Chapter 6.3 Cadmium

General description

Cadmium (Cd) is a soft, ductile, silver-white metal that belongs together with zinc and mercury to group IIb in the Periodic Table. It has relatively low melting (320.9 °C) and boiling (765 °C) points and a relatively high vapour pressure. In the air cadmium is rapidly oxidized into cadmium oxide. However, when reactive gases or vapour such as carbon dioxide, water vapour, sulfur dioxide, sulfur trioxide or hydrogen chloride are present, cadmium vapour reacts to produce cadmium carbonate, hydroxide, sulfite, sulfate or chloride, respectively. These compounds may be formed in chimney stacks and emitted to the environment. Several inorganic cadmium compounds are quite soluble in water e.g. acetate, chloride and sulfate, whereas cadmium oxide, carbonate and sulfide are almost insoluble *(1)*.

Sources

Cadmium is a relatively rare element (0.2 mg/kg in the earth crust) and is not found in the pure state in the nature. It occurs mainly in association with the sulfide ores of zinc, lead and copper. Cadmium has only been produced commercially in the twentieth century. It is a by-product of the zinc industry; its production is thus determined essentially by that of zinc. Before the First World War cadmium was not usually recovered from zinc plants or other nonferrous metals plants, which resulted in an uncontrolled contamination of the environment for decades. The average annual production of cadmium throughout the world increased from only 20 tonnes in the 1920s to about 12 000 tonnes in the period 1960–1969, 17 000 tonnes in 1970–1984; since 1987 it has fluctuated around 20 000 tonnes *(2,3)*.

The pattern of cadmium uses has changed in recent years. In the past cadmium was mainly used in the electroplating of metals and in pigments or stabilizers for plastics. In 1960, the engineering coatings and plating sector accounted for over half the cadmium consumed worldwide, but in 1990 this had declined to less than 8%. Nowadays, cadmium-nickel battery manufacture consumes 55% of the cadmium output and it is expected that this application will expand with the increasing use of rechargeable batteries and their potential use for electric vehicles. For instance, the demand for cadmium in nickel-cadmium batteries moved from 3000 tonnes in 1980 to 9000 tonnes in 1990. This rapid growth has more than offset declining trends for pigments (20%), plating (8%) and stabilizers (10%). In many respects cadmium has become a vital component of modern technology, with countless applications in the electronics, communications, power generation and aerospace industries *(4,5)*.

In the European Union and worldwide, approximately 85–90% of total airborne cadmium emissions arise from anthropogenic sources, mainly from smelting and refining of nonferrous metals, fossil fuel combustion and municipal waste incineration. The natural source of cadmium is volcanic emissions. The total atmospheric emission of cadmium in western Europe was estimated at 1144 tonnes/year in 1982. It is estimated that with the application of the best available technology to control emissions in nonferrous smelters this amount can be cut by 34% in the 1990s *(6)*. In European Union Member States, atmospheric emissions of cadmium in 1990 amounted to 158 tonnes/year distributed as follows: natural, 9.3%; nonferrous metal industry, 20.4%; oil combustion, 17.9%; waste incineration, 17.5%; iron and steel industry, 15.3%; coal combustion, 13.4%; cement manufacture, 4.4%; and others, 1.8% *(7)*.

Occurrence in air

Most of the cadmium that occurs in air is associated with particulate matter in the respirable range (diameter 0.1–1 μm). Cadmium is emitted to the atmosphere predominantly as elemental cadmium and cadmium oxide and from some sources as cadmium sulfide (coal combustion and nonferrous metal production) or cadmium chloride (refuse incineration). The residence time of cadmium in air is relatively short (days to weeks) but sufficient to allow long-range transport in the atmosphere. Information on the concentrations and deposition rates of atmospheric cadmium is available mostly from northern European countries, in particular Norway, Denmark, Sweden, Netherlands, Belgium and Germany. A recent review of measurement data available for the period 1980–1988 in these countries gives mean annual levels of around 0.1 ng/m^3 in remote areas (e.g. northern Norway), 0.1–0.5 ng/m^3 in rural areas, 1–10 ng/m^3 in urban areas and 1–20 ng/m^3 in industrial areas; levels of up to 100 ng/m^3 can be found in the proximity of emission sources. Mean annual wet deposition rates of cadmium are 0.02–0.08 mg/m^2 in remote areas, 0.04–0.4 mg/m^2 in rural areas, 0.2–3.3 mg/m^2 in urban areas, and 0.8–3.3 mg/m^2 in industrial areas. Mean annual dry depositions vary similarly from less than 1 mg/m^2 in remote areas up to more than 500 mg/m^2 around emission sources *(8)*.

A downward trend in both the air concentrations and the deposition rates of cadmium was observed from the mid-1970s to the end of the 1980s in several areas of northern Europe. For example, in Belgium, the mean annual concentration of cadmium around the main emission point (Hoboken lead smelter) decreased from 150 ng/m^3 in 1981 to 60 ng/m^3 in 1991. In the same area, total cadmium deposition decreased from about 160 to 20 mg/m^2 per year between 1983 and 1992 *(9)*. A similar trend is described by Ewers *(10)* for Germany, in the industrialized area of Stolberg and in the west Ruhr area. For example, atmospheric cadmium concentrations 1 km from the Stolberg smelter and in the west Ruhr area were close to 60 and 10 ng/m^3, respectively, in 1974 but had decreased to less than 20 and 5 ng/m^3, respectively, in 1988.

Routes of exposure

Air

Assuming a daily inhalation of 20 m^3 of air and indoor concentrations similar to those outdoors, the average amount of cadmium inhaled daily by humans in rural, urban and industrialized areas should not exceed 0.01, 0.2 and 0.4 μg, respectively. Deposition of inhaled cadmium in the lungs varies between 10% and 50% depending on the size of airborne particles. Absorption of cadmium in the lung depends on the chemical nature of the particles deposited. It is around 50% for cadmium oxide but considerably less for insoluble salts such as cadmium sulfide.

Cigarette smoking may represent an additional source of cadmium which may equal or exceed that from food. Depending on the brand (i.e. mainly on the origin of the tobacco), cigarettes produced in Europe or the United States of America contain cadmium at a concentration of 0.5–2 μg/g (dry weight) of tobacco, of which 10% can be absorbed *(2)*.

Drinking-water

Drinking-water contains very low concentrations of cadmium, usually in the range 0.01–1 μg/litre. In a survey in the Netherlands, about 99% of drinking-water samples in 1982 contained less than 0.1 μg/litre. Levels of up to 5 μg/litre have been reported occasionally

and, on rare occasions, of up to 10 µg/litre *(1)*. In polluted areas, well-water may contain very high concentrations of cadmium (exceeding 25 µg/litre) *(11)*. Such unusual situations excepted, the intake of cadmium via drinking- water, based on a water consumption of two litres per day is thus very low.

Food

For nonsmokers, food constitutes the principal environmental source of cadmium. The lowest concentrations are found in milk (around 1 µg/kg). The concentration of cadmium is in the range 1-50 µg/kg in meat, fish and fruit and 10-300 µg/kg in staple foods such as wheat, rice and potatoes. The highest cadmium levels (100-1000 µg/kg) are found in the internal organs (kidney and liver) of mammals and in certain species of mussels, scallops and oysters. When grown on a cadmium-polluted soil, some crops, such as rice, can accumulate considerable amounts of cadmium (more than 1000 µg/kg). The average daily intake of cadmium via food in European countries and North America is 15-25 µg but there may be large variations depending on age and dietary habits. In Japan, the average intake is generally 40-50 µg but may be much higher in cadmium-polluted areas. The gastrointestinal absorption of cadmium in humans amounts to about 5% but may be increased by nutritional factors (up to 15% in iron deficiency). The average amount of cadmium absorbed via food can thus be estimated at about 1 µg/day *(1,12)*.

Relative significance of different routes of exposure

Assuming a mean absorption of 15% for inhaled cadmium, and excepting special circumstances, such as living close to a cadmium emission source, the amount of cadmium absorbed daily by the pulmonary route in nonsmokers does not exceed 0.0015 µg in rural areas, 0.03 µg in urban areas and 0.06 µg in industrialized areas. These amounts are at least one order of magnitude lower than that absorbed via food (1 µg for the average European) or by smoking one pack of cigarettes per day (1.4 µg).

Cadmium in food comes to a large extent from atmospheric cadmium as a result of foliar absorption or root uptake of cadmium deposited on soils. Foliar absorption is determined by dry or wet deposition rates of cadmium and uptake of cadmium by plants from soils is primarily controlled by the concentration of cadmium in soil solution. In rural areas, 20–60% of the total plant cadmium may originate from foliar absorption *(8)*. It is important to stress that excessive levels of cadmium in soil (>1 mg/kg) presently found in some industrialized areas are largely the result of emissions in the past when smelters were operating under less stringent conditions and when cadmium was not recovered during zinc production. In Belgium, for instance, airborne cadmium emissions were estimated at 125 000 kg in 1950 and dropped to less than 130 kg in 1989 *(13)*. The transfer of cadmium from soil to the food-chain depends on a number of additional factors, such as the type of plant, the type and pH of the soil, and the zinc and organic matter content of the soil. These factors explain why a transfer of cadmium from soil to plants and humans has been demonstrated in some polluted areas in Europe *(14,15)* but not others *(16,17)*.

Another important source of contamination of soils is the use of commercial fertilizers derived from rock phosphate and sewage sludge. Studies in Denmark and the Netherlands indicate that current inputs of cadmium to the soil from this source exceed that of atmospheric deposition by 35–184% depending on the area. The yearly increases of cadmium in soil in Denmark and the Netherlands are estimated at between 0.4% (sandy soils) and 0.79% (clay soils) corresponding to doubling times of 250 and 125 years, respectively.

In summary, compared to smoking and dietary sources, the direct contribution of airborne cadmium to human exposure is very low and has probably declined during the last decade in parallel with the reductions in atmospheric emissions. However, as cadmium in air is ultimately deposited on soils, which determine future dietary intakes via plants, it is important to ensure that emissions of cadmium to air, added to the fertilizers inputs, does not lead to a progressive rise of the cadmium levels in cultivated soils.

Toxicokinetics

The main metabolic feature of cadmium is an exceptionally long biological half-life resulting in a virtually irreversible accumulation of the metal in the body throughout life. In blood, more than 90% of cadmium is found in cells. In adults not exposed to cadmium at work, the cadmium level in blood is usually less than 0.5 μg/100 ml. During exposure to cadmium, the blood concentration of cadmium is mainly an indicator of the absorption over the previous few months. In persons with previous high exposure (e.g. retired workers) the blood concentration may be predominantly influenced by the body burden if the amount of cadmium released from storage sites exceeds the amount currently being absorbed.

The two main storage sites for cadmium in the body are the liver and the kidney. Newborn infants are virtually free of cadmium but during their lifetime there is a considerable accumulation of cadmium in these two organs, which contain about 40–80% of the body burden. In the case of low-level exposures, such as those occurring in the general environment, about 30-50% of the cadmium body burden is stored in the kidneys alone, with concentrations in the cortex about 1.25 times higher than in the kidney as a whole. In non-occupationally exposed subjects, the concentration of cadmium in the liver increases continuously with age. The concentration also increases in the renal cortex but only until the age of 50-60 years, after which it levels off or even decreases. In Europe, mean concentrations of cadmium in the renal cortex in the age group 40-60 years are in the range 15-50 mg/kg. The highest values have been reported from the Liège area in Belgium. Concentrations are usually 50-100% higher in current or exsmokers than in nonsmokers. In industrial workers, the concentration of cadmium in the renal cortex may be as high as 300 mg/kg. However, when the integrity of renal structures is compromised by cadmium itself or by other factors (e.g. ageing), the cadmium concentration in the kidney decreases. In the tissues, cadmium is mainly bound to metallothioneine, a low-molecular-weight protein (MW 6.6 kD) rich in cysteine residues. The synthesis of this protein probably represents a defence mechanism against the toxic cadmium ion. It is also hypothesized that, by virtue of its small size, metallothioneine is involved in the transport of cadmium from the liver to the kidney, the cadmium-metallothioneine complex released from the liver being rapidly filtered through the glomeruli then reabsorbed by the tubules. Such a mechanism could explain the selective accumulation of cadmium in the renal cortex *(2,12)*.

Cadmium is eliminated from the organism mainly via urine. The amount of cadmium excreted daily in urine is, however, very small; it represents only about 0.005-0.01% of the total body burden which corresponds to a biological half-life for cadmium of about 20-40 years. In subjects non-occupationally exposed to cadmium, the urinary excretion of cadmium is usually less than 2 μg/g of creatinine. A substantial body of evidence derived from numerous human and experimental studies indicates that in the general population and in workers moderately exposed to cadmium, the urinary excretion of cadmium on a group basis

is a reliable indicator of the cadmium body burden. The urinary excretion of cadmium is thus proportional to the body burden and, in non-occupationally exposed subjects, increases with age at least up to 50-60 years. The main determinants of cadmium excretion in the general population are sex (tends to be higher in women than men), age, smoking habits and place of residence *(18)*. In urine, cadmium is partly bound to metallothioneine, which therefore has the same significance as urinary cadmium *(2,12)*.

Health effects

Effects on experimental animals and *in vitro* test systems

In experimental animals, cadmium can produce acute toxic effects on various organs, such as the kidney, liver, pancreas, testes and lung (by inhalation). In chronically intoxicated animals, cadmium gives rise to a nephropathy very similar to that described in humans and characterized functionally by the appearance of a tubular or mixed-type proteinuria, aminoaciduria, glucosuria and hypercalciuria and, morphologically, by lesions predominantly involving the tubules. It is noteworthy that the critical concentration of cadmium in the renal cortex associated with these changes in rats is around 200 mg/kg, as in humans. Other chronic effects which have been described in animals treated with cadmium include lung emphysema and inflammation (by inhalation), disturbances in calcium and vitamin-D metabolism resulting in bone lesions, hepatic damage and effects on the pancreas, testes or cardiovascular system. Cadmium can also produce embryotoxic, teratogenic and carcinogenic effects (see below) *(19)*.

Effects on humans

Toxicological effects

Short-term exposure to moderate concentrations (200-500 $\mu g/m^3$) of freshly generated cadmium fume during less than 1 hour may cause symptoms similar to those of the metal fume fever, usually with a complete recovery within a few days. More intense or prolonged exposure may lead, again after a latency period of several hours, to a chemical pneumonitis with death in 15-20% of cases. Chronic respiratory effects consisting of bronchitis, obstructive lung disease or emphysema have been described in the past in workers heavily exposed to cadmium (more than 20 $\mu g/m^3$ for more than 20 years). With the progressive reduction of acceptable exposure levels in industry, the risk of chronic lung effects in workers has almost disappeared *(20)*.

The kidney is the critical organ after long-term occupational or environmental exposure to cadmium. Since the first report by Friberg in 1948, numerous epidemiological studies on industrial workers or on inhabitants of cadmium-polluted areas have documented the constellation of renal effects that may be produced by this heavy metal *(12,19,21)*. Because of intrinsic differences in their sensitivity or the sequential involvement of specific sites of the nephron, these indicators become abnormal at different levels of cadmium body burden. This variable response is reflected by the thresholds of urinary excretion of cadmium (CdU) associated with an increased probability of renal changes. As shown in Table 1, some of these effects appear at CdU thresholds clearly inferior to that associated with the onset of classical

Table 1. Thresholds of cadmium exposure associated with an increased probability of renal effects

	Industrial workers			General population	
				Belgium	Japan
Renal effect	**CdU (µg/g of creatinine) *(22,23)***	**Cadmium in renal cortex (mg/kg) *(24)***	**Cadmium in air: cumulative exposure (µg/m^3-years) *(8)***	**CdU (µg/24 hours) *(26)***	**CdU (µg/g of creatinine) *(12,27)***
Increased urinary excretion of NAG-B	0.5–2	–	–	–	–
Biochemical alterations (Increased urinary excretion of sialic acid, prostaglandins)	2–3	110–127	–	–	–
Increased urinary excretion of tubular enzymes or antigens (total NAG, AAP, etc.) High-molecular-weight proteinuria (increased urinary excretion of albumin or transferrin) and increased serum ß2-microglobulin	4–6	139–157	–	1.9 (NAG)	–
Low-molecular-weight proteinuria (increased urinary excretion of serum ß2-microglobulin, RBP, CC16)	= 10	= 182	500–1000	2.9–3.1	3.8–6.3

Note: AAP = alanine aminopeptidase; CC16 = Clara cell protein; CdU = cadmium in urine; NAG = *N*-acetylglucosaminidase; NAG-B = NAG released with cell membranes; RBP = retinol-binding protein.

tubular proteinuria (10 µg/g creatinine). For some indicators, such as *N*-acetylglucosaminidase (NAG) released with cell membranes, the CdU threshold even falls into what is currently regarded as the normal range for populations exposed only to environmental cadmium *(22)*.

With respect to occupational exposure (which occurs mainly by inhalation), a recent study *(23)* has identified three main groups of thresholds: one at around 2 µg/g of creatinine mainly associated with biochemical alterations; a second at around 4–5 µg/g of creatinine for the onset of high-molecular-weight proteinuria and of some cytotoxicity signs (tubular antigens or enzymes); and a third at around 10 µg/g of creatinine for the development of tubular proteinuria. These findings have led to a reassessment of the critical concentration of cadmium in the renal cortex, which was estimated at about 200 mg/kg in the early 1980s. The recent reassessment was based on the relationship between urinary cadmium and the cadmium concentration in the renal cortex of workers, as determined by neutron activation analysis *(24)*. The average concentrations of cadmium in the renal cortex corresponding to CdU thresholds of 2, 4 and 10 µg/g of creatinine are 110, 139 and 182 mg/kg, respectively (Table 1). Only the effects associated with the CdU threshold of 10 µg/g of creatinine (182 mg/kg in the renal cortex) are known to be irreversible and to predict a faster decline of the renal function with age in occupationally exposed workers *(25)*. The health significance of effects occurring at lower thresholds and their link with the subsequent development of cadmium nephropathy are still unknown. A few epidemiological studies were carried out in Europe at the end of the 1980s to determine whether environmental pollution by cadmium gives rise to renal effects. The largest of these was the Cadmibel study, which was carried out in Belgium between 1985 and 1989 in areas where the soil was contaminated by past emissions of cadmium from zinc smelters *(26)*. The main conclusion was that subclinical renal effects are likely to occur in the general population with a probability of 10% (relative risk 2) when CdU exceeds 2–4.3 µg/24 hours. It was estimated that 10% of the general population in Belgium had a cadmium excretion higher than the threshold of 2 µg/24 hours. Similar findings were made in a cross-sectional study carried out in the Netherlands in the area of Kempenland, on the Dutch-Belgium border *(15)*. In Japan, renal effects consisting of low-molecular-weight proteinuria have been reported at CdU thresholds in the same range as those found in the Cadmibel study (Table 1) *(12,27)*. A recent study in Japan suggests that proteinuria induced by environmental exposure to cadmium has a poor prognosis and is associated with an increased mortality rate *(28)*.

Carcinogenic effects

In 1993, IARC classified cadmium and cadmium compounds as group 1 human carcinogens, having concluded that there was sufficient evidence of cadmium being carcinogenic to humans and animals. The evidence for carcinogenicity in humans was mainly based on the observation of excess lung cancer mortality among cohorts of workers in a United States cadmium recovery plant and from United Kingdom cadmium processing plants. In the United States cohort, a dose–response relationship was demonstrated between estimated cumulative exposure to cadmium and lung cancer risk. The latter was not thought likely to be due to confounding by cigarette smoking or exposure to arsenic. Excess mortality from prostatic cancer was found initially but the relative risk diminished and became insignificant during further follow up. In the United Kingdom cohort, there were suggested trends with duration of exposure and with intensity of exposure. The mortality from prostatic cancer was decreased. There was no control for confounding by concomitant exposure to other cancer determinants, including arsenic.

Classification of cadmium as an animal carcinogen was based mainly on two inhalation studies in rats showing a dose-related induction of malignant lung tumours by cadmium chloride, cadmium sulfide/sulfate, cadmium sulfate and cadmium oxide fume and dust. In making the overall assessment, IARC took into consideration the evidence that ionic cadmium produces genotoxic effects in a variety of eukaryotic cells, including human cells *(3)*.

Evaluation of human health risks

Exposure evaluation

It is not possible to carry out a dose–response analysis for cadmium in air solely on the basis of epidemiological data collected in the general population, since the latter is exposed to cadmium mainly via food or tobacco smoking. In addition, epidemiological studies which have recently reported renal effects in areas of Belgium and the Netherlands polluted by cadmium refer to historical contamination of the environment. Assuming, however, that the only route of exposure is by inhalation, an indirect estimate of the risk of renal dysfunction or lung cancer can be made on the basis of data collected in industrial workers.

Health risk evaluation

Pooled data from seven studies which have examined the relations between the occurrence of tubular proteinuria and cumulative cadmium exposure show that the prevalence of tubular dysfunction (background level 2.4%) increases sharply at cumulative exposure of more than 500 $\mu g/m^3$-years (8% at 400 $\mu g/m^3$-years, 50% at 1000 $\mu g/m^3$-years and >80% at more than 4500 $\mu g/m^3$-years) *(29)*. Some studies suggest that a proportion of workers with cumulative exposures of 100–400 $\mu g/m^3$-years might develop tubular dysfunction (prevalences increasing from 2.4% to 8.8%, increase above background from 200 $\mu g/m^3$-years). These estimates agree well with that derived from the kinetic model of Kjellström *(30),* which predicted that the critical concentration of 200 mg/kg in the renal cortex will be reached in 10% of exposed workers after 10 years of exposure to 50 $\mu g/m^3$ and in 1% after 10 years of exposure to 16 $\mu g/m^3$ (cumulative exposures of 500 and 160 $\mu g/m^3$-years, respectively).

With respect to the risk of lung cancer two risk estimates have been made, one based upon the long-term rat bioassay data of Takenaka et al. *(31)* and the other on the epidemiological data of Thun et al. *(32)*. Modelling of these data yielded risk estimates that did not agree. On the basis of the Taneka data, the unit risk is 9.2×10^{-2} $(\mu g/m^3)^{-1}$; the human data yielded a unit risk of 1.8×10^{-3} $(\mu g/m^3)^{-1}$. In general, the use of human data is more reliable because of species variation in response. However, there is evidence from recent studies *(33,34)* that this latter unit risk might be substantially overestimated owing to confounding by concomitant exposure to arsenic.

Some uncertainty exists with regard to the thresholds of exposure associated with effects on the kidney. This is primarily due to the limited number of subjects, methodological differences and inaccuracies in exposure data. An overall assessment of the data from industrial workers suggests that, to prevent tubular dysfunction, the 8-hour exposure level for cadmium should not exceed 5 $\mu g/m^3$. This corresponds to a cumulative exposure of 225 $\mu g/m^3$-years. Adopting the lowest estimate of the critical cumulative exposure to airborne cadmium (i.e. 100 $\mu g/m^3$-years), extrapolation to continuous lifetime exposure results in a permissible concentration of about 300 ng/m^3.

As indicated earlier, cadmium in ambient air is transferred to soil by wet or dry deposition and can enter the food-chain. However, the rate of transfer from soil to plant depends on numerous factors (type of soil and plant, pH, use of fertilizers, meteorology, etc.) and is impossible to predict.

Present average concentrations of cadmium in the renal cortex in the general population in Europe at the age of 40–60 years are in the range 15–40 mg/kg. These values are only 4–12 times lower than the critical levels estimated in cadmium workers for the induction of tubular dysfunction (180 mg/kg, Table 1) and very close to the critical level of 50 mg/kg estimated by the Cadmibel study in Belgium. Any further increase in the dietary intake of cadmium owing to an accumulation of the metal in agricultural soils will further narrow the gap to these critical levels. It is thus imperative to maintain a zero balance for cadmium in agricultural soils by controlling and restricting inputs from fertilizers (including sewage sludge) and atmospheric emissions. Since emissions from industry are currently decreasing, attention must be focused on the emissions from waste incineration which are likely to increase in the future.

Guidelines

IARC has classified cadmium and cadmium compounds as group 1 human carcinogens, having concluded that there was sufficient evidence that cadmium can produce lung cancers in humans and animals exposed by inhalation. However, because of the identified and controversial influence of concomitant exposure to arsenic in the epidemiological study, no reliable unit risk can be derived to estimate the excess lifetime risk for lung cancer.

Cadmium, whether absorbed by inhalation or via contaminated food, may give rise to various renal alterations. The lowest estimate of the cumulative exposure to airborne cadmium in industrial workers leading to an increased risk of renal dysfunction (low-molecular-weight proteinuria) or lung cancer is 100 $\mu g/m^3$-years for an 8-hour exposure. Extrapolation to a continuous lifetime exposure gives a value of around 0.3 $\mu g/m^3$. Existing levels of cadmium in the air of most urban or industrial areas are around one-fiftieth of this value.

The finding of renal effects in areas contaminated by past emissions of cadmium indicates that the cadmium body burden of the general population in some parts of Europe cannot be further increased without endangering renal function. In order to prevent any further increase of cadmium in agricultural soils likely to increase the dietary intake of future generations, a guideline of 5 ng/m^3 is established.

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