INTRODUCTION
Arsenic (As) is the 20th most abundant element on earth. Arsenic compounds occur in many rocks and find their way into soils, water and food. There are many different compounds of both inorganic and organic arsenic. Arsenic is present in all living organisms, mostly from dietary uptake.

USES
The greatest commercial use of inorganic arsenic is in wood preservatives with other major uses in pesticides (herbicides and termicides). Small amounts of inorganic arsenic are used in production of glass and non-ferrous alloys, and in the manufacture of electrical semiconductors. Arsenic oxide is produced primarily as a by-product from the smelting of copper and lead ores.

Organic arsenic is generally not used commercially. However, some organic arsenicals, such as derivatives of phenylarsenic acid are used as feed additives for pigs and poultry.

OCCUPATIONAL EXPOSURE
Occupational exposure occurs in smelters, timber treatment plants and among workers engaged in the manufacture and use of arsenical pesticides and preservatives.

The Worksafe Australia Exposure Standards for arsenic are:

<table>
<thead>
<tr>
<th>Time Weighted Average (TWA)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic &amp; soluble...........</td>
<td>0.05mg/m³ compounds</td>
</tr>
<tr>
<td>Arsine..........................</td>
<td>0.16mg/m³</td>
</tr>
</tbody>
</table>

Inorganic arsenic is recognised as an established human carcinogen ie. known to be carcinogenic in humans, and has been classified as a Category 1 carcinogen by Worksafe Australia (Worksafe Australia - Exposure Standards for Atmospheric Contaminants in the Occupational Environment, October 1995).

ENVIRONMENTAL EXPOSURE
For the general population, diet is the major source of arsenic exposure. Levels in air and drinking water are generally very low.

With the exception of seafood, and animal and poultry offal, arsenic in food is usually less than 0.25 ppm (parts per million or mg/kg). Animal and poultry offal often contain higher levels because of the use of organoarsenical feed additives. Levels in kidneys and livers from pigs and poultry often exceed

---

1 Exposure standards represent airborne concentrations of individual chemical substances which, according to current knowledge, should neither impair the health of nor cause undue discomfort to nearly all workers. They do not represent No Effect Levels which guarantee protection to every worker.

2 The average airborne concentration of a particular substance when calculated over a normal eight-hour working day, for a five-day working week. Providing the TWA is not exceeded, short term exposures should not exceed three times the TWA exposure standard for more than a total of 30 minutes per eight-hour working day and in no circumstances should the short term values exceed five times the TWA exposure standard.

3 There is sufficient evidence to establish a causal association between human exposure to these substances and the development of cancer.
An average smoker may inhale up to 20 μg of arsenic daily depending on the content of arsenic in the tobacco.

**HEALTH EFFECTS**

The acute and chronic toxicity of arsenic is affected by the valency form and route of absorption. Acute toxicity decreases in the order trivalent (+3) inorganic > pentavalent (+5) inorganic > organic arsenicals. Most organic derivatives have relatively low toxicity.

Symptoms of acute exposure by the oral route usually include metallic taste, burning of the mouth, vomiting, diarrhoea, dehydration, general vascular injury and muscular cramps. Death can occur at sufficient doses of exposure. Acute inhalation exposure causes irritation of the upper respiratory tract. Inhalation of sufficient amounts will cause the same effects as with ingestion.

Signs of arsenic toxicity, after chronic oral exposure, include gastrointestinal irritation, anaemia, neuropathy, skin lesions (hyperpigmentation, hyperkeratoses), vascular lesions, and hepatic and renal injuries. Severity of effects is usually a function of dose and exposure duration.

Dermal contact with arsenic may produce skin irritation but it is not associated with any systemic health effects. Arsenic is well absorbed after oral or inhalation exposure, but only very poorly absorbed through the skin.

Arsenic is also classed as a Category 1 carcinogen by the International Agency for Research on Cancer (IARC). This is based on sufficient evidence for carcinogenicity in humans and limited evidence in animals. Exposure by inhalation increases the risk of lung cancer, while ingestion increases the risk of skin cancer.

The epidemiological studies of populations drinking water with naturally elevated levels of inorganic arsenic which demonstrated the risks of skin cancer also suggested there is a threshold dose before which the skin lesions and cancers are not induced. It is on this basis that the FAO/WHO Joint Expert Committee on Food Additives set the PTWI for inorganic arsenic at 15 μg/Kg body weight/week.

Teratogenic and fetotoxic effects have been seen in studies with experimental animals. It appears that developmental toxicity is not a concern below exposure levels which cause maternal toxicity.

Arsenic possibly exerts its toxicity by reacting with sulfhydroly groups of cellular proteins, destroying their activity.

**MONITORING**

The biological half-life of arsenic is short. Blood levels do not correlate well with exposure levels. Arsenic is excreted in urine, faeces and by the dermis as shed skin, hair and nails. Most excretion is via the urine and after a single dose most is eliminated within a week. The hair and nails may be used to detect past, discontinued exposure.

Measurement of arsenic in urine is the method of choice for evaluating current exposure to arsenic. However, clearance of arsenic from the body is rapid and the concentration in urine is very time-dependent when there is occasional or intermittent exposure. Two methods of measuring arsenic in urine are available:

1. If the measurement done is the total arsenic content of the urine, interference may occur from low toxicity organic arsenicals, particularly from seafood. Thus, it is

---

4 The unit of time for PTWIs (one week) is longer than that used for the acceptable daily intake (ADI). The longer time interval is used because the chronic intake is of greater importance than acute intake for substances that accumulate in the body. Consumption of particular foods may result in a high intake of a contaminant in a single day; however, over a longer period, such as a week, consumption may not be excessive.
necessary to exclude seafood from the diet for the few days prior to collection of the urine sample if the total arsenic content is to be measured.

2. To overcome the problem of interference from low toxicity organic arsenicals from diet, it is preferable to measure the concentration of inorganic arsenic and its metabolites, monomethylarsonic acid and cacodylic acid, for evaluating current exposure to the more toxic inorganic arsenic.

Normal arsenic concentrations in the urine of adults (without occupational exposure) are <100 µg/L, with values of 20 to 50 µg/L being typical. Worksafe Australia recommends that in occupationally exposed persons, urine levels should not exceed 150 µg/L.

Arsenic in hair is usually less than 1 ppm.

Blood levels depend on dietary and other intakes, and do not correlate well with exposure. Thus, measurement in blood is not recommended.

LEGISLATION

Arsenic for human therapeutic use is in Schedule 4 of the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP). Arsenic in ant poisons containing 0.4% or less, in animal feed premixes containing 4% or less, or in preparations for the treatment of animals (except thiocetarsamide when included in Schedule 4) is in Schedule 6 of the SUSDP. Other uses and concentrations of arsenic are covered under Schedule 7 and persons must not possess or use such arsenicals unless authorised to do so by the appropriate authority.