“The scientific evidence strongly implicates cadmium as a major human toxicant ... and because it accumulates in the human body, there is a need to limit exposures to cadmium from as many sources as possible” (Mead, 2010).

HOW PREVALENT IS CADMIUM?

While cadmium is generally present in the earth’s crust and environment at low levels, human activity has greatly increased those levels above ambient. Cadmium is present in soils and rocks, in metal ores, in fossil fuels, in human and animal waste, and as an impurity in phosphate rock used to produce fertilizer.

Cadmium production in the US is declining; however, use across the world is still slightly increasing. Cadmium is used in many applications from batteries and photovoltaic cells to pigments and stabilizers, from making ceramics and glass to coating materials and components. Cadmium-coated (galvanized) products are often preferred for critical and safety applications in aerospace, defense, nuclear, mining, offshore, and electrical systems (ICdA).

Cadmium can be released to the environment through natural activities (volcanoes, wildfires, weathering and erosion of rocks), human activities (tobacco smoking, mining, smelting and refining, fossil fuel combustion, incinerating or recycling used products and waste, and manufacture of phosphate fertilizers). These releases can travel long distances by atmospheric or surface water transport. Atmospheric deposition of cadmium on arable lands along with the application of municipal sewage sludge and phosphate fertilizers has resulted in a gradual increase in cadmium levels in agricultural soils and crops (WHO, 2010).
HUMAN EXPOSURES AND HEALTH EFFECTS

Non-occupational exposure occurs mainly from active and passive inhalation of tobacco smoke and from consumption of contaminated food and water. Almost all humans carry a significant body burden of cadmium, generally increasing with age.

Cadmium, when introduced into the body, is stored in the kidneys, liver, pancreas, and bones, where it can do a significant amount of harm. The half-life in the human body has been estimated at one to four decades, so the metal tends to accumulate in bodies over time.

Cadmium can cause serious effects on renal function, bones, and the pulmonary system. It is classified by the International Agency for Research on Cancer (IARC) as a Group 1 known human carcinogen (lung by inhalation) based on human evidence and animal studies. It has also been implicated as a possible contributor to other cancers (kidney, bladder, pancreas, and hormone influenced cancers—such as breast, and uterine), and identified as a mutagen and reproductive toxin. Acute exposure may result in death from pulmonary edema. Chronic exposure is known to cause kidney damage that may increase the excretion of low molecular weight proteins and calcium. The loss of calcium and the direct effects of cadmium storage in the bones may result in loss of bone density and fractures. Although there is less conclusive evidence, cadmium has also been linked with hypertension and other cardiovascular problems, as well as neurotoxic, teratogenic, and endocrine-disrupting effects. Epidemiology studies suggest an association between cadmium exposure and the incidence and severity of diabetes.

“Environmental exposure to cadmium increases total mortality in a continuous fashion without evidence of a threshold... for each doubling of urinary cadmium concentrations, the relative risk of mortality increases 17%” (Nawrot, 2010). Men in the highest quartile of cadmium body burden were found to have a 15% higher mortality rate from all causes. The intense biomedical research on cadmium is showing the “safe exposure level” is steadily decreasing (ECHA, 2014).

Human Biomonitoring Findings

- Cadmium exposure is cumulative, the body burden of cadmium generally increases with age.
- Urinary cadmium is an indicator of cumulative, long-term exposure; the body burden of cadmium is usually estimated from urinary excretion.
- Blood levels of cadmium are indicators of recent exposure, except some times in people over 60, blood cadmium can better indicate long-term exposure than urinary cadmium.
- Smokers have the highest body burden of cadmium, smokers 50 years and older have more than double the levels found in nonsmokers.
- Vegetarians have the second highest body burden of cadmium.
- Women more efficiently absorb cadmium and have almost 50% higher urinary cadmium than men. Iron deficiency further elevates absorption.
- There are racial differences for cadmium in the US—Asians have the highest body burden of cadmium, followed by non-Hispanic blacks, non-Hispanic whites, all Hispanics, and the lowest is in Mexican Americans.
- Comparing the US and EU populations, the geometric mean for urinary cadmium is similar but the levels for the top 10% and the top 5% populations are significantly higher in the US (smokers at 1.85 μg/L and nonsmokers at 0.83 μg/L).
- Washington generally has higher cadmium levels than the US as a whole, but the top 5% population category has slightly lower levels in WA than those for the US. California has conducted two studies on specific groups—teachers and fire fighters—both show notably lower cadmium levels than the US as a whole.
It is not clear which endpoint - the adverse effects on the kidneys and bones, or carcinogenesis - should drive limit values. However, because cadmium accumulates primarily in the kidneys, the kidney is most frequently identified as the target organ. Accumulation may lead to renal tubular dysfunction and cascading effects. The critical level for the advent of tubular dysfunction in cadmium workers in Europe was estimated between 50 and 180 mg Cd per kg body weight. In the late 1990's, the average concentration of cadmium in the renal cortex in the general population in Europe between the ages of 40 and 60 years was estimated in the range of 15 to 40 mg/kg, a very narrow margin of safety (WHO 2000).

Recent studies indicate that kidney tubular damage starts at urinary cadmium concentrations of 0.5 to 2 μg Cd per gram creatinine and increased risk of osteoporosis at urinary concentrations below 1 μg/g creatinine. The top quartile of the US nonsmoking adult population has concentrations close to or higher than 0.5 μg/g creatinine (Nawrot et al., 2010).

The WHO, in partnership with the Joint Food and Agriculture Organization of the United Nations, developed cadmium guidelines for the protection of human health. These guidelines were based on preventing renal damage and loss of bone calcium. The European Food Safety Authority (EFSA) developed a tolerable weekly intake for cadmium using a similar approach but with a more conservative basis for converting urinary cadmium data to food intake. The current EFSA intake limit is less than half of the original WHO guideline.

California OEHHA established two safe harbor levels for cadmium—the no significant risk level (NSRL) based on cancer risk and the reproductive toxicity maximum allowable dose level (MADL) derived by taking 1/1000th of the no observed effects level (NOEL) (OEHHA, 2012). Proposition 65 safe harbor levels are the point below which no warning label is required. The safety factor of 1000 established for reproductive toxicity is considerably more conservative and more protective than guidelines established in an attempt to minimize increases in body burden of cadmium as people age.

**Cadmium Guidelines**

**World Health Organization**  
(WHO, 2010)
- Provisional tolerable monthly intake (PTMI) = 25 μg/kg body weight
- Drinking water = 3 μg/liter
- Ambient air = 5 ng/m3

**European Food Safety Authority Guidelines**  
(EFSA, 2011)
- Provisional tolerable weekly intake (PTWI) = 2.5 μg/kg body weight*  
  *reduced from 7 μg/kg body weight

**California Safe Harbor Levels**  
(OEHHA, 2012)
- NSRL = 0.05 μg/day (inhalation)
- MADL = 4.1 μg/day (all pathways)

**Pollution Matters**  
(NYC Health, 2004; McKelvey, 2007)

New York City conducted a survey in 2004 to measure metals in the blood of adult city dwellers. Blood cadmium concentrations were similar to the rest of the US, except for the foreign-born Chinese population. This group had significantly higher cadmium levels (9.67 μg/L for the highest) than the top 5% of current smokers (3.0 μg/L), but they also had higher mercury levels than the highest category of fish consumers and higher lead levels than the oldest New Yorkers, the highest categories for each of these pollutants. The Asian population had lower smoking rates than NYC as a whole.
Cadmium-contaminated food is the primary source of exposure for most people. Applying phosphate rock fertilizers and organic fertilizers from municipal sludge or animal waste has steadily increased soil levels over time. Cadmium may be incorporated into plants and animals as a result of the use of fertilizers containing elevated levels of cadmium (Mann, 2002). Plants readily take up cadmium from contaminated soil into their leaves, roots and tubers, and to a lesser extent their seeds, grains, and fruits.

Measurements of cadmium in common foods have shown cadmium at significant concentrations. More than 80% of dietary cadmium has been estimated to come from vegetables (especially leafy greens such as spinach, lettuce, and kale), root vegetables (especially potatoes and carrots) and cereals (especially rice and wheat) (Mead, 2010). Mushrooms are also particularly good at accumulating cadmium. Studies comparing vegetable concentrations of cadmium from conventional farming with organic farming demonstrate that, while there is variability depending on the soil type and chemistry, the differences in cadmium concentrations are not significant.

**Example European Maximum Limits in Food**

(EC 1881/2006 as amended and updated)

The EFSA set maximum limits for cadmium in food to reduce the presence of cadmium in food as much as possible. These limits are not set based on consumption rates but are set such that they are reasonably achievable by following good agricultural practices.

Maximum limits for cadmium in food are listed in mg Cd per kg of food (wet weight):

- Meat—bovine, sheep, pig, and poultry = 0.050 mg/kg (50 μg/kg)
- Fish = 0.10 to 0.30 mg/kg
- Crustaceans = 0.50 mg/kg
- Bivalve molluscs = 1.0 mg/kg
- Cereals excluding bran, germ, wheat, and rice = 0.10 mg/kg
- Bran, germ, wheat, and rice = 0.20 mg/kg
- Vegetables and fruit, excluding those below = 0.050 mg/kg
- Stem and root vegetables, potatoes = 0.10 mg/kg
- Leafy vegetables and fresh herbs = 0.20 mg/kg
- Mushrooms—common, oyster, and shiitake = 0.20 mg/kg
- Other fungi = 1.0 mg/kg
Surface waters can also collect significant cadmium loading from a variety of sources. Cadmium accumulates in many organisms. Shellfish and mollusks, especially oysters and scallops, are particularly adept at concentrating cadmium from water and sediments. Fish and other seafood have also been implicated in cadmium intake above allowable limits.

**An Oyster Problem**  
*(BC CDC, 2013)*

The North Pacific Ocean has a cadmium concentration of 100 ng/liter, a concentration 3 to 5 times greater than cadmium in the North Atlantic. It is believed that higher cadmium levels in the Pacific are primarily due to the geology and ocean currents, but with some local contributions from anthropogenic sources.

Pacific Northwest oysters have cadmium levels that range from 0.4 to 4.0 ppm wet weight. Alaskan oysters have the highest cadmium levels, followed closely by British Columbia, then Oregon and Washington.

Health Canada compared eating one typical 40 gram BC oyster to be the approximate equal of the cadmium dose of smoking 40 cigarettes. The current Health Canada policy recommends a maximum consumption rate of 12 oysters per month for adults and no more than 1.5 oysters per month for children.

**Dietary Intake > Body Burden > Health**

- A serving of 100 grams of spinach (3 cups raw or half a cup cooked) at the maximum EU limit for leafy vegetables would give you 20 μg of Cd, or, by contrast, 400 grams of meat (~ 14 ounces of steak) at the EU limit would give you 20 μg of Cd.
- Accumulation from a long-term dietary intake of 20 μg Cd per day until you reach middle age corresponds to a urinary excretion rate of about 0.8 μg Cd per liter (FEI, 2000).
- Adverse health effects are estimated to start when urinary cadmium levels are in the range of 0.5 to 1.0 μg/g creatinine (Nawrot et al., 2010).
- [Biomonitoring urinary cadmium data, measured in μg Cd/L is often normalized to μg Cd/g creatinine. This is done simply to correct for differences in hydration levels between people and in the same person over time. Data may be reported in either units but they are not directly comparable. The US National Biomonitoring Program reports the data both ways for the same sample sets. In the data sets in the years from 1999 to 2010, the ratio of μg/liter to μg/gram creatine varied from 0.98 to 1.79. Averaging, it was close to a 1.05:1 ratio for females and a 1.44:1 for males.]*
RISK MITIGATION

To decrease global environmental cadmium releases and to minimize human exposures and health impacts, the WHO made (and keeps repeating) the following recommendations (WHO, 2010):

1. **Prohibit smoking in public places.**
2. **Reduce as far as practicable emissions and discharges of cadmium from mining and smelting, from waste incineration, from application of sewage sludge to the land, and from use of phosphate fertilizers and cadmium-containing manure.**
3. **Develop techniques for the safe disposal of cadmium-containing wastes and effluents.**
4. **Promote effective measures to increase recycling of cadmium and to restrict non-recyclable uses.**
5. **Reduce occupational exposure by improving working conditions in the non-ferrous smelting industry.**
6. **Reduce exposure by disseminating information on the proper use of fertilizers.**
7. **Raise global awareness on the importance of minimizing waste discharges of cadmium.**

This report focuses on mitigating risk from the two primary sources of human exposure in the US: active or passive smoking, and contaminated food. Food may be contaminated by pollution – through leachate or by atmospheric deposition – or as a result of plant uptake of cadmium from fertilizers. In Europe, strong evidence implicates fertilizer as the dominant source of cadmium, but in places like China, with minimal pollution controls, industrial and energy sources may serve as more important factors in human exposure.

**Mitigating Smoking in Public Places**

As of December 31, 2014, in the United States 20.5% of adult males, 15.3% of adult females, and 12.7% of high school students smoke. In addition, 2.6% of adults and 5.7% of high school students use smokeless tobacco. Smoke-free legislation is limited, with none at the federal level except for government facilities. At the state level, there are restrictions in some state that prohibit smoking in specified buildings and on public transit, but there are no laws against smoking in outdoor public places. There are no fines for smoking in restricted areas and no funds dedicated for enforcement (WHO Americas online).
Mitigating Cadmium in Fertilizer, Soils, Crops, and from Leachate

Because much of the cadmium in agricultural soils comes from fertilizers, reducing cadmium content and better management of soil chemistry is necessary to inhibit plant uptake.

The average cadmium content in European phosphate fertilizers has been estimated at 138 mg Cd per kg phosphorus, but the phosphate fertilizers used in Finland are from igneous rock and have an average cadmium level of 2.5 mg Cd per kg P (FEI 2000). In Australian fertilizers, the range is from 6 to 68 mg/kg dry product [assuming a 45% P2O5 product, this translates to 30 to 344 mg Cd per kg P] (Mann, 2002). In North American fertilizers, the typical range is from 3 to 110 mg Cd per kg P but cadmium concentrations can exceed 300 mg Cd per kg dry product (about 1500 mg/kg P) (Grant, 2011).

Cadmium levels in phosphate rock from Florida average about 7.5 mg Cd kg rock. By contrast cadmium levels in phosphate rock from Idaho average 92 mg Cd per kg rock (range from 40 to 150 mg Cd per kg rock).

For ordinary single super phosphate (SSP) fertilizer and triple super phosphate (TSP) fertilizer, all of the cadmium in the phosphate rock is transferred to the fertilizer. In wet process phosphoric acid (WPA) processing, about 55 to 90% of the cadmium is transferred to the acid fraction that is used to produce mono-ammonium (MAP) and di-ammonium (DAP) fertilizer (Roberts, 2014).

Studies have shown that the application of phosphate fertilizers containing 20 to 50 mg Cd per kg led to significant increases in the concentration of cadmium in soil (Roberts 2014).

The many factors that affect cadmium soil concentrations – such as accumulation in soils, soil health, phytoavailability for crop uptake with subsequent accumulation in humans and animals, and leaching from soil that may affect surface waters – are interrelated, highly complex, and variable from one environment to another. For example, the amount of phosphorous in fertilizers
can complicate the bioavailability of cadmium through numerous changes in soil chemistry and the rate of fertilizer application can both increase at (moderate P levels) and decrease (at high P levels) cadmium uptake in plants (Grant, 2011).

Diet is the main source of human exposure to cadmium and the relative contribution of cadmium to agricultural soils from fertilizer is higher than that from atmospheric deposition in all but highly industrialized or polluted areas. Therefore, the use of cadmium-rich fertilizers can risk human health and requires careful management.

A number of governments have enacted legislation and regulations that restrict the cadmium content in fertilizers. These are listed in the table below.

<table>
<thead>
<tr>
<th>Country or State</th>
<th>Limit equivalent (mg Cd/kg P)</th>
<th>Limit equivalent (mg Cd/kg P₂O₅)</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Netherlands</td>
<td>40</td>
<td>17.5</td>
</tr>
<tr>
<td>EU Proposal*</td>
<td>137 → 91.6 → 45.8</td>
<td>60 → 40 → 20</td>
</tr>
<tr>
<td>Finland</td>
<td>50</td>
<td>21.5</td>
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<td>889</td>
</tr>
<tr>
<td>Canada</td>
<td>2040</td>
<td>889</td>
</tr>
</tbody>
</table>

*The European Commission launched an internet consultation on a draft proposal for the stepwise introduction of upper limits for cadmium in phosphate fertilizers. The proposal is well behind the initial timeline.
Only a few countries have limits that will likely protect the soil from long-term cadmium accumulation and potential exposures through food. Since current food limits in the EU are not based on health risk but on the as low as reasonably achievable (ALARA) philosophy, consuming food with cadmium levels near the upper limit may not meet dietary intake guidelines.

Finland, one of the countries with strict limits on cadmium in fertilizer, defends these limits in this way:

“In Finland, there is no margin of safety for the risk group (worst cases) between the estimated urinary levels and the critical levels that have been associated with adverse health affects caused by cadmium. Therefore, any additional human exposure to cadmium is considered unacceptable and should be avoided. While the atmospheric fallout of cadmium as well as the industrial uses of cadmium are decreasing, phosphate fertilizers are the only source of human exposure to cadmium, which will increase, in [the] case that Cd accumulates in cultivated soils.”

During the thirteen-year period between 1974 and 1987, the extractable cadmium concentration in Finnish cultivated soils increased by 30%. This was mainly caused by use of fertilizers corresponding to the average cadmium content in EU fertilizers in the year 2000 (138 mg Cd/kg P) (FEI, 2000).

As the protective thresholds continue to move downward, eliminating human exposure to cadmium becomes an increasing priority. Sufficient evidence exists to indicate little to no margin of safety between the measured lifetime accumulation (body burden) of cadmium of the top quartile of the US population and the potential onset of adverse effects. Because food is the primary exposure pathway for Americans that are not exposed through the workplace or using tobacco, more effort is needed to characterize cadmium levels in agricultural soils and foodstuffs and to establish more protective food standards and practices to reduce further cadmium entering our agricultural systems through fertilizers. §

Photo from Media Mike Hazard
REFERENCES


3. ECHA (European Chemicals Agency), 2014. “Member State Committee Support Document for Identification of Cadmium Chloride as a Substance of Very High Concern... adopted on 30 May 2014.”


