about arsenic, whose name comes from the Greek word "arsenikon" for potent or bold, which means orpiment form Latin auripigmentum. In the 16th an 17th centuries, red and white arsenic were put into amulets that were worn around the neck and close to the heart to ward off the plaques (Cullen 2008). Most arsenic is found in conjunction with sulfur in minerals such as arsenopyrite (AsFeS). Because of the association with ore and the stability of As in form like AsFeS, As was used as a "pathfinder element" in geochemical exploration for gold (Jones 2007).



Fig. 2. Acidum Arsenicosum Anhydricum bottle, Global Antiques

Through the centuries Arsenic was a common method of homicide. The death of the French emperor Napoleon Bonaparte, on 5 May 1821 was believed to be a victim after drinking arsenic-tainted wine that was served him (Leslie and Smith 1978; Lin, Alber, and Henkelmann 2004). Arsenic was a popular murder weapon because of the odorless and tasteless properties and the poisoning result in symptoms that can be confused with other natural disorders. In the Middle ages arsenic was a favorite poison and has been called the Poison of Kings and the King of Poison (Vahidnia, van, V, and de Wolff 2007).
Arsenic was also used as healing agents. The Greek physicians such as Hippocrates and Galen popularized it use for treating skin ulcers and tumors such as superficial epitheliomas. Arsenic has been used as topical pastes, as vapor inhalation, intravenous injection, orally in liquid or in solid form. A paste of the sulfides were used for treatment of neuralgia, rheumatism, arthritis and skin disease (Shen et al. 1997). Also Fowler´s solution, a 1% arsenic trioxide preparation, was widely used during the 19th century. Fowler´s original recipe was described as "64 grains arsenic oxide, 64 grains purest vegetable alkali, distilled water half pound. Heat until clear. Cool. Add half pound spirit of lavender and make up to 15 oz with water." (Cullen 2008).



Fig. 3. Fowler´s Solution made by Wisconsin Pharmacal Co

It was used to treat diseases like leukemia, Hodkin´s disease and pernicious anemia.
The first organic arsenical used therapeutically was Salvarsan, which was developed by Paul Ehrlich 1907. It was used to treat syphilis, until penicillin became available in the 1940s. A model representation from Salvarsan and a picture of Ehrlich adorned the 200 Deutschmark banknote.



Fig. 4. German 200 Deutschmark banknote with hologram and Paul Ehrlich

For centuries arsenic has been used for different purposes. Arsenic was an ingredient of a lot of consumer products e.g. wallpapers, toys, food wrappers, cosmetics, pigments in paints – known as "Paris green". William Withering, an english doctor, who discovered 1775 digitalis was a proponent of therapies with arsenic. He argued:" Poison in small doses are the best medicines; and the best medicines in too large doses are poisonous (Aronson 1994).
Arsenic-containing compounds have been used for cancer-treatment in both tradition Western and Chinese medicine. The first use of arsenic in the treatment of leukemia was in 1865 by Lissauer (Lissauer and H. 1865). With the development of modern medicine against cancer the use of arsenic in the western world diminished.

4. Epidemiology

Arsenic exposure occurs from inhalation, absorption through the skin and by ingestion. Arsenic is mainly transported in the environment by food, which contains both organic and inorganic As, but mostly accrue as relatively no-toxic organic compounds (arsenobentaine and arsenocholine). Seafood, fish and algae are the richest organic sources (Edmonds and Francesconi 1987). The following table shows an overview of the arsenic content of various foods.

Food	**Estimates of daily intake (µg/d)**
Milk	1,39
Fruits and vegetable	0,46
Meat	2,14
Cereals and bakery wares	6,57
Fish	34,9
Eggs	0,13
Sweeteners	0,2
Beverages	4,67

Table 2. Estimated of daily arsenic intake from diet (Sorvari et al. 2007)

Concentrations of arsenic vary in the environment, e.g. 0,03-025 ppm in soil, 0,023-0,35 ppm in plants, up to 55 ppm in groundwater, 0,0001-0,08 ppm in seawater, 4-170 ppm in fish, 0,008-0,85 ppm in wine and up to 0,00049 or 0,63 mg/ m^3 in urban air (2004; Jones 2007; Rahman 2006; Basu et al. 2001). Contamination of arsenic in ground water is a global problem and millions of people are at a risk of arsenicosis. People from countries in Asia (Taiwan, Bangladesh, West Bengal (India) and South America (Chile and Córdoba) get presented to inorganic arsenic in ground water with very high concentration. The arsenic poisoning from drinking As-contaminated underground water was often triggered by the introduction of deep tube-pump wells to replace surface water. The World health organization (WHO) and US environment protection agency (EPA) had set up the standard for drinking water known as maximum contamination level (MCL) which is 10 µg/ l (Effelsberg 1992). The WHO recommended 0.01 mg/ l of arsenic in drinking water as an allowable ranger for human consumption. Millions of people are compelled to use the drinking water higher arsenic level than MCL worldwide. In addition there are industrial exposures for workers, e.g. semiconductor workers and famers handle with arsenical herbicides. Arsenic has been used as feed additives e.g. poultry feeds.
It was found an increase in the prevalence of skin lesions at 0,005 mg As/ l in the drinking water, which is a lower level than the drinking water quality standard of WHO (Yoshida,

Yamauchi, and Fan 2004). The skin is very sensitive to As and skin lesions, which are As-induced, are the early effects to chronic As exposure.
Arsenic is released to the atmosphere from both natural and anthropogenic sources.
Tobacco smoke may contain arsenic, especially when the plants have been treated with arsenate insecticide.

Country	Daily dietary intake of total arsenic from diet (µg/d)
Bangladesh	515
Japan	182
USA	20-130
Spain	245
French	62
Germany	52
UK	66
Denmark	64

Table 3. Estimated daily intake of arsenic by the general population ((Devesa et al. 2001; Jelinek and Corneliussen 1977; Leblanc et al. 2005; Mohri, Hisanaga, and Ishinishi 1990; Saipan and Ruangwises 2009; Sorvari et al., 2007; Watanabe et al. 2004)

The principal natural source is volcanic activity, with minor contribution by exudates from vegetation and wind-blow dust. Man-made emissions to air arise from the smelting of metals, the combustion of fuels, especially of low-grade brown coal, and the use of pesticides. Because of the use of numerous arsenical pesticides the arsenic concentration raised in the soil.

5. Effects on human

The biological activity of arsenic in the body covers a broad spectrum from toxic to therapeutic agent. Not to forget- Arsenic is a human carcinogen (IARC 2004). A number of studies show that arsenic is an essential element for humans. Other studies have attempted to show that arsenic has not been demonstrated to be essential to humans (Ohtake 2000).
The major routes of arsenic absorption in the general population are ingestion and inhalation. Meat, fish and poultry account for 80 % of dietary arsenic intake (Edmonds and Francesconi 1987). Arsenic is absorbed in the small intestine by an electrogenic process involving a proton gradient. The absorbed arsenic undergoes hepatic biomethylation. The products are less toxic but not completely innocuous. About 50 % of the ingested dose may be eliminated in the urine in 3-5 days. Metabolism of As involves reduction of As V to a trivalent state and subsequent oxidative methylation.

5.1 Acute effects

After acute poisoning studies show that the highest concentration of arsenic is in the kidney and liver (Benramdane et al. 1999). Most cases of acute arsenic poisoning occur from accidental ingestion of insecticides or pesticides. Acute exposure to arsenic arise symptoms like abdominal pain, vomiting, diarrhea. The abdominal pain may mimic an acute abdomen (Mueller and Benowitz 1989). Other clinical features are muscular weakness and cramping, erythematous skin eruptions like diffuse skin rash and swelling of acrals. A progressive deterioration in the motor and sensory responses and toxic cardiomyopathy may also result

leading to shock and death. Depending on the quantity of arsenic, death usually occurs within 1-5 days. In acute poisoning the best indicator of recent ingestion (1-2 days) is urinary arsenic concentration. Dimethylarsinic acid is the dominant urinary metabolite compared with monomethylarsoonic acid (Hopenhayn-Rich, Smith, and Goeden 1993).

5.2 Chronic effects

Chronic ingestion of inorganic arsenic causes multisystem adverse health effects. The clinical features of chronic arsenic toxicity vary between individuals, population groups and geographic areas. In chronic arsenic ingestion, arsenic accumulates in the liver, kidneys, heart and lungs and smaller amounts in the gastrointestinal tract, spleen and muscles (Benramdane et al., 1999). High doses of arsenic cause characteristic skin manifestation, vascular, renal and neurological diseases, cardiovascular and chronic lung diseases and cancer of skin, lungs, liver, kidney and bladder. After about two weeks arsenic is deposited in the hair and nails. Levels between 0,1 and 0,5 mg/ kg on a hair sample indicate chronic poisoning. Various epidemiological studies have reported that arsenic exposure is associated with hypertension, atherosclerosis and endothelial dysfunction (Yang et al. 2007) (Chen et al. 2007) (Kwok et al. 2007). Increasing exposure of arsenic is also associated with non insulin dependent diabetes mellitus (Wang et al. 2003). Studies reported that arsenic is associated with the growth retardation in children (Wang et al. 2007).

5.3 Skin symptoms

Skin manifestation is the most common and initial sign of chronic arsenic exposure. Chronic ingestion of arsenic causes characteristic melanosis, keratosis, basal cell carcinoma and squamous cell carcinoma (Maloney 1996). Melanosis includes hyperpigmentation, spotted pigmentation, depigmentation and leucomelanosis. Keratosis is a late feature of arsenical-dermatosis and appears especially on palm as a uniform thickening or as discrete nodules (Wong, Tan, and Goh 1998b). Both palmar and solar keratosis are significant diagnostic criterion. Bowen´s disease is a precancerous lesion and predisposed to an increased incidence of the squamous cell carcinoma. Chronic ingestion of arsenic lead to accumulate in keratin rich areas of body and appears as white lines in the nails, called Mee´s lines (Fincher and Koerker 1987). The latency period of skin lesions of arsenic after first exposure varies



Fig. 5. Patient with plantar keratosis (2004; Wong, Tan, and Goh 1998c).



Fig. 6. Blackfoot diseases after chronic arsenic exposure (Better Life Laboratories, USA)

from 20 to 50 years (Haque et al. 2003). It is described that the latent period after exposure can be as long as 60 years, which has been reported in patients treated with Fowler´s solution, in vineyard workers using arsenical pesticides and from drinking contaminated wine (Everall and Dowd 1978).

Many different systems within the body are affected by chronic exposure. Some of these systems and their associated toxic effects from chronic arsenic exposure are listed in the following table.

System	**Effect and symptoms**
Skin	Skin lesions (melanosis, keratosis)
Cardiovascular	Blackfoot disease, atherosclerosis, hypertension
Hepatic	Hepatomegaly, fibrosis, cirrhosis, altered heme metabolism
Hematological	Bone marrow depression (anemia, leucopenia, thrombocytopenia)
Endocrine	Diabetes
Renal	Tubule degeneration, papillary and cortical necrosis
Nervous	Peripher and central neuropathy, encephalopathy
respiratory	Pulmonary insufficiency, emphysem
Gastrointestinal	Hemorrhage

Table 4. Human effects after chronic arsenic exposure (Singh, Kumar, and Sahu 2007; Schuhmacher-Wolz et al. 2009; Hughes 2002; Balakumar and Kaur 2009; Rahman, Ng, and Naidu 2009).

6. Toxicity

Arsenic compounds or arsenic-containing compounds vary in toxicity to mammalian cells. Arsenic does not directly react with DNA or cause gene mutations, except to a small extent at high dose. As can cause gene amplification and chromosomal damage at lower doses and can enhance mutagenesis by other agents, apparently by inhibiting DNA repair. The following table gives an overview over the modes of carcinogenic action of arsenic.

Modes of carcinogenic action of arsenic
Genotoxicity
Oxidative damage
Modification of cell signalling
Influence on DNA repair
Influence on DNA methylation
Changes in cell proliferation
Co-mutagenesis and transformation
Tumor promotion

Table 5. Modes of carcinogenic action of arsenic (Schuhmacher-Wolz, Dieter, Klein, and Schneider 2009; Hughes 2002).

The binding with sulfhydryl groups by arsenite compounds has the potential to influence a wide range of metabolic activities. Arsenic toxicity inactivates up to 200 enzymes. The effects of As occur through indirect alteration of gene expression via disruption of DNA methylation, inhibition of DNA repair, oxidative stress, or altered modulation of signal transduction pathways. Another indirect mechanism is the influence of growth-stimulating chemicals or cytokinesed generated in response to arsenic exposure. Biotranformation is the major metabolic pathway for inorganic arsenic in humans. Toxic inorganic arsenic species can be biomethylated by bacteria, algae, fungi and humans. The high affinity of arsenic for sulphydryl groups makes keratin-rich cells a target for arsenic.
The order of toxicity of arsenicals is:
Monomethylarsonic acid (MMA III) > Arsenite (III) > Arsenate (V) > MMA(V) (Singh, Kumar, and Sahu 2007).
In arsenic biotransformation the intermediate product MMA III is highly toxic than other arsenical, which might be responsible for the arsenic-induced carcinogenesis and other effects (Styblo et al. 2000). As III binds thiol or sulfhydryl groups in tissue proteins of the liver, lungs, kidney, spleen, gastrointestinal mucosa and keratin-rich-issues (skin, hair, nails). By binding a wide range of metabolic activities are influenced including cellular glucose uptake, gluconeogenesis and fatty acid oxidation (Jones 2007). Many other toxic effects of arsenic compounds are detailed by Abernathy et al in 1999 (Abernathy et al. 1999).
The acute toxicity is related to its chemical form and oxidation state. In the human adult the lethal range of inorganic arsenic is estimated at a dose of 1-3 mg As / kg (Schoolmeester and White 1980). The characteristics of acute arsenic toxicity in humans include gastrointestinal discomfort, vomiting, diarrhea, bloody urine, anuria, shock, convulsions, coma and death.

7. Pharmaceutical use

Arsenic has been used therapeutically for over 2000 years. During the 18th- 20th centuries arsenic compounds have been used as medicines, including arsphenamine and arsenic

trioxide. In 1910, Paul Ehrlich introduced the arsenic-based drug Salvarsan (arsenobenzol) as a remedy for syphilis in all stages, a sexually transmitted disease. It was efficient in various similar diseases such as relapsing fever, Vincent´s angina.

Arsenic trioxide is also known as an anti-bacterial and anti-cancer agent (Bardos, tta-Gupta, and Hebborn 1966). Inorganic As has been also used pharmacologically for the treatment of eczema, pemphigus and psoriasis under the name of Fowler´s solution. It was a 1 % solution of potassium arsenite, colored with a tincture of lavender-which contained a very high concentration of arsenic (Rahman 2006). Some arsenic containing drugs are still presently used to treat diseases like asthma rheumatism, cough, pruritus and itching (Ko 1999; Wong, Tan, and Goh 1998a).

In 2000, the US Food and Drug Administration approved the use of arsenic trioxide for treatment of relapsed or refractory acute promyelocytic leukemia (APL), a subtype of acute myeloid leukemia (AML) (Antman 2001). It is based on its mechanism as an inducer of apoptosis (programmed cell death) (Soignet et al. 1998).

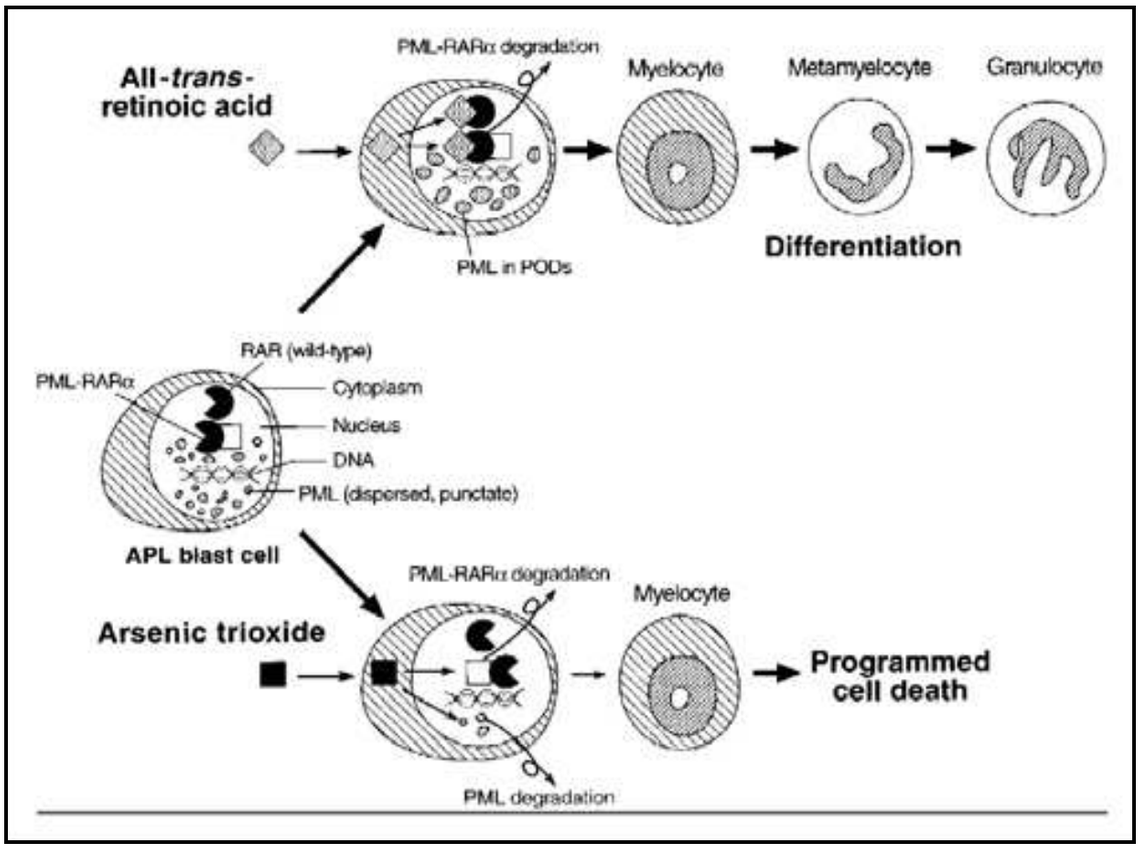


Fig. 7. Effects of all-trans-retinoic acid and arsenic trioxide in the blast cells of acute promyelocytic leukemia (APL) (Look 1998).

Traditional medicine products contain arsenic sulfides (realgar) and are available as pills and tablets. They are still used for psoriasis, syphilis, asthma, rheumatism, hemorrhoids, cough and pruritus and are rescribed as a health tonic, an analgesic, anti-inflammatory agent (Ko 1999; Wong, Tan, and Goh 1998d). In Korea arsenic is prescribed in herbal medicine for anal suffering such haemorrhoids (Mitchell-Heggs, Conway, and Cassar 1990).

8. Industrial use

In industry, arsenic is used to manufacture polants, fungicides, insecticides, pesticides, herbicides, wood preservatives, and cotton desiccants.
In most local hardware stores arsenic-containing herbicides are readily available, the most common are e.g. Disodium methylarsonate (DSMO), monosodium methylarsonate (MSMA), monomethyl arsenic acid (MMA(V)). These compounds can kill crabgrass and other unwanted grass types. Arsenic trioxide (AS_2O_3) is commonly used as an antisecticide. Arsenic acid and arsenous acid are common rodenticides. The major use for arsenic is in the form of chromated copper arsenate, which reduces termites and ants from wood.
Arsenic is used industrially as an additive to glass to reduce coloring, in semiconductors, in pigments such as Paris green (CuHAsO3) and in pesticides. Paris Green is a common name for copper (II) acetoarsenite, which is a toxic emerald-green crystalline powder. Other names for the chemical are Emerald Green, Vienna Green, Schweinfurt Green and Parrot Green. The use has been abandoned around 1960. The III-V semiconductors are very important in the fabrication of LED´s, tunnel diodes, infrared emitters, laser window and Hall-effect devices.

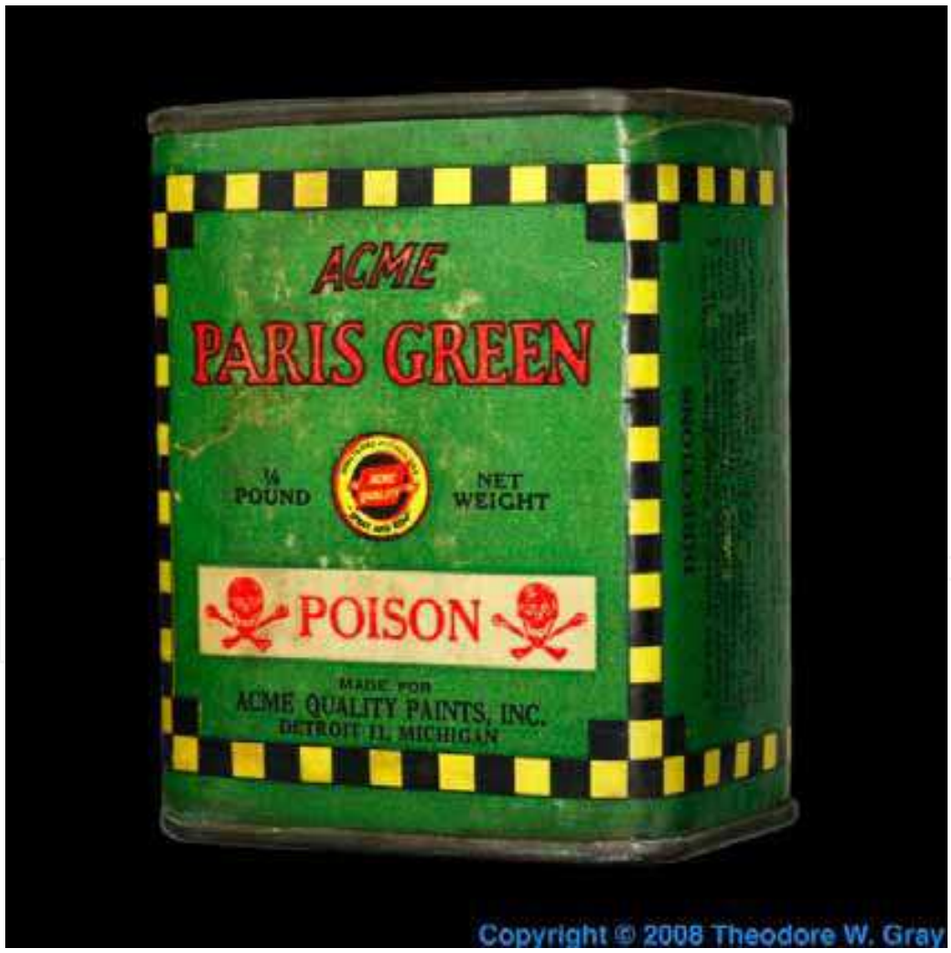


Fig. 8. Paris Green bottle
(http:/ / theodoregray.com/ periodictable/ Elements/ 033/ index.s7.html).

9. Arsenic, wine and profession

Arsenic was used in vineyards for only some years as a pesticide. It was officially introduced as a pesticide in viniculture in 1925. Its purpose was to protect the wine plants. But it was banned in 1942. It was used in Germany until the mid 1950ties (Shab, 2009). Consumption of the so-called wine-grower´s house drink led to severe symptoms and illnesses, especially liver damage. This homemade wine was produced by watering down the wine obtained from a second pressing of the grape skins. It was consumed in large quantities, which had a low alcohol content, from 3-5 %, but high arsenic content (Kunz and Kunz 2008). Exposure to arsenic has been reported to lead to cirrhosis and to angiosarcoma among famers exposed to arsenical insecticides. Chronic liver disease which can be caused by arsenic toxicity includes also steatosis and noncirrhotic portal hypertension (Von Hyman JZimmerman. 1999).

Not only at the vineyards there was an occupational exposure to arsenic. Other important occupational exposure opportunities exist for processing of metal ores, roasting of pyrites in the chemical industry, the production and use of arsenic colors and tints for glass, porcelain and ceramics industry, pesticides and wood preservatives as well as at the battery and semiconductor. Occupational diseases caused by arsenic and its compound can be recognized as an occupational disease (BK-Nr. 1108 in Germany) (1964).



Fig. 9. Wine glass with wine yard (Markus Ebert - photographer Heidelberg/ Potsdam)

10. Conclusion

From history to the present, the story of arsenic is double-edged: a poisonous edge and a medicinal edge. Arsenic has been mentioned mainly as a poison and public health problem

than as an effective anticancer drug. Arsenic is one of the most toxic metals derived from the natural environment. Inorganic Arsenic is a human carcinogen, but nowadays also acts as a beneficial chemotherapeutic agent. The major cause of human arsenic toxicity is from contamination of drinking water and from As-contaminated food through fertilization. Current uses of arsenic compounds are in the glass industry, as a wood preservative and in the production of semiconductor. Over the centuries, arsenic has been used for a variety of purposes. In industry arsenic is used as a potential weapon against insecticides concerning humans as a modern weapon. Arsenic compounds became available e.g. in Fowler´s solution as indication for skin conditions and treatment for acute and chronic diseases. Arsenic affects many cellular and physiological pathways, which is useful in treating malignancies like hematological cancer and solid tumors. The ability of arsenic trioxide to treat APL has changed the point of view.

Still today moderately elevated concentrations of inorganic arsenic in drinking water is a major public health concern as well as arsenic exposure from food, especially rice products (Sun et al. 2008).

Chronic arsenicism may lead to multiple benign skin diseases as well as potentially fatal skin and visceral malignancies e.g. lungs, bladder, liver kidneys. Pigmentation changes and hyperkeratosis are the earliest signs of toxicity from chronic exposure. People with chronic arsenicism should undergo regular skin and systemic examination. There are no evidence based treatments to reduce chronic arsenic poisoning, but antioxidants have been advocated: Pharmacological interventions such as vitamin C, folic acid, vitamin b12 have been identified to halt the development of arsenic-induced toxicity. More studies are needed. The essential and basic efforts for the reduction of chronic arsenic toxicity are prevention. Although current exposure to arsenic is decreasing, continual surveillance programs to detect unrestricted and unsupervised manufacture and sale of drugs that may contain inorganic arsenic must be implemented to prevent a potentially fatal disorder.

11. Acknowledgment

Arsenic is a fascinating element. We were inspired by treating patients having contact to arsenic. Quod vide:

Shab, C. Crößmann und C. Bayerl, „Multiple Basalzellkarzinome und aktinische Keratosen bei einem landwirtschaftlichen Arbeiter nach Arsen-Exposition: Immer nur BK 1108?“ Dermatologie in Beruf und Umwelt (Vol. 57,No. 4/ 2009(4. Quartal)).

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This book is a compilation of 29 chapters focused on: pesticides and food production, environmental effects of pesticides, and pesticides mobility, transport and fate. The first book section addresses the benefits of the pest control for crop protection and food supply increasing, and the associated risks of food contamination. The second book section is dedicated to the effects of pesticides on the non-target organisms and the environment such as: effects involving pollinators, effects on nutrient cycling in ecosystems, effects on soil erosion, structure and fertility, effects on water quality, and pesticides resistance development. The third book section furnishes numerous data contributing to the better understanding of the pesticides mobility, transport and fate. The addressed in this book issues should attract the public concern to support rational decisions to pesticides use.

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