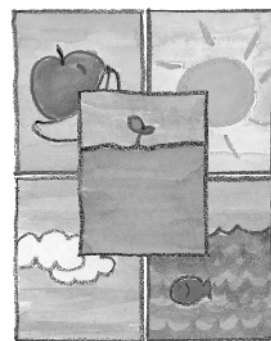


# The soiled environment: bubble, bubble, soil in trouble

Andrew J Langley



THE LAND has always been etched into the Australian identity. Revered by its original inhabitants and a source of inspiration for poets and rock stars, only recently has its contamination and its loss from soil erosion made headlines. While our national anthem extols our "golden soil", there is no mention of the high arsenic levels sometimes associated with gold deposits.

Soil contamination was largely ignored until the 1980s, when both regional contamination (around places such as Port Pirie, site of the world's largest lead smelter) and localised contamination (such as at cattle tick dip sites in Queensland and New South Wales), were recognised. Significantly elevated blood lead levels and subtle cognitive effects in children were detected in Port Pirie,<sup>1</sup> while exposure to pesticides, especially arsenic and DDT, was of concern near cattle tick dip sites.<sup>2</sup>

At the same time, urban consolidation was creating pressure to transform old inner-city industrial sites into residential areas. Bank foreclosures occurred on properties that then became onerous possessions because of contamination liabilities. This led to audits becoming part of transactions involving industrial land, the identification of many contaminated sites and, in the 1990s, the development of national guidelines<sup>3,4</sup> for assessing and managing contaminated sites in Australia and New Zealand.

There have been relatively few epidemiological studies, in Australia or overseas, of health effects related to contaminated sites. A UK study<sup>5</sup> of people living within a two-kilometre radius of a landfill (80% of the UK population falls into this category) detected increased risks of congenital anomalies and low birthweight. However, the study did not differentiate potential data artefacts and confounding effects from possible causal associations. Moreover, such studies often do not identify actual exposure pathways.

## The size of the problem

Little is known about the scale of soil contamination in Australia because of limited investigation and definitional issues. One estimate, based on extrapolation from Queensland data, is that there may be as many as 200 000 contaminated sites in Australia (a few examples are shown in Box 1).<sup>6</sup>

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## ABSTRACT

- There are many contaminated soil sites in Australia.
- Contamination may be from human activities (eg, smelters, industrial waste dumps, old gasworks) or from naturally occurring sources (eg, surface mineralisation).
- Concentrations of contaminants may vary markedly across a site. Their distribution may be localised or quite extensive.
- Common contaminants include lead, arsenic, cadmium, petrol and diesel products, and polycyclic aromatic hydrocarbons.
- People living on or near a contaminated site will often be concerned about potential effects on their health. Assessing their exposure potential and/or measuring levels of biological markers often allays concerns, but occasionally confirms them (eg, elevated blood lead levels and subtle cognitive effects have been found in some people living near the Port Pirie lead smelter).

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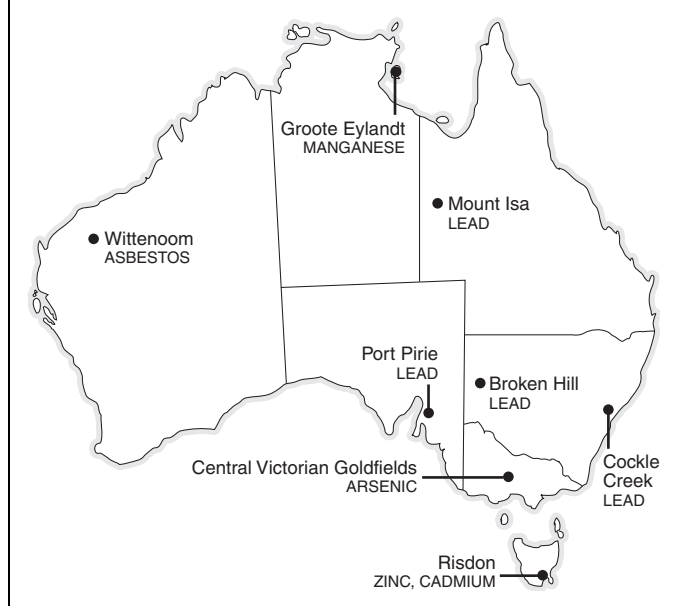
The amount of contaminated material can be considerable: at Port Pirie, where a smelter has operated since 1889, it is estimated that 80 000 tonnes of lead have been deposited on land and almost 90 000 tonnes in the marine environment.<sup>7</sup> Elevated lead concentrations have been detected in soil as far as 60 km downwind of the smelter.<sup>8</sup>

The most common toxic soil contaminants are lead (eg, from paint, scrapyards), arsenic (eg, from agricultural pesticides, weed clearance and tanneries), BTEX (benzene, toluene, ethylbenzene and xylenes from petroleum products), polycyclic aromatic hydrocarbons (eg, from combustion products, tars and gasworks) and persistent pesticides (eg, DDT used in cattle tick dips). At sufficient exposures, the first significant health effects noted from lead are cognitive effects, while other contaminants such as arsenic, benzene and some polycyclic aromatic hydrocarbons are regarded as carcinogens.

## Identifying contaminated sites

Many contaminants do not cause obvious soil discoloration unless they are in very high concentrations. There may, instead, be subtle signs of contamination, such as reduced or absent plant growth. Some sites have been identified by odour or by sickness in animals. For example, in the 1950s, cows belonging to the mayor of Murray Bridge, grazing under the bridge for which the city was named, were poisoned by lead from paint released by sanding and

**1: Australian examples of potential or identified sites with contamination related to mineral bodies, mining or smelting, and principal contaminants**



scraping of the bridge during routine maintenance.<sup>9</sup> In the 1980s, an Adelaide backyard contaminated in the past by lead sinker manufacture was identified after a vet diagnosed lead poisoning in a dog (Len Turczynowicz, Scientific Officer [toxicologist], South Australian Department of Human Services, personal communication).

Soil sampling may be performed at random locations, at regular spacings, or where contamination is thought to be most likely because of the site history — or using a combination of these approaches. Sampling is usually undertaken at different depths. As contamination concentrations often vary greatly across a site, isolated “hot spots” may be missed or randomly oversampled by the use of a grid.

**Routes of human exposure to contaminated soil**

Potential exposures are greater where there is regional, not just localised, contamination, as more exposure opportunities exist — at home, at childcare or shopping centres, and more generally from contaminated air, food and water.

**Direct soil ingestion**

Direct soil ingestion is usually the principal exposure route. People rarely deliberately consume “dirt”, but children often convey dirt to the mouth on their hands, and adults may ingest contaminated soil on vegetables or on the surface of cigarettes.

Children are the “key receptor” because of hand-to-mouth behaviours and because gastrointestinal absorption of heavy metals is higher in children than adults. Furthermore, children have heightened susceptibility to the effects of heavy metals because of their developing neurobehavioural system.

It is difficult to measure soil ingestion accurately. One surrogate measure is based on subtracting the quantity of tracer elements ingested in food from faecal amounts of the tracers. Children aged between one and four years living at a contaminated site in Montana, USA, have been estimated to ingest a median of 17 mg of soil per day.<sup>10</sup> Peak soil ingestion probably occurs around 15–24 months of age.<sup>1</sup> The condition of soil pica (the recurrent ingestion of unusually high amounts of soil) is considered to occur rarely in Australia, but may result in high soil ingestion by certain individuals. In some countries there is cultural geophagia.

**Inhalation**

For volatile contaminants, inhalation may be a significant exposure route. Dust inhalation is generally a minor exposure pathway, but may be the only exposure route for people living near a contaminated site.

**Dermal absorption**

Dermal absorption of many contaminants such as inorganic lead is very low. Elevated concentrations of allergic sensitisers, such as chromium VI and nickel, may present a dermal hazard.

**Exposure via food and water**

Contamination of homegrown fruit, vegetables and herbs is a common concern of people living on or near contaminated sites.<sup>11</sup> There are substantial differences in uptake of contaminants according to the type of contaminant and the species of plant. Cadmium is taken up more readily than lead, but lead may still adhere to the dust on green leafy vegetables.

Dust arising from the site may cause concerns about contamination of rainwater tanks, and measures to test rainwater and prevent contamination may be warranted.

**Clinical appraisal**

If a patient presents with concerns about exposure to toxic substances from a contaminated site, the following questions should be considered:

- Is there evidence that the site is contaminated?
- What are the likely contaminants?
- Are there significant exposure opportunities from ingestion, inhalation or dermal contact?
- Are biomarkers of exposure or effect available?
- Are short- or long-term protective measures or treatments available?

Environmental protection agencies, public health departments and local governments may be able to provide useful information in this regard (see case study in Box 2).

**Use of biomarkers**

Although over 20 million organic and inorganic substances are known, far fewer are in general commercial use, and validated biomarkers (used to measure exposures to, suscep-

tibilities to, or effects of environmental agents) with established normal ranges are available for only a few substances.

Blood lead level is a validated marker of lead exposure, and urinary arsenic level is the most appropriate indicator of arsenic exposure. However, as arsenic has a short biological half-life (several days), old exposures will not be detected. Furthermore, unless the level of "inorganic arsenic" is measured (rather than the commonly used "total arsenic" level), relatively high concentrations of naturally occurring organic arsenic from recent dietary seafood intake may confuse the interpretation.

Urinary cadmium is the best readily available marker of historical exposures (cadmium has a biological half-life of several years), but, with concentrations less than 10 parts per billion expected, sample contamination is a significant problem. This means that absolute cleanliness during collection and analysis is critical — do not accept urine specimens in Vegemite jars!

Where there is a mixture of contaminants it may be feasible to use a single validated biomarker, such as blood lead level, as a surrogate measure of exposure to a range of other toxic substances.

### Clinical management

A key principle is to identify exposure pathways and try to prevent or minimise further exposure. For example, in an environment with high lead contamination, doctors should also check for iron deficiency and establish whether pregnant women are receiving adequate calcium<sup>12</sup> (calcium competes with lead for absorption and retention). If indicated, children should be referred for neurodevelopmental and behavioural assessment. Detailed recommended interventions for various concentrations of elevated blood lead are available,<sup>13</sup> and may include paediatric assessment, environmental assessment and sampling (if the source is not obvious), and remediation. A large randomised controlled trial<sup>14</sup> concluded that chelation therapy was not indicated for children with blood lead concentrations below 45 µg/dL, as these children did not perform better in tests of cognition, behaviour and neuropsychological function after chelation therapy.

### Environmental management

To determine the need for intervention, the risk from a contaminated site needs to be assessed (Box 3).

For sites with localised contamination, interventions should be directed at remediating the site. Where there is regional contamination, environmental management will also need to focus on controlling air, food and water contamination and minimising contamination of living areas and children's play areas. Large mats at external doors can reduce the amount of soil being carried into houses on footwear, and wet mopping and carpet cleaning can reduce the burden in living areas. Using a standard vacuum cleaner can paradoxically worsen exposures, as the fine particulate fraction that will pass through the filter and be blown back

### 2: Case study: investigation of possible exposure to toxic substances

Mrs X presented to her general practitioner with her seven-year-old son, Oliver. She was concerned about a local newspaper report that a block of land where he often played was contaminated with high levels of lead and arsenic. She was anxious about her son's health and about the potential reduction in value of her property.

On examination, Oliver had no clinical signs of ill health or overt lead or arsenic poisoning (eg, pallor, lead lines on the gums, neuropathy).

Fortunately, the regional public health unit had recently sent a letter to all local GPs containing information from the local Environmental Protection Agency (EPA) about the site in question. The site was being proposed for housing redevelopment, and local authorities, alerted by the site history to possible contamination, had demanded an audited site assessment.

The site was a scrapyard that had been abandoned 20 years ago. It had been used to recover lead from batteries and to dump tannery waste with high chromium and arsenic content.

Recent tests undertaken by an environmental consultant on soil and groundwater (close to the sandy surface and used by locals for their vegetable gardens) showed the groundwater to be unaffected, but levels of lead and arsenic in the soil were higher than acceptable criteria. High levels of chromium III were found in some samples, but this is of low toxicity and concentrations did not exceed soil criteria.

After undertaking a health risk assessment, the public health unit's advice to GPs was that the lead and arsenic levels were only of concern for any children under five years actually living on the site, because of potential ingestion by hand-to-mouth behaviours. Dermal and inhalational exposures were considered negligible in this instance.

The mother was not readily reassured, and requested testing. The GP organised blood lead and urinary inorganic arsenic testing at a laboratory with expertise in biological monitoring. The results were within the normal population range for a child, and Mrs X was reassured.

The EPA also announced that remediation of the site would be undertaken in the near future. Contaminated soil would be removed to a managed landfill (with clay and geotextile lining) and replaced with clean soil. As an interim measure, the site would be fenced.

### 3: How risky is a site?

Precise estimates of the hazard risk of a contaminated site are often made, usually based on US Environmental Protection Agency (EPA) methodologies. However, quantitative estimates may be misleading<sup>15</sup> because of uncertainties arising from soil sampling processes and from exposure and toxicological assessments. These estimates have been particularly controversial in cases where the soil contaminants are classified as carcinogenic.

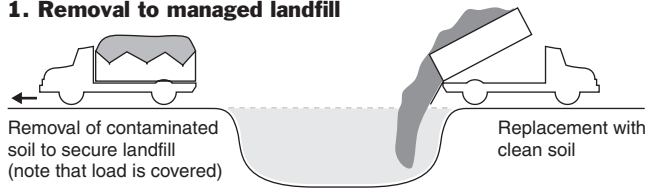
To address these problems, Australia has developed an approach to the environmental and health risk assessment of sites,<sup>4</sup> a method for assessing carcinogenic soil contaminants,<sup>16</sup> and environmental health risk assessment guidelines.<sup>15</sup> The guidelines set out five stages of investigation:

- Issue identification, to establish where risk assessment is of value;
- Hazard identification, to determine possible adverse effects;
- Dose–response assessment, to estimate the incidence of adverse effects at different exposure levels;
- Exposure assessment, to estimate the contributions of various exposure pathways to total population exposures; and
- Risk characterisation, to provide a qualitative and/or quantitative estimate of the nature, severity and potential incidence of effects in a population.<sup>15</sup>

**4: Remediation of contaminated sites**

The principal approaches to remediating contaminated sites are:

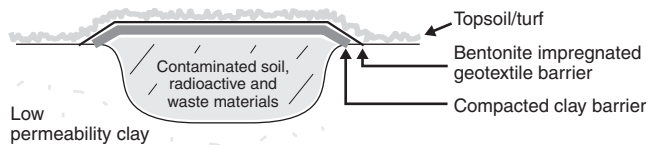
**1. Removal to managed landfill**



**2. Installing "cap" and changing landuse**

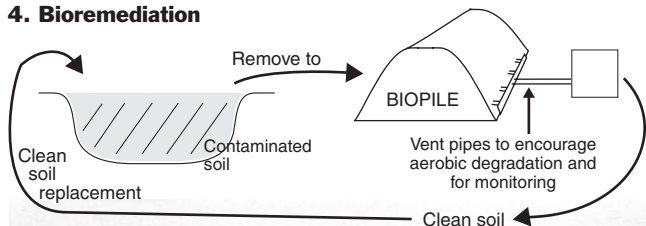


**3. Creating on-site repository**



A contaminated, abandoned mineral processing site. A containment cell is being constructed on site for depositing waste.

**4. Bioremediation**



Biopile for the remediation of diesel-contaminated soil from an old railway yard. Moist soil is stockpiled, and endogenous bacteria are stimulated to degrade diesel hydrocarbons by addition of inorganic nutrients.

into the surrounding environment will often contain a disproportionately high level of the contaminant. A vacuum cleaner with a HEPA (high efficiency particulate arresting) filter is preferred.

Environmental agencies have primary legislative responsibility for the assessment and management of contaminated sites. Importantly, this is underpinned by a range of State and Federal powers (eg, National Environment Protection Measures) that control the production, storage and disposal of waste and are designed to prevent further contamination.

**Remediation of contaminated sites**

Large sites with extensive contamination (including gasworks, railway yards and landfills) have been successfully remediated to enable safe use. The Sydney Olympics site at Homebush Bay, where a variety of methods were used (including clean soil capping over mounds of contaminated soil), is an example of large-scale remediation.

It has been more difficult to remediate contaminated groundwater that has often arisen from leaking underground storage tanks containing petroleum products. Contaminated groundwater plumes have in some instances extended a considerable distance off site. In these situations, benzene has often been the principal concern because of its ability to rapidly disperse in aquifers and its carcinogenicity.

The conventional approaches to remediating contaminated sites are shown in Box 4. Novel clean-up methods are also being considered. One involves growing crops that will bioaccumulate heavy metals from the soil — the crops can then be harvested and the contaminant extracted from them. Another is vitrification: an electric arc is generated between large buried electrodes, creating temperatures sufficient to turn the soil and its contaminants into a glass-like mass from which the contaminants cannot leach. This very expensive method has been used at the nuclear testing site of Maralinga.

Unfortunately, the high cost of remediation and the difficulty of identifying responsible parties have sometimes resulted in the persistence of unremediated wastelands.

**Conclusion**

In the coming years, we will refine our assessment and management of soil contamination and associated health risks (Box 5).

To minimise further loss of clean soil in the future, we must learn to treat it as a precious and limited resource and develop and apply improved legislative tools that will help to achieve this end.

**Competing interests**

None identified.

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**5: Reflections and predictions**

**Circa 1900**

- Natural "contamination" arising from alluvial deposits of some minerals (eg, arsenic in goldmining areas of Victoria; lead in Broken Hill) often exacerbated by mining and smelting operations.
- Older inner-city areas contaminated from many sources (eg, use of industrial waste for land reclamation; industrial debris from foundries, gasworks, incinerators, leaking underground storage tanks and flaking lead-based paints).
- Role of lead-based paint in childhood lead poisoning first recognised around this time. Turner (1897) related lead poisoning to habitation and Gibson (1904) identified source as paint on railings and walls.<sup>17</sup>
- Rural areas contaminated by use of persistent pesticides (eg, lead arsenate in orchards; arsenic in cattle dips).

**Circa 2100 (an optimistic view)**

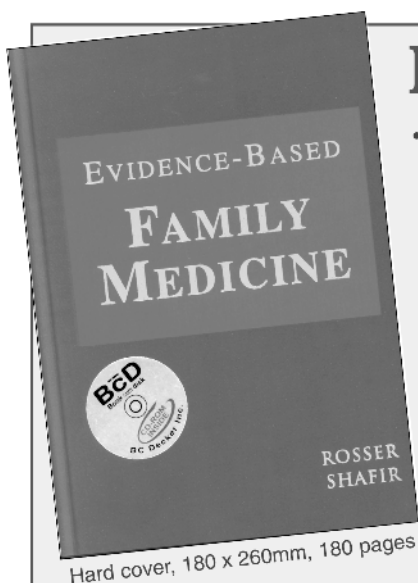
- Uncontaminated soil has become highly valued by the community. There is greater concern about contamination, with more stringent laws to control it.
- Contaminated sites have been identified and clearly mapped.
- Three-dimensional methods of site characterisation and new technologies allow contaminants to be extracted or degraded more accurately and efficiently, rather than retained in landfills.
- More accurate health-risk assessments allow better estimates of risk and better management of sites.
- Site characterisation, risk assessment and site remediation remain challenging and expensive.
- Increasing urban density, smaller block sizes and extensive paving mean that city dwellers have little exposure to soil.

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