Cadmium in Fertilizers
Risks to human health and the environment

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Abstract

The aim of this report was to estimate impacts of cadmium in phosphorus fertilizers on a cadmium content and cadmium balance of the cultivated soil, and to assess related human health and environmental risks under Finnish conditions.

The assessment was performed by applying, as appropriate, the EU principles on risk assessment of New and Existing Substances, and the guidance document prepared by Environmental Resources Management Limited for the risk assessment of cadmium in fertilizers. The work was carried out on the basis of the available literature and other information relating to the occurrence of cadmium in the environment in Finland, harmful environmental influences of cadmium, as well as its adverse health effects.

Based on the cadmium mass balances hundred-year scenarios were calculated at three different cadmium levels in P-fertilizers: 1) the present Finnish average cadmium level in fertilizers (i.e. 2.5 mg Cd/kg P), 2) the cadmium level corresponding to the national limit (i.e. 50 mg Cd/kg P), and 3) the cadmium level corresponding to an average European value (138 mg Cd/kg P).

At present, cadmium concentrations in the Finnish cultivated soils pose a risk to soil organisms. Also, the leaching of cadmium from the arable land is causing a risk to the aquatic environment. However, if the use of the low cadmium P-fertilizers will be continued in Finland, it will result in a balance between cadmium inputs and outputs or even in a gradual decrease in the cadmium concentrations of soil, crops and leachates. The use of P-fertilizers containing cadmium at a level of the Finnish national limit value and, especially at a level of the average European content, would increase the risk to the environment.

At present, a part of the elderly population in Finland is at risk from adverse health effects caused by cadmium. These risk groups consist of people whose dietary intake of cadmium has increased by using much of kidneys, liver, mushrooms, or molluscs in their diet, people whose gastrointestinal absorption of cadmium has increased because of deficiency of iron and heavy smokers. In these risk groups, the estimated urinary level of cadmium can be associated with high risk of kidney dysfunction and bone effects. An increase in the cadmium content of P-fertilizers would increase the size of the risk groups considerably.

Keywords	Cadmium, cultivated soil, environmental impacts, exposure, fertilizer, health effects, mass balance, modeling, risk assessment			
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FOREWORD

The aim of this report is to estimate the impact of cadmium in fertilizers on cadmium content and cadmium balance in cultivated soil, and to assess the related human health and environmental risks under Finnish conditions.

The assessment has been performed by applying, as appropriate, the adopted EU principles on risk assessment of New and Existing Substances, and the guidance document prepared for this purpose by Environmental Resources Management Limited (ERM). The work has been carried out on the basis of available literature and other information relating to the occurrence of cadmium in the environment in Finland, the environmental hazards of cadmium, as well as its adverse health effects.

The report updates and supplements the corresponding Finnish report which was published in 1997 (Cadmium in Fertilizers; Risks to Human Health and the Environment, Publications of the Ministry of Agriculture and Forestry 9/1997).

The work has been commissioned by the Finnish Ministry of Agriculture and Forestry. Persons and institutions in charge include:

Senior Adviser Jukka Malm, Finnish Environment Institute: Project coordination, environmental risks, Senior Advisers Jaana Pasanen & Virpi Virtanen, Finnish Environment Institute: Environmental risks, Dr. Kimmo Louekari, Finnish Occupational Health Institute: Risks for human health, Senior Scientist Ritva Mäkelä-Kurtto, Agricultural Research Centre of Finland: Cadmium in agroecosystem and in accumulation module, Professor, Dr. Jouko Sippola, Agricultural Research Centre of Finland: Model calculations in cadmium accumulation module.

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EXECUTIVE SUMMARY

The aim of this report is to estimate the impact of cadmium in fertilizers on cadmium accumulation in cultivated soils and plants and cadmium leaching in waters from the soil, and to assess the related human health and environmental risks under Finnish conditions.

The assessment has been performed by applying, as appropriate, the adopted EU principles on risk assessment of New and Existing Substances, and the guidance document prepared for this purpose by Environmental Resources Management (ERM). The work has been carried out on the basis of available literature and other information relating to the occurrence of cadmium in the environment in Finland, the environmental hazards of cadmium, as well as its adverse health effects.

In the assessment, special emphasis has been put on vulnerable environments and risk groups in human populations, representing realistic worst case situations.

Cadmium in fertilizers and in cultivated soils

The main anthropogenic sources of Cd in cultivated soils are phosphorus-fertilizers, atmospheric deposition, animal manures, and to a smaller extent liming agents, sewage sludge and biowaste. In Europe, fertilizers have given significant, although highly variable inputs from 0.03 to 38 g/ha/year to cultivated soils.

The average cadmium content in European fertilizers is 138 mg/kg phosphorus. Presently, the P-fertilizers used in Finland are manufactured from phosphate rock of igneous origin, and have a cadmium concentration from 1 to 5 mg/kg P. Due to this low Cd level, fertilizers only contribute about 4% to the total Cd input to Finnish cultivated soils.

The mean total cadmium content in Finnish cultivated soils is 0.21 mg/kg, which is the same level as in Sweden and Denmark. The average total Cd content of cultivated soils in Europe is 0.5 mg/kg. There are distinct regional differences in the soil Cd level in Finland. Cultivated soils in Southern Finland contain nearly twice as much cadmium as the soils in Northern Finland, which can be attributed to intensive agriculture, larger atmospheric fallout, and to differences in soil types.

The mean AAAc-EDTA extractable Cd concentrations in the five Finnish cultivation zones in 1987 vary from 0.127 in the south to 0.049 mg/kg in the north.

Cadmium is relatively soluble or easily solubilizable in soil, with the pH being the principal factor determining the degree of solubility. Other factors leading to increased solubility include for example low organic matter content, low clay content and low cation exchange capacity. Cadmium from fertilizers seems to be more soluble in the soil than Cd from deposition, manure, or Cd from soil parent material. In general, cadmium is extremely easily taken up by plants as compared to many other trace elements.

Trends of cadmium accumulation in soil and crops

Hundred-year scenarios based on cadmium mass balances were calculated at three different cadmium levels in P-fertilizers: i) present Finnish average Cd level in fertilizers (i.e. 2,5 mg Cd/kg P), ii) Cd level corresponding to the national limit (i.e. 50 mg Cd/kg P), and iii) Cd level corresponding to average European values (138 mg Cd/kg P).

The average EU scenario would lead to radical increase in concentrations of cadmium in soil and crops, and in cadmium leaching. The highest impacts would be 125 % increase in total soil Cd concentrations in potato fields, 34% increase in the concentrations of wheat grains, and 123% increase in cadmium leaching. Also increase in the Cd content of fertilizers up to the present national limit value would lead to significant increases in

soil and crop concentrations, and to Cd leaching. The corresponding figures would be 43%, 12% and 42%, respectively.

Figures estimated in this report can be compared with the observed trend found in Finland in a period of 13 years (1974-1987). The measured concentrations of AAAc-EDTA extractable Cd in cultivated soils increased by 30% during that period. The increase was mainly due to exceptional use of Cd-rich fertilizers (corresponding average EU levels) between 1975-1981, and, to a lesser extent, to atmospheric fallout.

Environmental aspects

The toxicity of cadmium to terrestrial organisms shows a variable pattern. Plants grown in soil are generally considered insensitive to the effects of cadmium, although some exceptions do exist. Cadmium exhibits moderate or low toxicity to avian species in subacute exposure. Microorganisms and terrestrial invertebrates show moderate or high sensitivity to cadmium.

The predicted no-effect concentration (PNEC) was derived with the assessment factor method. Statistical method for PNEC derivation was not used, as there is a considerable amount of issues under discussion within EU in relation to its use. A PNEC of 0.06 mg/kg (total Cd) for agricultural soil was derived. Also an indicative PNEC based on soil pore water concentration was established which, however, is associated with considerable uncertainty.

Cadmium is toxic or very toxic to a variety of aquatic organisms both in short-term and in long-term exposure. Invertebrates are usually considered as the most sensitive group. Cadmium is more toxic to aquatic species at low pH, low salinity, and in soft water, due to increased bioavailability. A soft water PNEC of 0.0085 µg/l was derived, also in this case with the assessment factor method.

In risk characterization for metals in the soil the PEC and the PNEC should represent the same level of availability. Due to the uncertainties in determining the soil pore water concentrations the results based on them are not preferred. Instead, the most reliable comparison of exposure and no-effect levels in this study can be performed using total Cd-concentrations for effect studies, which usually apply soluble Cd-compounds, and extractable (AAAc-EDTA) concentrations for soil concentrations. In general, 40% of the total soil cadmium is in extractable form in Finnish soils.

Based on the available data the current Cd concentrations in Finnish agricultural soils present a risk for the soil environment. This conclusion can be reached both by using mean extractable Cd concentrations and 90 percentile values in the five Finnish cultivation zones for the year 1987. Only in the most northern zone the PEC/PNEC ratio using the mean concentration is below 1. Risk ratios for the different cultivation zones vary from 1.2 to 2.8.

The increasing soil concentrations based on trend calculations, as described above, would lead to greater risks, accordingly. Risk ratios with the average EU Cd level in fertilizers would increase from 40 to 130 per cent. The highest risk ratio (3.2) after 100 years would be found on potato fields.

There are two remarkable differences in the soil characteristics between Finland and most other EU Member States affecting the cadmium content and solubility. They are soil pH and soil organic matter. The mean pH of Finnish cultivated soils is only 5.8, in spite of intensive liming. Mostly, the pH values of cultivated soils in the European countries are around 7. The other important difference is the high organic matter content of Finnish soils, due to the slow decomposition rate of organic matter as a result of the cold and humid climate. The mean organic carbon content of cultivated soils in Finland is 9% and consequently a mean organic matter content 15%. Typical soil organic matter contents in other European countries vary between 1% and 4% These two factors mean that Finnish soils are capable to accumulate large amounts of cadmium in a soluble or easily solubi-

lizable form, and thus can be generally regarded as vulnerable environments.

The exposure of aquatic environment to cadmium leaching from agricultural soils was assessed based on soil solution concentrations, and by taking into account dilution and background concentrations in Finnish waters.

Risk characterization reveals that there is risk for aquatic environment for all calculated scenarios, both at present time and in the future. In practise this means that no margin of safety can be established, and any increase to the natural background concentrations is likely to present a risk for the aquatic environment.

The bioavailability, and consequently toxicity, of cadmium in Finnish surface waters is increased by some specific features of the Finnish environment. Firstly, the pH value of Finnish lakes is generally low (median pH 6.6, pH < five in 10% of lakes). Secondly, the organic matter content is high, and thirdly, Finnish waters are very soft (mean hardness ≤ 10 mg CaCO3). Hence, also the Finnish aquatic environment is particularly vulnerable for adverse effects caused by cadmium.

Human health aspects

Diet is the main source of human exposure to cadmium. Cereals, vegetables and potatoes cause most of the average dietary intakes of cadmium in Finland (10 µg/day). In some sub-populations, which prefer certain types of food such as liver, kidney, mushrooms, and molluscs in their diet or which have a high caloric intake, a long-term dietary intake of cadmium is 20-30 µg/day in Finland. This corresponds a urinary cadmium level of about 0.8-1.2 µg/l in the middle-aged and elderly population.

Normally, only 5% of the cadmium in food is absorbed in the gastrointestinal tract. However, studies on humans and experimental animals show that a deficiency of iron, calcium or protein significantly increases (up to 10%) the rate of absorption. The most relevant nutritional deficiency in Finland, is a low iron status, which is not uncommon in women.

Heavy smoking remarkably increases the body burden of cadmium, because a significant proportion of the cadmium in tobacco smoke is absorbed from lungs. Body burden and urinary cadmium of heavy smokers is two- or threefold as compared with nonsmoker. Drinking water and certain consumer goods (e.g. utensils, decorated pots) may occasionally increase the total cadmium-exposure.

The three main factors of total cadmium exposure: high dietary intake of cadmium, increased absorption and smoking are considered in the risk assessment. The corresponding urinary levels of cadmium and the critical urinary levels that are associated with health effects are compared. It is concluded that for high exposure groups (i.e. in worst cases), the urinary Cd level is about 2-3 µg/l. This is also the level of urinary excretion of cadmium associated with high risk of cadmium-induced kidney dysfunction and bone effects, which are due to the increased excretion of cadmium. In some populations, 10% of those having an elevated Cd level in urine suffer from cadmium-induced health effects. There are studies, which indicate that the critical U-Cd level in respect to increased calcium excretion may be lower.

Based on the present risk assessment, a part of the elderly population in Finland is at risk of adverse health effects caused by cadmium. It is estimated that about one thousand individuals are at risk. Use of phosphate fertilisers, which contain 138 mg Cd/mg of P (the average level in Europe), would increase the dietary intake of cadmium from 9.5 to 13 µg/day in hundred years, (based on algorithms recommended by ERM). Due to distribution of cadmium exposure in the Finnish population, this causes that the size of the risk groups would be several times greater, approximately 5 000 -10 000. These results are not based on epidemiological studies in Finland. However, consideration of population studies made elsewhere and estimated on the urinary cadmium levels in

Finland justifies, with reasonable certainty, the conclusions presented in this risk assessment.

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 effects 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 fertilisers are the only source of human exposure to cadmium, which will increase, in case that Cd accumulates in cultivated soils.

As compared to many other risk assessments, the present assessment is based on extensive human and epidemiological data and not only on toxicological experiments with animals. Therefore, the results can be considered to be very relevant in terms of human populations.

Overall conclusions

At present, cadmium concentrations in Finnish agricultural soil pose a risk for soil organisms. Also, the leaching of cadmium from the agricultural soil is causing a risk for the aquatic environment. However, if the current situation with low cadmium content in fertilizers continues, cadmium concentrations in soil, crops and leachates will gradually decrease in potato and sugar beet cultivation. In wheat cultivation, cadmium inputs and outputs will stay in balance. If phosphorus fertilizers with average cadmium content in the EU (138 mg Cd/kg P) were to be used in Finland, the cadmium balance in agricultural soils would be radically disturbed, according to model calculations. Over 100 years, cadmium concentrations in soil on potato fields would increase by 125%, wheat grain concentrations by 34%, and an increase of 123% in cadmium leaching from potato fields would take place. These figures can be compared with the observed trend in Finland between 1974 and 1987. During that 13-year period the measured extractable cadmium concentration in the Finnish cultivated soils increased by 30%. This was mainly caused by exceptional use of fertilizers with high cadmium content, corresponding the average EU fertilizers. Both soil and aquatic environments in Finland are particularly vulnerable to adverse effects caused by cadmium.

There is a risk of adverse health effects as a result of the present total cadmium exposure in the Finnish population. While the average cadmium intake from food alone does not pose a risk in Finland, certain parts of the population are at risk because of high dietary intake, increased absorption and/or smoking. For these high exposure groups the estimated urinary level of cadmium can be associated with high risk of kidney dysfunction and bone effects. If phosphorus fertilizers containing average EU level of cadmium were to be used in Finland the dietary intake of cadmium would increase by more than 40% over 100 years, based on model calculations. Due to the shape of statistical distribution of the dietary cadmium exposure in the Finnish population this increase would lead to a much higher increase in the size of the risk groups.

The present assessment is a combination of measured and estimated data, epidemiological information and results based on experimental studies. It is noteworthy that compared to most chemicals the number of epidemiological and other human studies, which form a consistent basis of this assessment, is large. Despite the uncertainties associated with the estimations, the assessment is deemed to provide a sufficient basis for the conclusions drawn above.

ABBREVIATIONS AND DEFINITIONS

AA	Aminoacids
AAAc	Acid ammonium acetate
AAAc-EDTA	Acid ammonium acetate-ethylenediaminetetracetic acid
AAP	Alanine aminopeptidase
Acute toxicity	The adverse effect occurring within a short time following the administration of a single dose or multiple doses given within this short time period
Adsorption	The adhesion of molecules to the surfaces of solids
Alkalinity	The acid-neutralizing (i.e. proton-accepting) capacity of water; the quality and quantity of constituents in water which shift the pH towards the alkaline side of neutrality
AR	Aqua regia
BCF	Bioconcentration factor: the ratio of the test substance concentration in the test organism (e.g. fish, plant) to the concentration in a medium (e.g. water, soil) at steady-state conditions.
Bioaccumulation	The net result of the uptake, distribution, and elimination of a substance due to all routes of exposure, i.e. exposure to air, water, soil/sediment and food
Bioconcentration	The net result of the uptake, distribution and elimination of a substance due to water-borne exposure
Biomagnification	The accumulation and transfer of chemicals via the food web (e.g. algae-invertebrate-fish-mammal) due to ingestion, resulting in an increase of the internal concentration in organisms at the succeeding trophic levels
Cadmibel	A large epidemiological study on health effects of cadmium on the general population in Belgium, reported in various journals in 1990-1996
Chronic toxicity	Extended or long-term exposure to a stressor. Long-term effects related to the changes e.g. in metabolism, growth, reproduction, or ability to survive. Exposure concentrations are usually low
DOC	Dissolved organic carbon
d.w.	Dry weight

EC_{50}	Median effective concentration: 1. the concentration resulting in a 50% change in a parameter (e.g. algal growth) relative to the control, 2. the concentration at which a particular effect (e.g. daphnia immobilization) is observed in 50% of the organism population relative to the control
f.w.	Fresh weight
GM	Geometric mean
GSD	Geometric standard deviation
IARU	International Agency for Research of Cancer
LC_{50}	Median lethal concentration: a statistically derived concentration that can be expected to cause death in 50% of animals exposed for a specified time
LOEC(L)	Lowest observed effect concentration (level). The lowest concentration of a material used in a toxicity test that has a statistically significant adverse effect on the exposed population of a test organism compared with the control
NAG	N-acetyl-b-glucosamidase
NHANES II	U.S. National Health and Nutritional Examination Survey II
NOAEL	No observed adverse effect level
NOEC	No observed effect concentration. The highest concentration of a test substance to which organisms are exposed that does not cause any observed and statistically significant adverse effects on the organism compared with the control
PEC	Predicted environmental concentration: the expected concentration in an environmental compartment. The PEC can be based on either measured or calculated data
PNEC	Predicted no effect concentration: environmental concentration which is regarded as a level below which the balance of probability is that an unacceptable effect will not occur
PTWI	Provisional tolerable weekly intake
RBT	Retinol binding protein
Safety factor	A factor applied to an observed or estimated toxic concentration or dose to arrive at a criterion or standard that is considered safe. Safety factor, assessment factor and uncertainty factor (UF) are often used synonymously.

Contents

1 GENERAL SUBSTANCE INFORMATION

Identification of the substance

CAS No.:	7440-43-9
RTECS:	EU 98000
EINECS-No.	231-152-8
Symbol:	Cd
Atom mass:	112.41

Cadmium is a soft silverish white metal that does not occur unadulterated in nature. In the environment Cd occurs as a free hydrated ion and also complete with inorganic ligands (forming e.g. chloride, carbolated, sulphide and hydroxide-complexes). Humic-, fulvine-amino- and nucleinic acid function as organic ligands (Ros and Slooff 1988).

Physico-chemical properties

Melting point:	321 °C
Boiling point:	765 °C at 1 bar
Density:	8.64 g/cm^3 at 20 °C

Standard electrode potential: -0.402 V ($Cd^{2+} + 2e^- \rightarrow Cd$)
Water solubility: insoluble

Metallic cadmium is essentially insoluble in water; however, several cadmium compounds are freely soluble {such as cadmium chloride ($CdCl_2$), cadmium bromide ($CdBr_2$), cadmium iodide (CdI_2), cadmium nitrate [$Cd(NO_3)_2$], and cadmium sulphate ($CdSO_4$)} (Weast 1986, Budavari et al. 1989). The water-insoluble compounds of cadmium, such as cadmium oxide (CdO), cadmium sulphide (CdS), cadmium carbonate ($CdCO_3$), cadmium *ortho*-phosphate [$Cd_3(PO_4)_2$], and cadmium fluoride (CdF_2) (Weast 1986) may solubilize under strong oxidizing or acidic conditions (WHO 1992).

2 GENERAL INFORMATION ON EXPOSURE

2.1 Sources of cadmium in agricultural soil

Sources of Cd in the soils are both natural and anthropogenic. One of the most important natural sources of Cd is eruptions of volcanoes (Nordic Council of Ministers 1992). In general, the main anthropogenic sources of Cd to the cultivated soils are fertilizers, atmospheric deposition, animal manures and to smaller extent liming agents, sewage sludges and other wastes (table1).

2.1.1 Atmospheric deposition

Scandinavian moss monitoring (Nordic Council of Ministers 1992) indicates that Cd deposition has clearly decreased in the Nordic countries during the years 1969-1985. During the period of 1990-97, the annual atmospheric emissions of cadmium in Finland decreased by 85%, from 6.3 to 1.1 tonnes (Melanen et al. 1999).

According to air quality measurements made during 1997-99 in eight background stations in Finland (Finnish Meteorological Institute 1997, 1998, 1999) annual bulk deposition of cadmium was the highest in southern Finland, 0.3 g/ha, and decreased gradually to the north being in central Finland 0.2 and in northern Finland 0.1 g/ha (Table 2).

Table 1. Mean Cd inputs from various sources in Finnish agricultural soil at the national level.

Input	g/ha/a	%
Atmospheric deposition	0.2	33.1
Fertilizers	0.025	4.1
Liming	0.035	5.8
Manures	0.322	53.2
Other organic wastes	0.023	3.8
Total	0.605	100

2.1.2 Fertilizers

Since 1995, the Finnish agro-environmental programme according to the Council regulation (2078/92) has limited the use of P-fertilizers. In the fertilizer year 98/99, the use of P-fertilizers was 11,0 kg (Table 3) and in the fertilizer year 99/00 about 10.4-10.5 kg P/ha (Hero 2000). During the whole 1990's, the Cd concentration of the P-fertilizers on Finnish markets has varied from 1 to 5 mg/kg P (Syvälahti 1996, Hero 2000). Today, the annual Cd load from the P-fertilizers per hectare is 0.025 g (= 10 kg x 2.5 mg).

2.1.3 Liming agents

In 1999, a total sale of various liming agents was 1.266 million tonnes (Table 3) of which 85-90 % was the lime (Kalkitusyhdistys 2000) and the rest were the slags of steel industry and the wastes of sugar industry. In the same year, an area in production was 2.000 million ha. Thus, the mean use of different liming agents was 633 kg per hectare. According to the unpublished data material of the Agricultural Research Centre of Finland, the Institute of Soils and Environment, lime contains Cd 0.056 mg/kg. Thus, the annual Cd load from liming agents is approximately 0.035 g/ha (=0.056 mg x 633 kg/ha). According to the decision of Ministry of Agriculture and Forestry, Nr 46/1994, the maximum Cd concentration in liming agents is 3 mg/kg f.w.

2.1.4 Animal manures

In 1997, the amount of manure generated in Finland was calculated on the basis of the numbers of various farm animal species (Siika-aho 2000). The manure amounts were the following: cattle 18 000 000, pigs 2 830 000, sheep and geese 123 000, horses 190 000, poultry 223 000 and fur animals 96 000 (f.w.) tons, ie. all together 21 462 000 tons containing from 10 to 20% of dry matter (d.m.). As a dry matter the manure amount is 3 219 300 tons. A mean concentration

Table 2. Annual bulk depositions of cadmium in eight Finnish background stations measured by Finnish Meteorological Institute during 1997-99.

	Location	Location	1997	1998	1999	1997-99
Background station	**N**	**E**	**g/ha**	**g/ha**	**g/ha**	**g/ha**
Southern Finland						
- Utö	59° 47'	21° 23'	0.28	0.28	0.27	0.28
- Virolahti	60° 32'	27° 41'	0.27	0.41	0.38	0.35
- Kotinen	61° 14'	25° 04'	0.17	0.27	0.22	0.22
Average			0.24	0.32	0.29	0.28
Central Finland						
- Hietajärvi	63° 10'	30° 43'	0.11	0.23	0.20	0.18
- Hailuoto	65° 00'	24° 41'	0.09	0.11	0.26	0.15
Average			0.10	0.17	0.23	0.17
Northern Finland						
- Pesosjärvi	66° 18'	29° 30'	0.08	0.12	0.09	0.10
- Matarova	68° 00'	24° 15'	0.05	0.12	0.13	0.10
- Vuoskojärvi	69° 44'	26° 57'	0.04	0.11	0.06	0.07
Average			0.06	0.12	0.09	0.09
Mean			0.14	0.21	0.20	0.19

Table 3. Use of fertilizers and lime in 1990's in Finland.

	1990	1994	1996	1997	1999
Fertilizers:					
total use (M kg)	1 218	861	880	826	
nutrients, kg/ha	200	153	143	130	
- nitrogen (N) kg/ha	111	94	92	86	
- phosphorus (P) kg/ha	31	19	16	12	11
- potassium (K) kg/ha	58	40	34	32	
Lime total use (M kg)	1 238	1 217	1196	1266	
Area in production, in thousands, ha	2 088	1 797	1 968	2 000	

of Cd in manures of various animal species is 0.2 mg/kg d.m. (Mäkelä-Kurtto and Kemppainen 1993). Thus, the Cd load from manure to the whole area in production (2 000 000 ha) is 0.644 tons and to one field hectare 0.322 g/a, on the average. However, in practice the manure has not been spread evenly to all cultivated fields. A portion of manure from the total annual Cd load to the cultivated fields is 45.6%.

One of the main reasons contributing to the Cd content of animal manures are feeds which may contain Cd as impurity. In feeds, the maximum acceptable Cd content in Finland is 10 mg/kg feed (the Decision of the Ministry of Agriculture and Forestry, Nr 184/1994). For other feeds, feed mixtures or concentrates, the Cd limit values vary from 0.5 to 5.0 mg/kg feed (containing 12% water). In many countries, there is no limit value for Cd in animal feeds at all, although farmyard manure may give a significant Cd input similar to or higher than some of the fertilizers (Jensen and Bro-Rasmussen 1990).

2.1.5 Sewage sludge and other organic wastes

Municipal sewage plants produce annually about 1 million tons (f.w.) of sewage sludge which is about 150 000 tons as dry matter. The concentration of Cd has remarkably decreased during the 1970s and 1980s in Finland. In 1992, the median of Cd concentration of sewage sludges was 0.83 mg/kg d.w. (and the mean 1.78 mg/kg d.w.) (Levinen 1994). The highest concentration of Cd acceptable in sewage sludges to be used in agriculture is 3 mg/kg d.w. (Council of State decision Nr 282/1994). A maximum annual Cd load to the cultivated soils is 3 g/ha. Today, about 60% of the total amount of municipal sewage sludges is recycled and about one third, i.e. 50 000 tons of dry matter, is used in agriculture. Consequently, a mean annual Cd load from sewage sludges based on the median concentration is 0.042 tons to the whole fertilized area and 0.023 g to one hectare.

The highest acceptable concentration of Cd in the cultivated soils for application of sewage sludge is 0.5 mg/kg d.w. (Council of State decision Nr 282/1994). As with manure, sewage sludge is not evenly spread to agricultural fields. Recycling of sewage sludges is planned to be higher up to 70% by the year 2005 (Ministry of Environment 1995).

A mean Cd concentration of the composted biowaste is 0.57 mg/kg d.w. (Mäkelä-Kurtto and Sippola 1995). A maximum allowable Cd concentration in composts and other soil improvers in Finland is 3 mg/kg f.w. (decision of Ministry of Agriculture and Forestry, Nr 46/1994). Until today, recycling of composted municipal biowaste has been minor in agriculture and has had nearly no importance on the Cd load in the cultivated soils in Finland. Finnish municipalities generate annually totally 2.1 million tonnes (f.w.) of wastes. About 0.84 tonnes, ie. 40% of all municipal wastes, is organic. Approximately 10% of this amount has been composted and recycled in plant production, mainly in domestic gardens. According to the Finnish waste strategy (Finnish Ministry of Environment 1995) recycling of municipal biowaste has to be increased to 75% by the year 2005.

2.2 Cadmium in fertilizers

The cadmium content of the P-fertilizers depends on the Cd concentration of phosphate rock from which P-fertilizers have been derived and processed. The Cd content of phosphate rock of igneous origin is relatively low, generally below 5 mg/kg, but these rocks constitute only 15% of world phosphate production. Sedimentary phosphate rocks contain Cd between 3 and 120 mg/kg (Ghoshesh et al. 1996).

In Finland, a phosphate mine was opened at the beginning of the 1980´s at Siilinjärvi. This mine produces phosphate rock of igneous origin, like the Kola mine does in Russia. The Cd content of Siilinjärvi rock phosphate is internationally exceptionally low (Figure 1, Ref. Kemira 1991). During the 1990´s, the Cd concentration of P-fer-

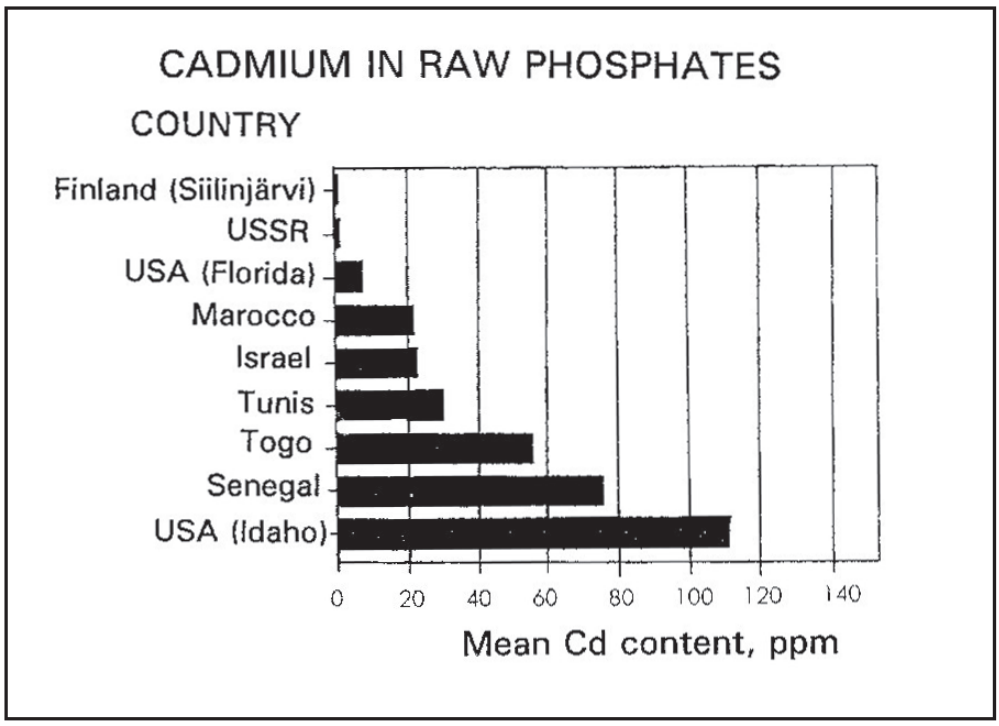

Figure 1. Mean cadmium contents (ppm) of raw phosphates from different countries (Source: Phosphorus and Potassium 1989, ref. Kemira 1991).

tilizers on the Finnish markets have varied from 1 to 5 mg/kg P (Syvälahti 1996, Hero 2000). The cadmium concentration in the P-fertilizers available in the Finnish markets for crop and garden production has been limited to 50 mg/kg P and the limit value for the Cd concentration in the fertilizers not containing P is 3 mg/kg DM (the decision of the Ministry of Agriculture and Forestry, Nr 45/1994). An average Cd content in European fertilizers is 138 mg/kg P (Davister 1996).

Mortvedt and Osborn (1982) conducted experiments to characterize the chemical form or forms of Cd in phosphate fertilizers. Results suggest that the chemical form of cadmium in triple superphosphate (TSP) and diammonium phosphate (DAP) is $Cd(H_2PO_4)_2$, $CdHPO_4$, or a mixture of these salts, which are Cd analogues of the P compounds in commercial P fertilizers.

Although high Cd contents in soils and plants may be partly due to high native Cd contents in soils or to airborne sources, several studies have reported an increase in the Cd content of plants and soils in the cultivated soil layer following the application of high rates of phosphates (e.g. Kaarstad 1991; Mortvedt et al. 1981; Mulla et al. 1980; Williams and David 1976). This is the result of the presence of Cd in the phosphatic fertilizers.

The large world soil and plant data material and fertilization documents of Sillanpää and Jansson (1992) indicate a close relationship of plant and soil Cd to the fertilization rate of P (Figure 2) and to the P level of soil (Figure3). The plants and soils in the countries which traditionally use high amounts of phosphate fertilizers clearly have higher Cd contents than in developing countries where the large scale use of fertilizers has not yet been practiced.

Also, the Finnish research results (Sippola 1997, unpublished data of Agricultural Research Centre of Finland) show that AAAc-EDTA -extractable Cd in soil has significant correlations with both AAAc-extractable P in soil (Figure 4) and with acid ammonium oxalate extractable P in soil (Figure 5). In addition to this, McLaughlin et al. (1996) has indicated a very clear relationship between EDTA-extractable Cd in soil and H_2SO_4-extractable P in soils under pasture in South Australia.

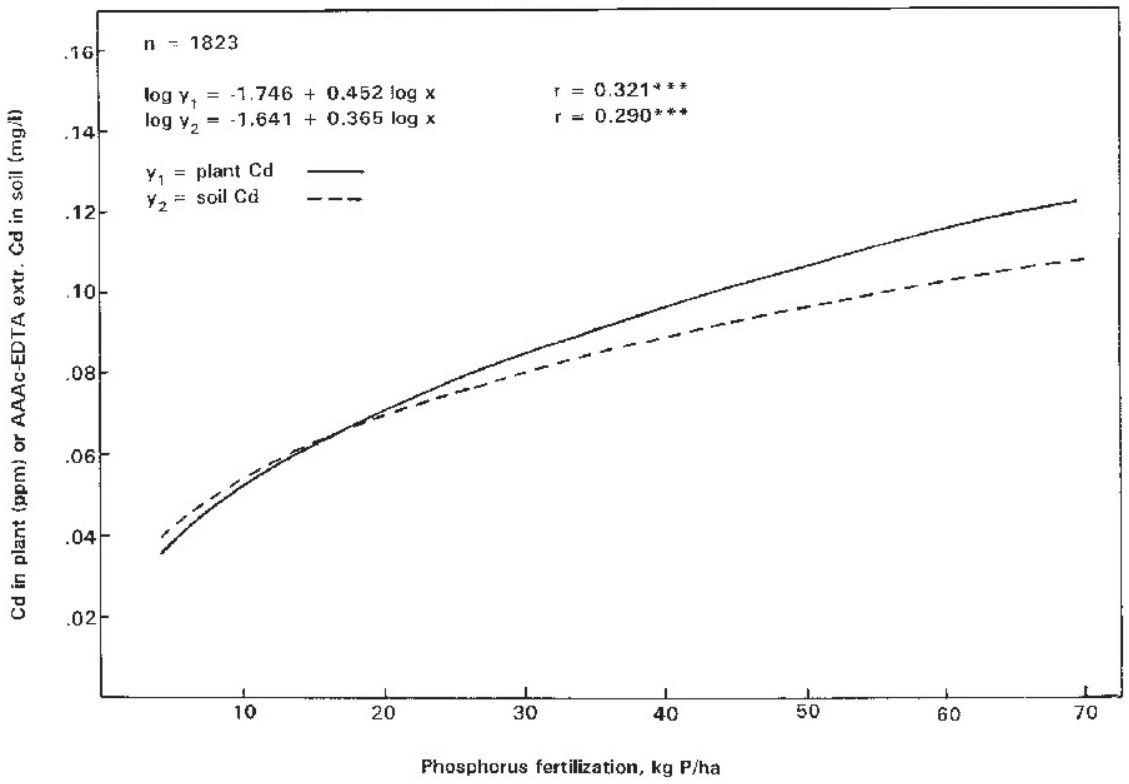

Figure 2. Plant Cd and soil Cd as functions of phosphorus fertilization (Sillanpää and Jansson 1992).

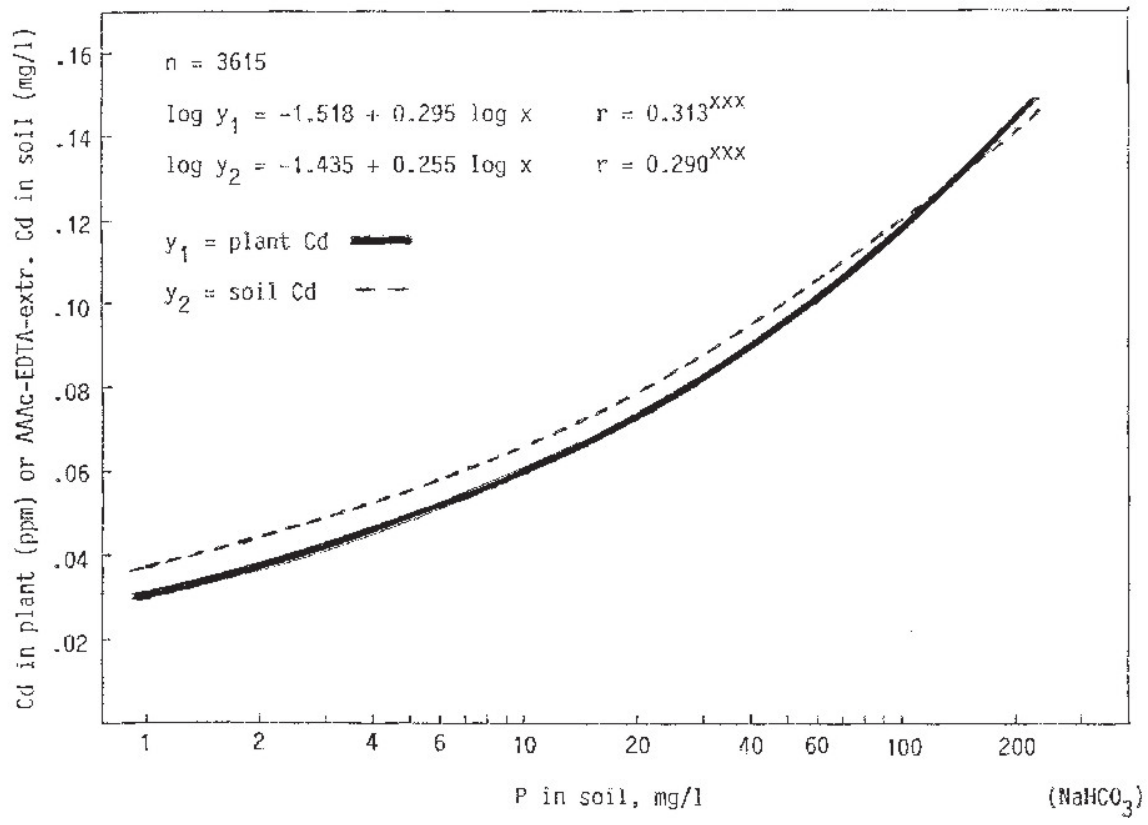

Figure 3. Plant Cd and soil Cd as functions of $NahCO_3$ - extractable soil phosphorus (Sillanpää and Jansson 1992).

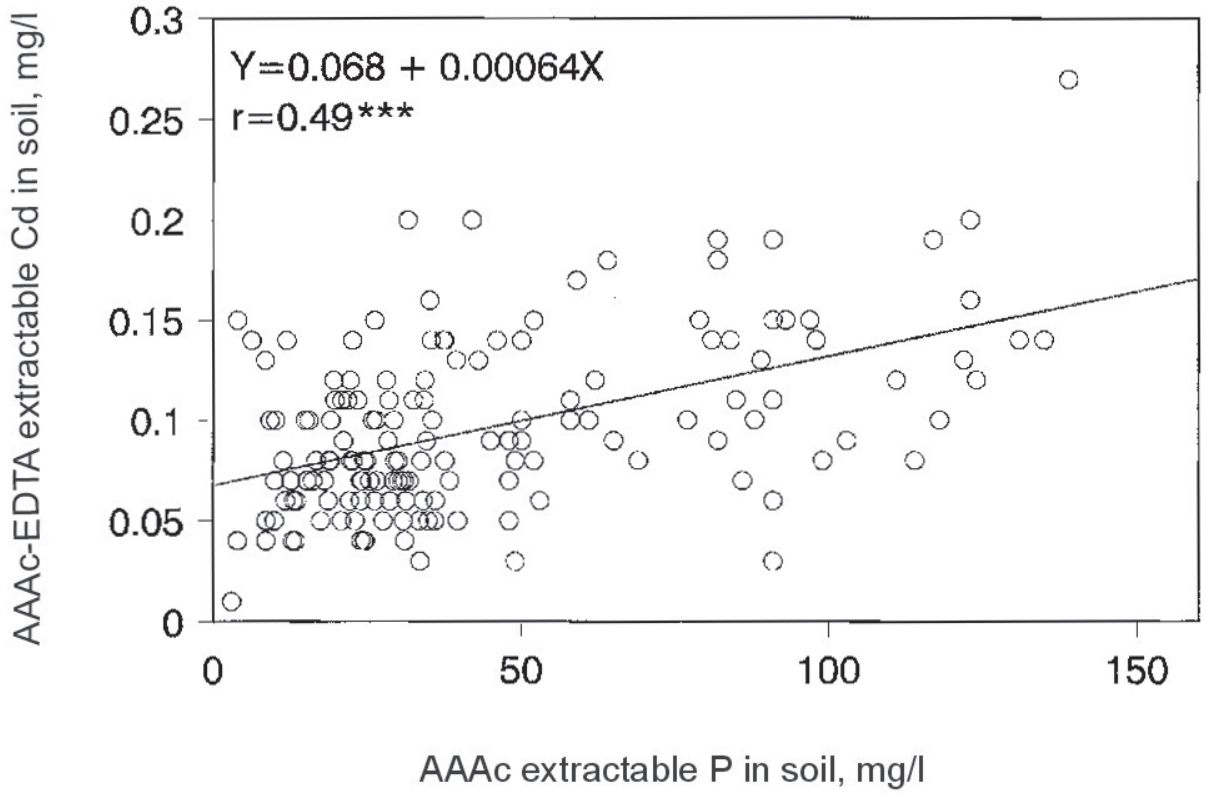

Figure 4. Dependence of AAAc-EDTA extractable Cd on AAC extractable P in the plough layer of Finnish cultivated soils, n=152 (Sippola 1997, unpublished).

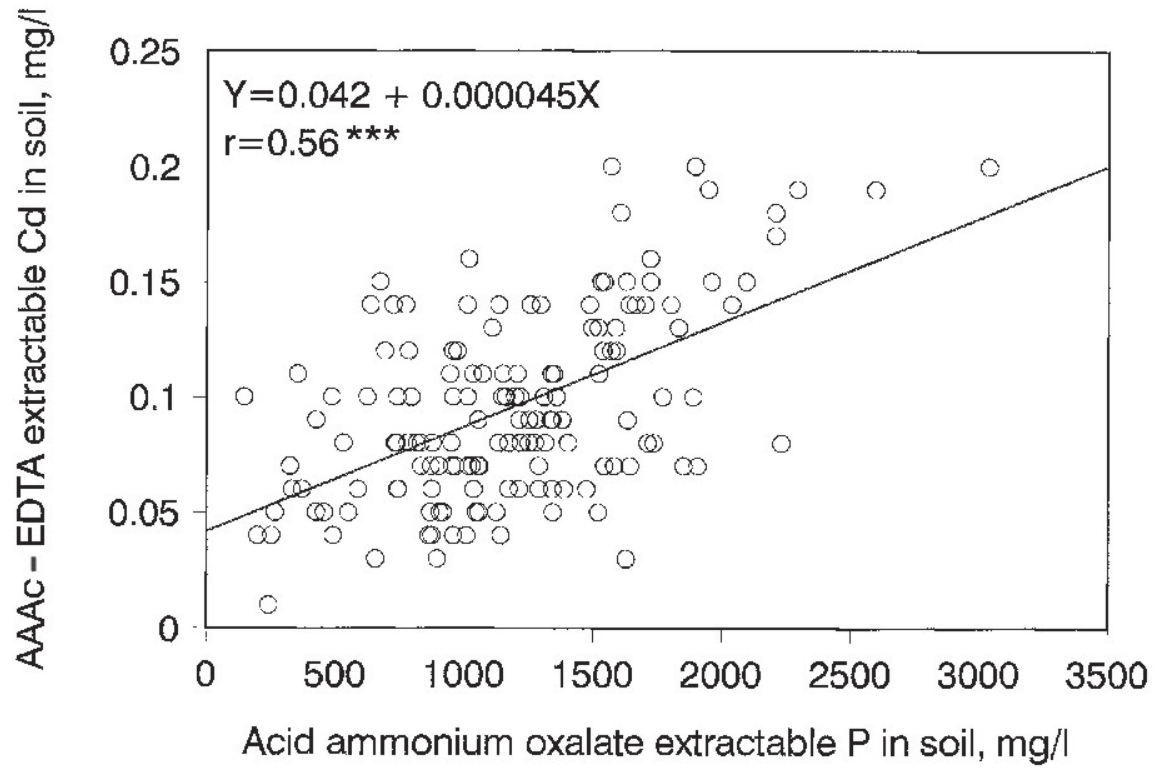

Figure 5. Dependence of AAAc-EDTA extractable Cd on oxalate extractable P in Finnish cultivated soils, n=150 (Sippola 1997, unpublished).

2.3 Cultivated soils

2.3.1 Soil types

The soil in Finland has developed mainly from acid magmatic rocks, pulverized by the continental ice sheet during the glacial period and from organic materials during the postglacial period (Sippola and Tares 1978). Soil types of the cultivated fields vary considerably within Finland. About 35% of the Finnish cultivated soils can be classified as clays (clay content > 30%), 32% silts, 18% as coarse mineral soils and 15% as organic soils (Puustinen et al. 1994). In the plough layer, the dominant soil types are clay in southern and south-western Finland (Figure 6), and till in the Central lake districts; fine sand in parts of western and eastern Finland; and peat in northern Finland (Kurki 1972). A distribution of the soil types in the subsoils of the cultivated fields is to a great extent similar to that in plough layer (Kurki 1972).

2.3.2 General characteristics and fertility

Dissimilarity between the soil types in different parts of Finland is reflected in the fertility i.e. in the soluble amounts of mineral elements present in the soils (Sippola and Tares 1987). The clay in southern Finland contains calcium, potassium and magnesium in abundance. On the other hand, in the glacial tills of the central areas and peat soils of northern Finland, mineral elements occur in soluble form to a lesser extent than in clay. The minerals from acid rocks contain smaller quantities of trace elements than those from so-called basic rocks, which only occur to a very limited extent in the Finnish bedrock. As minerals weather slowly in the cool climate of Finland, elements are released in smaller quantities in plant-available form than under warmer conditions.

There are two remarkable differences in the chemical characteristics in the cultivated soils between Finland and many other countries. One of the main distinguishing feature is the low pH of Finnish cultivated soils. Mostly, the pH(H_2O) values of cultivated soils in the European countries are around 7 (Sillanpää 1982, FAO/Trace element network 1987). Because of acid parent material, the soils in Finland are very acidic, perhaps the most acidic in Europe. According to Kähäri (1989) the most acidic fields of Scandinavia are cultivated in Finland.

Since 1955, the mean pH(H_2O) of the soils has increased from 5.5 to 5.8 very slowly in spite of intensive liming (Mäntylahti 1997) (Figure 7). The subsoils are still more acid than the plough layer of the cultivated fields (Kurki 1972).

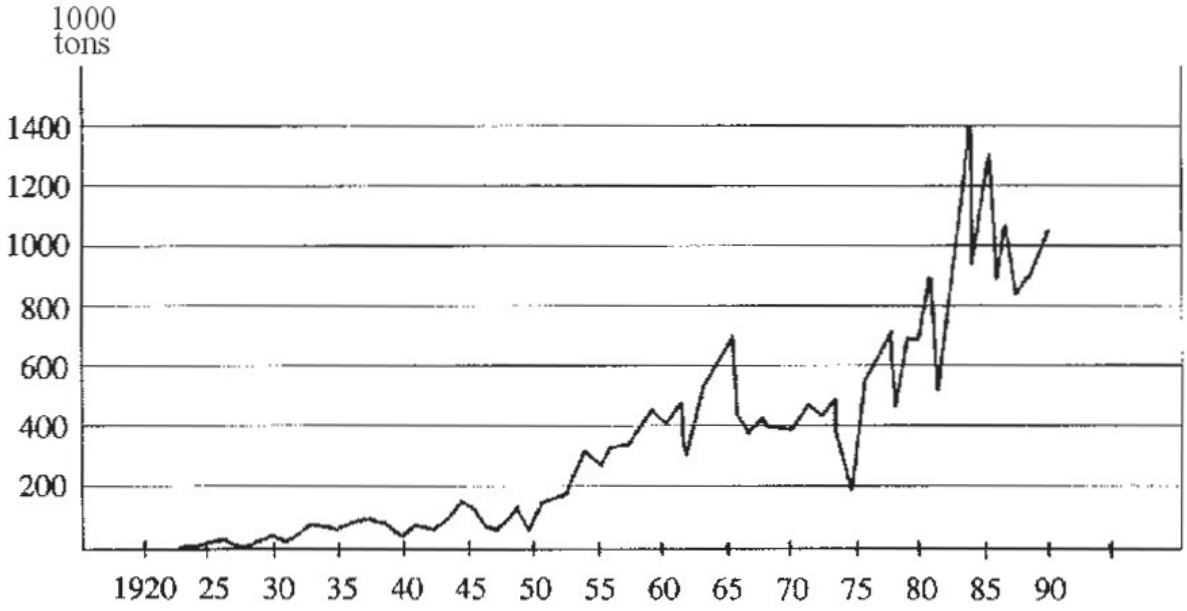

Figure 7. Total annual use of liming agents for the whole fertilized area in Finland during the years 1923-90 (Maatalouskeskusten liitto 1991).

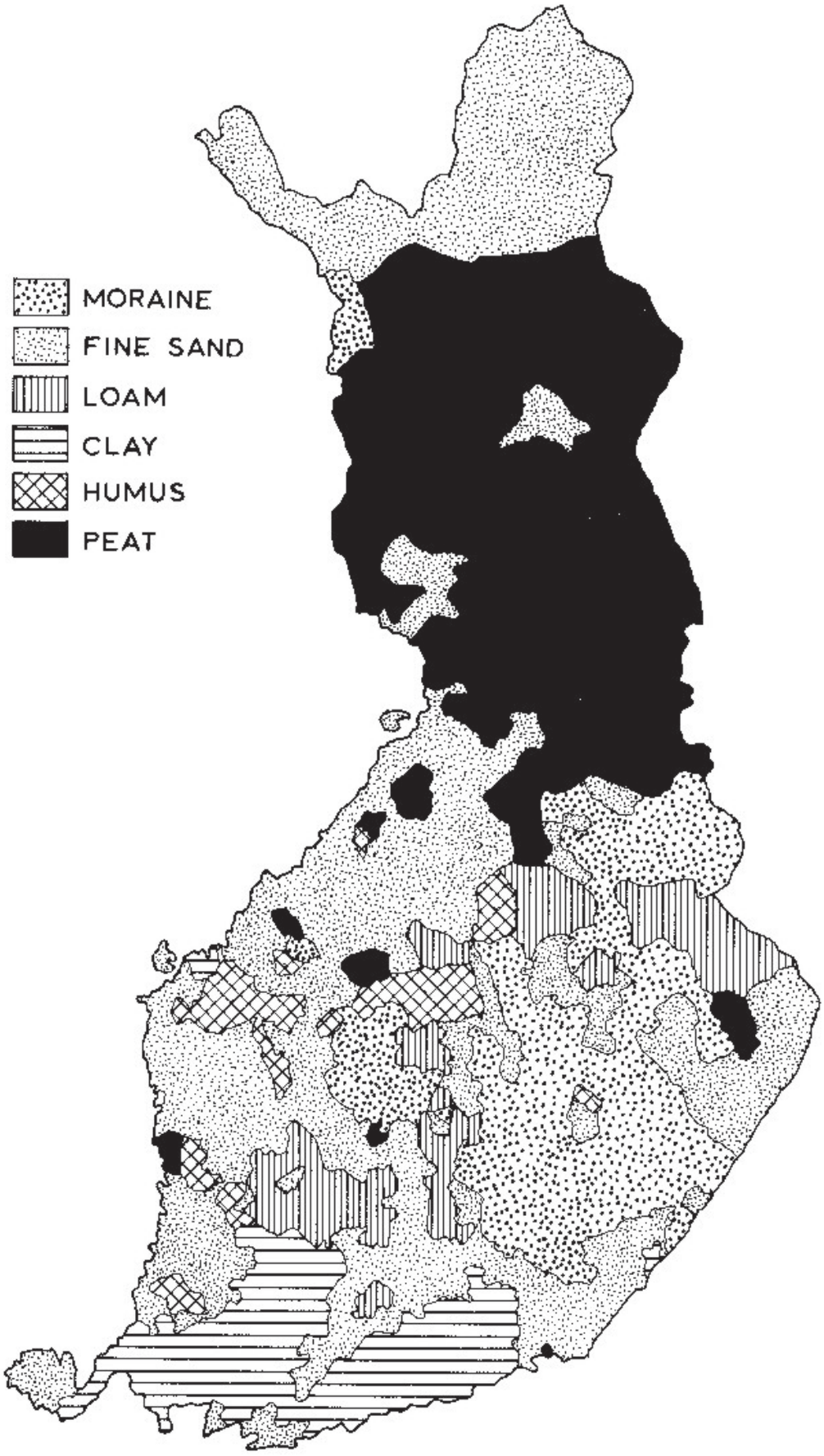

Figure 6. Dominant soil types in Finland (Kurki 1972).

The other remarkable difference in the chemical characteristics of the cultivated fields between Finland and many other countries is the high organic matter content of Finnish cultivated soils. It is a result of the abundance of peat soils and of the slow decomposition rate of organic matter due to a cool and humid climate. In 1987, a mean organic carbon content of Finnish cultivated fields was 9% and consequently a mean organic matter content 15%. Typical soil organic matter contents in other European countries vary between 1- 4% (Sillanpää 1982, FAO/Trace element network 1987).

2.3.3 Plant crops, areas and yields

Only about ten of the one hundred most important food crops in the world can be grown in Finland. With the exception of certain grasses and to some extent barley and potatoes, no commercial crop can survive in the northern third of the country. Natural vegetation is essentially boreal coniferous forests with a zone of treeless tundra in the north and small areas of temperate mixed forests in the south-west (Ministry of Foreign Affairs and Ministry of Agriculture and Forestry 1992).

Finnish agricultural production is mainly based on livestock. Milk is the most important product of Finnish agriculture. Only 15% of arable land is used for crop production for human consumption, about 2/3 of which consists of bread grains and the rest mainly of rapeseed, sugar beets and potatoes. However, more than half of arable land is used for the production of grains when feed grains such as barley and oats are taken into account (Ministry of Foreign Affairs and Ministry of Agriculture and Forestry 1992).

In 1990's, the total area of cultivated land in Finland varied between 2.1 and 2.3 million ha of which 1.8-2.1 million ha was in production. Annual plant crops, areas and yields are shown in Table 4.

Table 4. Plant crops, areas and yields in Finland in 1990's.

	1990	1994	1997	1998
Wheat area (in 1 000 ha)	180	89	125	135
- total yield (M kg)	490	337	464	397
- yield kg/ha	3 480	3 800	3 720	2940
Rye area (in 1 000 ha)	81	9	23	31
- total yield (M kg)	274	22	47	49
- yield kg/ha	3 010	2 580	2 070	1 570
Barley area (in 1 000 ha)	486	505	583	552
- total yield (M kg)	1 720	1 858	2 004	1 316
- yield kg/ha	3 540	3 690	3 440	2 390
Oats area (in 1 000 ha)	453	333	369	377
- total yield (M kg)	1 662	1 150	1 243	975
- yield kg/ha	3 670	3 460	3 370	2 590
Sugar-beets area (in 1 000 ha)	30	34	35	33
- total yield (M kg)	995	1 097	1 355	897
- yield kg/ha	33 040	32 360	38 820	27400
Oil plants area (in 1 000 ha)	65	67	61	58
- total yield (M kg)	117	108	93	64
- yield kg/ha	1 790	1 600	1 530	1110
- Peas area (in 1 000 ha)	3	6	6	4
- total yield (M kg)	3	14	13	4
Potatoes area (in 1 000 ha)	41	37	33	32
- total yield (M kg)	490	726	754	591
Vegetables total yield (M kg)	..	..	258	209
- tomatoes	32	35	33	31
- cucumber (greenhouse)	23	26	27	28
- cabbage	22	23	29	19
- carrots	31	59	69	53
- onions	18	15	14	18

Source: Information Centre of the Ministry of Agriculture and Forestry (TIKE)

3 ENVIRONMENT EXPOSURE

3.1 Release into and occurrence in the environment

3.1.1 Cadmium concentrations in soils

Cadmium is one of the elements which occur at very low concentrations in the lithosphere and in topsoils. The average total contents of Cd in soils lie between 0.07 and 1.1 mg/kg. However, the background Cd level in soils apparently should not exceed 0.5 mg/kg, and all higher values reflect the anthropogenic impact on the Cd status in topsoils (Kabata-Pendias and Pendias 1984).

The main factor determining the Cd content of the soil is the chemical composition of the parent rock. In addition to this, the age of the soil has a significant influence on the concentrations of total and soluble Cd in soil. The more aged and older the soil, the less may be the influence of the parent material. According to Bergseth (1989), chemical and mineralogical analysis of soil samples from 10 European countries indicate that the samples from Portugal and Spain have the lowest total Cd content and the highest degree of weathering. The samples from Finland and Norway seem to have higher total as well as easily extractable Cd content than the samples from southern Europe. Although the bedrock is old in Finland, the soil is young as compared to the soil in southern Europe, because Finnish soil is mainly formed after the last ice age. However, the soil samples from strongly industrialized countries, Germany, United Kingdom and the Netherlands, seem to have the highest total Cd content, mainly due to extensive pollution and large supplies of Cd-containing phosphate fertilizers.

The mean total content of Cd in the Finnish cultivated soils is 0.21 mg/kg (Sippola and Mäkelä-Kurtto 1986), when extracted with aqua regia (AR). The total Cd content of cultivated Finnish soils approximates to the values reported in other Northern European countries, like in Sweden 0.22 mg/kg HNO_3 extractable Cd (Andersson 1977) and in Denmark 0.25 mg/kg concentrated HNO_3 extractable Cd (Tjell and Hovmand 1978). Relatively high total mean Cd contents occur in the soils of Germany (0.52 mg/kg) and France (0.74 mg/kg) (Angelone and Bini 1992). In the U.K., a mean Cd content of agricultural soils reaches 1.0 mg/kg (Jackson and Alloway 1992) indicating anthropogenic pollution. According to Davister (1996) the average Cd content of cultivated soils in Europe is 0.5 mg/kg.

There are distinct regional differences in the Cd content of the soils (Figure 8). Cultivated soils in southern Finland contain nearly twice as much Cd as the soils in northern Finland (Sippola and Mäkelä-Kurtto 1986). That can be attributed to intensive agriculture and industry in southern Finland; and the long-range transport of Cd from Central Europe; and to the differences in the soil types. Also higher concentrations of AAAc-EDTA -extractable Cd are evident in the cultivated soils in southern Finland than in northern Finland (Table 5 and Figure 9). The AAAc-EDTA extractable Cd concentration of the Finnish soils (n = 1320) studied in 1987 averaged 0.076 mg/l (Erviö et al. 1990). The extraction power of AAAc-EDTA is about 40% of the extraction power of AR (Sippola and Mäkelä-Kurtto 1986, Mäkelä-Kurtto et al. 1992).

Table 5. Mean AAAc-EDTA-extractable Cd concentrations (mg/l and mg/kg) of soils by plant cultivation zones in 1987 (Erviö et al. 1990).

Year	1987				
Cultivation zone	I	II	III	IV	V
pH	6.05	5.79	5.78	5.65	5.49
org. C %	5.0	6.3	7.7	13.1	14.4
Bulk dens.	0.94	0.91	0.94	0.83	0.85
Cd (mg/l)	0.119	0.095	0.077	0.061	0.042
Cd (mg/kg)	0.127	0.104	0.082	0.073	0.049

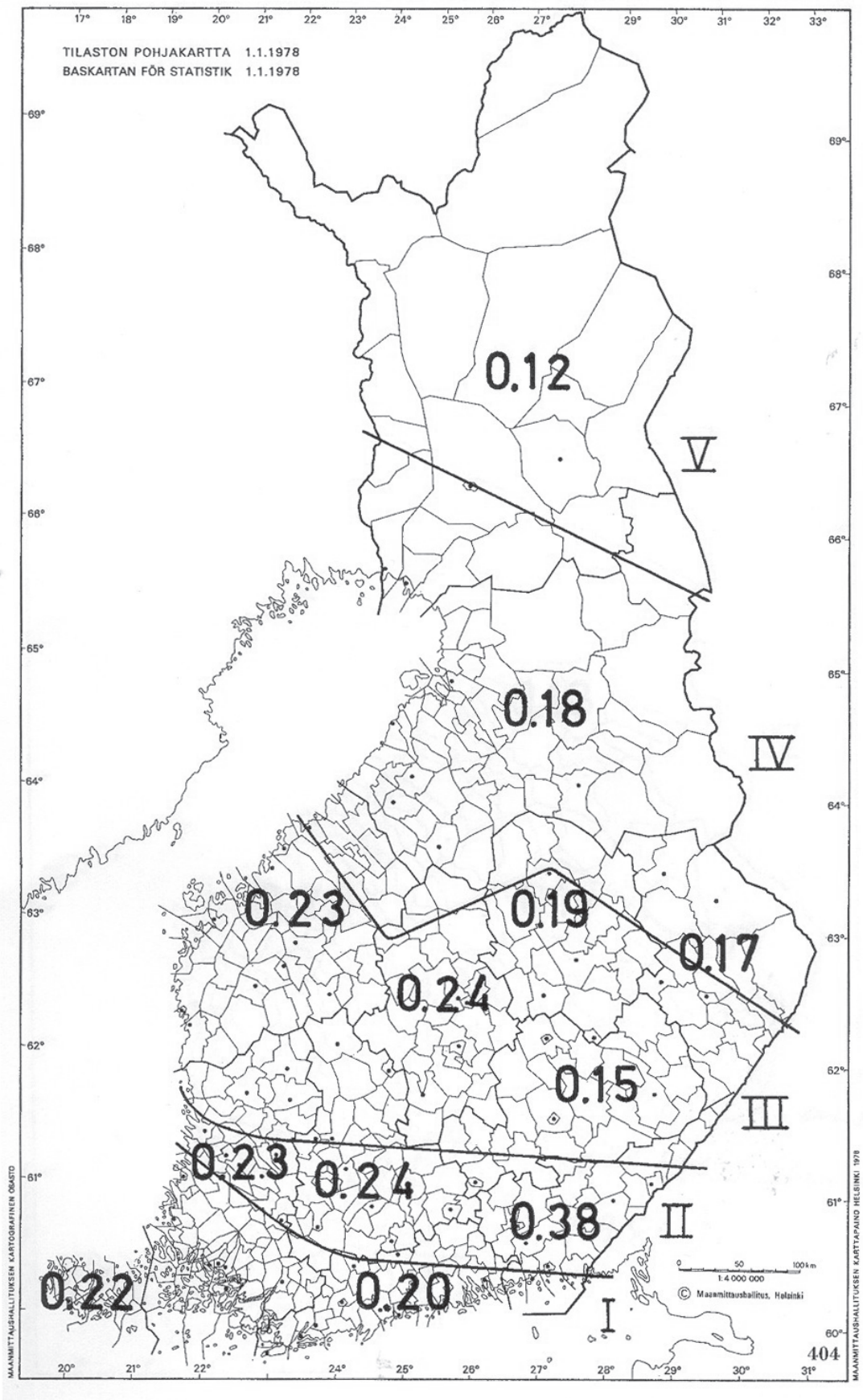

Figure 8. Total cadmium contents (mg/kg) of cultivated soils by locality. Cultivation zones from I to V are indicated by dividing lines (Sippola and Mäkelä-Kurtto 1986).

It is difficult to compare the extractable Cd levels of the soils between Finland and other countries, because many various extraction solutions with different extraction power have been used and are still in use in the world. However, Sillanpää and Jansson (1992) have reported on the AAAc-EDTA (acid, pH 4.65, ammoniumacetate-EDTA) extractable Cd levels in the agricultural soils of 30 countries under FAO-project. Analytical results of this study are very comparable, because all the soil samples were analysed with the same method and in the same laboratory. According to these researchers, the national mean of AAAc-EDTA extractable Cd concentration of wheat fields in southern and central Finland was 0.11 mg/l. This is the same low level as measured in wheat and/or maize fields in the developing countries and lower than the corresponding soil Cd concentration in developed countries (Figure 10). The world mean of extractable Cd was 0.101 mg/l. The highest national mean, 0.36 mg/l, was in the cultivated soils of Belgium. In this international soil material, the highest AAAc-EDTA extractable soil Cd values exceeded the lowest by about 125-fold.

Various methods have been used for analysing and extracting Cd from soil and the analytical results have been expressed in various ways. Generally, the total content is expressed on a weight basis, i.e. mg/kg soil. From the plant uptake point of view, analytical results are more comparable when expressed on a volume basis, i.e. mg/l of soil, especially in the case of soils which have a broad range of organic matter. Therefore both ways are used in Finland.

3.1.2 Trend measured in soil for 1974–1987

In Finland, monitoring of the same sites of cultivated fields indicates that Cd concentrations of cultivated soils increased by 30% from 1974 to 1987 (Erviö et al. 1990). Soil monitoring which started in 1974 and earlier than in many other countries shows that the mean concentration of AAAc-EDTA-extractable Cd in the fields (n = 142) increased from 0.061 mg/l to 0.080 mg/l during 13-year period (Erviö et al. 1990). During the same period, the levels of soil pH and soil phosphorus were increased, too. An annual Cd increase was 2.4%, on average. In different cultivation zones, the increase was of the same magnitude, 0.020 mg/l.

The change was mainly due to exceptionally Cd-rich P-fertilizers in use during 1975-1981. According to Kivioja (1982) and the personal communication of Heikki Hero at Kemira Oy (1988), the Cd load from fertilizers in Finland varied annually between 1974 and 1987 as follows: 1.7, 2.6, 2.3, 4.0, 4.6, 7.4, 6.2, 3.6, 1.8, 0.9, unknown, 1.6, 0.5, and 0.5 g/ha, respectively. On average, the total cadmium load from fertilizers was 37.7 g/ha during the whole study period and 2.9 g/ha annually. Annual variations in the Cd load between 1960 and 1990 from fertilizers are presented in Figure 11 (Kemira Oy 1991). Before 1975, Finnish P-fertilizers were mainly manufactured from Russian Kola-apatite which contained very little Cd. During 1975-1987, P-fertilizers were manufactured from African raw phosphates rich in Cd. Since 1981, Finnish Siilinjärvi-apatite, the Cd concentration of which is also exceptionally low was used more and more for the production of domestic P-fertilizers. Since 1986, P-fertilizers in use in Finland had mainly been produced of the domestic, nearly Cd free, raw phosphate and consequently, the annual Cd load from fertilizers has decreased rapidly to 0.5 g/ha, on average. During the 1990´s, the mean annual Cd load from fertilizers has been less than 0.1 g/ha (Syvälahti 1996, Hero 2000).

Also deposition of Cd from the atmosphere was a reason for an increase in the soil Cd. Based on the measurements in southeastern Finland during 1974-1987 (Finnish Meteorological Institute 1985-1988), an average Cd bulk deposition was annually about 2 g/ha for the winter season and about 1 g/ha for the summer season. If the annual Cd deposition is assumed to have been 1.5 g/ha, then the total Cd load from the atmosphere

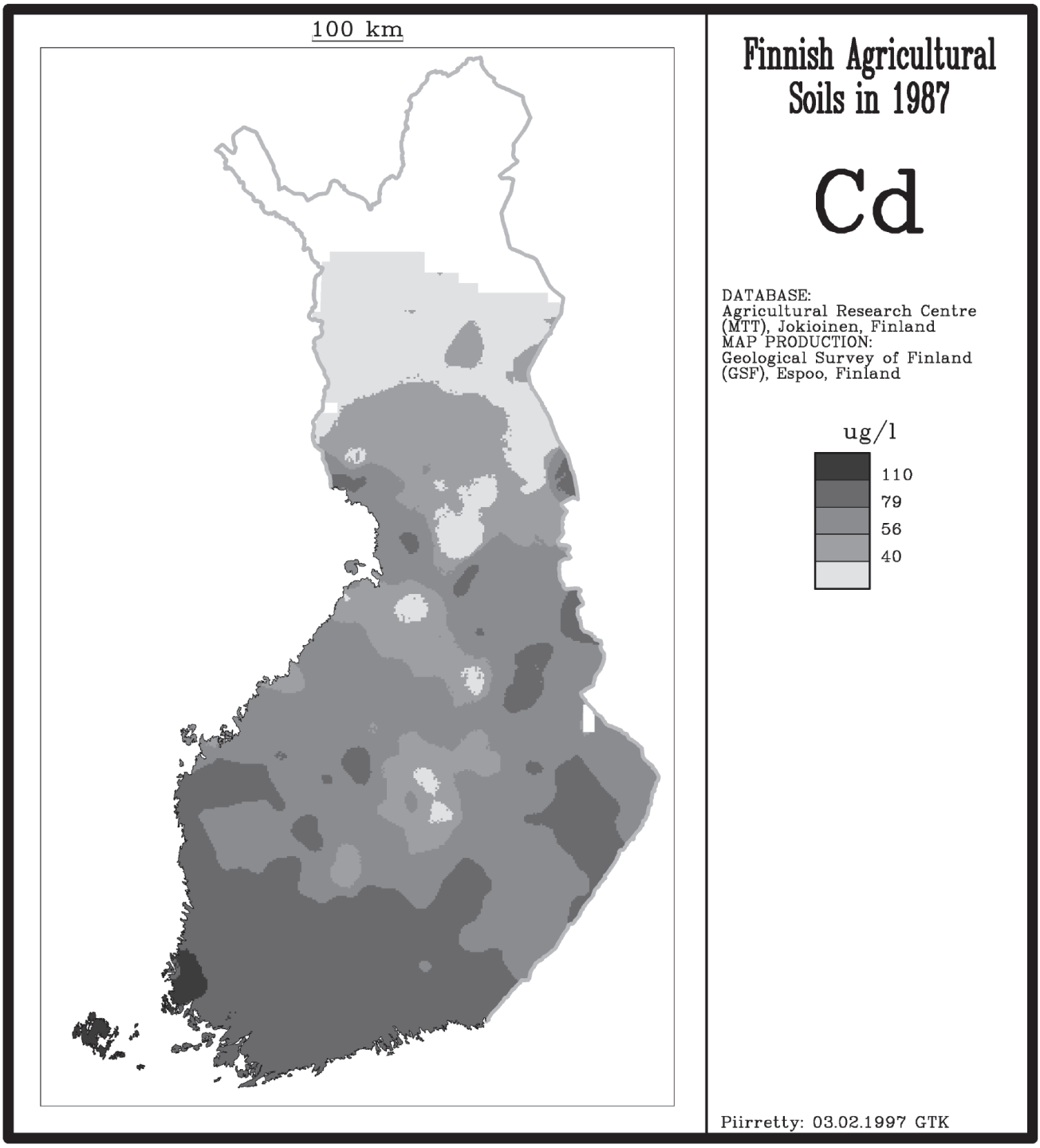

Figure 9. Areal distribution of AAAc-EDTA extractable cadmium in Finnish cultivated soils (n=1320) in 1987 (Erviö et al. 1990).

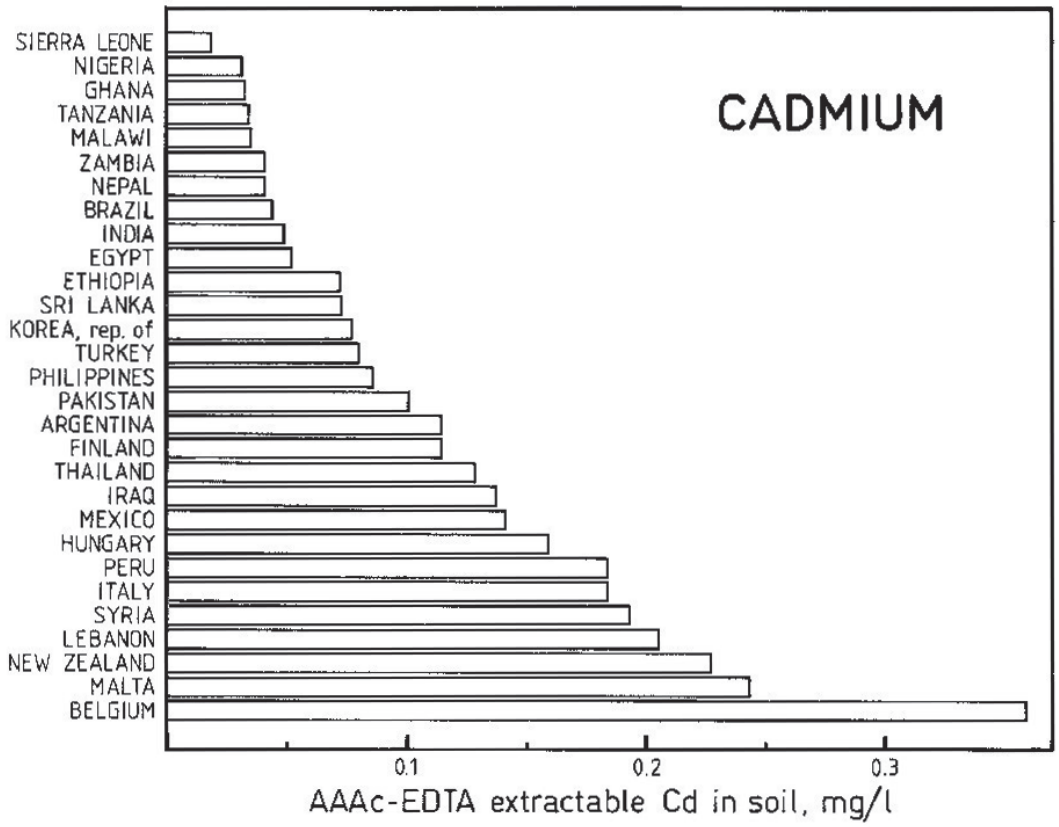

Figure 10. AAAc-EDTA extractable cadmium in cultivated soils of 30 countries (Sillanpää and Jansson 1992).

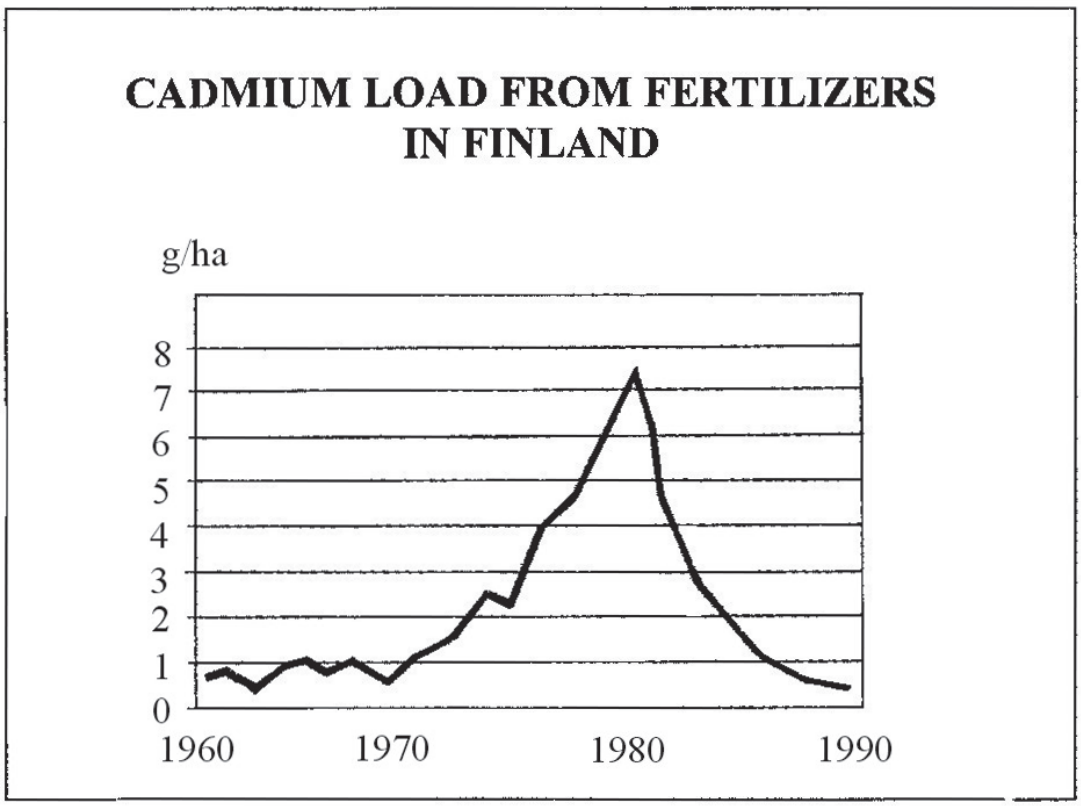

Figure 11. Mean annual cadmium loads (g/ha) from fertilizers into cultivated soils in Finland between 1960-1990 (Kemira 1991).

was 19.5 g/ha over the study period 1974-1987. On the basis of this, the Cd load from fertilizers was nearly twice that from the atmosphere.

A total increase in AAAc-EDTA-extractable Cd in the plough layer (0.2 m) between the years 1974 and 1987 was 38 g/ha. Respectively, cadmium inputs from fertilizers and the atmosphere together was 57.2 g/ha. Obviously, about two thirds of the total Cd input came from fertilizers. According to Andersson (1984), the Cd inputs to Swedish soils has been approximately three times higher than the outputs. Hence, Cd is gradually accumulating in the soil.

Total concentrations of Cd are reported to increase in the cultivated soils annually by 0.3-0.4% in Sweden (Andersson 1984) and by 0.6% in Denmark (Tjell et al. 1981). Furthermore, it is assumed (Andersson 1984) that the more soluble and plant-available soil Cd fraction will increase at a higher rate than the total soil Cd. According to a review by Jensen and Bro-Rasmussen (1990) there is an evidence that the increase in soil Cd has been fast during 1960´s, 1970´s and 1980´s and that also a significant long-term increase has been demonstrated in the Cd content of the harvested crops in some cases.

A mass balance estimation (Table 6) indicates that during the period 1974-1987, Cd inputs to the plough layer were annually together 4.78 g/ha, on average. The measured increase (30%) in the AAAc-EDTA extractable Cd was much faster than the estimated increase (14%) in aqua regia (AR) extractable Cd for the same period. If the annual accumulation rate would have been constant for 100 years, then the soil AR-extractable Cd would have been increased to 0.434 mg/kg that is more than twice the initial concentration.

3.1.3 Cadmium concentrations in crops

Cadmium contents in the dry matter (d.m.) of 16 plant crops grown side by side in two years at nine locations in various parts of Finland have been determined and compared by Sillanpää and Jansson (1991). The variation between the Cd contents of different plant species and plant parts is very wide (Figure 12). The Cd contents are the highest in sugar beet tops which exceed by a hundredfold those of the lowest in pea seeds. Also within individual plants, the difference in Cd concentrations is considerable. A very great difference (19-fold) between different parts of the same plant is evident in the pea but differences may also occur in cases of wheat, turnip rape, swede and sugar beet by 3-4 -fold.

In general, cereal grains have low Cd contents, the mean values varying from 0.03 to 0.09 mg/kg, but their straws contain considerably more Cd, from 0.13 to 0.26 mg/kg in d.m. (Sillanpää and Jansson 1991). These values are in good agreement with the results of other Finnish researchers, Syvälahti and Korkman (1978) and Varo et al. (1980).

The Cd contents in hay crops, including red clover are about equal or only slightly higher than those in cereal grains (Sillanpää and Jansson

Table 6. Cadmium mass balance in Finnish cultivated soils during 1974-1987 (13 years).

Inputs, g/ha/a		
Deposition	1.5	
Fertilizers	2.9	
Liming	0.035	
Manure	0.322	
Other wastes	0.023	
Together	4.78	
Outputs, g/ha/a		
Crops	0.138	
Waters	0.06	
Erosion	0.11	
Together	0.308	
Balance, g/ha/a	4.472	
	AAAc-EDTA -Cd	AR -Cd
Cd_{soil} 1974	0.061	0.210
Cd_{soil} 1987	Detected 0.080	Estimated 0.239
Cd_{soil} 2074	Estimated 0.146	Estimated 0.434

1991). The Cd averages of various cuts of timothy varying from 0.034 to 0.058 mg/kg are somewhat higher than those (aver. 0.017 mg/kg) reported by Sippola and Mäkelä-Kurtto (1986) from Finland but lower than the mean Cd contents of grasses in eight countries reported by Kabata-Pendias and Pendias (1984): West Germany 0.07, Poland 0.08, Iceland 0.21, DDR 0.27 and Czechoslovakia 0.6 mg/kg in d.m.

In the international FAO plant sample (n=3664) material (Sillanpää and Jansson 1992) from 30 countries, the world Cd mean of wheat shoot samples taken at mid-tillering stage and maize shoot samples taken at 5 to 6-leaf stage were 0.103 and 0.120 mg/kg d.m, respectively. The highest Cd contents in plants exceeded the lowest as much as about 1500-fold.

Root crops are very effective Cd collectors. They generally have higher mean Cd contents (0.1-0.7 mg/kg d.m.) both in their tops and roots than many other crops (Sillanpää and Jansson 1991). The high availability of soil Cd to plants may be an explanation as to the high Cd contents present in the roots of the root crops. The still higher Cd contents in the tops of the root crops may be explained so that the broad leaves receive much more exposure to external Cd contamination from the air than those of the other crops.

According to Niemi and Hallikainen (1993) the concentration of Cd in the Finnish potato tubers was 0.01 mg/kg f.m. (dry matter content 22%), i.e. 0.045 mg/kg d.m. and according to Venäläinen et al. (1999) less than 0.01 mg/kg f.m.

Kabata-Pendias and Pendias (1984) have reported background levels of Cd in common crop plants in various countries. The grand mean values for all cereal grains range between 0.013 to 0.22, for grasses between 0.07 to 0.27 and for legumes between 0.08 to 0.28 mg/kg d.m. The Cd contents of the Finnish crops (Sillanpää and

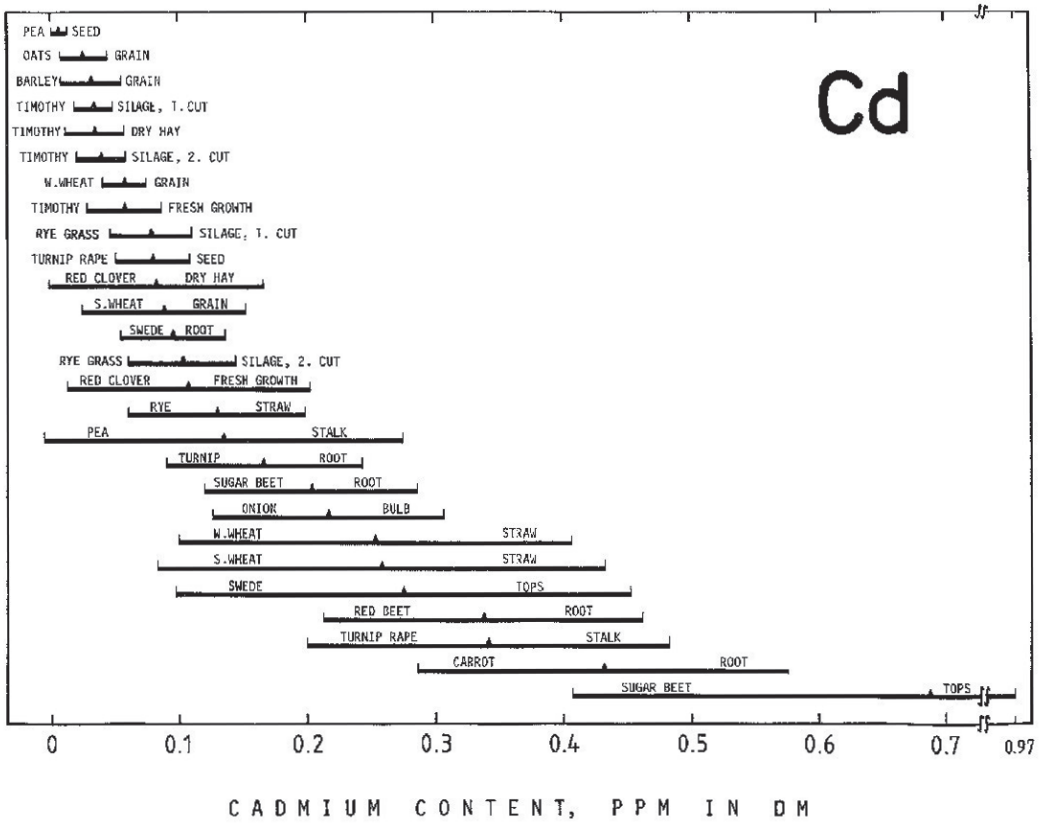

Figure 12. Two-year averages of cadmium contents of different parts of 16 crops grown side by side (Sillanpää and Jansson 1991).

Jansson 1991) are very low when compared to these international background values.

In general, vegetables, especially lettuce and spinach, and other plant species grown in the garden contain much more Cd than cereals. Furthermore, cultivated plants may take up Cd and other heavy metals at excessive levels, too, if they are grown inpolluted soil as indicated by the preliminary research results on the previous pot experiments conducted by the Agricultural Research Centre of Finland.

3.1.4 Cadmium concentration in ground water

Geological Survey of Finland has monitored the quality of groundwater in Finland since 1969. During that time period, a decreasing trend in the groundwater cadmium was observed both in till and in sand aquifers (Backman et al. 1999). The decrease was drastic during the 1980s. A mean and median concentration of cadmium in Finnish groundwater, ie. springs and captured springs, is 0.05 μg/L and 0.03 μg/L, respectively. Aquifers in the areas of intensive agriculture, big cowhouses or pastures generate groundwater with cadmium concentrations higher than averages.

3.2 Biogeochemical behaviour of cadmium

3.2.1 Transformation and distribution in soil

Soil is composed of three phases, solid (mineral and organic), liquid and gaseous, and exhibits properties resulting from the physical and chemical equilibrium of these phases. A knowledge of the association of cadmium with particular soil phases and its affinity to each soil constituents is the key to a better understanding of the principles governing Cd behaviour in soil (Kabata-Pendias and Pendias 1984).

In the soil, Cd is not evenly distributed between various soil phases (solid, water and air), between various soil constituents (mineral and organic constituents) or between various soil profiles i.e. various soil layers.

Usually, cadmium is present:

- as a part of the soil parent material or soil minerals of secondary origin;
- precipitated with other compounds of the soil;
- sorbed on exchange sites. Metal oxides or hydroxides, clay minerals and organic matter can serve as exchangers;
- dissolved in the soil solution, either in the aquaform or complexed with inorganic or organic ligands;
- embodied in micro-organisms, plants or animals (Schmitt and Sticher 1991).

The behaviour of Cd ions in the soil depends on pedological factors such as pH, humus content, cation exchange capacity, redox potential, as well as external factors such as temperature, precipitations, erosion, land use practice etc. Furthermore, the degree of activity, bioavailability and mobility of Cd is influenced by other factors, like competition with other metal ions, ligation by anions, composition and quantity of the soil solution.

The major physico-chemical reactions of Cd in the soil are sorption, complexation, precipitation, biosynthesis, biodecomposition and uptake by the roots. The principal chemical species of Cd in acid soil solution under oxic conditions are Cd^{2+}, $CdSO_4$, $CdCl^+$ and in alkaline conditions Cd^{2+}, $CdCl^+$, $CdSO_4$, $CdHCO_3^+$. Soluble, exchangeable and chelated species of Cd are the most mobile in soils and these factors governs its migration and phytoavailablity (Schmitt and Sticher 1991). The knowledge of the speciation of cadmium in natural and anthropogenically sourced materials, and the chemical species´ bioavailability is incomplete (Garrett 1996).

3.2.2 Adsorption and mobility in soil

The distribution of Cd between the soil solid phase and solution phase is considered by Christensen et al. (1990) to be the key in the evaluation of environmental consequences of Cd when applied to land. Gerritse et al. (1983) showed that the uptake of Cd by the plants is highly correlated to Cd concentration in the soil solution.

The dominating mechanism governing the distribution of Cd between soil and soil solution is sorption (Christensen et al. 1990), which includes adsorption, absorption, chemisorption, ionexchange and surface complexation. Precipitation processes, like in terms of carbonates, phosphates and sulphides, are supposedly - at environmentally relevant concentrations - only of significance in strongly anaerobic, sulphide containing environments. Many soil components, like clay minerals, sesquioxides, organic matter and calcite, are able to sorb Cd.

Cadmium sorption in soils is from an environmental point of view a fast process reaching equilibrium within 1 hour, the sorption process approximately is fully reversible and the distribution of Cd between soil and solute is independent of its origin (Christensen et al. 1990). This indicates that although the soil may have a significant capacity to sorb Cd, the soil is no permanent sink and previously sorbed Cd may be released upon changes in the soil solution composition.

The main factors affecting the distribution of Cd between soil and solute are pH, Ca concentration of the solute and maybe the presence of Zn in the soil solution (Christensen et al. 1990). Soil pH is the dominating factor accounting for 72% of total variation of the distribution of Cd between the solid and solute. Calcium, if significantly increased, may to some extent compete with Cd for sorption sites, while Zn at concentrations above 0.3 mg/l soil solution may compete significantly with Cd for sorption sites.

As compared to other toxic heavy metals, like lead and mercury, Cd is exceptionally soluble in soil. Soluble, exchangeable and chelated species of Cd are the most mobile in soils and this governs its migration and phytoavailablity. Cadmium is relatively mobile between the pH levels of 4.2-6.6 and only moderately mobile between the pH levels of 6.7-8.8 (Schmitt and Sticher 1991).

In several experimental studies, including field trials, Cd has been found to transfer from the surface soil (0-25 cm) to the lower soil horizons as reviewed by Jensen and Bro-Rasmussen (1990). Mobility of the Cd in the soil seems to be dependent on the source. Sequential extraction analysis of Cd by Chlopecka et el. (1996) gives support to the view that metals from anthropogenic sources are in general more mobile than those from native origins, like soil parent materials. The results of Jones et al. (1987) on the field experiment since the mid-1800´s to the present day indicate that Cd entering the soil in phosphate fertilizers is not retained in the soil to the same extent as that from atmospheric deposition and from manure.

The current EU policy is to set aside a significant part of the area of agricultural soils because of costly overproduction. In Finland, totally 200 000 ha of arable land has been afforested during the last thirty years (Hynönen and Hytönen 1998). Dutch research results referred by del Castilho et al. (1996) show that the changing of agricultural land into forest land may have significant implications for the solubility and mobility of Cd in the ecosystem. After the termination of liming, the soil pH decreased to 3.5-4 within 3 to 4 decades, indicating a rapid acidification of the topsoil which lead to a significant increase in the Cd concentrations in the soil solution. Especially below pH 5, dissolved Cd concentration increased rapidly due to pH-dependent desorption from the soil surface. Mobility of Cd was increased also in the deeper soil layers 40-80 cm. This indicates that soil pH is one of the most important factors determining not only the short-term, but also the long-term variations in the mobility of Cd in the soil.

3.2.3 Transformation in water

Heavy metal concentrations in headwater lakes and streams are heavily influenced by bedrock lithology, soil granulation and chemistry of the catchment, land-use such as cultivation, fertilization and ditching in the catchment area and the main hydrogeochemical parameters such as pH, alkalinity and total organic carbon (TOC) (Tarvainen et al. 1997).

In the aquatic environment Cd is found in various forms: Cd^{2+} is the most important dissolved species in freshwater whereas in seawater the dissolved chloride-complexes are the most important.

The pH and redox potential of water have a major effect on the physical and chemical forms of metals and metal compounds in an aquatic environment. Increasing the acidity increases the free metal ion concentration in solution.

Water hardness, contributed in most natural waters by calcium (Ca^{2+}) and magnesium (Mg^{2+}), affects bioavailability of metals by competing with metal cations for binding sites of anions in the water. The ionic competition results in a redistribution in the concentrations of metal salts, sometimes changing the ratio of a dissolved to solidphase metal by forming insoluble salts (ICME 1995).

Organic ligands can play a major role in aquatic systems. Metals bind to dissolved organic ligands primarily at carboxylic and phenolic functional groups.

Cd may be bound to suspended particles. This depends on the content of the suspended material, the particle size distribution, pH, temperature and on the complexants present. The proportion of the cadmium bound to suspended material is large in fresh inland waters (>50%), small in the coastal waters (10-20%) and minor in the open ocean (1%) (Ros and Slooff 1988).

Also, it has been well established that naturally occurring dissolved organic compounds such as humic acids have a substantial effect on the bioavailability of metals (ICME 1995). Complexation of metal ions usually reduces their effective concentration in water and, in many cases, also their biological availability and toxicity to animals. On the other hand, there is contradictory information on the interrelationships between the decreased bioaccumulation and toxicity of cadmium to fish and invertebrates in humic waters (Ramamoorthy and Blumhagen 1984, Winner 1984, Oikari et al. 1992).

The interface between the sediment and water column represents an important environment governing the fate of metals in an aquatic environment. As particulate matter falls through the water column, metals are scavenged and incorporated into the bottom sediment (ICME 1995). This phenomenon increases when large quantities of calcium carbonate percipitate. If the sediment gets anaerobic and sulphate reduction takes place, the cadmium will be bound as insoluble Cd sulphide. Remobilisation occurs when the pH decreases; as a result of the microbial process; possibly at very high sulfide concentration (>30 mg/l); by oxidation; and by decomposition of organic material (Ros & Slooff 1988).

Bioturbation of sediments by benthic organisms disrupts the water/sediment interface and brings oxygen in the sediment, thus enabling Cd to redissolve.

In Finland lake water is typically very soft (mean hardness $\leq$ 10 mg $CaCO_3$). The median pH in Finnish lakes is 6.6 (Mannio and Vuorenmaa 1996). The proportion of acidic lakes with a pH $\leq$ 5 was in 1987 about 10% (Forsius et al. 1990). More than half of Finnish lakes can be classified to be brown coloured, humus lakes (colour > 40 mg Pt/l) (Mannio and Vuorenmaa 1996). Concentration of aquatic humic substances in Scandinavian inland waters varies, given as dissolved organic carbon (DOC) generally ~ 5-20 mg C/l (Oikari et al. 1992). The high concentrations of organic matter decrease the pH values of the lakes and the humic lakes are on average more acidic than the clear water lakes (Kortelainen et al. 1989). In humic waters, eleva-

ted concentrations of especially land-derived metals are to be expected. Due to their complexing ability and their acidity, humic substances can also transport metals which in other conditions would be retained in the soils of the catchment (Mannio et al. 1993). All these typical characteristics of Finnish waters make Cd more soluble and more bioavailable to aquatic organisms.

3.3 Uptake by plants

It is generally thought that the chemical form of Cd taken up by plants is the free uncomplexed Cd^{2+} ion present in soil solution. Thus, any treatment or changes in soil conditions which affect the concentration or activity of the Cd^{2+} will affect the plant accumulation of Cd. The factors affecting the uptake of Cd by plants can be summarized as follows (modified from McLaughlin et al. 1996 and Chaney and Hornick 1978):

* Atmospheric factors
  - depositions, dry and wet
* Plant factors
  - plant species
  - plant cultivar
  - plant part
  - age of the plant or plant part
  - root excretes
  - depth of roots in the soil
* Soil factors
  - soil parent material (age, texture, $CaCO_3$)
  - total amount and solubility of soil Cd
  - amount and nature of clay
  - amount and nature of organic matter
  - soil salinity
  - soil pH
  - redox potential
  - cation exchange capacity
  - interactions between other elements (Zn, etc.) and substances
* Weather and climatological factors
  - temperature
  - precipitation
* Cultivation management practices
  - fertilization (N, P and their forms)
  - liming agents
  - soil improving agents (wastes: ashes, sewage sludges, composts etc.).

Soil factors that increase the uptake of Cd by plants are low pH, high salinity, high Cd concentration, low organic matter content, low cation exchange capacity, low clay, Fe and Mn oxides concentration, Zn deficiency, presence of NH^{4+}, high temperature. In spite of Zn fertilization (100-200 g Zn/ha/a), a great deal of Finnish cultivated soils, especially clay soils in southern Finland, are deficient of Zn that imposes a risk for the high Cd uptake by the plants.

Plant species and plant parts differ in their Cd contents. Usually, the concentration of Cd decreases in various plant species and plant parts in the following order: leafy vegetables > root vegetables > cereals > fruits. Older plants or plant parts contain more Cd than younger ones.

The most important factor influencing the uptake of Cd by the plants is the pH level of the soil. The amount of Cd present in soil solution is mainly (70%) governed by the soil pH (Christensen 1984). For instance, it has been shown in Sweden (Statens livsmedelsverk 1989; Eriksson et al. 1990) that the Cd content of oat grains has a clear connection with the soil pH. When the pH of the soil was increased by one pH unit in the pH range of 4.7-7.3, the Cd content of grains was decreased to about a half. Uptake of Cd by the plants can be regulated with the aid of costly liming, but not always and not for all plant species and plant parts. According to Jackson and Alloway (1991) liming of the soil was not able to reduce the Cd content of potato tubers.

The availability of cadmium to plants depends not only on the pH of the soil, but also on the pH of the rhizosphere. The plant roots have ability to acidify their surroundings i.e. rhizosphere in order to solubilize elements for enhancing uptake. Plant species differ in their ability to acidify the rhizosphere (Youssef and Chino 1991). For in-

stance, the roots of the beans can solubilize and take up metals much better than the roots of barley. Dicotyledonous plants generally take up metals much more than monocotyledonous plants do (Sauerbeck 1991).

Uptake of Cd by the plants are effected by the application rate and the form of fertilizers, especially the form of nitrogen. If the N is applied to the soil in ammonium form, the soil will be acidified by the roots through the uptake of nutrients. After harvesting three crops, the pH of the soils which received N-fertilization in ammonium form may be three pH units higher than the pH of the soils which received N-fertilization in nitrate form (Dijkshoorn et al 1983).

Cadmium contents of the crops mainly depend on the purity of the soils, because the greatest part of Cd in the plants is mostly absorbed through the roots from the soil and only a smaller part of Cd is of air born origin. According to some estimates, 20 to 60%, with an average of about one third, of plant Cd may be airborne falling down as either wet or dry depositions (e.g., Hovmand et al. 1983; Christensen 1983), but of course, its proportion varies greatly from one location to another. Thus, about two thirds of plant Cd is normally taken up through the roots from the soil.

Cadmium concentrations of the plants are, in general, very high as compared to the low Cd concentrations of the soils (Kabata-Pendias and Pendias 1984). Bioaccumulation of Cd is exceptionally intensive by the plants from the soil as compared to many other trace elements.

Cadmium is generally considered a non-essential element for both plants and animals. Its importance lies in its toxicity. Many plants can accumulate relatively large concentrations of Cd without adverse effect on their growth. Thus, concern is not only for the effect of Cd on plant health, but particularly plant Cd as a source of Cd for herbivores and humans. Consequently, differences in the adsorption rate of cadmium between the plant species are important from both an environmental and health standpoint.

Area-weighted mean Cd offtakes by Finnish plant crops is based on the following national data: the Cd concentration in Finnish crops (Figure 12, Sillanpää and Jansson 1991), the yields and areas of various crops in Finland in 1995 (Table 7), Information Center of Ministry of Agriculture and Forestry 1995). The Cd concentration of potato was reported by Niemi and Hallikainen (1993) and Venäläinen et al. (1999).

3.4 Bioaccumulation

Elemental metals and solid phase inorganic metal compounds are generally not bioavailable as such and therefore, the bioaccumulation of inorganic metal compounds is not a useful parameter for their hazard identification. However, it is their constituent soluble metal cations that undergo a biological uptake (Technical workshop 1996).

Bioconcentration factors for several groups of water organisms studied under laboratory conditions are shown in Table 8. BCF range from 16 to 130 000 and do not seem to show any consistent pattern (WHO 1992). Some species accumulate metals to high levels (e.g. zooplankton), while other species such as fish closely regulate internal concentrations or sequester the metal with cellular binding proteins (e.g. metallothioneins). Filter-feeding aquatic organisms generally accumulate more than any other group (ICME 1995).

Bioconcentration factors and biomagnification factors were calculated for the freshwater isopod *Assellus aquaticus* by van Hattum et al. (1989, ref. in WHO 1992). Much of the cadmium (added as the chloride) was taken up from the water (BCF 18000), but there was little uptake from food (BCF 0.08). Experiments conducted at two different pHs (5.9 and 7.6) revealed no significant effect of pH on uptake of cadmium by the isopod. Metals are not generally known to biomagnify in aquatic food chains (ICME 1995).

Table 7. Data on which mean national area-weighted Cd offtakes by various Finnish crops are based.

Crop	Cd	Dry matter	Cd	Yield in 1995	Cd uptake	Area in 1995	Total Cd uptake
	mg/kg DM	%	mg/kg DW	kg/ha	g/ha	1000 ha	kg
Wheat	0.06	86	0.0516	3770	0.195	101	19.6
Rye	0.03	86	0.0258	2770	0.071	21	1.5
Barley	0.03	86	0.0258	3600	0.093	516	47.9
Oat	0.025	86	0.0215	3330	0.072	329	23.6
Sugar beet roots	0.2	23	0.046	31900	1.467	35	51.4
Oil rape seeds	0.08	92	0.0736	1500	0.110	84	9.3
Pea seeds	0.01	86	0.0086	2422	0.021	4.5	0.1
Potato	0.045	22	0.01	22167	0.222	36	8.0
Hey	0.035	83	0.02905	3780	0.110	287.1	31.5
Forage grass 1.	0.035	23	0.00805	18720	0.151	300.9	45.3
Forage grass 2.	0.04	23	0.0092	8410	0.077	40.6	3.1
All together						1755.1	241.3
Mean of 11 crops					**138**		

Although metals do bioaccumulate in terrestrial food-chains, their BAFs (biomagnification factor) are generally low. Field-based BAF for cadmium in plant and plant-frugivore food-chains was less than one (Hunter and Johnson 1982, ref. in ICME 1995). In another field study, the BAF was 0.06 for the cadmium in kidneys of a herbivore (Pascoe et al. 1994, ref. in ICME 1995). However, in another study, insect herbivores, detrivores and predators bioaccumulated cadmium at factors 1.1 to 5.4 times higher than their food items (Hunter and Johnson 1982).

3.5 Accumulation module

The total Cd content (Cd_{tot}) in agricultural soils is the result of inputs of Cd from several sources, such as parent material (Cd_p); atmospheric deposition (Cd_a); fertilizers (Cd_f); agrochemicals (Cd_{ac}); wastes (Cd_w) minus Cd removed in crop material (Cd_c); and Cd in losses through leaching, erosion and volatilization (Cd_l) as described by the following formula:

$$Cd_{tot} = (Cd_p + Cd_a + Cd_f + Cd_{ac} + Cd_w) - (Cd_c + Cd_l)$$

One of the best ways to predict the trend of Cd concentration of the soils for the future is a mass balance calculation which considers the inputs of Cd into the soil and outputs of Cd from the soil.

Different approaches to assessing heavy metal balances are presented by Moolenaar (1999b). A static balance is comparable to a black box model that finds relationships between the input and output of a system without knowing the system´s structure and behaviour. In a static balance, a record is kept of the input and output flows, where the output flows are assumed to be unrelated to the metal content in the soil. The change in heavy metal content in the plough layer is therefore the result of the net difference be-

Table 8. Bioconcentration of cadmium in organisms in laboratory studies.

Organism	Size	Stat/ flow	Organ	Temp. (°C)	Duration (days)	Exposure (μg/l)	BCF	Reference
Freshwater alga (*Chlorella vulgaris*)					10	10	3000 dw	Ferard et al. 1983
Freshwater alga (*Scenedesmus obliquus*)		stat		20-22	14	250	4940 daw	Cain et al. 1980
Freshwater diatom (*Asterionella formosa*)		flow	whole body		23	10	40000	Conway 1978
American oyster (*Crassostrea virginica*)	4.9-5.1 g	flow	whole body	16-20	21	10	116 ww 4280 aw	Eisler et al. 1972
	8.1 g	flow	soft tissues	. 2.8-22.6	280	5	2376 ww 18472 dw	Zaroogian & Cheer 1976
Mussel (*Mytilus edulis*)	32-34 mm	flow	soft tissues	13	166	10	50802 dw	Riisgard et al 1987
Scallop (*Aquipecten irradians*)	6.8-7.7 g	flow	whole body	16-20	21	10	131 ww 3970 aw	Eisler et al. 1972
Bay scallop (*Argopecten irradians*)	0.51-0.73 g	flow	soft tissues	9.5-16	42	60	20400	Pesch & Steward 1980
Crab (*Pandalas montaqui*)	2-4 g		whole body	10	14	37	152 dw	Ray et al. 1980
Grass shrimp (*Palaemonetes pugio*)	20-33 mm	flow	whole body	9.5-16	42	60	223	Pesch & Steward 1980
Lobster (*Homarus americanus*)	160-169 g	flow	whole body	16-20	21	10	21 ww 10 aw	Eisler et al. 1972
Fathead minnow (*Pimephales promelas*)		flow	whole body	13.9-15.3	21	49	190	Sullivan et al. 1978
Red maple (*Acer rubrum*)			leaves	15-27	45	0.5	14400 dw[a]	Mitchell & Fretz 1977
			roots	15-27	45	0.5	131800 dw[a]	-"-
			leaves	15-27	101	2.6 mg/kg	0.76 dw[b]	-"-
			roots	15-27	101	2.6 mg/kg	12.5 dw[b]	-"-
White pine (*Pinus strobus*)			leaves	15-27	66	0.5	3400 dw[a]	Mitchell & Fretz 1977
			roots	15-27	66	0.5	118400 dw[a]	-"-
			leaves	15-27	365	2.6 mg/kg	1.2 dw[b]	-"-
			roots	15-27	365	2.6 mg/kg	10.4 dw[b]	-"-

[a] The medium was a cadmium-enriched nutrient solution and [b] a cadmium-amended soil mix,dw = dry weight; ww = wet weight; aw = ash weigt, daw = dry ash weight

tween input and (constant) output rates. Because a static balance does not address the dependance between soil content and output flows, it cannot realistically simulate the heavy metal soil content in time. However, it is an attempt to clarify long-term effects of Cd inputs to the development of the cadmium concentration in soil. For simulating the fate of metals in time, a dynamic balance may be calculated in which the relationship between heavy metal soil content and output flows is explicitly included. A dynamic balance equation could thus be derived in terms of heavy metal content by relating the output rates (e.g., by leaching and crop uptake) to the metal content in soil. Also according to Moolenaar (1999a) heavy metal balance studies tend to suffer from a lack of recent and accurate data and hence unreliable, averaged, or estimated numbers are being used. Leaching is one of the most difficult flows to quantify reliably, by measurements or models. Therefore, in most balance studies, leaching is either neglected or simulated in a simplified way.

3.5.1 Methodology

Methodological procedures used in the accumulation module was directly taken from the report of ERM (2000). Estimations in the module were areally applied in southern Finland, cultivation zones I-III, where 70-80% of all the Finnish arable land is situated. Estimations were focused to an average Finnish field hectare and to wheat, potato and sugar beet fields in the same area. These cultivation systems were selected for module estimations, because wheat and potato crops form of a major portion of foodstuff consumption of the Finnish population. In Finland, 45% of cadmium intake via food comes from wheat and nearly 60% from the cereals together. In addition, more than a half of the Finnish arable land area is under cereal cultivation. Instead, sugar beet represents an exception among the plants because it has a relatively high ability to take up cadmium from the soil.

Current general characteristics of the fields assessed in the accumulation module are presented in table 9.

3.5.1.1 Cadmium inputs

Cadmium inputs from various sources and from P-fertilizers with different Cd concentrations assessed in accumulation module are presented in table 10. The mean atmospheric deposition of cadmium in the area assessed in the accumulation module was 0.3 g/ha/a and was based on the measurements of the Finnish Meteorological Institute in 1997-99.

Today, an average field hectare in Finland annually receives about 10 kg of P. Application rates (kg/ha/a) of P to wheat, potato and sugar beet were based on the current national fertilizer recommendations and the Finnish agro-environmental programme according to the Council regulation (2078/92) and were 15, 40 and 30 kg/ha/a, respectively. Three various concen-

Table 9. General characteristics of the fields assessed in accumulation module.

	Cultivation zone	pH (H_2O)	OM %	Zn mg/kg d.m.	D.m. yield kg/ha/a	Cd in crop mg/kg d.m.	Cd uptake g/ha/a
Average field	I-III	5.8	10		3275	0.042	0.138
(mean of 11 crops)							
Wheat field	I-III	5.8	10	60	3242	0.06	0.195
Potato field	I-III	5.8	10		4877	0.045	0.222
Sugar beet field	I-III	6.4	10		7337	0.2	1.467

Table 10. Cadmium inputs from various sources and from P-fertilizers of different Cd concentration to fields assessed in accumulation module.

Cadmium inputs	Use of P kg/ha/a	Atm. dep. g/ha/a	P-fertilizer g/ha/a	Liming g/ha/a	Manure g/ha/a	Wastes g/ha/a	Total inputs g/ha/a
Average field							
Cd 2.5 mg/kg P	10	0.3	0.025	0.035	0.322	0.023	0.705
Cd 50 mg/kg P	10	0.3	0.500	0.035	0.322	0.023	1.180
Cd 138 mg/kg P	10	0.3	1.380	0.035	0.322	0.023	2.375
Wheat field							
Cd 2.5 mg/kg P	15	0.3	0.0375	0.035	0	0	0.373
Cd 50 mg/kg P	15	0.3	0.7500	0.035	0	0	1.085
Cd 138 mg/kg P	15	0.3	2.0700	0.035	0	0	2.405
Potato field							
Cd 2.5 mg/kg P	40	0.3	0.10	0	0	0	0.400
Cd 50 mg/kg P	40	0.3	2.00	0	0	0	2.300
Cd 138 mg/kg P	40	0.3	5.52	0	0	0	5.820
Sugar beet field							
Cd 2.5 mg/kg P	30	0.3	0.075	0.035	0	0	0.410
Cd 50 mg/kg P	30	0.3	1.500	0.035	0	0	1.835
Cd 138 mg/kg P	30	0.3	4.140	0.035	0	0	4.475

trations of cadmium in P-fertilizers were selected for assessing in the accumulation module: 1) the current Finnish mean cadmium concentration 2.5 mg/kg P, 2) the Finnish limit value 50 mg/kg P and 3) the European mean value 138 mg/kg P.

An input value from liming, 0.035 g/ha/a (Table 6), was based on the mean national use of liming agents for an average field hectare and for wheat and sugar beet fields. Instead, there was no input from liming for potato cultivation. Hence potato can be cultivated in rather acidic soils, too.

In the accumulation module, animal manure and other organic wastes were used only in the average field hectare. In other cases, only mineral fertilizers were applied.

3.5.1.2 Distribution coefficient (K_D) of cadmium

For the determination of distribution coefficient (K_D) of cadmium in the soil the following algorithms proposed by the ERM (2000) were applied and tested in the accumulation module:

1) algorithm of Anderson and Christensen (1988)

$$\log K_D = -1.22 + 0.56\ (\%C) + 0.55\ pH$$

where

%C = percentage organic carbon

This algorithm was developed for 38 soils from 13 different agricultural experimental stations, with low Cd concentrations. Electrolyte contained 0.001 M $CaCl_2$. Other soil properties also included. Tested in Denmark.

2) algorithm of Christensen (1989)

$\log K_D = - 1.35 + 0.587\ pH + 0.157\ (\%OM)$

where

%OM = percentage organic matter

This algorithm was developed for 63 agricultural soil with pH of 4.1-7.7, OM of 0.3-4.1%, and K_D range 15-2450 L/kg. Electrolyte contained 0.001 $CaCl_2$. pH is for soil solution. 0.4 should be added to pH of soil to convert to pH of soil solution. Tested in Denmark.

3) algorithm of McBride et al. (1997)

$\log Cd_l = 3.62 - 0.5\ pH + 0.96 \log Cd_T - 0.45 \log (OM)$

where

Cd_l = cadmium concentration in soil solution (μg/L).
Cd_T = total cadmium content of soil (mg/kg dry weight)

This algorithm was reworked of previously published data (Gerritse and van Driel 1984) and developed for 31 agricultural soils, many with elevated cadmium concentrations, range 0.1-90 mg/kg, pH range 4.3-7.8, OM range 11-340 g/kg. Soil solution data used were obtained by water extraction. Tested in French, Dutch and UK soils. A conversion factor of 0.85 was taken into account of the transition from Cd^{2+} in order to achieve Cd_L (Wenzel 1999).

4) algorithm of Romkens (pers. comm., 2000)

$\log K_D = -1.00 + 0.44\ pH + 1.03 \log (\%OM)$

where

%OM = percentage organic matter

This algorithm was reworked of data in Romkens and Salomons (1998) to develop an algorithm with pH and OM. $R^2 = 0.76$. Tested in the Netherlands.

3.5.1.3 Annual precipitation excess

In the accumulation module, the annual precipitation excess (F) was calculated using the following formula:

$$F = P - (E+S)$$

where:

P = the rate of precipitation (m/a)
E = the rate of evapotranspiration (m/a)
S = the rate of surface run off (m/a)

Long-term averages for annual precipitation and evapotranspiration in southern Finland, particularly for cultivation zones I to III, are 0.600 and 0.300 m, respectively (Atlas of Finland 1960, Turtola and Jaakkola 1986). The annual surface run off was evaluated to be 0.100 m. Thus, the annual precipitation excess was 0.200 m.

3.5.1.4 Dynamic mass balance model

In order to calculate temporal trend in cadmium accumulation in soil, a dynamic mass balance model developed by Harmsen (1992), Boekhold and van der Zee (1991), Vissenberg and van Grinsven (1995), Moolenaar et al. (1995, 1997) and Tiktak et al. (1999) and recommended by ERM (2000) was used in accumulation module. The mass balance equation applied was the following:

$$Cd_s(t) = Cd_s(0)\ e^{-(kp + kl + ke)\,t} + \{ki / (10 \rho d_p)(kp + kl + ke)\}\{1 - e^{-(kp + kl + ke)\,t}\}$$

where

$Cd_s(0)$ = initial (present day) concentration of Cd in soil (mg/kg d.m.)
$Cd_s(t)$ = cadmium concentration in the plough layer at time t years (mg/kg d.m.)
ki = input rate of cadmium (g/ha/a)

kp = cadmium offtake rate by plants (g/ha/a)
kl = cadmium leaching rate (g/ha/a)
ke = cadmium erosion rate (g/ha/a)
t = time (a)
þ = soil bulk density (1000 kg/m3)
d_p = depth of plough layer (0,2 m)

A cadmium concentration of the soil at the steady state (SS) was calculated according to the following formula:

$$Cd_s(SS) = k_i / \{(10 þ d_p)(k_p + k_l + k_e)\}$$

3.5.1.5 Determination of plant Cd as a function of soil properties

Plant cadmium as a function of soil properties were estimated for the fields under wheat, potato and sugar beet cultivation by using the following algorithms:

1) for wheat grains algorithm of Eriksson et al. (1996):

$$Cd_p = 78.8 - 7.26\ pH - 1.58\ (\%OM) + 0.8\ (\%Clay) + 184.8\ Cd_s - 0.73\ Zn_s$$

where

%Clay = 45

2) for potato tubers algorithm of Eriksson et al. (1996):

$$Cd_p = 193 - 24.1\ pH - 0.94\ (\%OM) + 39\ Cd_s$$

3) for sugar beet roots algorithm of Sippola (2000):

$$Cd_p = 905 - 120\ pH + 300\ Cds$$

3.5.2 Scenarios for a 100-year period

In general, scenarios estimated in the accumulation module resulted in a large variation in the cadmium distribution coefficient (K_D) depending on the algorithm used for the determination. For demonstrating this, all the scenarios estimated are presented in this report. However, McBride´s and Romkens´ algorithms developed for the contaminated soils or soils with elevated Cd concentrations could not be considered suitable for agricultural soils with low cadmium concentration in Finland. Instead, algorithms of Anderson´s and Christensen´s were developed for cultivated soils of low cadmium content. Therefore, these algorithms, particularly Christensen´s algorithm, could be believed to indicate realistically the future trends in Finland.

3.5.2.1 Cadmium concentration in pore water/leachate

Cadmium concentrations in the pore water/leachate obtained by the various algorithms differed from each others by more than 500 to 1000 times depending on the time and the Cd content of P-fertilizers (Table 11). At the initial time (0), the concentration order from the lowest to the highest values by the algorithms was the following: by Anderson´s algorithm 0.000-0.001; by Christensen´s algorithm 0.022-0.050; by McBride´s algorithm 0.087-0.174; and by Romkens´ algorithm 0.299-0.550 µg/L. These concentrations could not be compared to any Finnish real values, because there are no scientific measurements available in Finland on the Cd concentrations in the soil water. In order to select the most probable algorithm for further scenario calculations, some theoretical speculations were made. A mean and a median Cd concentration in the ground water for springs and captured springs in Finland are 0.050 µg/L and 0.030 µg/L, respectively (Backman et al. 1999) and are of the same magnitude as the values obtained by the Christensen´s algorithm. However, the cadmium concentration in the soil solution of the plough layer of cultivated fields could be higher than that in the groundwater, because the topsoil is a direct subject of high inputs. On the other hand, Finnish cultivated soils have high levels of clay and organic matter which may effectively accumulate cadmium. In addition, cultivated soils are regularly limed in Finland.

If about 40% of total cadmium in cultivated soils is considered to be mobile or mobilizable in

the Finnish conditions (Sippola and Mäkelä-Kurtto 1986 and Mäkelä-Kurtto et al. 1992) it means that 168 g of the total cadmium amount (420 g = 0.21 mg/kg x 2 000 000 kg) in the plough layer (0.2 m) could be soluble or solubilizable. If water content in soil is 20%, the amount of soil water in the plough layer in the area of one hectare is 400 000 L. Then the Cd concentration in soil water could be 0.042 µg/L. Also this value is the closest to the respective values obtained by Christensen´s algorithm.

The Cd concentrations obtained by Anderson´s algorithm seem to be unrealistic low as compared to the Cd concentrations in the Finnish ground water. Because McBride´s algorithm was developed for the soils with elevated Cd content, this algorithm could not be considered as a proper selection for the Finnish cultivated soils with low Cd concentration. Romkens´ algorithm gave still higher pore water concentrations than McBrides´s algorithm did. These were the reasons why the Finnish scenario calculations will

Table 11. Cadmium concentrations in pore water/leachate, µg/L, at different times and at different Cd contents of P-fertilizers and changes (%) over 100 years.

ACCUMULATION MODULE							
Cd concentration in pore water, µg/L							
		Cd 2.5 mg/kg P		**Cd 50 mg/kg P**		**Cd 138 mg/kg P**	
Time elapsed, in years	**0**	**100**		**100**		**100**	
	µg/L	**µg/L**	**%**	**µg/L**	**%**	**µg/L**	**%**
Wheat field							
Anderson	0.001	0.001	8	0.002	25	0.002	50
Christensen	**0.050**	**0.050**	**0**	**0.058**	**16**	**0.072**	**45**
McBride	0.174	0.164	-6	0.191	10	0.240	38
Romkens	0.550	0.438	-20	0.518	-6	0.665	21
Potato field							
Anderson	0.001	0.001	0	0.002	100	0.003	200
Christensen	**0.050**	**0.049**	**-2**	**0.071**	**42**	**0.112**	**124**
McBride	0.174	0.162	-7	0.234	35	0.363	109
Romkens	0.550	0.428	-22	0.646	18	1.045	90
Sugar beet field							
Anderson	0.001	0	-30	0.001	6	0.001	68
Christensen	**0.022**	**0.150**	**-31**	**0.023**	**5**	**0.037**	**67**
McBride	0.087	0.059	-33	0.089	2	0.139	60
Romkens	0.299	0.172	-43	0.276	-8	0.451	50
Average field							
Anderson	0.001	0.001	10	0.002	22	0.002	42
Christensen	**0.050**	**0.054**	**8**	**0.060**	**20**	**0.070**	**40**
McBride	0.174	0.178	2	0.196	13	0.230	32
Romkens	0.550	0.477	-13	0.531	-3	0.632	15

be focussed mainly on the results obtained by Christensen´s algorithm.

Cultivation system had an effect on the Cd concentration of the pore water/leachate. In general, the highest Cd concentrations occurred in the potato cultivation and the lowest ones in sugar beet cultivation. The reasons for the low concentrations in the sugar beet cultivation obviously were the high soil pH and the high Cd uptake by sugar beet roots. Instead, in the potato cultivation, soil pH was low, Cd uptake by potato tubers was low and an application rate of P-fertilizers was high.

Particularly, the cadmium content of P-fertilizers has a marked effect on the cadmium concentration in the pore water/leachate. Scenario calculations (Table 11) indicated that during a 100-year period, the cadmium concentrations in the pore water/leachate will change from the value at the initial time (0) by -31 to 8%, 5 to 42% and 40 to 124 % when the Cd contents of P-fertilizers were 2.5, 50, 138 mg/kg P, respectively. If the P-fertilizers with cadmium content equal to the current Finnish mean value will be annually used to the fields, an increase in the cadmium concentration in pore water/leachate will be 8% in the average Finnish field. In the wheat field, no change will occur. Instead, in the potato fields after a 100-year period pore water cadmium will decrease by 2% and in the sugar beet fields even by 31%.

If the P-fertilizers contain cadmium 50 mg/kg P which is a Finnish limit value, the cadmium concentration in pore water/leachate will increase during 100 years in all the cultivation systems: in sugar beet cultivation by 5%, in wheat cultivation by 16%, in the average field by 20% and in potato cultivation by 42% when the scenarios were calculated with the aid of Christensen´s algorithm. P-fertilizers of European quality will lead to increases of 67%, 45%, 40% and 124%, respectively.

In conclusion, Christensen´s algorithm seemed to give the most realistic values for pore water/leachate cadmium because they had the best accordance with the respective values obtained by the theoretical calculations. Thus, the figures based on the Christensen´s algorithm were considered to be the most suitable for the Finnish conditions and will be mainly focussed in this report. The Finnish pure P-fertilizers have only a slightly increasing or even decreasing effect on the cadmium concentration of pore water/leachate during a 100-year period. Instead, P-fertilizers meeting the Finnish limit value will cause an increase of 5-42% and P-fertilizers of European quality will increase the cadmium concentration of pore water/leachate by 40-124% depending on the cultivation system. This will lead to higher cadmium concentrations in drinking, ground and surface waters.

3.5.2.2 Cadmium leaching rate

The present leaching rates (at the time 0) based on the algorithm of Christensen varied from 0.044 to 0.100 g/ha/a depending on the cultivation system (Table 12). According to the current groundwater figures, a mean 0.05 μg/L and a median 0.03 μg/L) and to the annual precipitation excess 0.2 m (= 2 000 000 L/ha) the outputs of cadmium via leaching in Finland could be 0.100 and 0.060 g/ha/a, respectively. These values are quite similar to the respective figures obtained by the Christensen´s algorithm. In the Netherlands, Moolenaar and Lexmond (1998) evaluated cadmium losses via leaching and resulted in a leaching rate of 0.060 g/ha/a. Their adsorption model was the following: $c = 0.19 q_i^{1.47}$ where c = Cd concentration in solution (mg m^{-3}) and q_i = the initial Cd concentration in soil (mg kg^{-1}). High pH and $CaCO_3$ content were the key factors for this low cadmium leaching in the Netherlands. An application of this formula to the Finnish conditions gives Cd leaching rate of 0.057 g/ha/a. In Finland,low pH of the cultivated soils has an increasing effect on the cadmium leaching, but a high organic matter content, a high clay content and regular liming have a decreasing effect on it.

According to Christensen´s algorithm leaching rate will increase by 8% in the average field, and decreased by 2% in the potato field, and by 31% in the sugar beet field while no changes will occur in the wheat field, if the typical Finnish P-fertilizers will be used for 100 years. A clear increase, from 5 to 42%, was observed, if the cadmium content of P-fertilizers will be the maximum allowable in Finland. If the European type fertilizers will be used an increase in leaching rates will vary between 40-123%. The highest leaching rate will be found in the potato cultivation.

In conclusion, the Finnish pure P-fertilizers seem to be an efficient way to manage the leaching rate of cadmium from the soil to the waters. However, P-fertilizers meeting the Finnish cadmium limit requirement may clearly increase cadmium leaching rate when applied no longer than for 100 years. Respectively, typical European fer-

Table 12. Cadmium leaching rate, g/ha/a, from soil at different times and at different Cd contents of P-fertilizers and changes (%) over 100 years.

ACCUMULATION MODULE							
Cd leaching from soil, g/ha/a							
		Cd 2.5 mg/kg P		**Cd 50 mg/kg P**		**Cd 138 mg/kg P**	
Time elapsed, in years	**0**	**100**		**100**		**100**	
		g/ha/a	**%**	**g/ha/a**	**%**	**g/ha/a**	**%**
Wheat field							
Anderson	0.003	0.003	25	0.003	20	0.004	48
Christensen	**0.100**	**0.100**	**0**	**0.115**	**15**	**0.144**	**44**
McBride	0.348	0.328	-6	0.382	10	0.481	38
Romkens	1.100	0.876	-20	1.035	-6	1.330	21
Potato field							
Anderson	0.002	0.003	50	0.004	100	0.006	200
Christensen	**0.100**	**0.098**	**-2**	**0.142**	**42**	**0.223**	**123**
McBride	0.347	0.324	-7	0.467	35	0.726	109
Romkens	1.100	0.856	-22	1.292	17	2.090	90
Sugar beet field							
Anderson	0.001	0.001	-20	0.001	20	0.002	90
Christensen	**0.044**	**0.031**	**-31**	**0.047**	**5**	**0.074**	**67**
McBride	0.174	0.117	-33	0.178	2	0.278	60
Romkens	0.599	0.344	-43	0.553	-8	0.901	50
Average field							
Anderson	0.003	0.003	12	0.003	20	0.004	44
Christensen	**0.100**	**0.108**	**8**	**0.119**	**20**	**0.140**	**40**
McBride	0.348	0.356	2	0.393	13	0.460	32
Romkens	1.100	0.954	-13	1.063	-3	1.263	15

tilizers may more than double the cadmium leaching rate. That will be a harmful trend for the water environment.

3.5.2.3 Cadmium content in soil

When the current Finnish P-fertilizers will be used no changes will occur in the soil cadmium content in the wheat fields during a 100-year period (Table 13). However, in the average field the Cd content will increase by 9%, obviously due to the Cd inputfrom manure. Instead, soil cadmium will decrease in the potato fields by 1% and in the sugar beet fields even by 31%.

By using the medium-Cd P-fertilizers, the soil cadmium content will increase by about 40% in the potato fields, by about 20% in the wheat and average fields and by 5% in the sugar beet fields. In the case of high-Cd fertilizers, increases in the soil cadmium varied from 40% in the average fields to 125% in the potato fields.

Table 13. Cadmium contents of soil, mg/kg d.m., at different times and at different Cd contents of P-fertilizers and changes (%) over 100 years.

Cd ACCUMULATION IN SOIL, mg/kg d.m.							
		Cd 2.5 mg/kg P		**Cd 50 mg/kg P**		**Cd 138 mg/kg P**	
Time elapsed, in years	**0**	**100**		**100**		**100**	
	mg/kg d.m.	**mg/kg d.m.**	**%**	**mg/kg d.m.**	**%**	**mg/kg d.m.**	**%**
Wheat field							
Anderson	0.21	0.215	2	0.250	19	0.314	50
Christensen	**0.21**	**0.210**	**0**	**0.244**	**16**	**0.305**	**45**
McBride	0.21	0.198	-6	0.232	10	0.278	32
Romkens	0.21	0.167	-20	0.198	-6	0.254	21
Potato field							
Anderson	0.21	0.212	1	0.307	46	0.481	129
Christensen	**0.21**	**0.207**	**-1**	**0.301**	**43**	**0.473**	**125**
McBride	0.21	0.195	-7	0.287	37	0.455	117
Romkens	0.21	0.163	-22	0.247	18	0.401	91
Sugar beet field							
Anderson	0.21	0.147	-30	0.224	7	0.354	69
Christensen	**0.21**	**0.145**	**-31**	**0.221**	**5**	**0.352**	**68**
McBride	0.21	0.138	-34	0.214	2	0.343	63
Romkens	0.21	0.120	-43	0.194	-8	0.317	51
Average field							
Anderson	0.21	0.232	10	0.255	21	0.298	42
Christensen	**0.21**	**0.228**	**9**	**0.251**	**20**	**0.295**	**40**
McBride	0.21	0.216	3	0.238	13	0.281	34
Romkens	0.21	0.182	-13	0.203	-3	0.242	15

At the end of 100-year period, the soil cadmium content, 0.473 mg/kg d.m., estimated for the potato cultivation at the high-Cd P-fertilizers was close to the Finnish limit value (0.5 mg/kg d.m.) for cultivated soils receiving sewage sludge (Council of State decision Nr 282/1994).

Soil cadmium concentrations shown by the 100-year scenarios based on Christensen´s algorithm, but also based on Anderson´s algorithm, were in a good accordance with the respective values obtained in the static mass balance calculations (Tables 14-17) in which the mean value for Cd leaching, 0.060 g/ha/a, was based on a Swedish report (Andersson 1992).

In conclusion, the Cd content of P-fertilizers will have a remarkable influence on the soil Cd contents. Due to high application rate of P-fertilizers and low cadmium concentration of potato tubers, cadmium will be accumulated exceptionally fast in the soil under potato cultivation. By using the medium-Cd fertilizers in the potato cultivation the soil Cd will be doubled during 100 years. That must have an impact on soil ecosystem. Instead, sugar beet seems to clean the soil from cadmium, if the fertilizers will be of the Finnish good quality. Even in the case of the low-Cd P-fertilizers, the soil Cd content will increase, if also animal manure and/or other wastes will be applied to the fields.

Cadmium in soil at steady state

At the low-Cd P-fertilizers, the soil Cd content at the steady state (SS) will be equal to the present soil Cd content, 0.21 mg/kg d.m., in the wheat cultivation (Table 18). In the potato and especially in the sugar beet cultivation, the steady state soil Cd will be lower than the present soil Cd content. In the average field which will receive manure, the soil Cd (SS) will be 0,433 mg/kg d.m. This is near to the

Table 14. Static mass balances of cadmium in fields of southern Finland (including plant cultivation zones of I-III) in 2000 by using P-fertilizers containing cadmium 2.5, 50 or 138 mg/kg P and accumulation of cadmium in cultivated soils by 2100.

AVERAGE FIELD			
SOUTHERN FINLAND			
Inputs, g/ha/a	**Cd 2.5 mg/kg P**	**Cd 50 mg/kg P**	**Cd 138 mg/kg P**
Deposition, S. Finland	0.3	0.3	0.3
Fertilizers, P 10 kg/ha	0.025	0.5	1.38
Liming	0.035	0.035	0.035
Manures	0.322	0.322	0.322
Other wastes	0.023	0.023	0.023
All together	0.705	1.18	2.375
Outputs, g/ha/a			
Crops, mean of 11 crops	0.138	0.138	0.138
Waters	0.06	0.06	0.06
Eroded soil	0.11	0.11	0.11
All together	0.308	0.308	0.308
Net mass balance, g/ha/a	0.397	0.877	1.752
Soil Cd, in 2000	0.21	0.21	0.21
Soil Cd, in 2100	0.23	0.25	0.3

Table 15. Static mass balances of cadmium in wheat fields of southern Finland (including plant cultivation zones of I-III) in 2000 by using P-fertilizers containing cadmium 2.5, 50 or 138 mg/kg P and accumulation of cadmium in cultivated soils by 2100.

WHEAT FIELD			
Southern Finland			
Inputs, g/ha/a	**Cd 2.5 mg/kg P**	**Cd 50 mg/kg P**	**Cd 138 mg/kg P**
Deposition, S. Finland	0.3	0.3	0.3
Fertilizer, P 15 kg/ha	0.0375	0.75	2.07
Liming	0.035	0.035	0.035
All together	0.373	1.085	2.405
Outputs, g/ha/a			
Crops	0.195	0.195	0.195
Waters	0.06	0.06	0.06
Eroded soil	0.11	0.11	0.11
All together	0.365	0.365	0.365
Net mass balance, g/ha/a	0.008	0.715	2.04
Soil Cd, in 2000	0.21	0.21	0.21
Soil Cd, in 2100	0.21	0.23	0.312

Table 16. Static mass balances of cadmium in potato fields of southern Finland (including plant cultivation zones of I-III) in 2000 by using P-fertilizers containing cadmium 2.5, 50 or 138 mg/kg P and accumulation of cadmium in cultivated soils by 2100.

POTATO FIELD			
Southern Finland			
Inputs, g/ha/a	**Cd 2.5 mg/kg P**	**Cd 50 mg/kg P**	**Cd 138 mg/kg P**
Deposition, S. Finland	0.3	0.3	0.3
Fertilizer, P 40 kg/ha	0.1	2	5.52
All together	0.4	2.3	5.82
Outputs, g/ha/a			
Crops	0.222	0.222	0.222
Waters	0.06	0.06	0.06
Eroded soil	0.11	0.11	0.11
All together	0.392	0.392	0.392
Net mass balance, g/ha/a	0.008	1.908	5.428
Soil Cd, in 2000	0.21	0.21	0.21
Soil Cd, in 2100	0.2104	0.305	0.4814

Table 17. Static mass balances of cadmium in sugar beet fields of southern Finland (including plant cultivation zones of I-III) in 2000 by using P-fertilizers containing cadmium 2.5, 50 or 138 mg/kg P and accumulation of cadmium in cultivated soils by 2100.

SUGAR BEET FIELD			
Southern Finland			
Inputs, g/ha/a	**Cd 2.5 mg/kg P**	**Cd 50 mg/kg P**	**Cd 138 mg/kg P**
Deposition, S. Finland	0.3	0.3	0.3
Fertilizer, P 30 kg/ha	0.075	1.5	4.14
Liming	0.035	0.035	0.035
All together	0.41	1.835	4.475
Outputs, g/ha/a			
Crops	1.467	1.467	1.467
Waters	0.06	0.06	0.06
Eroded soil	0.11	0.11	0.11
All together	1.637	1.637	1.637
Net mass balance, g/ha/a	-1.227	0.198	2.838
Soil Cd, in 2000	0.21	0.21	0.21
Soil Cd, in 2100	0.149	0.22	0.352

soil limit value presented in the national sewage sludge regulation (Council of State decision Nr 282/1994).

When medium- or high-Cd P-fertilizers will be used the soil Cd contents at the steady state will be very high and will vary between 0.238-1.054 or 0.581-2.667 mg/kg d.m., respectively. These figures are 2-13 times greater than the present soil Cd contents. All the Cd concentrations in soils that will exceed 0.5 mg/kg d.m. can be considered too high for the cultivated soils.

3.5.2.4 Cadmium output via erosion

Finnish measurements on Cd in eroded soil particles are not available from cultivated soils. An annual loss of total solids from the Finnish fields may vary from 50 to 7000 kg/ha depending on many factors like soil type, slope of the fields, precipitation etc., as reported by Uusi-Kämppä (1989). If the mean annual loss of total solids from cultivated soil is supposed to be 500 kg/ha, the amount of Cd eroded annually is 0.105 g/ha based on the mean total concentration of Cd in Finnish cultivated soils,0,21 mg/kg d.m. (Sippola and Mäkelä-Kurtto 1986). This erosion value was used as an initial value in the accumulation module.

The 100-year scenarios show that Cd outfluxes via erosion (Table 19) will vary between 0.073 and 0.235 g/ha/a and will relatively increase in the same proportion as the soil Cd content will do. The outflow of cadmium from the soil to the surface waters via erosion is directly depended on the soil Cd content.

In conclusion, the rate of Cd outfluxes from the soil to the surface waters via erosion directly depends on the soil Cd content.

Table 18. Soil cadmium contents, mg/kg d.m., at steady state at different Cd contents of P-fertilizers.

ACCUMULATION MODULE			
SOIL Cd CONTENTS, mg/kg d.m., AT STEADY STATE			
	Cd 2.5 mg/kg P	Cd 50 mg/kg P	Cd 138 mg/kg P
	mg/kg d.m.	mg/kg d.m.	mg/kg d.m.
Wheat field			
Anderson	0.284	0.826	1.832
Christensen	**0.210**	**0.611**	**1.354**
McBride	0.125	0.364	0.691
Romkens	0.057	0.166	0.368
Potato field			
Anderson	0.233	1.337	3.384
Christensen	**0.183**	**1.054**	**2.667**
McBride	0.119	0.684	1.731
Romkens	0.058	0.331	0.838
Sugar beet field			
Anderson	0.055	0.245	0.597
Christensen	**0.053**	**0.238**	**0.581**
McBride	0.049	0.221	0.538
Romkens	0.040	0.177	0.433
Average field			
Anderson	0.604	1.011	1.765
Christensen	**0.433**	**0.724**	**1.264**
McBride	0.249	0.416	0.727
Romkens	0.110	0.185	0.322

3.5.2.5 Cadmium content of crops

The algorithm of Eriksson et al. (1996) for wheat grains gave the same Cd content as presented in Finland by Niemi and Hallikainen (1993) but for the potato tubers to some extent higher than that reported by the same researchers (Table 20). The algorithm of Sippola (2000) resulted in the same Cd concentration for the sugar beet roots as measured by Sillanpää and Jansson (1991).

The 100-year scenarios indicated that if Cd inputs from the fertilizers will increase, the Cd contents will increase relatively more slowly in the crops than in the soil. However, the clearly fastest increase will occur in wheat grains which are the main source of Cd intake for human population in Finland and in Europe.

With the current low Cd input rate from the P-fertilizer, the Cd contents of the Finnish wheat grains and potato tubers will not increase during the 100-year period, but a clear decrease will occur in the sugar beet roots. If the medium-Cd fertilizers will be used, the Cd contents of wheat,

Table 19. Cadmium output rates via erosion, g/ha/a, at different times and at different Cd contents of P-fertilizers and changes (%) over 100 years.

ACCUMULATION MODULE							
CADMIUM ERODED, g/ha/a							
		Cd 2.5 mg/kg P		Cd 50 mg/kg P		Cd 138 mg/kg P	
Time elapsed, in years	0	100		100		100	
		g/ha/a	%	g/ha/a	%	g/ha/a	%
Wheat field							
Anderson	0.105	0.107	2	0.125	19	0.156	49
Christensen	**0.105**	**0.105**	**0**	**0.121**	**15**	**0.152**	**45**
McBride	0.105	0.099	-6	0.116	10	0.139	32
Romkens	0.105	0.084	-20	0.099	-6	0.127	21
Potato field							
Anderson	0.105	0.106	1	0.152	45	0.239	128
Christensen	**0.105**	**0.104**	**-1**	**0.150**	**43**	**0.235**	**124**
McBride	0.105	0.098	-7	0.143	36	0.226	115
Romkens	0.105	0.081	-23	0.123	17	0.200	90
Sugar beet field							
Anderson	0.105	0.074	-30	0.112	7	0.177	69
Christensen	**0.105**	**0.073**	**-30**	**0.111**	**6**	**0.175**	**67**
McBride	0.105	0.070	-33	0.107	2	0.171	63
Romkens	0.105	0.060	-43	0.097	-8	0.158	50
Average field							
Anderson	0.105	0.116	10	0.127	21	0.149	42
Christensen	**0.105**	**0.114**	**9**	**0.126**	**20**	**0.147**	**40**
McBride	0.105	0.108	3	0.119	13	0.140	33
Romkens	0.105	0.091	-13	0.101	-4	0.121	15

potato and sugar beet will increase by 12, 7, and 2%, respectively. The use of the high-Cd fertilizers will result in the increases of 34, 20, 21%, respectively.

In conclusion, the algorithms tested in this study estimated rather well the Cd contents of the crops, in general. The Finnish low-Cd P-fertilizers will keep the Cd content of wheat constant in the future. In the case of potato and sugar beet, the Cd contents will decrease to some extent. Instead, medium-Cd fertilizers will increase the Cd content of wheat by 12%, potato by 7% and sugar beet by 2%. Respectively, the high-Cd fertilizers will cause increases of 34, 20 and 21%.

3.5.2.6 Cadmium uptake by plants

At the initial time (0), Eriksson´s algorithm underestimated Cd uptake by wheat (Table 21) as

Table 20. Cadmium contents in crops, µg/kg d.m., at different times and at different Cd contents of P-fertilizers and changes (%) over 100 years.

ACCUMULATION MODULE							
CADMIUM ACCUMULATION IN CROPS, µg/kg d.m.							
		Cd 2.5 mg/kg P		Cd 50 mg/kg P		Cd 138 mg/kg P	
Time elapsed, in years	0	100		100		100	
	µg/kg d.m.	µg/kg d.m	%	µg/kg d.m	%	µg/kg d.m	%
Wheat grains							
Anderson	51.9	52.8	2	59.1	14	70.9	37
Christensen	**51.9**	**51.9**	**0**	**58.0**	**12**	**69.3**	**34**
McBride	51.9	49.8	-4	55.9	8	64.3	24
Romkens	51.9	44.0	-15	49.6	4	60.0	16
Potato tubers							
Anderson	52.0	52.1	0,2	55.8	7	62.5	20
Christensen	**52.0**	**51.9**	**-0,2**	**55.5**	**7**	**62.2**	**20**
McBride	52.0	51.4	-1	55.0	6	61.5	18
Romkens	52.0	50.2	-3	53.4	3	59.4	14
Sugar beet roots							
Anderson	200.0	181.2	-9	204.0	2	242.9	21
Christensen	**200.0**	**180.6**	**-10**	**203.3**	**2**	**242.1**	**21**
McBride	200.0	178.8	-11	201.3	1	239.6	20
Romkens	200.0	173.2	-13	195.2	-2	231.8	16

compared to the real value, 0.195 g/ha/a, while Eriksson´s algorithm overestimated the uptake by potato to some extent as compared to the value, 0.222 g/ha/a, measured in practice. Instead, Sippola´s algorithm gave for the sugar beet a Cd uptake which was precisely the same as calculated according to the real Cd concentration and the real yield of sugar beet roots. According to the scenarios the cadmium uptake by plants will directly increase in the same proportion as the Cd contents of the crops will increase. Cadmium outflows via crops will vary by wheat between 0.168-0.225 g/ha/a, by potato 0.254-0.303 g/ha/a and by sugar beet 1.467-1.776 g/ha/a depending on the plant Cd contents. Cadmium output via crops from the soil can be remarkable, because cadmium outflow via the crops can be greater than Cd outflow via leaching and erosion together.

In conclusion, cadmium uptake by the crops will directly increase in the same proportion as the Cd contents of the crops will increase. Cadmium output via crops from the soil can be remarkable, because cadmium outflow via the crops can be greater than Cd outflow via leaching and erosion together.

Table 21. Cadmium uptake rates by crops, g/ha/a, at different times and at different Cd contents of P-fertilizers and changes (%) over 100 years.

ACCUMULATION MODULE							
CADMIUM UPTAKE BY CROPS, g/ha/a							
		Cd 2.5 mg/kg P		Cd 50 mg/kg P		Cd 138 mg/kg P	
Time elapsed, in years	0	100		100		100	
		g/ha/a	%	g/ha/a	%	g/ha/a	%
Wheat grains							
Anderson	0.168	0.171	2	0.192	14	0.230	37
Christensen	**0.168**	**0.168**	**0**	**0.188**	**12**	**0.225**	**34**
McBride	0.168	0.162	-4	0.181	8	0.208	24
Romkens	0.168	0.143	-15	0.161	-4	0.195	16
Potato tubers							
Anderson	0.254	0.254	0	0.272	7	0.305	20
Christensen	**0.254**	**0.253**	**-0,4**	**0.271**	**7**	**0.303**	**19**
McBride	0.254	0.251	-1	0.268	6	0.300	18
Romkens	0.254	0.245	-4	0.260	2	0.290	14
Sugar beet roots							
Anderson	1.467	1.329	-9	1.497	2	1.782	21
Christensen	**1.467**	**1.325**	**-10**	**1.492**	**2**	**1.776**	**21**
McBride	1.467	1.312	-11	1.477	1	1.758	20
Romkens	1.467	1.271	-13	1.432	-2	1.701	16

3.5.3 Conclusions on accumulation module

Dynamic model and scenario estimations in the cadmium accumulation module were carried out according to the methodology recommended by ERM. The algorithm of Christensen for determining the distribution coefficient (K_D) of cadmium in soil was considered to be the most suitable for the Finnish circumstances. Results presented here are based on that algorithm and on the 100-year time scale. Results from these dynamic scenario estimations were in a good accordance with the respective results from the static mass balance calculations.

By using the P-fertilizers with current Finnish cadmium content of 2.5 mg/kg P, cadmium concentrations in soil, soil water and crop will remain unchanged in the wheat cultivation in which cadmium inputs and outputs are balanced at present and will be in the future, too. In the potato cultivation, the cadmium concentrations will even decrease a little, but in the sugar beet cultivation, they will decrease remarkably. It means that perhaps potato, but most likely sugar beet fields will be purified gradually.

A clear increase in the concentrations of soil, soil water and crop will occur in all the cultivation systems studied, if the cadmium content of P-fertilizers will meet the Finnish cadmium limit value, 50 mg/kg P. The increase in crops will be the highest in wheat grains.

Summary table 22. Changes (%) over 100 years in cadmium concentrations of soil, soil water and crops at different cadmium contents of P-fertilizers and soil cadmium concentrations at steady state, respectively.

ACCUMULATION MODULE				
CHANGES IN %				
		Cd 2.5 mg/kg P	**Cd 50 mg/kg P**	**Cd 138 mg/kg P**
Time elapsed	**Fertilization**			
years		**100**	**100**	**100**
Wheat field	P 15 kg/ha/a			
Soil Cd		0	16	45
Soil water Cd		0	16	45
Crop Cd		0	12	34
Soil Cd at SS	mg/kg d.m.	0.210	0.611	1.354
Potato field	P 40 kg/ha/a			
Soil Cd		-1	43	125
Soil water Cd		-2	42	124
Crop Cd		-0.2	7	20
Soil Cd at SS	mg/kg d.m.	0.183	1.054	2.662
Sugar beet field	P 30 kg/ha/a			
Soil Cd		-31	5	68
Soil water Cd		-31	5	67
Crop Cd		-10	2	21
Soil Cd at SS	mg/kg d.m.	0.053	0.238	0.581
Average field ha	P 10 kg/ha/a + manure			
Soil Cd		9	20	40
Soil water Cd		8	20	40
Crop Cd				
Soil Cd at SS	mg/kg d.m.	0.433	0.724	1.264

If the P-fertilizers will contain cadmium 138 mg/kg P which is a European mean, the cadmium concentration in soil, soil water and crops will radically increase in all the cultivation systems. The increase will be the highest in the wheat crops and potato fields. Scenario estimations indicated that wheat and potato cultivation will be exceptionally sensitive to cadmium inputs. Extra-pure P-fertilizers will be needed to prevent contamination of wheat crops and potato fields.

The outfluxes of Cd via leaching and erosion were directly related to the accumulation of Cd in the soil and the offtakes of Cd by plants were directly related to the accumulation of cadmium in the crops. Cadmium output via crops from the soil can be remarkable, because it can be greater than Cd outflow via leaching and erosion together.

3.6 Predicted environmental concentrations (PEC)

3.6.1 PEC in agricultural soil

1) PEC in soil

Measured monitoring data (from the year 1987) i.e. measured mean extractable concentrations of cadmium (mg/kg) have been used in derivation of PEC in soil. In addition, 90-percentile values of the measured data as recommended in the EU Technical Guidance Document for New and Existing Substances (TGD) have also used in derivation of PEC (see Table 23). According to the TGD the 90-percentile values of the measured data are of highest preference and can be considered as a realistic worst case, whereas the average concentrations may underestimate the risk. Mobile or easily mobilizable AAAc-EDTA-extractable cadmium has been considered to represent better than total cadmium the bioavailable cadmium fraction in soil.

2) PEC in soil pore water

The most relevant exposure route for many soil organisms is through soil pore water. It is also generally agreed that uptake of cadmium by plants takes place via soil pore water. Therefore, also PEC in soil pore water (cadmium concentration μg/l) was derived by using Christensen´s algorithm with three different concentrations of cadmium in fertilizers, in four different fields and for a100 years period (Table 24, see also Table 11).

3.6.2 PEC in water

No relevant measured data are available on cadmium contents in surface waters nearby agricultural fields. The background concentrations of cadmium in Finnish lakes is generally low. According to NIVA (1999) a median concentration of cadmium in Finnish lakes located throughout Finland and selected at random is 0.01 μg/l. The 90-percentile value for cadmium in surface waters is 0.03 μg/l. Elevated cadmium concentrations are found in north of and in west of Finland. Cadmium concentration in Finnish rivers is typically between 0.01 - 0.04 μg/l (Tarvainen et al. 1997).

ERM (2000) does not give any detailed instructions on how to calculate PEC in water. PEC in water has been derived from soil pore water concentrations by using dilution factor 10 and by adding the background concentration to the local concentration, i.e. soil pore water concentration as recommended by the TGD. 90-percentile value of 0.03 μg/l has been used as a background concentration representing the realistic worst case situation (Table 24).

3.7 Effects assessment

Data on the effects on terrestrial and aquatic organisms are basically taken from reviews of cadmium made by WHO (1992), CEPA (1994) and Ros and Slooff (1988).

Table 23. PECs in soil in five different cultivation zones in Finland (AAAc-EDTA -extractable Cd (mg/kg).

Cultivation zones 1987	**I**	**II**	**III**	**IV**	**V**
Mean	0.127	0.104	0.08	0.073	0.049
90-percentile	0.17	0.15	0.12	0.09	0.07

Table 24. PECs in soil pore water and in water based on Christensen's algorithm

Cd content in fertilizers	**Cd 2.5 mg/kg P**				**Cd 50 mg/kg P**		**Cd 138 mg/kg P**	
Time elapsed in years	**0**		**100**		**100**		**100**	
Cadmium concentrations	**Soil pore water µg/l**	**Water µg/l**	**Soil pore water µg/l**	**Water µg/l**	**Soil pore water µg/l**	**Water µg/l**	**Soil pore water µg/l**	**Water µg/l**
Wheat field	0.05	0.04	0.05	0.04	0.06	0.04	0.07	0.04
Potato field	0.05	0.04	0.05	0.03	0.07	0.04	0.112	0.04
Sugar beet field	0.02	0.03	0.02	0.03	0.02	0.03	0.04	0.03
Average field ha	0.05	0.04	0.05	0.04	0.06	0.04	0.07	0.04

3.7.1 Agricultural soil

The uptake and toxicity of cadmium in terrestrial organisms is greatly influenced by a number of environmental factors effecting the bioavailability, such as soil pH, cation exchange capacity (CEC), organic matter (OM), clay content, redox potential, the presence of metals and other ions and the binding species of cadmium (Chardon, W.J. 1984, ref. in Ros and Slooff 1988). The bioavailability of Cd is higher at lower pH, lower CEC and lower OM content.

3.7.1.1 Toxicity to microorganisms

In CEPA (1994) eight studies were identified on the effects of cadmium on soil metabolic processes. A level of 2.9 mg Cd/l (cadmium compound not spesified) caused a 60% reduction in nitrification (soil pH = 6.4, clay content = 7.7 %) over 60 days (Kobus and Kurek 1990, ref. in CEPA 1994).

Data reviewed by Ros and Slooff (1988) show that soil microbial process may be inhibited: carbon transformation at ≥ 7 mg Cd/kg of soil, nitrogen transformation at ≥ 50 mg Cd/kg of soil, and the activity of sensitive enzymes at ≥ 4 mg/kg of soil. Bewley and Stotzky (1983, ref. in WHO 1992) investigated the effect of cadmium (100 and 1000 mg/kg soil) on carbon mineralization and on the mycoflora in glucose-supplemented soils amended with clays (kaolinite or montmorillonite) at 9%. Cadmium had no significant effect on the length of the lag period, carbon dioxide evolution or on the amount of carbon mineralized.

Naidu and Reddy (1988, ref. in WHO 1992) incubated cotton soil (0.8% organic carbon, 55% clay) for up to 8 weeks in the presence of $CdCl_2$ at concentrations of between 10 and 500 mg Cd/kg. The ammonical nitrogen (NH_4-N) concentration increased for the first week at all treatment levels and then decreased at the concentrations of 50 mg/kg or less. The rise in NH_4-N levels led to an increase in nitrate nitrogen (NO_3-N) levels. At all cadmium concentrations there was a significant accumulation of nitrite nitrogen (NO_2-N) in every sample time, suggesting, according to the writers, that cadmium might be toxic to soil nitrification. At all exposure levels cadmium

significantly depressed both bacterial and fungal populations. Concentrations of 10 or 50 mg Cd/kg had no effect on soil actinomycetes, but both 100 and 500 mg/kg significantly reduced the population.

Levels of cadmium in soil (cadmium chloride) of 2.0 mg Cd/kg (d.w.) and 5.0 mg/kg (d.w.) inhibited colonization of ectomycorrhizae on white pine (*Picea glauca*) roots by 62% and 87%, respectively (Dixon and Buschena 1988, ref. in CEPA 1994).

3.7.1.2 Toxicity to terrestrial invertebrates

The toxicity of cadmium to terrestrial invertebrates is summarized in Table 25.

In the study of Schmidt et al. (1991) the LOEL for exposure to cadmium ($CdCl_2$) in soil (sandy substrate) was 2 mg $CdCl_2$/kg (d.w.) (1.23 mg Cd/kg). This concentration caused a significant reduction (15%) in egg hatching of the grasshopper (*Aiolopus thalassinus*). Also, the growth rate of adults was significantly impaired (8.5 to 12.5%) over a two generation soil test at the same cadmium concentration.

3.7.1.3 Toxicity to agricultural crops

Cadmium has been shown to have an adverse effect on plant growth and yield in laboratory experiments. However, plants grown in soil are generally insensitive to the effects of cadmium except at high doses. Effects are only seen when cadmium is given in nutrient solutions rather than in soil, where the cadmium is bound and is therefore less available to the plants. Cadmium is only available to plants in solution in soil (WHO 1994).

Toxicity studies on plants are related mainly to effects on growth and yield and on some physiological parameters. The effects largely depend on plant species and soil properties; 25% yield decrement is observed at 2-250 mg Cd/kg soil and at 15-160 mg Cd/kg plant tissue. Toxic symptoms in potatoes are observed at 5 mg Cd/kg in a sandy clay soil, not in sandy and clay soils (Ros and Slooff 1988).

Table 25. Toxicity of cadmium to terrestrial invertebrates.

Organism	Toxic effect	Reference
Grasshopper (*Aiolopus thalassinus*)	LOEL 1.23 mg/kg (15 % reduction in egg hatching)	Schmidt et al. 1991
Nematode (*Aphelenchus avenae*)	22-d NOEC 1 mg/kg feed (exposed via fungi)	Doelman et al. 1984 (ref. in WHO 1992)
Collembola (*Orchesella cincta*)	9-week LC_{50} 179.8 mg/kg NOEC 4.7 mg/kg	Van Straalen et al. 1989 (ref. in WHO 1992)
Collembola (*Platynothrus peltifer*)	9-week LC_{50} 817.2 mg/kg NOEC 2.9 mg/kg	Van Straalen et al. 1989 (ref. in WHO 1992)
Snail (subadult) (*Helix aspersa*)	30-d NOEC 10 mg/kg diet	Russell et al. 1981
Earthworm	NOEC 10 mg/kg d.w. soil	Ros & Slooff 1988
Sringtail	NOEC 4.5 mg/kg feed*	Ros & Slooff 1988
Woodlice	NOEC 2.0 mg/kg feed*	Ros & Slooff 1988

* According to the writers, this Cd level in leafy materials may be reached at ≥ 0.5 mg Cd/kg d.w. soil.

Mahler et al. (1978, ref. in WHO 1992) treated eight soils, the pH values of which ranged from 4.8 to 7.8 with 1 % (by weight) sewage sludge containing added cadmium sulphate, leading to cadmium concentrations in the soil ranging from 0.1 to 320 mg/kg. Two plants, lettuce (*Lactuca sativa variety longifolia)* and Swiss chard (*Beta vulgaris variety cicla*) were grown in the soils in pots. The EC_{50} (yield) for lettuce was 214 and 139 mg/soil for acid and calcareous soils, respectively. The EC_{50} values for chard were 175 and 250 mg/kg for acid and calcareous soils. The corresponding tissue concentrations of cadmium associated with these effects were 470 and 160 mg/kg for lettuce and 714 and 203 mg/kg for chard.

In CEPA 1994 data on the toxicity of cadmium to terrestrial plants were identified for 17 species from six families. The cadmium levels in soil that elicited adverse effects on these species ranged from 4.0 to 171 mg Cd/kg (d.w.). Spinach was identified as the most sensitive species; the LOEC of 4.0 mg Cd/kg ($CdSO_4$) caused a 30% reduction in the yield during a 90-day study (soil pH 6.6, clay content 69%)(Coppola et al. 1988, ref. in CEPA 1994). Even lower values were obtained in the study reviewed by Krajnc et al. (1987), where EC_{10} values for growth inhibition of spinach of 0.9 - 1.5 mg Cd/kg sand, 0.8 - 1.8 mg Cd/kg sandy clay and 2.4 - 3.5 mg Cd/kg clay could be derived. In this study Cd was applied to soil as $Cd(NO_3)_2$.

The yield dependent-toxicity-limit for Cd content in sensitive crops is about 1-5 mg/kg (Ros and Slooff 1988).

3.7.1.4 Toxicity to birds

In WHO (1992) data were reviewed on the subacute toxicity of cadmium salts to four species on birds (summarized in Table 26). Dosing for 5 days, followed by 3 days of clean diet, resulted in LC_{50} values ranging from 767 to > 5000 mg/kg diet.

Altered kidney morphology or function are considered to be the most widely accepted endpoints of toxicity in both wild birds and mammals. According to CEPA 1994 a renal concentration of 100 mg of Cd/kg (f.w.) is the best estimate of treshold toxicity in wild birds. Wood ducks (*Aix sponsa*) fed cadmium in their diet for 3 months showed widespread renal pathological changes at an average renal concentration of 132 mg of Cd/kg, but not at 62 mg/kg (Mayack et al. 1981, ref. in CEPA 1994).

Captive mallard ducks exposed to cadmium in their diet exhibited moderate to severe tubular degeneration over a renal cadmium concentration range of 88 to 134 mg of Cd/kg (White et al. 1978, ref. in CEPA 1994).

Nicholson and Osborn (1983, ref. in CEPA 1994) detected necrosis of renal proximal tubule cells in free-living seabirds from Britain and experimentally in starlings (*Sturnus vulgaris*) at kidney concentrations of 10 to 70 mg Cd/kg (f.w.). However, Elliott et al. (1992, ref. in CEPA 1994) examined several species of seabirds collected on the Atlantic Coast of Canada and found no renal lesions in birds up to 83 mg Cd/kg (f.w.) in the kidney.

3.7.1.5 PNEC for terrestrial species

PNEC-value based on 5th percentile of a log-logistic distribution of NOEC -values as recommended by ERM (2000) has not used in the PNEC -derivation as the statistical method is not yet validated in EU but is still under debate. Furthermore, the principles and criteria of choosing the studies in the Belgian draft Cd risk assessment, to which ERM (2000) is referring, are not clear enough.

Therefore, in this study PNEC was derived by applying the assessment factor method. PNEC for terrestrial species was derived from a study on grasshoppers, which gave the second lowest NOEC -value in terrestrial compartment. According to the Technical Guidance Document a LOEC can be used to derive a NOEC with the following procedure: if the effect percentage of the LOEC

Table 26. The toxicity of cadmium salts to birds.

Species	Age (days)	Salt	Duration	LC_{50} (mg Cd/ kg diet)	Reference
Japanese quail (*Coturnix coturnix japonica*)	14 14	cadmium chloride cadmium succinate	5 + 3 days 5 + 3 days	2440 2052	Hill & Camardese 1986
Pheasant (*Phasianus colchicus*)	10 14	cadmium chloride cadmium succinate	5 + 3 days 5 + 3 days	767 1411	Hill et al. 1975
Bobwhite quail (*Colinus virginianus*)	14	cadmium succinate	5 + 3 days	1728	Hill et al. 1975
Mallard duck (*Anas platyrhynchos*)	10 10	cadmium chloride cadmium succinate	5 + 3 days 5 + 3 days	> 5000 > 5000	Hill et al. 1975
Leghorn chicken	2 week	cadmium chloride	20 days	565	Pritzl et al. 1974
Mallard duck (*Anas platyrhynchos*)	1 adult	cadmium chloride cadmium chloride	12 weeks ?	NOEC 10 NOEC 20	Cain et al. 1983 White et al. 1978

is >10 and <20%, NOEC can be calculated as LOEC/2. So, NOEC for *Aiolopus thalassinus* would be 1.2/2 mg/kg = 0.6 mg/kg. Applying an assessment factor of 10 gives a PNEC of 0.06 mg/kg. The factor 10 was chosen because long-term data are available for more than three trophic levels.

Also according to the TGD an EC_{10} for a long-term test which is obtained by extrapolation using appropriate statistics can be considered as a NOEC. Hence, the EC_{10} of 0.8 mg/kg for growth inhibition of spinach could be considered as NOEC. Althoug the NOEC for spinach is lower than the NOEC for grasshopper, the NOEC for grasshopper was selected for deriving the PNEC for the terrestrial compartment because grasshoppers are considered to represent better soil species exposed in agricultural land. In addition, plants grown in soil are generally considered insensitive to the effects of cadmium and spinach was not considered to be representative species of the plants grown in agricultural soil.

$PNEC_{soil}$ = 0.06 mg/kg

Although ERM (2000) does not give much instructions for calculation of PNEC representing a bioavailable form, an attempt was made to calculate PNEC in soil porewater. The PNEC of 0.06 mg/kg which is the total cadmium concentration in the soil was transformed to cadmium concentration in soil pore water by using the adsorption algorithm of Christensen. However, there were very little information available about the characteristics of the soil used in the study. The study report only stated that the soil used was standardized sandy substrate. Therefore the default values for pH and organic matter content (%) were assumed to be 7 and 3, respectively. In terrestrial ecotoxicological studies the pH of the soil is recommended to be adjusted close to neutral (pH 6.5 -7.5).

$PNEC_{soil\ pore\ water}$ = 0.035 μg/l

This value, however is subjected to considerable uncertainty because the calculation of PNEC in soil pore is very much dependent on pH-values used in the algorithm. The lower the pH-value was the higher the PNEC in soil pore water was obtained. Organic matter content did not influence that much.

In Ros and Slooff (1988) two different theoretical procedures were used to calculate “safe levels” for the terrestrial environment. Application method of Kooijman (1985) led to a level of

0.005 μg Cd/kg. Application of the method of van Straalen led to a "safe level" of 0.23 mg Cd/kg. The third method of van Straalen and Denneman (1989) took soil organic matter and clay content into account and a concentration protecting 95% of soil invertebrates was estimated as 0.16 mg Cd/kg for a standard soil.

3.7.2 Aquatic compartment

The literature relating to the toxicity of cadmium in the aquatic environment is very extensive. In the evaluation of the tests most of the results presented here have been taken from the detailed reviews without assessing the original papers. However, only those studies in which proper control was used and abiotic parameters were reported (e.g. pH, hardness) were considered in this assessment report. Regardless of the original cadmium compound used in laboratory exposures, the free ion is considered to be the most bioavailable and toxic chemical species (Rainbow 1990, ref. in CEPA 1994).

Cadmium uptake from water by aquatic organisms is extremely variable and depends on the species and various environmental conditions as water hardness, salinity, temperature, pH and an organic matter content.

In general, cadmium is more toxic to aquatic species at low pH (5 to 7), low salinity (< 15 ‰) and in soft water (<100 mg/l calcium carbonate) (Wong 1987, Sprague 1987, Langston 1990, Voyer and McGovern 1991, all ref. in CEPA 1994). However, some species have shown no pronounced differences in toxicity over various ranges in the above-noted physical/chemical parameters (Canton and Slooff 1982, ref. in CEPA 1994).

Cadmium forms complexes with natural humic substances. Complexation of metal ions usually reduces their effective concentration in water and, in many cases, also their biological availability and toxicity to animals. In the case of cadmium, however, there are studies that show that humic acid in test water increases the acute toxicity of cadmium (Winner 1984, Virtanen et al. 1989). The acute toxicity (48-h LC_{50}) of Cd to neonatal *Daphnia magna* was in humic-free control water (pH 6.5, hardness 0.5 mmol/l) 99 μg/l compared to the LC_{50} of 12 μg/l in natural humic water (pH 6.5, hardness 0.1 mmol/l) (Virtanen et al. 1989). According to Penttinen et al. (1995) enhanced toxicity of cadmium was due to uptake rates two times faster than those of humic-free reference water. Since the depuration rates were equal, an acutely lethal body burden was reached faster in the humic than in the reference water. The humic water, however, was softer than the reference water and in the study of Penttinen et al., the effect of water hardness on accumulation of Cd was more significant than the effect of dissolved organic carbon (DOC). Nevertheless, the effect of water hardness is not the only explanation for the altered toxicity, since the interaction between water hardness and DOC was also very significant.

3.7.2.1 Acute toxicity to fish

In CEPA (1994) data were reviewed on the acute and chronic toxicity of cadmium compounds to 22 species on freshwater fish, representing eight families. In WHO (1992) data were reviewed on the acute toxicity of cadmium compounds to 11 species on freshwater fish. The acute toxicity of cadmium to fish is summarized in Table 27. The salmonids appeared to be the most sensitive group as a whole with a lowest 96-hour LC_{50} of 0.7 μg/l (*Salmo gairdneri*). The 96-h LC_{50} of the other species appears to be in the range of 0.001-11.1 mg/l.

3.7.2.2 Chronic toxicity to fish

In the long-term toxicity tests the salmonids appeared to be the most sensitive group (as in acute tests, too) with the lowest LOECs of 0.2 and 0.47 μg/l. The summary of the results of the long-term toxicity tests is shown in Table 28.

Table 27. Toxicity of cadmium to freshwater fish.

Organism	Size/ age	Static/ flow	Temp. (°C)	Hardness[a] (mg/l)	pH	Duration (h)	LC_{50}[b] (mg/l)	Reference
Chinook salmon (*Onchorhynchus tshawytscha*)	juvenile	flow	11-13	20-22	7.0-7.3	96	0.001 m	Finlayson & Verrue 1982
Rainbow trout (*Salmo gairdneri*)	1-6 g	flow	14.5-17.5	9	4.7	96	0.028 m	Cusimano et al. 1986
	1-6 g	flow	14.5-17.5	9	5.7	96	0.0007 m	Cusimano et al. 1986
	1-6 g	flow	14.5-17.5	9	7.0	96	<0.0005[c] m	Cusimano et al. 1986
	5-15 g	stat	8.5-10.7	61-65	7.4	48	2.9 m	Pascoe et al. 1986
	5-15 g	stat	8.5-10.7	61-65	7.4	96	1.3 m	Pascoe et al. 1986
Fathead minnow (*Pimephales promelas*)	adult	stat	25	20	7.5	48	1.09 n	Pickering & Hendersson 1966
			25	20	7.5	96	1.05 n	Pickering & Hendersson 1966
			18-22	190-210	7.7	48	0.1 n	Hall et al. 1986
			18-22	190-210	7.7	96	0.09 n	Hall et al. 1986
Bluegill sunfish (*Lepomis macrochirus*)	adult	stat	25	20	7.5	48	2.76 n	Pickering & Hendersson 1966
						96	1.94 n	Pickering & Hendersson 1966
Goldfish (*Carassius auratus*)	adult	stat	25	20	7.5	48	2.62 n	Pickering & Hendersson 1966
						96	2.34 n	Pickering & Hendersson 1966
Guppy (*Poecilia reticulata*)	adult	stat	25	20	7.5	96	1.27 n	Pickering & Hendersson 1966
	3-4 weeks		23-25	100			3.8 m	Canton & Slooff 1982
							(NOEC 0.6)	Canton & Slooff 1982
	3-4 weeks		23-25	200			11.1 m	
							(NOEC 5.2)	
Green sunfish (*Lepomis cyanellus*)	adult	stat	25	20	7.5	96	2.84 n	Pickering & Henderson 1966
Puntius arulius	2.4 g	semistat	23-27	60-70	7.5	96	39 n	Shivaraj & Patil 1988
Zebra fish (*Brachydanio rerio*)	6 months	flow	19-21	170		48	4.2 m	Canton & Slooff 1982
							(NOEC 2.0)	
Killifish (*Oryzias latipes*)	4-5 weeks	semistat	23-25	100		96	0.35 m	Canton & Slooff 1982
							(NOEC 0.006)	
				200			0.13 m	
							(NOEC 0.03)	

[a] Hardness expressed as mg $CaCO_3$/l unless stated otherwise,[b] Fish exposed to cadmium as chloride unless stated otherwise; m=measured & n=nominal concentration,[c] The LC_{50} at pH 7.0 could not be differentiated from the backround Cd levels because all but the lowest exposure concentration of 0.4 μg/l produced total mortality

3.7.2.3 Toxicity to invertebrates

Cadmium is moderately to highly toxic to aquatic invertebrates. Cadmium is lethal to freshwater invertebrates (*Daphnia* sp.) following static short-term exposures (48 hours) in hard water to cadmium chloride concentrations as low as 3.6 μg Cd/l (Baird et al. 1991, ref. in CEPA 1994). The 48-h LC_{50} of the other species varies in the range of 0.02 - 4.25 mg/l (see Table 29).

When Schuytema et al. (1984, ref. in WHO 1992) exposed *Daphnia magna* to cadmium, the mean 48-h LC_{50} value was 39 μg/l in water and 91 μg/l in a watersediment slurry.

3.7.2.4 Chronic toxicity to invertebrates

In CEPA 1994, data on lethality, and the effects of cadmium compounds on reproduction and growth were examined for 28 species of freshwater invertebrates representing six broad taxonomic groups. Crustaceans were the most Cd-sensitive group of invertebrates, while aquatic insect larvae were the most tolerant. The summary of the results of the chronic toxicity studies is shown in Table 30.

The laboratory-derived chronic threshold is strongly supported by field work in experimentally-contaminated lakes in northwestern Ontario where the abundance of two Cladoceran species (*Daphnia galeata mendotae* and *Holopedium gipperum*) was reduced by 39% and 28%, respectively, following a 14-day exposure to 0.2 μg Cd/l ($CdCl_2$) using *in situ* flow-through containers. In a whole lake contamination experiment, exposure to cadmium chloride concentrations at and below 0.08 μg Cd/l had no observed effects on the composition or abundance of most zooplankton species (Malley and Chang 1991, ref. in CEPA).

3.7.2.5 Toxicity to aquatic microorganisms

Canton & Slooff (1982) exposed the bacterium *Salmonella typhimurium* to the cadmium in the form of the chloride, and calculated an 8-h EC_{50} (growth inhibition) of 10.4 mg/l and NOEC of

Table 28. Summary of the results of the long-term toxicity tests with Cd^{2+}.

Test organism	Exposure time	Endpoint	Hardness (mg /l)	LC_{50} (μg/l)	NOEC (LOEC) (μg/l)	References
Pimephales promelas	11 m	Hatching	200		37	Pickering & Gast 1972
Jordanella floridae	100 d 30 d	Reproduction Fry survival	44 45		4.1 3	Spehar 1976 McKim 1977
Brachydanio rerio	36 d	Reproduction	100		1	Bresch 1982
Salvelinus fontinalis	3 gen.	Fry survival, growth	44		1.7	Benoit et al 1976
Salmo salar	46 d	Growth of alevins (12 % reduction)	soft		(0.47)	Rombough & Garside 1982
Salmo gairdneri	18 m	Fry survival (effect procent ?)	102		(0.2)	Birge et al. 1981
Oryzias latipes	18 d	Mortality Mortality + abnormal behavior	2 1 2 1	50 40 20 30	30 6 6 3	Canton & Slooff 1982

Table 29. Acute toxicity of cadmium to freshwater invertebrates.

Organism	Size/ age	Stat./ flow	Temp. (°C)	Hardness[a] (mg/l)	pH	Duration (h)	LC_{50}[b] (mg/l)	Reference
Snail	adult	stat	20-22		6.7	48	4.25 n	Wier & Walter 1976
(*Physa gyrina*)	adult	stat	20-22		6.7	96	1.37 n	Wier & Walter 1976
	immature	stat	20-22		6.7	48	0.69 n	Wier & Walter 1976
	immature	stat	20-22		6.7	96	0.41 n	Wier & Walter 1976
Snail	10-12 mm	flow	12	128-176	7.7	48	2.1 m	Williams et al. 1985
(*Physa fontinalis*)	10-12 mm	flow	12	128-176	7.7	96	0.8 m	Williams et al. 1985
Isopod	8-10 mm	flow	12	128-176	7.7	48	3.6 m	Williams et al. 1985
(*Asellus aquaticus*)	8-10 mm	flow	12	128-176	7.7	96	0.6 m	Williams et al. 1985
Scud	8-12 mm	flow	12	128-176	7.7	48	0.4 m	Williams et al. 1985
(*Gammarus pulex*)	8-12 mm	flow	12	128-176	7.7	96	0.02 m	Williams et al. 1985
Water flea (*Daphnia hyalina*)	adult	stat	10	0.85 meq/l	7.2	48	0.055[c] n	Baudouin & Scoppa 1974
Water flea	< 1 day	stat	18-22	51	7.5	48	0.0099 m	Chapman et al. 1980
(*Daphnia magna*)	< 1 day	stat	20-22	110-130	7.8	48	0.04 n	Hall et al. 1986
	< 1 day	stat	20-22	190-210	7.7	48	0.08 n	Hall et al. 1986
Water flea	< 1 day	stat	20-22	110-130	7.8	48	0.07 n	Hall et al. 1986
(*Daphnia pulex*)	< 1 day	stat	20-22	110-130	7.7	48	0.1 n	Hall et al. 1986
	< 1 day	stat	19-22		7.7	96	0.047 n	Bertram & Hart 1979
Copepod (*Cyclops abyssorum*)	adult	stat	10	0.85 meq/l	7.2	48	3.8[c] n	Baudouin & Scoppa 1974
Copepod (*Eudiaptomus padanus*)	adult	stat	10	0.85 meq/l	7.2	48	0.55[c] n	Baudouin & Scoppa 1974
Crayfish (*Orconectes virilis*)		flow	19-21	24-28	6.7-7.0	96	6.1 m	Mirenda 1986
Mayfly (*Ephemerella grandis grandis*)		flow	10		7.8	96	28 m	Clubb et al. 1975
Midge (*Chironomus riparius*)	10-12 mm	flow	12	128-176	7.7	96	300 m	Williams et al. 1985
Stonefly (*Pteronarcella badia*)		flow	10		7.8	96	18 m	Clubb et al. 1975
Stonefly (*hydropsyche angustipennis*)	10-15 mm	flow	12	128-176	7.7	96	520 m	Williams et al. 1985

[a] hardness expressed as mg $CaCO_3$/l unless stated otherwise, [b] organisms exposed to cadmium added as cadmium chloride unless otherwise stated, [c] organisms exposed to cadmium as cadmium sulphate

0.65 mg/l (hardness of 0.5 mmol/l). Chapman & Dunlop (1981, ref. in WHO 1992) estimated the 8-h LC_{50} for the freshwater protozoan *Tetrahymena pyriformis* to be less than 1 mg/l. However, this value increased with increasing water calcium concentration; at a value of 500 mg calcium/l, the LC_{50} was 19 mg/l.

3.7.2.6 Toxicity to algae

In freshwater species (Table 31) toxic effect were demonstrated mainly below 1 mg Cd/l. NOEC levels were in the range of 0.7 - 3.1 mg/l. Some species showed reduced growth at relatively low Cd concentrations, the diatom *Asterionella formosa* at 0.002 mg/l and the green alga *Scenedesmus quadricauda* at 0.006 mg/l.

The diatom algae, *Tabellaria flocculosa*, is the most sensitive freshwater algae tested; morphological changes in this species following a 14-day static renewal exposure to cadmium ($CdNO_3$) was reported at 1 µg Cd/l (=LOEC) (Adshead-Simonsen et al 1981).

3.7.2.7 Toxicity to aquatic plants

Hutchinson & Czyrska (1975, ref. in WHO 1992) exposed a common duckweed *Lemna minor*, to cadmium concentrations of between 0.01 and 1.0 mg/l for up to 3 weeks. Growth was reduced at all concentrations but especially at 0.05 mg/l or more. At concentrations 0.5 and 1.0 mg/l *Lemna* plants died. Loss of green coloration (chlorosis) was a common symptom of cadmium toxicity.

In a study by Nir and al. (1990, ref. in WHO 1992), water hyacinth plants were exposed to cadmium concentrations between 0.05 to 1.0 mg/l for 7 days. Concentrations of 0.1 mg/l or less had no significant effect on wet or dry biomass gain or on chlorophyll *a* content. Concentrations of 0.4 or 1.0 mg/l significantly reduced both biomass gain and chlorophyll *a* content but had no significant effect on dry biomass gain. After 3 weeks of exposure, the chlorophyll *a* levels in plants exposed to 0.4 mg/l were 75% lower than in control plants.

3.7.2.8 PNEC for the aquatic species

PNEC-value based on the 5th percentile of a log-logistic distribution of NOEC-values and PNEC-value calculated as a function of water hardness as recommended by ERM (2000) are not used in this report because the statistical method is not yet validated in EU but is still under debate and the principles of choosing the studies into the extrapolation are not enough clear. Besides, the soft waters of Finland are not sufficiently taken into account in the PNEC-value calculated as a function of water hardness. Therefore, in this study PNEC was derived by applying the assessment factor method.

Long term NOEC data are reported for fish, Daphnia and algae. For Daphnia two 20 days NOECs of 0.37 µg/l and 0.5 µg/l based upon mortality and reproduction, respectively, is reported. The NOEC value of 0.37 µg/l is the extrapolated value; effects could still be observed in the lowest test concentrations, the EC_{20} value was considered as NOEC. According to the TGD an EC_{10} for a long-term test can be considered as a NOEC. For Daphnia a 21-day LOEC of 0.17 µg/l based upon reproductive output is reported for *Daphnia magna*. According to the Technical Quidance Document a LOEC can be used to derive a NOEC with the following procedure: if the effect percentage of the LOEC is >10 and <20%, NOEC can be calculated as LOEC/2. According to this, the NOEC for the Daphnia would be 0.17/2 µg/l = 0.085 µg/l. For fish a 46-day LOEC of 0.47 µg/l based upon growth of *Salmo salar* alevins is reported. According to the same calculation method as with Daphnia, the NOEC for the fish would be 0.47/2 µg/l = 0.24 µg/l.

The PNEC for the aquatic compartment is extrapolated from the lowest NOEC of 0.085 µg/l for *Daphnia magna* using an extrapolation factor of 10. This factor is chosen because chronic data are available for three trophic levels and, additionally, these NOECs seem to cover the most sensitive taxonomic groups. The extrapolation leads to a PNEC for the aquatic environment of 0.0085 µg/l.

Table 30. The results of the chronic toxicity of cadmium to aquatic invertebrates.

Test organism	Exp. time (days)	Endpoint	Hardness (mg/l)	LC_{50} (μg/-l)	NOEC/ LOEC (μg/l)	Reference
Daphnia magna	14	Mortality		24	NOEC 3.2	van Leeuwen et al. 1985
	21	Mortality		14	NOEC 3.2	van Leeuwen et al. 1985
	20	Mortality	200	0.67	NOEC 0.37[a]	Canton & Slooff 1982
	20	Reproduction	200	4.2	NOEC 0.5	Canton & Slooff 1982
	21	Reproduction	48.5		LOEC 0.17	Biesinger & Christensen 1972
	21	Reproduction			NOEC 0.6	Kuhn et al. 1989
	21	Reproduction?	53		LOEC 0.29	Chapman et al. 1980
	21	Reproduction?	103		NOEC 0.16	Chapman et al. 1980
	21	Reproduction?	209		NOEC 0.21	Chapman et al. 1980
Hyalella azteca	6 weeks	Mortality			LOEC 1	Borgmann et al. 1989
Cammarus fasciatus	6 weeks	Mortality			LOEC 3.2	Borgmann et al. 1989

[a] Extrapolated value; as still effects could be observed in the lowest test concentration, the EC_{20} value was considered as the NOEC.

Table 31. Effects of cadmium on algae.

Species	Toxic conc. (mg/l)	Effect	Referencies
Chlorella pyrenoidosa	3.1	NOEC	Slooff et al. 1983
	0.1	Inhibit. primary prod.	Burnison et al. 1975
Chlorella vulgaris	3.7	EC_{50}(96h)	Canton & Slooff 1982
	1.5	NOEC	
Scenedesmus pannocinus	0.9	NOEC	Slooff et al. 1983
Selenastrum capricornutum	0.7	NOEC	Slooff et al. 1983
Tabellaria flocculosa	0.0001	LOEC	Adshead-Simonsen et al. 1981
Asterionella formosa	0.002	Growth inhibition	Conway & Williams 1979

$PNEC_{aquatic}$ = 0.0085 μg/l

For cadmium short term L(E)C_{50}s are reported for fish, invertebrates, algae and microorganisms. All data are from validated sources. The lowest acute LC_{50} reported is for fish (*Salmo gairdneri*) with a 96 -h LC_{50} of 0.7 μg/l.

In Table 32 a summary is given of the lowest LC_{50} and NOEC values taken into a account in deriving the PNEC values. Because the toxicity of cadmium is clearly affected by water hardness, the PNECs have been derived for both soft water ($\leq$50 mg $CaCO_3$/l, typical for Finnish waters) and hard water > 50 mg $CaCO_3$/l. In Table 32 also the calculated "safe" levels presented in Ros and Slooff 1988 are included.

3.8 Risk characterization

The risk for the environment caused by the chemical is assessed by comparing the environmental exposure (expressed as PEC) with the threshold

Table 32. PNEC values for Cd for aquatic species based on the lowest LC_{50} and NOEC-values.

	Lowest LC_{50} or NOEC (μg/l)		Method
	≤ 50 mg $CaCO_3$/l	> 50 mg $CaCO_3$/l	
lowest LC_{50} PNEC (applying AS[a] = 100)	0.7 **0.007**	90 **0.9**	Technical Guidance Document 1996
lowest NOEC PNEC (applying AS = 10)	0.085 **0.0085**	0.5 **0.05**	Technical Guidance Document 1996
lowest EC_{50} NOEC (ecosystems) applying UF[b]	2 0.49 **0.0058**	20 3.19 **0.037**	Slooff et al. 1986 (ref. in Ros & Slooff 1988)
lowest NOEC NOEC (ecosystems) applying UF	0.17 0.946 **0.028**	0.15 0.85 **0.025**	Slooff et al. 1986 (ref. in Ros & Slooff 1988)
lowest calculated LC_{50} "safe" NOEC	**0.00053** **0.00002**	**0.018** **0.0000003**	Kooijman 1985 (ref. in Ros & Slooff 1988)

AS = assessment factor
UF = uncertainty factor

concentration for harmful effect in the environment (expressed as predicted no-effect concentration, PNEC). Thus, an estimate of the environmental risk is the ratio PEC/PNEC. If the PEC/PNEC of a chemical is greater than 1, there is a risk for adverse effects in the environmental compartment concerned.

3.8.1 Agricultural soil

When comparing the measured levels of cadmium in Finnish cultivation zones as presented in table 23 with a PNEC of 0.06 mg/kg, the PEC/PNEC-ratios below are obtained (Table 33).

In the EU Technical Guidance Document (1996) it is stated that it is very important that both PEC and PNEC are based on similar levels of availability. It is also recommended that for the metals the PEC/PNEC ratio should be calculated with the concentrations of the relevant bioavailable metal species. As the study on which the PNEC was based, was carried out in a sandy substrate with easily soluble Cd-salts. Hence, the total concentration of cadmium in the study can be assumed to be totally as a bioavailable form. In this case the bioavailable PEC (= extractable cadmium concentration) was divided with total PNEC, because if the total PEC was used, the risk would be overestimated.

Another approach is to compare PEC and PNEC based on concentrations in soil pore water. Therefore, an attempt was made to calculate PEC/PNEC-ratio in soil pore water (Table 34). However, it must be kept in mind that the calculated PNEC in pore water (0.035 μg/l) was highly dependent on pH-value. The default pH value of 7 was chosen in calculations, but when choosing a lower pH-value there would be no risk. On the other hand, the PEC in soil pore water is propably higher today than the values based on data from the year 1974 represent.

On the basis of the data reported cadmium would present a risk to the agricultural soil.

3.8.2 Aquatic compartment

The quantitative PEC/PNEC ratio has been calculated for aquatic compartment on the basis of soil pore water concentration taking into account

Table 33. PEC/PNEC in soil in five different cultivation zones in Finland.

Cultivation zones 1987	**I**	**II**	**III**	**IV**	**V**
PEC/PNEC (mean values)	2.1	1.7	1.3	1.2	0.8
PEC/PNEC (90-percentiles)	2.8	2.5	2	1.5	1.2

Table 34. PEC/PNEC in soil pore water

Cd content in fertilizers	**Cd 2.5 mg/kg P**		**Cd 50 mg/kg P**	**Cd 138 mg/kg P**
Time elapsed in years	**0**	**100**	**100**	**100**
Wheat field	1.4	1.4	1.7	2.1
Potato field	1.4	1.4	2	3.2
Sugar beet field	0.6	0.4	0.7	1.1
Average field ha	1.4	1.5	1.7	2

Table 35. PEC/PNEC in water

Cd content in fertilizers	**Cd 2.5 mg/kg P**		**Cd 50 mg/kg P**	**Cd 138 mg/kg P**
Time elapsed in years	**0**	**100**	**100**	**100**
Wheat field	4.1	4.1	4.2	4.4
Potato field	4.1	4.1	4.4	4.8
Sugar beet field	3.8	3.7	3.8	4
Average field ha	4.1	4.2	4.2	4.3

dilution and background concentration. No measured concentrations of cadmium from rivers or lakes representing cadmium concentration from agricultural areas are available (Table 35).

On the basis of the data reported cadmium would present a risk to the surface waters.

When considering the low background concentrations of Cd in Finnish lakes i.e. the 90-percentile value of 0.03 µg/l and the PNEC-value of 0.0085 µg/l obtained for aquatic species, it can be noticed that from the theoretical point of view, a "safe" level can not be established. This means that all extra Cd load to surface waters is likely to present a risk to the aquatic compartment.

3.9 Conclusions

The toxicity of cadmium to terrestrial organisms shows a variable pattern. Plants grown in soil are generally considered insensitive to the effects of cadmium, although some exceptions do exist. Cadmium exhibits moderate or low toxicity to avian species in subacute exposure. Microorganisms and terrestrial invertebrates show moderate or high sensitivity to cadmium

The predicted no-effect concentration (PNEC) for soil was derived with the assessment factor method. Statistical method for PNEC derivation was not used, as there is a considerable amount of issues under discussion within EU in relation to its use. A PNEC of 0.06 mg/kg (total Cd) for agricultural soil was derived. Also an indicative PNEC based on soil pore water concentration was established which, however, is associated with considerable uncertainty.

In risk characterization for metals in the soil the PEC and the PNEC should represent the same level of availability. Due to the uncertainties in determining the soil pore water concentrations the results based on them are not preferred. Instead, the most reliable comparison of exposure and no-effect levels in this study can be performed using total Cd-concentrations for effect studies, which usually apply soluble Cd-compounds, and extractable (AAAc-EDTA) concentrations for soil concentrations. In general, 40% of the total soil cadmium is in extractable form in Finnish soils.

Based on the available data the current Cd concentrations in Finnish agricultural soils present a risk for the soil environment. This conclusion can be reached both by using mean extractable Cd concentrations and 90 percentile values in the five Finnish cultivation zones for the year 1987. Only in the northernmost zone the PEC/PNEC ratio using the mean concentration is below 1. Risk ratios for the different cultivation zones vary from 1.2 to 2.8 (90 percentiles values).

Also the indicative risk ratios based on soil pore water concentrations demonstrate a risk for the soil environment, with the exception of sugar beet fields. For other type of fields the risk ratio is 1.4.

The increasing soil concentrations based on trend calculations, as described in the accumulation module, would lead to greater risks, accordingly. Risk ratios with the average EU Cd level in fertilizers would increase from 40 to 130 per cent. The highest risk ratio (3.2) after 100 years would be found on potato fields.

There are two remarkable differences in the soil characteristics between Finland and most other EU Member States affecting the cadmium content and solubility. They are soil pH and soil organic matter. The mean pH of Finnish cultivated soils is only 5.8, in spite of intensive liming. The other important difference is the high organic matter content of Finnish soils, due to the slow decomposition rate of organic matteras a result of the cold and humid climate. The mean organic carbon content of cultivated soils in Finland is 9%. These two factors mean that Finnish soils are capable to accumulate large amounts of cadmium in a soluble or easily solubilizable form, and thus can be generally regarded as vulnerable environments.

Cadmium is toxic or very toxic to a variety of aquatic organisms both in short-term and in long-term exposure. Invertebrates are usually considered as the most sensitive group. Cadmium is more toxic to aquatic species at low pH, low salinity, and in soft water, due to increased bioavailability. A soft water PNEC of 0.0085 μg/l was derived, also in this case with the assessment factor method.

The exposure of aquatic environment to cadmium leaching from agricultural soils was assessed based on soil solution concentrations, and by taking into account dilution and background concentrations in Finnish waters.

Risk characterization reveals that there is risk for aquatic environment for all calculated scenarios, both at present time and in the future. Risk ratios vary from 3.7 to 4.8. It is noteworthy that the aquatic PNEC value (0,0085 μg/l) is coarsely at the same level as the Finnish cadmium background values (median 0,01 μg/l). In practice this means that no margin of safety for aquatic exposure to cadmium can be established, and any increase to the natural background concentrations is likely to present a risk for the aquatic environment.

The bioavailability, and consequently toxicity, of cadmium in Finnish surface waters is increased by some specific features of the Finnish environment. Firstly, the pH value of Finnish lakes is generally low (median pH 6.6, pH < 5 in 10% of lakes). Secondly, the organic matter content is high, and thirdly, Finnish waters are very soft (mean hardness $\leq$ 10 mg CaCO3). Hence, also the Finnish aquatic environment is particularly vulnerable for adverse effects caused by cadmium.

4 HUMAN HEALTH

4.1 Introduction

In this review, the recent data on cadmium exposure and evidence of health effects of cadmium are summarized. The emphasis is on exposure of the general population, i.e. those not occupationally exposed and on the nephrotoxicity and effects on bones since these are the most relevant issues in terms of public health risks caused by soil Cd-contamination. The most relevant subpopulation is the middle aged and the elderly, due to long-term accumulation of cadmium in the human body. Evaluation of carcinogenicity of cadmium, published by International Agency for Research of Cancer (IARC) is also presented.

The overall goal of the risk assessment of human exposure to environmental pollutants is to ascertain that the exposure does not cause adverse health effects. The assessment of exposure should cover all relevant groups of the population, especially those who are exposed to high levels of chemicals ("realistic worst case"). These principles have been recognised in the Commission Regulation (EC) 1488/94 on Risk Assessment for Existing Substances and the Technical Guidance Document (TGD) supporting the regulation. According to the Technical Guidance Document 1996, upper estimates/maximum exposure and averages are both needed and "the exposure assessment should be focused on those uses for which the highest exposure is expected to occur on a regular basis".

The critical step in the risk assessment procedure of the TGD is a comparison of the estimated exposure level and/or the absorbed dose with the No Adverse Effect Level (NOAEL), which is usually obtained from the animal experiments. Since there is much data on the health effects of cadmium in human, the term "critical levels" is used in following to refer to the e.g. urinary level of cadmium, which have caused adverse effects in humans.

4.2 Human exposure to cadmium

4.2.1 General discussion

For cadmium as well as with most other environmental pollutants, there is a considerable variation in human exposure between individuals as well as between population groups. The factors that cause variation of exposure level and absorbed dose are numerous; the most important factors are listed below:

INTAKE:
Food consumption habits
Local food contamination
Smoking
Drinking water
Ambient air
Occupation

UPTAKE (absorption):
Interactions in pulmonary or intestinal absorption
Nutrient status, e.g. deficiency of iron, calcium

OTHER FACTORS:
Age
Gender

In this report the emphasis is given to dietary intake of cadmium and exposure caused by smoking. In Finland, the average exposure to cadmium via drinking water and via ambient air are 0.1 and 0.02 μg/day, respectively (Louekari et al. 1989). These are of less importance as compared with the average dietary intake (10 μg/day) and therefore exposure from drinking water and ambient air are not considered in detail in this risk assessment.

When considering the exposure to cadmium it is important to note that there are several methods available and each of the methods has got certain merits and disadvantages (Louekari 1992). Obviously, estimates of high regular exposure are of great

importance. Unfortunately, distribution or 90 percentile of the urinary cadmium or the dietary intake of cadmium, which would give an indication of realistic high exposures, are very seldom reported. Often only the range of these parameters is reported (see tables 34 and 36).

In the following, the dietary cadmium intake is discussed, since diet is the main source of cadmium exposure in general population. Smokers are exposed to cadmium in cigarette smoke, which significantly contributes to the total exposure. Uptake i.e.the absorbed and accumulated amount of cadmium in human body is measured by analysing the samples of blood and urine. Most relevant published studies on these measurements are also summarised below.

4.2.2 Dietary intake of cadmium

The dietary exposure to cadmium is measured using four different methods, namely the total diet method, the market basket method, the duplicate diets method or the faecal cadmium content method.

The average dietary intake of cadmium in the different countries is between 8.5 (Sweden) and 49 μg/day (Japan) (see Table 36). Environmental contamination and food habits cause remarkable differences among the countries where dietary intake has been studied. Elevated dietary exposure is observed in several countries near the non-ferrous metal industry, the smelters and the mines.

The high exposures (maximum of the range) in the studies presented in Table 36 are relatively close to the respective PTWI-value of WHO (60-70 μg/day for an adult). It seems that the study population (sample size) is often insufficient to reflect the intake variations and high exposure levels.

Some of the studies only reveal intra-individual (day to day) variation of cadmium intake, but it is the inter-individual and long-term variation that is relevant for risk assessment.

Only a few trend analysis are available on the dietary intake of cadmium. Ruick (1991) analysed food samples and dietary intake of cadmium in Sachsen, Germany during 1985-1989. An increasing trend was observed in both, the dietary intake increased from 12 to 19 μg/day.

The maximum values of the long-term dietary intake of cadmium are 2-3 fold when compared with the average intake (WHO 1992). Applied to Finland, where the average exposure in adults is about 10 μg/day (Kumpulainen 1991, Louekari 1988), this implies that the maximum dietary intake is 20-30 μg/day. Some studies (Morgan et al. 1988, Louekari et al. 1989, Coomes et al. 1982) suggest that for about 5% of the population, the dietary intake of cadmium is at least two-fold as compared with the average intake, i.e. 20 μg/day for Finland. Vahter et al. (1991) found that in 3 out of 105 duplicate diets of 15 women (27-46 years of age) the cadmium content exceeded 20 μg/day. According to the data on distribution of exposure in the Finnish population, maximum intakes are about 2.5-fold when compared with the average (Louekari et al. 1989).

Vahter et al (1992 and 1996) has found that in Sweden vegetarians as well as women who had diets rich in cereals and root vegetables or shellfish had a higher intake of cadmium (17 μg of Cd/10 MJ) than women eating a mixed diet (14 μg of Cd/10 MJ). Due to rather similar food culture and habits, these observations are likely to apply to Finland as well. Whether a vegetarian diet constitutes a special risk factor is uncertain. Some food items, not vegetables but those of animal origin (liver, kidneys, shellfish), have especially high concentration of cadmium. In fact, Vahter et al. (1996) also found that women who had a diet rich in shellfish had two-fold dietary

In Finland, Cd limit values for plants are: for potatoes 0.05 mg/kg f.w.; for other vegetables 0.1; for raw cereals and cereal preparations 0,1; and for bran, embryos and durum wheat 0.15 mg/kg f.w. (the decision of Ministry of Trade and Industry Nr 134/1996). In addition to this, there are Cd limits for some sea originating food stuffs, too.

Table 36. Dietary intake of cadmium in different countries.

DIETARY INTAKE (μg/day)		n	Method	Reference (Country/area)
Average	Variation			
14	Range: 3-3.5	1348	Total diet method, 3 days food record	Louekari et al. 1989 (Finland, Kuopio, Turku) *distribution provided*
8.5 (nonsmokers, F)	Range: 5.7-14 SD: 2.1	15	Duplicate diets One week average	Vahter et al. 1991 (Sweden, Stockholm)
	Range: 5-45	110 diets	Total diet study	Coomes et al. 1982 (UK) *distribution provided*
	Range 5-50	260 diets	Duplicate diets	Coomes et al. 1982 (UK) *distribution provided*
13.7 (18 years old)	SD: 6.1	272	Total diet method 48-hour recall	Mykkänen et al. 1986 (Finland/five cities) *distribution provided*
13.8	Range: 7.1-33.3	24	Duplicate diets	Dabeka et al. 1087 (Canada/five cities)
30 F	SD: 8	26	Duplicate diets 4 days for all subjects	Schelenz 1983 (Germany/Karlsruhe)
15.8 M	Range: 6.6-30.3 SD: 5.9	40	Total diet method 24-hour recall	Louekari et al. 1988 (Finland/Jyväskylä)
8.2	Range: 2.0-25.1 SD: 4.9	40	Duplicate diets based on 24-h recall	Louekari et al. 1988 (Finland/Jyväskylä)
14.5 F	Range: 7.2-23.8	42	Total diet method 14 days food diary	Louekari et al. 1991 (Finland/Helsinki)
8.5	5.7-14	15	Duplicate diets of 15 women for 7 days	Vahter et al. 1990 and 1991 (Sweden)
49	-	thousands of households	Total diet method Food record for 7-14 days	Ohmomo 1981
33	theoretical max. 75		Total diet method	Andersen 1981 (Denmark)
18	Range: 2.1-88.1			Buchet 1983 (Belgium)
12.59	Range: 0.6-66.9	199	Fecal Cd	Kowal et al. 1979 (USA/Chicago)
DIETARY INTAKE, CONTAMINATED AREAS (μg/day)				
32	Range: 16-79 95 percentile: 71	75	Diary estimate	Morgan et al. 1988 (families, 4 weeks)
26	Range: 1-151	65	Duplicate diets (individuals, one week)	Morgan et al. 1988

SD= standard deviation
M= male, F= female

Trend of dietary exposure to cadmium

If dietary exposure to cadmium will increase, the number of people suffering from renal and bone effects would increase consequently.

It is estimated in chapter 3.5.2.3, based on Finnish data, that cadmium concentration in wheat fields would increase from 0.21 mg/kg to 0.305 mg/kg in 100 years if Cd content in phosphate fertilizers is 138 mg/kg of P (Table 13). This Cd content in fertilizer represents the average current level in Europe. Based on the Finnish data and algorithms recommended by the Commission/ERM, the corresponding Cd content in wheat is about 70 μg/kg, whereas the present mean concentration is 52 content in μg/kg (see Table 20). It is assumed that the increase of Cd content in other cereals is comparable to that of wheat ie. 35%.

For potato fields, the current and predicted soil Cd concentrations are 0.21 and 0.473 mg/kg (Table 13), respectively. For the potato tuber, the current and predicted Cd contents are 52 and 62.5 μg/kg, respectively (Table 20). This figures do not represent the concentrations measured in the Finnish potato, but apply for algorithm calculations only. The increased of Cd in other vegetable is assumed to be the same as in potatoes ie. 20%.

Based on a relatively large number of samples (totally 250), Kumpulainen (1998) found a clear difference in average cadmium content in Finnish and imported potatoes, ie. 5 and 30 μg/kg, respectively. This difference is likely to be due to different soil parameters and also to the accumulation of cadmium in soils of the countries where potatoes are imported.

It is assumed that the increase of soil Cd - in 100 years caused by fertilizers which contain 138 mg of Cd/kg of P - results in about 20% increase of Cd content of all other food items. From these data, it is calculated that the dietary intake of Cd would increase from 9.5 to 13.0 μg/day (Table 37).

4.2.3 Cadmium concentration in blood

The average blood cadmium concentration in the non-smoking population and in groups including both non-smokers and smokers is 0.3-1.6 μg/L (see Table 38). This is consistent with the estimation of pooled reference values by Alessio et al. (1994). In the three subgroups of smokers the blood cadmium was 1.33 - 2.7 μg/L. Among the smokers the range of blood cadmium is relatively wide. Smoking and age but not dietary habits seem to have a significant influence on the blood cadmium level (Maranelli et al. 1990). Furthermore, reference values of blood cadmium according to age group and sex have been presented by Apostoli (1992) and Alessio et al. (1990).

The range of blood cadmium also seems to depend on number of subjects in the study (n). When n is small (10-50), the maximum of the range is also small, ie. 2.5 -fold as compared with the average. When n is greater (50-2500) the maximum of the range is 3-20 times higher than the average. This indicates that variation of the cadmium exposure is large and is not observed in the studies with small number of samples. The exposure assessment should cover the groups of population, which have a high exposure to cadmium for various reasons. This is further discussed in the chapter 4.5.

As in the studies on dietary intake of cadmium, 90- or 95-percentiles and distributions of blood cadmium level are seldom reported. Therefore, data on high exposures is incomplete.

Unfortunately, no studies on blood cadmium level in the general population in Finland is available.

4.2.4 Cadmium concentration in urine

Data on urinary cadmium in the general population, in populations environmentally or occupationally exposed is available. In Finland, the concentration of cadmium in urine has not been analysed in the general population.

Table 37. Mean consumption (g/person/d) and cadmium concentration (µg/kg) of the Finnish foodstuffs and mean cadmium intake (µg/person/d and %), (Venäläinen et al. 1999). For the prediction of the Cd concentration in food items and the prediction of the intake in the year 2100, it was assumed that the Cd concentration in fertilisers is 138 mg/kg P.

Food stuff	Mean Consumption (g/person/d)	Current mean Cd Concentration (µg/kg)	Current Cd intake (µg/person/d)	Predicted mean Cd concentration (µg/kg) in the year 2100	Predicted Cd Intake (µg/person/d) in the year 2100
Wheat	96	45	4.3	61	5.9
Rye	66	10	0.7	14	0.9
Other	22	20	0.4	27	0.6
Vegetables	108	<10	0.5	12	1.3
Root	29	21	0.6	25	0.7
Potato	122		0.6	6.6	0.8
Fruits	183	<1	0.1		2.8 [5)]
Berries	39	10	0.4		
Meat	62	1	0.06		
Internal	3	135 [1)]	0.4		
Fish	39	12 [2)]	0.5		
Milk	442	<2 [3)]	0.04		
Oils and fats	37	<20	0.4		
Alcohol	155	<2 [4)]	0.2		
Sugar and sweets	31	<10	0.2		
Tea drinks	109	<0.1	<0.01		
Coffee	433	<0.2	0.04		
Totally			9.5		13

9,5 µg/person/d = 1,1 µg/kg body weight/week
PTWI = 7 µg/kg body weight/week
PTWI = Provisional Tolerable Weekly Intake

1) **Mean concentration of cadmium in bovine and porcine liver and kidney.**
2) **Mean concentration of cadmium in Baltic herring and rainbow trout.**
3) **Concentration unit, mg/kg d.w.**
4) **Mean concentration of cadmium in wine and beer.**
5) **This estimate is based on the assumption that the intake from all other food items except cereals and potatoes (ie. 2,35 µg/day), will increase by 20 % to 2,8 µg/day.**

In Table 39 urinary cadmium is expressed as µg/L, µg/24 hour, µg/g of creatinine or µmol/mol of creatinine. These units can not directly be transformed. However, with reasonable assumptions, transformations having sufficient accuracy can be made (see table 40).

In the studies presented in Table 41, the average urine cadmium concentration in general population was 0.2-2.3 µg/L. The maximum urinary cadmium levels measured in general population are 5.0, 6.88, 5.7 and 17.44 µg/L.

Variation of exposure as measured in urinary cadmium concentration seems greater than the variation of dietary intake. This is due to smoking, and the deficiency of calcium and iron in some parts of he population, which increases the gastro-intestinal absorption of cadmium, and other factors mentioned in section 4.2.1. Therefore, when the exposure of the general population to cadmium is compared with critical levels, dietary intake data does not give enough information. Urinary cadmium is reported in most epidemio-

Table 38. Blood cadmium concentration and its variation.

Blood Cd (μg/l)		**n**	**Reference (Country/area)**
Average	**Variation**		
$0.3_{nonsmoking}$	Range: 0.11-0.86 SD: 0.17	15	Vahter et al. 1991 (Sweden/Stockholm)
2.0	Range: 0.5-5.0	10	Ellis et al. 1983 (USA/Upton)
1.2 (GM)	GSD: 1.95	143	Friberg & Vahter 1983 (Belgium/Brussels)
0.41nonsmokers 0.63 smokers		124	Nordberg et al.1983 (Sweden/Stocholm)
0.9 (GM)	GSD: 3.43	192	Friberg & Vahter 1983 (Yugoslavia/Zagreb)
0.85	SD: 0.9 Range: <0.2-6.5	579	Brockhaus et al. 1983 (Germany/Cologne)
1.2	SD: 0.7	30	Fontana et al. 1986 (USA/Chicago)
1.6 (median)	Range: 0.3-4.1	38	Kraus et al. 1988 (Germany/Munchen)
$0.37_{nonsmokers}$ $0.57_{ex\text{-}smokers}$ $1.33_{smokers}$	SD: 0.24 SD: 0.37 SD: 0.95 Range all: 0.1-5.1	140 86 214	Moreau et al. 1983 (France/Paris) *distribution provided*
10.0 (GM)	GSD: 287	396	Carvalho et al. 1986 (Brazil/Santo Amaro City) contam. from a lead smelter *distribution provided*
1.0	Range: 0.21-3.3 GSD: 16.5	168	Kowal et al. 1979
$2.7_{smokers,\ F}$ $0.5_{ex\text{-}smokers,\ F}$ $0.5_{nonsmokers,\ F}$	Range: 0.1-12.7 Range: 0.2-1.1 Range 0.1-2.0	45 3 84	Telisman et al. 1986 (Yugoslavia,Zagreb) *distribution provided*
$0.9_{nonsmokers}$	SD: 0.28	17	Sharma et al. 1983 (New Zealand)
0.5	Range: -11.5	2545	Krause et al. 1989 (Germany)
$0.36_{nonsmokers}$	SD: 2.06	275	Alessio et al. 1993
$0.5_{GM,nonsmokers}$ $1.5_{GM,smokers}$	90 percentile 1.12 90 percentile 4.12	1319 665	Alessio et al. 1993 (general cases in the literature)
Occupationally exposed, blood Cd (μg/l)			
14.1	Range: 5.0-59.9	18	Hassler et al. 1983
-,M	Group means: 1.5-6.8	83	Alessio et al. 1993

Table 39. Cadmium in urine in different countries/areas and sub-populations with information on variation.

NOT-OCCUPATIONALLY EXPOSED			
Urine Cd (μg/l)		**n**	**Reference (Country/Area)**
Average	**Variation**		
2.3	Range: 0.1-5.0	10	Ellis et al 1983 (USA)
0.863 females 0.728 males		1000	Kowal 1988 (USA)
0.636	Range: 0.02-2.06	189	Kowal et al. 1979 (USA)
0.2		35	Järup et al. 1975
$0.62_{nonsmokers}$	SD: 0.34	16	Sharma et al. 1983 (New Zealand)
0.1 (?)	Range: - 6.88	2545	Krause et al. 1989 (Germany)
1.22	Range: 0.01-17.44 GSD: 3.07	378	Ikeda et al. 1995 (Japan/13 prefectures)
1.8 M 1.59 F	GSD: 2.0 GSD: 2.06	109 131	Kido et al. 1992 nonexp. (Japan/Ishikawa)
Urine Cd (μg/24h)			
	0.1-8.0	1699	Buchet et al. 1990 (Belgium)
$1.5_{nonsmoking\ F}$	0.06-6.6	58	Lin et al. 1995
Urine Cd (μmol/mol creatinine)			
3.4	90 percentile: 8.0	85	Bebedetti et al. 1992 (Canada/Quebec)
OCCUPATIONALLY EXPOSED			
Urine Cd (μg/l)			
	Group means: 2.5-20.6	83	Alessio et al. 1993

logical studies where the adverse health effects of cadmium in the general population have been studied. Thus, it is concluded that urinary cadmium is a most suitable estimate of long-term exposure and the body burden.

There is a relation between tubular kidney damage and the increase of urinary cadmium concentration. As summarized by Berglund and Vahtera (1998), tubular kidney damage causes that the normal re-absorption of cadmium-metallotionein complex (Cd MT) decreased and consequently the urinary cadmium level increases. Thus when accumulation of cadmium damages the kidney, the excretion of cadmium increases, and paradoxically the cadmium concentration of kidney gradually and slowly decreases.

Also for urinary cadmium, 90 or 95 percentiles and distributions are seldom reported.

4.2.5 Conclusions on human exposure to cadmium

The average dietary intake of cadmium in Finland is about 10 µg/day. The realistic "worst case" dietary intake is 20-30 µg/day. Increased absorption of the cadmium in diet and smoking increase the exposure of cadmium in some population-groups. Measurements of blood and urinary cadmium in other countries indicate that the variation of total cadmium exposure is remarkable. Based on those data the realistic worst case exposure is 4-5-fold as compared with the average exposure level.

Some factors that either cause variation of the cadmium exposure or increase the susceptibility among certain groups of population are presented in Figure 13. There are population groups attributed with more than one of these factors, but in Finland the level of cadmium exposure or urinary excretion of cadmium in those groups has not been studied. In large population studies, the maximum levels of e.g. urinary Cd or urinary $ß_2$-microglobulin are probably caused by the fact, that for a part of the population, several risk factors simultaneously apply. Variation of exposure to cadmium and most important risk factors are also dealt with in chapter 4.4 "Risk characterization".

4.3 Health effects of cadmium exposure

The present review aims to summarise recent finding on the toxicological effects of cadmium in humans. It is considered that the experimental studies in animals are not of great relevance, since large-scale epidemiological studies are available and are to be preferred for animal studies.

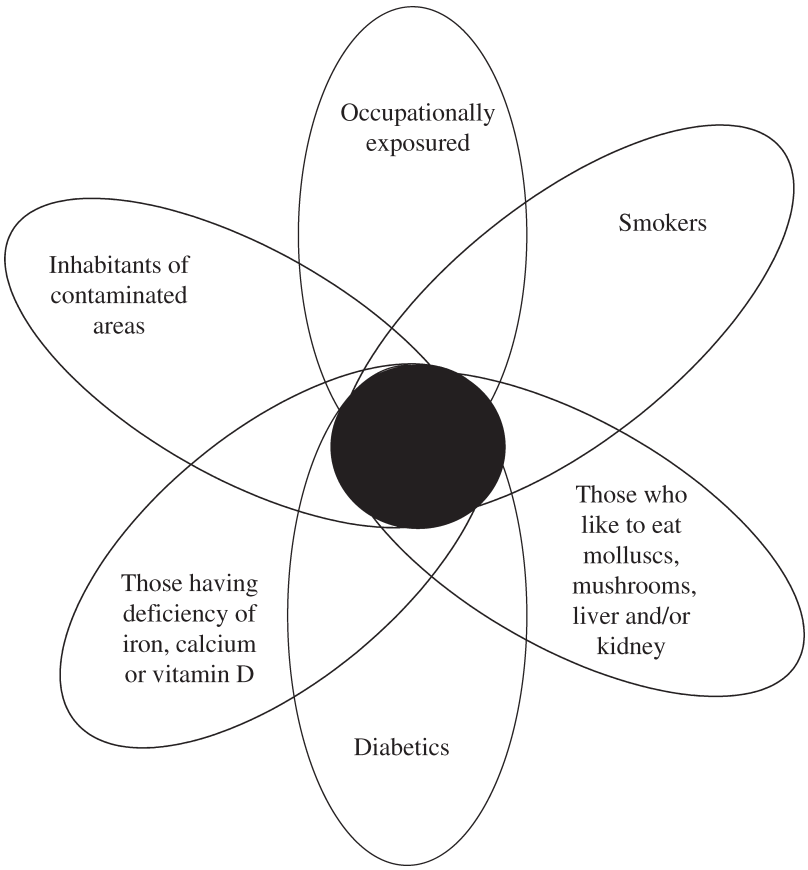

Figure 13. Risk groups. The size of the groups of populations attributed with several of these "risk factors" has not been systematically studied.

In the following, an overview of the studies on the health effects of cadmium published 1988-1999 is presented and the main results are tabulated. It is recognised that excellent reviews of toxicity of cadmium are available, e.g. the monograph of the WHO published in 1992. That review, however, refers only occasionally to articles of late 80's and 90's and omits the discussion of results of the largest epidemiological study available, ie. the Cadmibel study from Belgium. Therefore a summary of more recent results is considered valuable as a part of the risk assessment of cadmium.

4.3.1 Kinetics

Cadmium enters the body mainly by inhalation and by ingestion. Fractional intestinal absorption, which normally is about 5%, is influenced by dietary factors and increases with dietary cadmium concentration. Pulmonary fractional absorption depends partly on the solubility in vivo of the compound.

Kowal (1988) studied the effect of dietary iron and cadmium on the urinary cadmium and $ß_2$ - microglobulin using the data provided by National Health and Nutritional Examination Survey II (NHANES II). He found that urinary Cd was negatively and significantly correlated with dietary iron and dietary calcium. This indicates that cadmium absorption is increased in the general population of the United States by the low dietary intake of iron and calcium.

Cadmium induces synthesis of metallothionein, a low-molecular-weight protein that binds cadmium primarily in the liver and kidney. Metallothionein production can also be induced for example by zinc. When metallothionein-bound cadmium is released into the blood, it is filtered through the glomeruli and is then reabsorbed in the proximal tubules.

Most of the body burden of cadmium is retained in the kidneys and the liver. The half-life of cadmium in human kidneys is probably 10-20 years. Cadmium concentrations in whole blood are affected by both recent exposure and body burden. Excretion occurs mainly via the urine.

Urinary excretion of cadmium by individuals without renal dysfunction primarily reflects the amount of cadmium retained in the kidneys. The target organs for cadmium toxicity depend on the type of exposure. Inhalation of cadmium can lead to chronic obstructive airway disease. Following long-term dietary or respiratory exposure, renal tubular and glomerular dysfunction can develop.

4.3.2 Renal effects

The currently applied critical level of cadmium in renal cortex is 200 mg/kg, which corresponds urinary excretion of about 10 μg/24h. This critical level, however, has been obtained from studies on occupationally exposed men. It has been suggested, that the critical level is lower in the general population (2-4 μg/24h) and that application of the critical level biased by the "healthy worker effect" may lead to the underestimation of the health risk in the general population (Buchet et al. 1990). More recently, Elinder et al. (1998a) have suggested that the critical level of cadmium in the renal cortex is remarkably lower. They consider that 50 mg/kg (w.w.) in renal cortexcauses an excess prevalence of renal tubular damage and corresponds the U-Cd level of 2.5 μg/l and the dietary intake of about 50 μg/day.

In a large-scale population study with 1699 subjects in Belgium (the Cadmibel study), renal dysfunction caused by cadmium exposure have been studied. Some of the study areas (North-Kempen and Liege) are heavily polluted by the non-ferrous metal industry, especially zinc refining plants. The average cadmium content in soils of the non-polluted and polluted areas included in the Cadmibel study were 1.2 ppm and 7.4 ppm, respectively. Cadmium body burden in the residents of the most polluted area (Northern Kempen) was 50-80% higher that in the less polluted areas (Lauwerys at al. 1991 Acta clin Belg). The recent exposure of the population living in the

contaminated areas of the Cadmibel study is probably near to that of control areas, since the residents of contaminated areas have been advised to avoid consumption of well water and locally grown vegetables. In fact a negative correlation between cadmium concentration in soil and blood cadmium in the Cadmibel study was observed (Staessen et al 1996), probably showing that the advise given was followed. For young children, direct ingestion of dust containing cadmium contributes temporarily (PTWI has been set for lifetime exposure) to the cadmium exposure (Lauwerys at al. 1991). The subjects of the Cadmibel study had never been occupationally exposed to cadmium.

Several urinary parameters, urinary excretion of retinol-binding protein, N-acetyl-b-glucosamidase, $ß_2$ -microglobulin, aminoacids and calcium were statistically significantly associated with urinary excretion of cadmium. This suggests the presence of tubular dysfunction caused by cadmium in a part of this population. There was a 10% probability of abnormal values of these renal parameters, when cadmium exceeded 2-4 μg/24h. (Buchet et al. 1990). 10% of the non-smokers and about 30 % of the whole group of study subjects reached this threshold. However, for some urinary parameters, a dose-response relation was observed also when urinary excretion was below 2 μg/24h. Therefore a no-effect-level of cadmium in terms of renal effects is difficult to determine on the basis of this study.

The findings of the Cadmibel study on renal tubular function have been confirmed by a similar study in contaminated areas in the Netherlands (Kreiss 1990).

In the Cadmibel study, it was observed that diabetic patients may be more susceptible to the toxic effect of cadmium on the renal proximal tubule. The cadmium body burden measured as daily urinary excretion of cadmium and diabetes had a synergistic effect on two variables: urinary excretion of N-acetyl-b-glucosamidase, $ß_2$-microglobulin (Buchet et al. 1990).

In the subjects of the Cadmibel study, the body burden of cadmium in non-smoking females was higher than that of males. This might be caused by higher gastro-intestinal absorption of dietary cadmium due to decreased iron stores in women (Buchet et al. 1990).

Since a dose-response relation was also observed at relatively low levels of accumulated cadmium exposure, it was considered difficult to suggest such cadmium body burden that carries no risk for the general population. However, in one of the reports based on the Cadmibel study it was suggested that when the urinary excretion of cadmium is below 2 μg/24h, the risk of occurrence of renal effects remains low. The critical level, however, may be lower for diabetic subjects (Buchet et al. 1990).

The morbidity associated with the changes in the renal proximal tubule and calcium metabolism observed in the Cadmibel study remains to be assessed in the follow-up studies (Lauwerys at al. 1991).

In the Cadmibel study, also a decrease of serum zinc (12.2 vs. 12.6 µmol/l) and creatinine clearance (87 vs. 92 ml/min) were observed when the inhabitants of the polluted and control areas were compared. Creatinine clearance also had a negative correlation with the cadmium content of soil as well as the cadmium concentration in celery and beans (Staessen et al. 1994). Although cadmium emissions from zinc smelters and refiners have decreased from 125 tonnes in 1950 to 0.13 tonnes in 1989, soil contamination by cadmium causes a persistent adverse effect on human food supply.

Markers of early renal changes in workers exposed to cadmium (n=377) and non-exposed control workers (n=50) were studied by Roels et al. (1993). The content of cadmium in the blood and urine of exposed workers were 5.5 μg/l and 5.4 μg/l, respectively. Subjects of this study were only moderately exposed to cadmium, since only seven of them had blood and urine levels of cad-

mium exceeding previously proposed critical levels (10 μg/l and 10 μg Cd/g creatinine, respectively). Exposure to cadmium produced an alteration in several indicators of nephrotoxicity: urinary level of low and high molecular weight proteins (albumin, transferrin, $ß_2$-microglobulin), kidney derived antigens and enzymes (e.g. N-acetyl-b-D-glucosaminidase=NAG), protanoids, glucosaminoglycans and sialic acid.

Based on dose-effect relations in the study subjects, three thresholds are proposed: one around 2 μg Cd/g creatinine, associated with biochemical alterations (increased urinary 6-keto-PGF and sialic acid), second around 4 μg Cd/g creatinine, based on excretion of high molecular weight proteins and some tubular antigens or enzymes (e.g. albumin, transferrin, NAG), and third around 10 μg Cd/g creatinine for low molecular weight proteins and other indicators. This is roughly in accordance with the recommendation of the American Conference of Governmental Industrial Hygienists (ACGIH) of 5 μg Cd/g creatinine in urine as the biological exposure limit. For some of the effect observed around this threshold, link with subsequent development of overt Cd nephropathy has been established. Follow up studies in workers who have increased excretion of low molecular weight proteins (threshold around 10 μg Cd/g creatinine), have shown that the appearance of persistent microproteinuria is the forerunner of progressive deterioration of renal function, ie. age related exacerbation of the age related decline of the glomerular filtration rate (Roels 1993). Concentration of 4 μg Cd/g creatinine corresponds to the cadmium kidney cortex level of about 100 mg/kg, which is about half of the critical level (200 mg/kg) applied so far.

Considering the mechanisms of cadmium nephrotoxicity Roels et al. (1993) has suggested that the enhanced excretion of tubular enzymes might be due to exfoliation or increased turnover of tubular cells, or to a metabolic disturbance. Depending on the underlying mechanism some of these changes could be reversible. The exact mechanism of cadmium toxicity is not known. High affinity of cadmium for thiol groups in macromolecules is certainly relevant (Savolainen 1995).

In a group of workers who had been exposed to cadmium (for 7-42 years, mean 25 years), a progressive reduction of glomerular function was observed during the five years after the workers were removed from the exposure (Roels et al. 1989). Mean urine and blood cadmium levels of the subjects were 18.0 μg/l and 9.7 μg/l, respectively. In comparison, the maximum urinary level observed in the Cadmibel study of people environmentally exposed to cadmium was 8.0 μg/24h (Buchet et al. 1990) which is about 8.9 μg/l. The most important finding is considered to be a significant increase of creatinine and $ß_2$ -microglobulin concentration in serum, indicating a progressive reduction of glomerular filtration rate during the five years follow up after the removal of the workers from exposure. It is noteworthy that during the follow up period the concentration of cadmium in urine and in blood significantly decreased. The reduction of the glomerular filtration rate is about five times greater than that caused by ageing (Roels et al. 1989). It was also confirmed by this study that proteinuria induced by cadmium is not reversible.

Furthermore, this effect is not more pronounced in workers with impaired renal function at the start of the study, but is as clear in those workers showing only sub-clinical signs of renal damage (Roels et al. 1989). It seems reasonable therefore to assume that, since renal damage caused by cadmium is progressive, environmental exposure to cadmium, when associated with preclinical renal effects (increased urinary excretion of $ß_2$-microglobulin, retinol binding protein or albumin) can also cause impaired renal function ie. reduced glomerular filtration rate, at the older age. Due to this effect on the age related change of the renal function, the early pre-clinical renal changes are considered to be adverse by Roels et al. (1989).

As pointed out in recent reviews, significant renal effects of cadmium, usually only after occupational exposure to cadmium, include also glucosuria and phosphaturia (WHO 1992, Savolainen 1995).

Renal function of people who were not occupationally exposed to cadmium but had varying environmental exposure due to local contamination in Germany (Stolberg, Duisburg and Dusseldorf) were studied by Ewers et al (1985). 65 and 66 year old women, who had lived most of their life in one of these areas were selected for the study. Levels of cadmium in serum and in urine were significantly different, the highest levels were found in Stolberg, where cadmium concentrations of soil and plants were also higher than those in other districts. Serum creatinine levels were significantly higher in the residents of Stolberg as compared with Duisburg and Dusseldorf. Other renal parameters (proteinuria, phosphaturia, aminoaciduria) did not show significant differences between the study populations. The authors consider, that for the Stolberg group a synergism of ageing and cadmium with respect to the decline of glomerular function can not be excluded.

Jung et al. (1993) investigated the following groups of individuals: 1) controls; 2) environmentally exposed, and 3) occupationally exposed to cadmium. The serum creatinine and ribonuclease values, indicators of glomerular effect, did not differ between these groups. In the occupationally exposed group indicators of tubular damage (e.g. retinol binding protein, a1-microglobulin) were increased. In the environmentally exposed group, alanine aminopeptidase, alkaline phosphatase and N-acetyl-beta-D-glucosaminidase (NAG) levels were increased. The a1-microglobulin level in urine was increased in individuals with urinary cadmium excretion of >5 µmol/mol creatinine (corresponding 4 μg Cd/l of urine). This supports the results of the Cadmibel study indicating that changes in markers of renal effects are observed at the urinary level of 2-4 μg Cd/l . In 30% of the individuals in environmentally exposed group, a1-microglobulin or N-acetyl-beta-D-glucosamidase (NAG) levels exceeded the corresponding upper reference limits. These two analytes were recommended for screening and detection of cadmium-induced renal dysfunction.

Järup et al. (1995) studied 72 persons living near to cadmium polluting industry in Southern Sweden. Those living within 500 m of the plant had significantly elevated U-Cd (average around 1 μg/g creatinine) and also displayed a high prevalence of elevated NAG in urine.

A nine year follow-up study of 3178 persons living in a Japanese cadmium- contaminated area was conducted by Nakagawa et al. (1993). The standardised mortality ratios of the urinary $ß_2$-microglobulin positive subjects (excreting more than 1 000 μg/g creatinine) of both sexes were higher that those of the general Japanese population, whereas the cumulative survival curves were lower than those of the urinary $ß_2$ -microglobulin negative group. Furthermore, mortality rates increased in proportion to increases in the amount of $ß_2$-microglobulin excretion. These results suggest that the prognosis of cadmium exposed subjects with proximal tubular dysfunction is unfavourable. The mortality tended to become higher as the severity of renal dysfunction progressed. A higher mortality rate was observed when the urinary excretion of $ß_2$-M was above 300 μg/g creatinine. Using data reported by Nordberg et al. (1997) this corresponds urinary cadmium level of approximately 4-5 μg/l.) Among the environmentally exposed people of the Cadmibel study urinary $ß_2$ -M of 286 μg/24 h (corresponding 203 μg/g creatinine) was exceeded by 25% by the high exposure group (n=331). Maximum values of urinary $ß_2$-M for low exposure and high exposure groups were 3985 and 5004 μg/24h, respectively (Staessen 1994). This suggests that for a part of the residents of polluted areas in Belgium, the level of cadmium exposure is at a level that has been associated with increased mortality in population studies in Japan.

Table 40. Conversion factors for Cd concentration in urine used in this report:

1 µmol Cd/mol creatinine = 0.81 μg Cd/g creatinine **1 μg Cd/24h = 0.7 μg/g creatinine** **1 μg Cd/24h = 0.7 μg/l** **1 μg Cd/l = 1.4 μg/24h** **1 μg Cd/l = 1 μg/g creatinine** **1 μg β_2-M/24h = 0.7 μg β_2-M/g creatinine**
Factors are based on the following figures and estimates: *Volume of urine: 1400 ml (600–2500 ml)/24h* *Excretion of creatinine: 1.4 (1.0–1.8 g)/24h* *1 mol Cd=96 g, 1 mol creatinine=118 g*

These results were confirmed by a 15 years follow-up study by Nishijo, Nakagawa et al. (1995) on 2408 individuals living in the same polluted area (Kakehashi River basin). A significant relationship was seen between urinary excretion of RBP and mortality in both sexes. The observed increases of mortality are due to heart failure and renal diseases. Based on the comparison of urinary ß$_2$-M values, the environmental exposure to cadmium in this area in Japan seems to be somewhat higher than in the contaminated areas in Europe. Whether increased excretion of RBP could be caused by factors other than cadmium exposure remains to be evaluated.

Urinary Cd, NAG and creatinine levels were also analysed for 400 inhabitants of Cd -polluted areas in Annaka City, Japan (Kawada et al. 1992). The geometric means of urinary cadmium in five districts of this area were 3.0, 2.6, 2.5, 2.0 and 1.7 μg/l (see table 42). There were statistically significant differences in NAG excretion among the five groups but no difference could be found between two groups using multiple comparison. However, the study indicates an association of urinary Cd and NAG levels in Cd-exposed population whose geometric mean urinary Cd concentration is about 2 μg/l. Thus, the authors recommend a continuous survey of the minimum health effects of Cd-pollution using urinary excretion of Cd and proteins, e.g. NAG. These results also support the results of the Cadmibel study indicating that changes in indicators of the renal effects caused by cadmium are observed at the urinary level of 2-4 μg Cd/l.

Yamanaka et al. (1998) found that among people living in non-polluted area in Japan, total urinary protein, urinary ß$_2$-M and NAG were associated with urinary cadmium. The authors consider that their results are in agreement with studies of Buchet et al. (1990) and Lauwerys et al. (1994), who proposed 2 μg Cd/l as the maximum tolerable internal dose of Cd for the general population. Buchet et al. (1990) estimated that this urinary concentration corresponds a renal cadmium concentration of about 50 mg/kg.

According to Nogava (1992), a lifelong intake of about 2 g of cadmium, corresponding average daily intake of 80μg, would bring about a U-Cd value of 4-5 μg/l, which would elicit a significant rise in prevalence of tubular damage. However, several recent studies indicate that the threshold for renal effects is lower, 2-4 μg/l, which would correspond a daily intake of 40-80 μg.

Reversibility of the cadmium-induced renal lesions was studied by Iwata et al. (1993). They found that while daily dietary intake was dropped from about 200 μg to 53-106 μg (due to replacement of polluted soil of rice fields) there was no

evidence that renal lesions were reversible. On the contrary, the tubular damage had, in most cases, been aggravated. However, it is noteworthy, that the urinary concentration in population was very high, during the follow-up it decreased from 8.5 to 6.0 μg/l. In another Japanese study by Kido et al. (1990), in most of the examined subjects, serum creatinine, indicative of more severe glomerular damage, tended to increase.

The urinary excretion of cadmium and $ß_2$-microglobulin were studied for 260 residents of Walsall, England (Tennant 1991). The individuals participating grew a part of their vegetable diet in urban garden soils. For non-smoking, non-occupationally exposed residents, urinary excretion of cadmium was found to be up to 34% of the biological exposure index given by American Conference of Governmental Industrial Hygienists. Thus, it was concluded that when occupational exposure to cadmium is assessed, dietary intake from urban grown produce should be considered as an additional source of exposure.

Müller et al (1989) studied the level of indicator enzymes NAG and alanine aminopeptidase (AAP) in two groups: those with urinary cadmium levels less than 2.0 μg/l and those with Cd-U greater or equal to 2.0 μg/l. The mean NAG and AAP levels in urine were significantly higher in the high exposure group. The results indicate that markers indicating sub-clinical renal dysfunction, reveal changes already when urinary cadmium level is below 10 μg/l, which has been recommended as an upper limit by the 1980 World Health Organisation Study group. These results suggest, similarly to the observations of several other studies cited above, that the threshold level of renal effects of cadmium is close to 2 μg Cd/l of urine, above which first signs of nephrotoxicity can be measured.

Ishizaki et al (1989) studied the dose-response relation between urinary cadmium and $ß_2$-microglobulin. 3178 inhabitants over 50 years of age in the Cd-polluted Kakehashi River basin, Japan and a smaller group of inhabitants in non-polluted areas were studied. Urinary Cd and $ß_2$-microglobulin were significantly higher in the Cd-exposed subjects. Prevalence of ß-microglobulinemia increased proportionally with increasing urinary Cd concentration, thus confirming the dose-response relation. Biological threshold values for environmentally exposed people suggested on the basis of these results were 3.8-4.1 μg Cd/g creatinine, corresponding to the urinary cadmium level of 3.8-4.1 μg/l.

Nordberg et al. (1997) studied the urinary cadmium level, urinary $ß_2$-M and urinary albumin in three areas in China. Urinary cadmium in the high exposed area, the medium exposed areas and control area were 10.7, 1.62 and 0.40 μg/l, respectively. There was a clear increased of the $ß_2$-M and the urinary albumin in the heavily exposure group as compared with the control group and a slight increase in the medium exposed group. Statistically significant dose-response was found between cadmium in urine and urinary $ß_2$-M.

Studies on occupational exposure to cadmium and renal effects are not reviewed here in detail. An adequate overview is that dose-response data based on epidemiological studies is confirmed by the occupational evidence of cadmium toxicity. For example results from Swedish battery workers (Järup et al. 1994) indicates that depending on age, the prevalence of $ß_2$-microglobulinuria was 5% to 15% at U-Cd levels above 3 nmol/mmol creatinine. In this study , a 10% prevalence of tubular proteinuria already at 1.5 nmol Cd/mmol creatinine was found in an older age group. This suggests that elderly people are more susceptible to the adverse renal effects caused by cadmium.

The critical concentration of cadmium in urine and in the kidney cortex

As a risk characterisation based on several studies, Elinder et al (1998a) concluded that in the general population, an average U-Cd level of

2.5 μg/l is related to an excess prevalence of renal tubular damage of about 4%.

Elinder et al. (1998a) estimated that "in order to prevent renal tubular damage that can proceed to clinical disease and perhaps contribute to early death, cadmium levels in the kidneys and in urine should be kept below 50 mg/kg and 2.5 μg/l, respectively." Elinder et al (1998b) present a "best guess" of the relation of cadmium in kidney cortex, urinary cadmium and prevalence of the tubular effects in the population. These are simplified in table 41. The relation of these three parameters is obviouslynot mechanistic/deterministic. Not all who have a given kidney concentration of cadmium will develop renal insufficiency. There are susceptibility factors, for example, age, gender, use of nephrotoxic drugs, which complicate the mechanism by which the body burden of cadmium leads to renal effects.

It is necessary to discuss here, whether the existing evidence justifies the conclusions drawn by Elinder et al. (1998a). First question is the relation between early signs of renal effects and clinical disease and the second question is whether there is enough evidence of increased mortality caused by high exposures to cadmium. First, our evaluation is that there is some evidence of early onset of renal insufficiency (glomerular damage) associated with urinary cadmium level of about 4 μg/l and manifested e.g. by increased level of creatinine in serum. This seems to be the second phase of the development of renal failure which starts as increased excretion of high-molecular weight proteins, which as such could be considered to be a pre-clinical symptom. It is likely that exposure to cadmium which causes clinical disease (e.g. impaired excretion of creatinine) is higher that the exposure that causes e.g. increases urinary level $ß_2$-microglobuline and NAG.

Second, two Japanese studies (Nakagawa et al. 1993, Nishijo et al. 1995) suggest that renal effects caused by cadmium are associated with increased mortality due to heart failure and renal disease. These observation are preliminary, and this type of studies should be repeated in contaminated and uncontaminated areas of Europe. The level of exposure in these studies is relatively high (U-Cd about 4-5μg/l) and probably apply to 1) those living in highly contaminated areas and to 2) smokers, who have increased absorption of cadmium and diet, which is rich of cadmium. The observations of increased mortality are serious, since it was also found that the mortality tended to become higher as the severity of renal dysfunction progressed during the follow-up study (Nakagawa et al. 1993).

4.3.3 Calcium excretion and bone injuries

It has been observed that exposure to cadmium may cause higher urinary excretion of calcium. This was confirmed by the Cadmibel study, where

Table 41. Summary of the "best guess" presented by Elinder et al. (1998b) of prevalence of tubular effects and respective cadmium concentration in kidney cortex and urinary cadmium level.

Cadmium in kidney cortex (mg/kg)	Urinary cadmium (μg/l)	Prevalence of renal effects in the respective group of population
<50	<2.5	0
51-60	2.75	1
91-100	4.75	5
141-150	7.25	14
191-200	9.75	30
>200	>10.25	>35

a relatively low level of cadmium body burden increased the urinary level of calcium and caused calcium wasting. Significantly different calcium excretion/24h were (2.0 and 2.6 mmol of Ca/24h) found between groups which had 0.9-1.4 μg Cd/24h and 1.4-8.0 μg of Cd/24h (corresponding 1.0-5.6 μg/l). Also elevation of serum alkaline phosphatase activity was observed in the subjects of the Cadmibel study. These results suggest that environmental exposure to cadmium may be sufficient to influence calcium homeostasis and bone metabolism (Lauwerys at al. 1991). However, it is also stated by authors of the Cadmibel study that the biological significance of this observation still remains to be elucidated. The authors consider, that further studies are needed to show, whether this effect could exacerbate age-related osteoporotic changes especially in women with low dietary calcium (Buchet et al. 1990).

Also Järup (unpublished data) found in a recent study on women in Stockholm (50-70 years of age), a significantly increased urinary excretion of calcium was found in those, who excreted more than 0.81 μg Cd/l, versus women who had a lower U-Cd. This finding is in accordance with the Belgian results cited above.

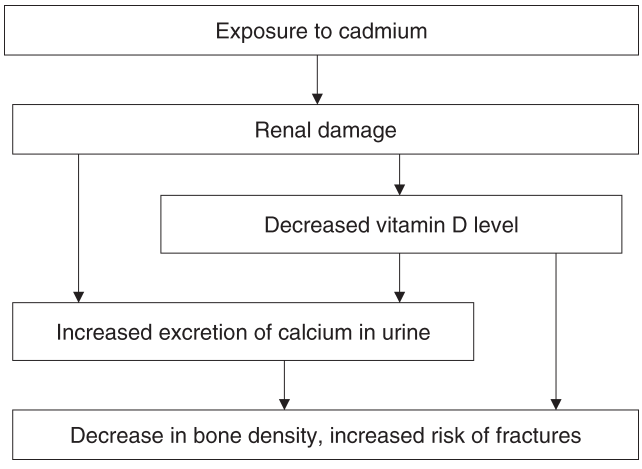

Figure 14. Probable sequences of effects, which could increase the risk of bone fractures, based on Järup (1998), Roels et al. (1999) and Tsuritani et al. (1992).

Elinder et al. (1998b) summarised that there is probably an important causative link between bone effects of cadmium and toxicity on the kidneys. Notably, vitamin D is activated in the kidney to calcitrol, which functions as a hormone with an important role in the absorption of calcium from the gut and in the calcification of bone.

In a 6.6 years follow-up in Belgium, Roels et al. (1999) found that long-term low-level environmental exposure to Cd may accelerate the demineralisation of the skeleton, especially in post-menopausal women, which in turn may lead to greater bone fragility and an increased risk of fractures. The relative hazard rate associated with a doubling of urinary Cd was 1.73 (p=0.007) for fractures in women and 1.60 (p=0.08) for stature loss in men. It is noteworthy that 6 of the 10 districts studied bordered on zinc/cadmium smelters, and the exposure levels are higher than in Finland. The urinary levels of cadmium were not sufficiently reported.

There is also experimental evidence that cadmium disturbs calcium homeostasis. In long-term exposures, or when repeated doses of Cd-MT are given with short intervals, irreversible changes in the urinary excretion of calcium are seen (Leffler et al. 1996).

Whether a small effect on calcium metabolism may lead to morbidity in the general population remains to be studied. In heavily polluted area in Shipham, Britain, cadmium exposure was not associated with excess mortality from fractures (Inskip et al. 1982). However, bone lesions have been experimentally induced by cadmium in laboratory animals. Combination of the administration of cadmium, and deficiency of vitamin D was particularly adverse (Kjellström 1985). Some epidemiological evidence supports these findings, namely several cases of osteomalacia and osteoporosis have been observed in workers exposed to cadmium and about 150 of the itai-itai patients have developed bone lesions.

Elderly people often suffer from decalcification of bones which can be attributed to ageing or hormonal or nutritional deficiencies. Therefore, further studies should definitely be undertaken to see whether environmental exposure to cadmium may contribute to osteoporosis and its consequences (Staessen et al. 1996). The significance of the findings of the Cadmibel study in terms of morbidity and mortality are studied in the prospective PheeCad Study in Belgium (Staessen et al. 1996).

Roels et al. (1993) suggest that interference of cadmium body burden with prostanoids observed at the level of 2 μg Cd/g creatinine should be studied further, since it might play a part in the toxicity of Cd in bone. Prostanoids are hormones with many biological activities. It has been suggested, based on a study on cultured osteoblasts like cells, that Cd can stimulate bone resorption via an increased production of prostaglandin E2. If renal synthesis of prostaglandins is affected also in vivo, this observation together with the results of the Cadmibel study, suggests that environmental pollution by cadmium may contribute to bone decalcification and osteoporosis in the general population (Roels et al. 1993).

In a group of workers who had been exposed to cadmium, serum alkaline phosphatase activity increased significantly during the five year follow up period, which may reflect the interference of cadmium with bone metabolism (Roels et al. 1989). Mean urine and blood cadmium levels of the subjects were 18.0 μg/l and 9.7 μg/l, respectively. In comparison, the maximum urinary level observed in the Cadmibel study of people environmentally exposed to cadmium was 8.0 μg/24h (Buchet et al. 1990) which is about 8.0 μg/l.

The quantitative dose response relation for the effects of cadmium on calcium and bone metabolism were elucidated by Staessen et al (1991) in the context of the Cadmibel study; urinary and blood samples of 1987 individuals were studied. The results showed that serum alkaline phosphates activity and urinary excretion of calcium significantly correlated with urinary excretion of cadmium. In men also serum total calcium concentration correlated negatively with the urinary excretion of cadmium. The slopes of these relations showed that whilst U-Cd doubles serum alkaline phosphatase and urinary calcium rise in both sexes by 3-4% and 0.25 mmol/24h, respectively, the serum calcium in men decreased by 6 µmol/l. These findings suggest that environmental exposure to cadmium calcium metabolism is gradually affected. The linear regression found between urinary excretion of cadmium and the parameters of calcium metabolism suggest in fact that no threshold exists andthat the effects on calcium metabolism develop gradually when cadmium accumulates in the body. The effect may be due to the dysfunction of renal tubules or development of vitamin D resistance (Staessen et al. 1991).

Recent experimental studies in animals support these observations made in humans. The effects of diet that was low in calcium, vitamin D and vitamin E, and added for cadmium on the bone density of the hind legs of mice for experimental period of 29 days - 24 months was investigated by Imai (1995). Using X-rays and a densitometer it was observed that diets deficient of calcium and vitamins D and E promoted the effect of cadmium on the ageing of bone.

4.3.4 Blood pressure and cardiovascular disease

In the Cadmibel study, where the population studied had a wide variation of cadmium body burden due to heavy local contamination by cadmium releases, no statistical association was observed between environmental exposure to cadmium and blood pressure elevation of the prevalence of cardiovascular cases (Lauwerys at al. 1991)

Also in another large scale epidemiological study, the Second National Health and Nutrition Examination Survey (NHANES II) in the USA, no significant correlation between urinary cadmium and blood pressure (Whittemore et al. 1991) was observed after the hypertensive patients were removed from the analysis.

4.3.5 Carcinogenicity

The following chapters on carcinogenicity and genotoxicity of cadmium are mostly based on the recent evaluation of IARC (1993).

Occupational exposure to cadmium

Among the important compounds of cadmium are cadmium oxide (used in batteries, as an intermediate and catalyst and in electroplating), cadmium sulphide (used as a pigment), cadmium sulphate (used as an intermediate and in electroplating) and cadmium stearate (used as a plastics stabiliser). Use of cadmium-pigments and cadmium-stabilisers of plastic have decreased. Trends in the total use of cadmium have been increasing but are much dependent on the demand of nickel-cadmium batteries, which now amounts 55% of the total consumption of cadmium (OECD 1994).

Occupational exposure to cadmium and cadmium compounds occurs mainly in the form of airborne dust and fume. Occupations in which the highest potential exposures occur include cadmium production and refining, nickel-cadmium battery manufacture, cadmium pigment manufacture and formulation, cadmium alloy production, mechanical plating, zinc smelting, soldering and polyvinylchloride compounding. Although levels vary widely among the different industries, occupational exposures have decreased in the last two decades. Urinary and blood cadmium concentrations are generally much higher in occupationally exposed peopleas compared with those non-occupationally exposed, for whom the most important sources of exposure are cigarette smoking and food.

Human carcinogenicity data

Following a report on the occurrence of prostatic cancers in a small group of workers employed before 1965 in a plant manufacturing nickel-cadmium batteries in the United Kingdom, a series of cohort analyses were undertaken. These did not confirm the excess among the remaining workers, but an increase in mortality rates from lung cancer was detected. A small cohort working in the same industry was studied in Sweden: no excess of prostatic cancer was detected, but a non-significant increase in mortality from lung cancer was found among workers who had the longest duration of employment and latency. Two small copper-cadmium alloy plants were studied in the United Kingdom. The rate of mortality from lung cancer increased in one of them but decreased in the other. A case-control analysis of lung cancer did not show any association with exposure to cadmium. No increase in mortality from prostatic cancer was found in these two plants, while in a similar plant in Sweden a non-significant excess was detected (IARC 1993).

Excess mortality from lung cancer was reported among workers employed in a US cadmium recovery plant, and a dose-response relationship was demonstrated between estimated cumulative exposure to cadmium and lung cancer risk. The latter was unlikely to be confounded by cigarette smoking. In a large cohort of workers from 17 cadmium processing plants in the United Kingdom, mortality from lung cancer was increased in the overall cohort and there were suggested trends linked to duration of employment and with intensity of exposure. The increase in lung cancer risk was stronger in the small proportion of workers with high cadmium exposure. Confounding by concomitant exposure to other cancer determinants, including arsenic, was not controlled (IARC 1993).

A number of early studies reported an increased risk for prostatic cancer among cadmium workers, but the results of later studies are not consistent. Early and recent studies provide consistent evidence that the risk for lung cancer is increased among workers exposed to cadmium. Constraints that influence the assessment of both lung and prostatic cancer risk are that the number of long-term, highly exposed workers is small, and the historical data on exposure to cadmium are limited, particularly for the non-US plants.

Confounding by cigarette smoking in relation to lung cancer was addressed directly only in the

study from the USA, but some other studies provided analyses based on internal comparisons, which are not likely to be affected by this problem. Control of the confounding effect of co-exposure to other metals, particularly arsenic and nickel, was limited. However, the analyses of results obtained from US cadmium exposed workers indicated that the increase in lung cancer risk was unlikely to be explained by exposure to arsenic.

Animal carcinogenicity data

Several adequate studies on orally administered cadmium chloride in rats and mice are available. Cadmium chloride has produced dose-related increases in the incidences of leukaemia, interstitial-cell tumours of the testis and proliferative lesions of the prostate. In another study on cadmium chloride in rats, no increase in tumour incidence was seen. In two inhalation studies in rats, malignant lung tumours were produced by cadmium chloride, cadmium sulphide/sulphate, cadmium sulphate and cadmium oxide fume and dust at low levels of exposure for short duration (IARC 1993).

In one inhalation study in mice cadmium oxide fume or dust had increased incidences of lung tumours. In an inhalation study in hamsters of cadmium chloride, cadmium sulphide/sulphate, cadmium sulphate and cadmium oxide fume and dust, no increase in the incidence of lung tumours was found.

Other relevant data

Cadmium can suppress cell-mediated immune responses in vitro. Furthermore, in a recent study on workers who are chronically exposed to high levels of cadmium, an effect on the levels of some cytokines was observed (Yucesoy et al.1997). This could cause immuno-suppression and contribute carcinogenesis although these results should be considered tentative. Parenteral administration of cadmium salts produces adverse effects on the testes, ovaries, placenta and embryo in experimental animals; many of these effects have been shown to be preventable by the administration of zinc compounds. Administration of cadmium at doses that affect placental morphology or function induces foetal anaemia, growth retardation, teratogenicity and embryonic and foetal death in experimental animals. Reproductive and developmental toxicity have been reported following exposure to cadmium compounds by oral and inhalation routes, but the effects are generally much less severe than after parenteral administration.

Genotoxicity

In three of five studies, the frequencies of chromosomal aberration were increased in peripheral blood lymphocytes of workers exposed to cadmium in the metal industry, where they were usually also exposed to other metals. No effect of cadmium was observed in a limited study of workers from a Swedish alkaline battery factory. In two studies of cadmium pigment plant workers, no increase in the frequency of chromosomal aberrations was observed. No increase in the frequency of sister chromatid exchange was seen in one study of workers exposed to cadmium. In one of two limited studies of itai-itai patients, increased frequency and severity of chromosomal aberrations were observed. In one study, no increase in sister chromatid exchange frequency was observed in people living in a cadmium-polluted region of Japan. In a study of subjects living in a cadmium-polluted region of China, there were small but significant increases in chromosomal aberration frequency. A significant dose-effect relationship between urinary levels of cadmium and chromosomal aberration frequency was also observed, and more severe aberration types were observed in individuals with high urinary levels of cadmium. In those studies inwhich significant responses were observed, the chromosomal aberrations tended to occur in the more heavily exposed groups and were of more complex types.

Chromosomal aberrations and aneuploidy were observed in animals exposed to cadmium chloride in vivo. Dominant lethal mutations were

generally not induced in mice. Cadmium chloride damages DNA of human cells in vitro. In the few studies available, chromosomal aberrations were observed in human cells treated with cadmium sulphide but not in those treated with cadmium chloride. Indications of aneuploidy were observed in human fibroblasts after treatment with cadmium chloride. Studies using cultured animal cells show that exposure to cadmium compounds damages genetic material. DNA strand breaks, mutations, chromosomal damage and cell transformation have been observed in vitro. Cadmium compounds inhibit the repair of DNA damaged by other agents, thereby enhancing their genotoxicity. Mutations have generally not been observed in Drosophila or bacteria; however, a weak response was observed in some studies in bacteria and there is evidence of cadmium induced DNA damage in bacteria.

According to findings of Prozialek et al. (1995), Cd can selectively induce disruption of the junctions between the cells and this could have implications regarding the mechanism of carcinogenic effects of cadmium. Prozialek et al. (1995) found that exposure of LLC-PK1 cells to low micromolar concentration of cadmium caused disruption of the adhering and occluding junctions between the cells. This effect was observed in these cells independently and at lower concentrations than apoptosis. At the time cadmium induced junctional changes there was no increase of apoptotic cells or evidence of DNA fragmentation. Thus, it seems that inhibition of intercellular communication could be a non-mutagenic mechanisms of carcinogenicity of cadmium, not excluding genotoxic activity of cadmium.

Evaluation of the IARC

There is sufficient evidence in humans for the carcinogenicity of cadmium and cadmium compounds. There is sufficient evidence in experimental animals for the carcinogenicity of cadmium compounds. There is limited evidence in experimental animals for the carcinogenicity of cadmium metal. In making the overall evaluation, the Working Group of the IARC took into consideration the evidence that ionic cadmium causes genotoxic effects in a variety of types of eukaryotic cells, including human cells. The overall evaluation is that cadmium and cadmium compounds are carcinogenic to humans (Group 1).

The conclusion of the IARC is that cadmium has been implicated in the development of lung and prostate cancer in exposed workers and in animals under various exposure conditions. However, there is presently no epidemiological and experimental evidence that exposure to cadmium via food may be associated with an increased risk of cancer.

The National Toxicology Programme (NTP) of the USA has recently upgraded the listing of cadmium and cadmium compounds from "Reasonable Anticipated to be a Human Carcinogen" to "Known to be a Human Carcinogen".

4.3.6 Summary of health effects of cadmium in light of recent epidemiological evidence

Renal effects

The recent studies have not dramatically changed the understanding of the toxicological profile of cadmium. When the health of the general population is considered, renal effects and effects on bones and calcium metabolism still deserve the greatest attention. However, the knowledge on dose-effect relation and thresholds for these effects has improved. Several studies indicate that the safe level, in terms of early renal effects and bone effects of cadmium exposure as expressed in the urinary concentration, is not below 10 μg of Cd/l urine suggested and applied earlier, but about 2-4 μg/l (see table 42). There is also evidence that the sub-clinical renal effects might proceed even after cessation of the exposure and may also impair the glomerular function leading to renal insufficiency at an older age.

When the results of the Cadmibel study are considered (see Table 42) it should be noted that

there was a 10% probability of abnormal values of renal parameters, when cadmium exceeded 2-4 μg/24h. (Buchet et al. 1990). Among the non-smokers 10% of the study subjects reached this threshold. However, for some urinary parameters, a dose-response relation was observed when urinary excretion was below 2 μg/24h. Therefore a no-effect-level of cadmium in terms of renal effects would be difficult to determine.

Since it has been shown that renal damage caused by cadmium is progressive, it also should be noted that environmental exposure to cadmium, when associated with pre-clinical renal effects (increased urinary excretion of ß-microglobulin, NAG, retinol binding protein or albumin) can cause impaired renal function ie. reduced glomerular filtration rate, at an older age. Due to this effect on the age related change of the renal function, the early pre-clinical renal changes are considered to be adverse by Roels et al. (1989).

Excretion of calcium

It has been observed that exposure to cadmium may cause higher urinary excretion of calcium. This was confirmed by the Cadmibel study (Buchet et al. 1990), where a relatively low level of cadmium body burden increased the urinary level of calcium and caused calcium wasting. Elevation of serum alkaline phosphatase activity was observed in the subjects of the Cadmibel study, and Lauwerys et al. (1991) assessed that environmental exposure to cadmium may be sufficient to influence calcium homeostasis and bone metabolism. Also Järup (unpublished data) found in a recent study on women in Stockholm (50-70 years of age), a significantly increased excretion of urinary calcium in those who excreted more than 0.81 μg/l, versus women who had a lower U-Cd. This finding is in accordance with the Belgian results.

The latest and most serious evidence in this respect comes from a Belgian follow-up, made after Cadmibel study. Roels et al. (1999) found that long-term low-level environmental exposure to Cd may accelerate the demineralisation of the skeleton,especially in post-menopausal women, which in turn may lead to greater bone fragility and an increased risk of fractures.

Increased mortality

There is also some evidence that environmental exposure to cadmium observed as increased excretion of ß-microglobulin increases mortality among the exposed population (Nakagawa et al. 1993). The standardised mortality ratios of the urinary $ß_2$-microglobulin positive subjects (excreting more than 1 000 μg $ß_2$-M/g creatinine) of both sexes were higher that those of the general Japanese population. Mortality rates increased in proportion to increases in the amount of, $ß_2$-microglobulin excretion. These results suggest that the prognosis of cadmium exposed subjects with proximal tubular dysfunction is unfavourable. The mortality rate tended to increase as the severity of renal dysfunction progressed. These results were confirmed by a fifteen year follow-up study by Nishijo et al. (1995). The observed increases of mortality are due to heart failure and renal diseases.

In these Japanese studies, higher mortality rate was observed when the urinary excretion of $ß_2$-M was above 300 μg/g creatinine. Among the environmentally exposed people of the Cadmibel study, urinary $ß_2$-M of 286 μg/24 h (corresponding 203 μg/g creatinine) was exceeded by 25% in the high exposure group (n=331) (Staessen 1994). This suggests that for a part of the residents of polluted areas in Belgium, the cadmium exposure is at a level associated with increased mortality in the cited Japanese studies.

It remains to elucidated whether also in contaminated areas and in risk groups in other countries of Europe, cadmium exposure is associated with higher mortality due to renal diseases. It is possible that, in the studies cited above all the confounders were not considered. Therefore, our estimate that U-Cd of 4-5 μg/l is associated with increased mortality, is preliminary. This urinary level cadmium and higher have been reported in the general population (non-contaminated areas, no occupational exposure), from Belgium, Germany, Japan and USA (see table 42). The group

Table 42. Health effects of cadmium caused by environmental (non-occupational) exposure and the respective urinary level of cadmium (U-Cd).

HEALTH EFFECTS	Critical urinary level of Cd *	Reference
Renal effects/dysfunction		
Increased urinary excretion of RBP, NAG, β2-M, AA, Ca	1.4-2.8 μg/l	Buchet et al.1990
Increased urinary excretion of albumin, transferrin, β_2-M, NAG, prostanoids, sialic acid	5.4 μg/l **	Roels et al. 1993
Age related renal dysfunction, decline of glomerular filtration rate	10 μg/l	Roels et al. 1993
Decrease of creatinine clearance	2.8-7.0 μg/l	Staessen et al. 1994
Increase of serum creatinine	1.6 μg/l ***	Ewers et al. 1985
Increase urinary excretion of AAP, Alkaline phosphatase, NAG, a1-microglobulin	4.0 μg/l	Jung et al. 1993
Increased urinary excretion of NAG	3 μg/l	Kawada et al. 1992
Increased urinary excretion of NAG and AAP	2 μg/l	Muller et al. 1989
Increased urinary excretion of β2-M and albumin	1.62 μg/l	Nordberg et al. 1997
Increased urinary excretion of β2-M	3.8-4.1 μg/l	Ishizaki et al. 1989
Effects on calcium wasting		
Increased urinary excretion of Ca	1.0-5.6 μg/l	Buchet et al.1990
Increased urinary excretion of Ca	0.81 μg/l	Järup (unpublished data)
Stimulation of bone resorption via increased level of prostaglandin E2	2 μg/l	Roels et al. 1993
Decreased level of serum Ca in men	2.8-7.0 μg/l	Staessen et al. 1991

RBP=retinol binding protein; NAG=N-acetyl-b-glucosamidase; β_2 -M = urinary β_2 -microglobulin; AA= urinary aminoacids; Ca= urinary calcium; AAP= alanine aminopeptidase
*) These critical levels apply to groups and not to every individual. When the critical level is reached certain significantly increased number of people will suffer from renal effects or increased excretion of calcium.
**) The study group included also workers.
***) geometric mean.

of population characterised by the high levels of U-Cd is small. Approximates concerning Finland and Sweden are discussed later.

4.3.7 Conclusion on health effects of cadmium

Among the population group having the urinary cadmium of 2-4 μg/l, 1-5% of people will suffer of renal or bone effects caused by cadmium. The renal effects start as increased excretion of high-molecular weight proteins in urine. There is evidence that the renal effects are progressive and that renal failure may follow (e.g. increased serum level of creatinine). Furthermore there is some evidence of increased mortality due to renal diseases associated with high exposure to cadmium. This would apply to a part of the elderly population which has a high body burden of cadmium.

There is some evidence that even at the urinary cadmium level of about 1.0 μg/l, excretion of cadmium will increase, which will decrease the bone density and increase the risk of fractures. These data should be substantiated, before the U-Cd level of about 1.0 μg/l can be used in the risk characterisation of cadmium. We consider, however, that these data emphasise the need for a conservative risk characterisation.

It is our conclusion that urinary Cd level of 2-4 is associated with a risk of serious health effects. Although the evidence on the progression of cadmium induced renal effects and evidence of increased risk of bone fractures is not conclusive, we consider that the recent results have increased the weight of evidence and there is a concern of impairment of public health particularly among the elderly population.

4.4 Risk characterisation

4.4.1 General

In most epidemiological studies, the health effects have been related to urinary levels of cadmium. In some cases also blood cadmium concentration is reported. In very few cases, dietary intake and other sources of exposure have been reported in the epidemiological studies. On the other hand urinary cadmium has often been studied without an epidemiological setting. Therefore, a comparison of exposure and health effects is made using the urinary level of cadmium (μg of Cd/l of urine).

From Table 37 it is concluded that the average level of urinary cadmium in many countries is about 1-1.5 μg/l, which is lower that most of the thresholds presented in Table 42. In Sweden (0.2 μg/l) and most likely also in Finland the average U-Cd is lower. The maximum values of urinary cadmium reported from different countries are at a level of 6 - 8 μg/g. These figures often, but not always reflect the situation of residents of cadmium-polluted areas.

These levels of Cd in urine should be compared with the threshold for early signs of renal effects and the effect on calcium homeostasis, which have been reported when urinary cadmium concentration is 2-4 μg/l (Table 42). This conclusion is supported by recent risk characterisation by Elinder et al. (1998a). They assess that in the general population, an average U-Cd level over 2.5 μg/l is related to an excess prevalence of renal tubular damage. This U-Cd corresponds to cadmium concentration of over 50 mg/kg in renal cortex, which would be result of long-term dietary intake of over 50 μg/day. For smokers and for those having iron deficiency, the critical level of dietary intake would be lower.

It seems likely that a remarkable proportion of the populations studied in various countries, is environmentally exposed to a level of cadmium (2-4 μg Cd/l urine) that causes renal dysfunction as well as calcium wasting, which may induce effects in bones.

Since the range of cadmium body burden (measured as U-Cd) in most studies exceeds the critical level of 2-4 μg/l, it would be of great relevance to clarify the distribution of cadmium exposure in the population, since that would result in estimates concerning the number of people,

who may have the risk of cadmium-induced renal dysfunction or altered calcium homeostasis.

4.4.2 Risk characterisation concerning Finland

Dietary intake

In Finland, the average dietary intake of cadmium in adults is about 10 μg/day (Kumpulainen 1991, Mustaniemi et al. 1994, Louekari et al. 1988). Several studies (Morgan et al. 1988, Louekari et al. 1989, Coomes et al. 1982, Vahter 1991) suggest that for about 5% of the population, the dietary intake of cadmium is at least two-fold more as compared with the average intake, ie. 20 μg/day for Finland.

According to data on distribution of exposure in the Finnish population maximum intakes are about 2.5-fold more as compared with the average (Louekari et al. 1989) (see Figure 15). Data on distribution of cadmium intake in children showed that the 95 percentile was about two-fold more as compared with the average (Mykkänen et al. 1986), which supports the results cited above.

In Finland, cadmium concentrations in the crops play a very important role in the food chains, because the greatest part, i.e. on average 86% of the total Cd taken in by the Finns from the diet originate from plant crops. Sixty per cent of the mean total Cd intake comes from cereals, 9% from potatoes, 9% from vegetables and 8% from fruits and berries (see Figure 16) (Tahvonen 1994).

In Finland, sound agricultural and environmental practices have lowered the amount of Cd cycling in the food chains. As a result, the Cd contents of food stuffs and the Cd intake by the Finnish population from the diets have decreased slightly in Finland (Tahvonen 1994). Decreasing trends in Cd contents of food stuffs have been observed also in Belgium (Van Assche and Ciarletta 1993) and in the United Kingdom. In these two countries the major reason for a decreasing trend is obviously a decrease in Cd deposition from the air.

On the other hand, there is a serious Cd trend in Swedish winter wheat, because at present about 10% of winter wheat grains produced in Sweden contain a level of Cd which is more than 100 μg/kg (Eriksson et al. 1995) which is the highest acceptable concentration for cereal grains as set by FAO/WHO in 1993.

Urinary Cd-concentration corresponding the average dietary intake in Finland

In the non-occupationally exposed population, according to reports from Sweden and USA, the cadmium concentration in urine is about 0.2-0.6 μg/l (see table 37). This is in agreement with reference values for the general population proposed by Nordberg (1992), 0.7 μg/l which is also applied in Finland. In Sweden and in the USA, the level of dietary intake of cadmium is likely to be close to that of Finland (10 μg/day). In Finland, Sweden and USA contamination of agricultural soils by cadmium is not at the same level as in some European countries, where the average dietary intake is also elevated. Limited number of urine samples from non-occupationally exposed non-smokers have normally contained less than 0.5 μg Cd/l in Finland (Kiilunen unpublished data) It is assumed for this evaluation that the average urinary level of cadmium in Finland is 0.4 μg/l. This is a basis for a conservative risk assessment, which needs to be confirmed or refuted by an adequate analytical study with sufficient number of non-occupationally exposed subjects. The high dietary intake of 20-30 μg Cd/day would thus correspond to 0.8-1.2 μg/l of urinary concentration of cadmium (see table 43).

Increased absorption

Flanagan et al. (1978) showed that women with low body iron stores had on average two-fold higher gastrointestinal absorption (10%) of cadmium as compared with the control group. The highest individual absorption rate was about

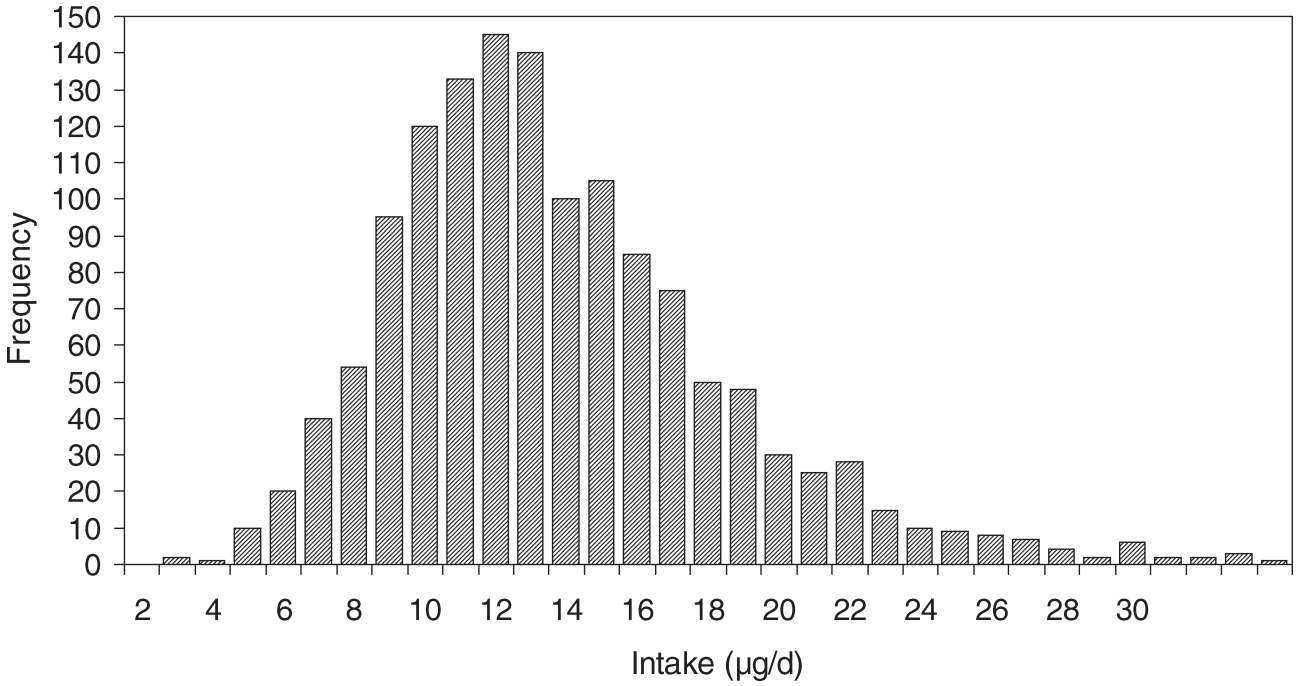

Figure 15. Distribution of dietary intake of cadmium among 1348 Finnish individuals aged between 25-64 years, Based on a 3-day food record and analytical data on food samples (Louekari et al. 1989).

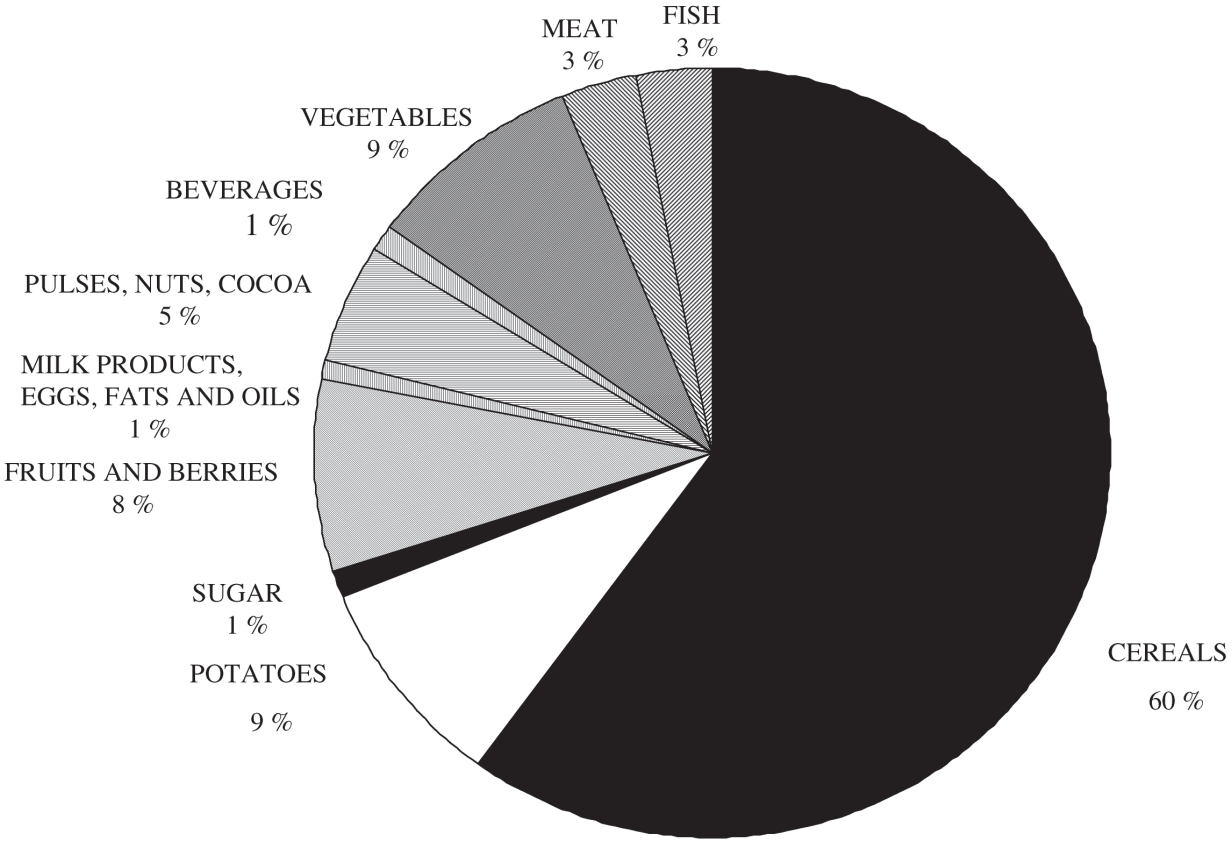

Figure 16. Intake of cadmium from different food groups (Tahvonen 1994).

20%. In experimental animals Friberg et al. (1975) observed a three-fold increase in absorption of cadmium due to low levels of dietary calcium and protein. Kowal (1988) also found in a large nutritional survey (NHANES II) that urinary Cd was negatively and significantly correlated with dietary iron and dietary calcium. Also Berglund et al. (1994) found that reduced body iron stores (serum ferritin) were highly associated with higher B-Cd concentrations. Bäcklund et al. (1996) found that women 50-55 years of age had higher B-Cd levels (0.5 μg/l) than men of the same age (0.3 μg/l). The difference is probably caused by increased absorption of dietary cadmium in women with low iron status before menopause. Berglund et al. (1998) has assessed that 175 000-700 000 women in Sweden would have empty iron stores and consequently increased B-Cd levels, by about factor of 2. Furthermore, they estimated that, in Sweden about 250 000 smoking women with low iron stores are likely to have 4-6 times the kidney cadmium concentration as compared with non-smokers with adequate iron stores.

This indicates that low intakes of iron and calcium can cause increased absorption of cadmium and thus consequently increased body burden and excretion. Although no quantitative estimates of increased absorption of cadmium in Finland are available, it is assumed that for the iron-deficient female sub-population the absorbed amount of cadmium is two-fold higher when compared with the average. Consequently, for those having a high dietary intake and increased absorption, the urinary levels of cadmium would be 1.6-2.4 μg/l.

Smoking

Heavy smoking increases the U-Cd remarkably. In Sweden it has been observed that the Cd-concentration in kidneys reflecting the long-term exposure is about 2-3 fold higher for smokers than for non-smokers (Elinder et al. 1976, Nilsson et al. 1995). Luoma et al. found that blood cadmium level of smokers was three times higher than in non-smokers. Based on the above data and review on blood and urinary cadmium levels by Berglund et al. (1998) it is estimated that U-Cd of heavy smokers is about 2.5-fold as compared non-smokers with average dietary intake of cadmium. This implies that U-Cd of heavy smokers is about 1.0 μg/l. For heavy smokers, who have a high dietary intake of cadmium the urinary level of cadmium would be about 1.4-1.8 μg/l (see Table 43).

This is in agreement with results of an epidemiological study, where food consumption and smoking data were used to calculate the total absorbed daily amount of cadmium. It was found that the maximum level of absorbed cadmium was about five-fold more as compared with that caused by the average dietary intake (Figure 17).

Total exposure and body burden of cadmium

Three main factors contribute to the body burden of cadmium, namely the dietary intake with remarkable variation, increased absorption caused by low dietary iron and possibly also dietary calcium, and smoking. The effect of these risk factors is also demonstrated by the ranges of the urinary cadmium level (a measure of cadmium body burden) in table 37, which shows that the maximum levels of urinary cadmium often are several times higher than the average. This is especially the case in studies where the number of people studied is relatively great (>100 individuals).

The concentration of cadmium in renal cortex is another indicator of total exposure and the accumulation of cadmium. Ala-Opas et al. (1995) found that in the renal cancer patients (n=13) cadmium concentration in kidney cortex in women and men were 9.4 mg/kg (3.4-14.0) and 14.7 (3.2-21.2), respectively. These values are low as compared with the critical concentration of 50 mg/kg. Since the number of samples is very small the wide variation of total exposure could not be observed in this study. Elinder et al. (1976) studied samples of victims of sudden death and found that among 292 persons in Stockholm the kidney cortex of non-smokers and smokers contained 11 and 23 mg Cd/kg, respectively, and that about 10% of subjects had concentration above 50 mg/kg.

Considering all the three risk factors, it is assessed that for heavy smokers who have a high dietary intake of cadmium and increased absorption, the urinary level of cadmium would be about 2.2-3.0 μg/l.

4.4.3 Conclusions on risk characterisation concerning Finnish population.

From the table 43, it is obvious that the cadmium-exposure of the vast majority of the Finnish population (on the average 0.4 μg/l urinary cadmium for non-smokers) is below critical level, which is 2-4 μg/l.

The Joint FAO/WHO Expert Committee on Food Additives (JECFA), which also considers food contaminants, has scheduled the Provisional Tolerable Weekly Intake (PTWI) of cadmium for re-evaluation. It seems probable that the current PTWI value (60-70 μg/day) will be lowered. It is noteworthy that Elinder et al. (1998b) estimated that the urinary cadmium level of 2.5 μg/l corresponds to a dietary intake of about 50 μg/day.

In table 43, exposure to cadmium in the general population is presented as well as exposure in some risk groups. A realistic worst case scenario is that some parts of the population are attributed to all three risk factors specified above and in table 43. For those individuals, the body burden and urinary cadmium concentration (2.2-3.0 μg/l) exceeds many of the critical concentrations specified in table 42. Therefore, it is probable that this sub-population suffers a risk of renal effects (increased urinary excretion of several indicator proteins and enzymes, symptoms which may later develop into renal dysfunction) and calcium wasting caused by cadmium. In Finland, the size of all the sub-populations has not been studied.

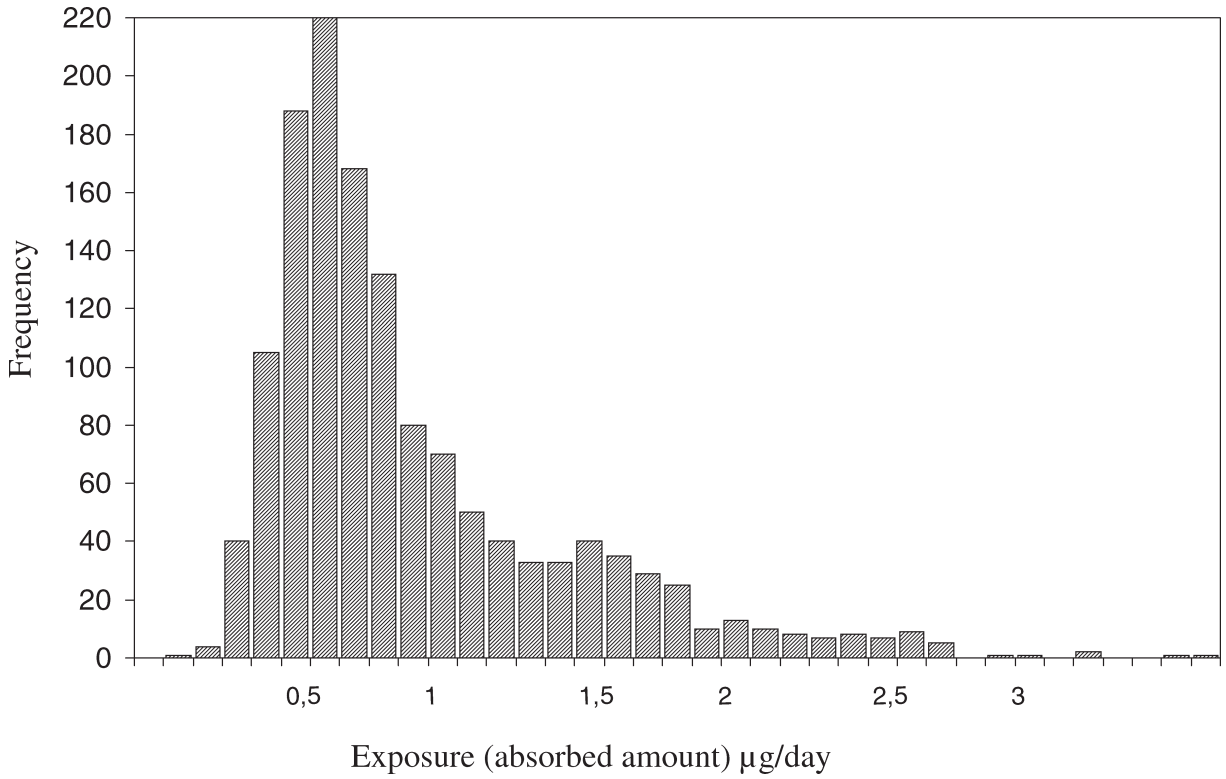

Figure 17. Distribution of total cadmium exposure among 1348 individuals, includes the intake from food (about 5% is absorbed) and smoking (about 25% absorbed) (Louekari et al. 1989).

The figures presented in Table 43 indicate that the size of each specified risk group (smokers, those with iron deficiency and those having high dietary intake) is large. It is likely that one risk factor does not cause that the critical dose level is reached. It is estimated that the "worst case scenario" applies to some thousands of individuals, at the most. This is in accordance with the assessment of Elinder et al. (1998a), in which it is estimated that adverse renal effects (tubular damage) will develop in several thousands people. Furthermore, according to Elinder et al (1998a) in case the average daily dietary intake would increased from 15 to 30 μg/day, the prevalence of tubular damage would be 100 000 persons in Sweden.

Elinder et al. (1998a) have assessed that doubling of the dietary intake of cadmium (from 15 to 30 μg/day) "will give rise to cadmium-induced renal tubular damage in about 1% of the general population". In risk groups (women with low iron stores) the percentage would be up to 5% corresponding to about 100 000. The predicted increase of the dietary intake of cadmium in Finland - in 100 years caused by fertilizers which contain 138 mg of Cd/kg of P - would be from 9.5 to 13.0 μg/day (see Table 43). It is assessed that due to this increased dietary intake of cadmium, the size of risk group would increase to about 5 000-10 000 persons in Finland. This estimate should be regarded as preliminary, since there is a lack of data on the urinary level of cadmium in Finland and because the size of the all the risk groups has not been studied. In the light of recent epidemiological evidence of bone effects, i.e. increased prevalence of osteoporosis associated with renal effects and potential increased renal mortality due to high cadmium exposure, this scenario causes a concern and should be prevented.

Table 43. Exposure to cadmium in Finland and corresponding urinary levels of cadmium

Sources of exposure and risk factors	**Corresponding urinary Cd-concentration (μg/l)**	**Size of (sub-)population in Finland**
Average dietary intake 10 μg/day	0.4	< 5 000 000
High dietary intake 20-30 μg/day	0.8-1.2	250 000
Average dietary intake + smoking	1.0	180 000
Average dietary intake + increased absorption	0.8	100 000 – 400 000
High dietary intake + increased absorption 20-30 μg/day with two-fold higher absorption	1.6-2.4	?
High dietary intake + smoking	1.4-1.8	?
Smoking + increased absorption	About 1.4	
High dietary intake + increased absorption + smoking	2.2-3.0	?

4.5 Conclusions

The three main factors of total cadmium exposure: high dietary intake, increased absorption and smoking are considered for risk assessment in this report. The corresponding urinary levels of cadmium and some of the critical urinary levels that are associated with health effects are compared in Table 44. From that table, it can be concluded that for high exposure groups (ie. in worst cases), the urinary Cd-level is about 2-3 μg/l. This is also the level of urinary excretion of cadmium in those sub-populations which are at risk of cadmium-induced kidney dysfunction. Early signs of kidney dysfunction and increased excretion of calcium in urine (calcium wasting) have both been observed when urinary Cd is about 2-3 μg/day.

Based on the present risk assessment of cadmium, which was made according to Technical Guidance Document and the ERM recommendations submitted by the Commission of the EU, in Finland a part of the elderly population is at risk from adverse health effects caused by cadmium. These pre-clinical effects should be considered adverse, since there is evidence that even a mild kidney dysfunction caused by cadmium is not reversible, and moreover, it progresses after cessation of exposure. Also there is some indication that mortality in sub-populations environmentally exposed to cadmium has been increased. The effect of cadmium on the calcium metabolism is considered to be serious since cadmium exposure might enhance calcium deficiency, osteoporosis and thus increase the risk of fractures which impair the health of old people.

Table 44. Comparison of estimated urinary levels of cadmium in Finland with the critical urinary concentrations in the middle-aged and elderly population (see also Tables 40, 41 and 43).

Risk Group/ Exposure	Corresponding urinary Cd-concentration (μg/l)	Size of (sub-)population in Finland	Increased risk of health effects Specified below
High dietary intake	0.8-1.2	250 000	None
High dietary intake + increased absorption (20-30 μg/day with two-fold absorption)	1.6-2.4	?	Increased urinary excretion of RBP, NAG, β_2-M, AA, Ca Stimulation of bone resorption via increased level of prostaglandin E2
High dietary intake + increased absorption + smoking	2.2-3.0	?	Increased urinary excretion of RBP, NAG, β_2-M, AA, Ca Decrease of creatinine clearance Increased urinary excretion of AAP, Alkaline phosphatase, a1-microglobulin Stimulation of bone resorption via increased level of prostaglandin E2 Decreased level of serum Ca in men

RBP=urinary excretion of retinol binding protein; NAG=urinary excretion of N-acetyl-b-glucosamidase, β_2-M=urinary β_2-microglobulin, AA=urinary aminoacids, Ca=urinary calcium, AAP=alanine aminopeptidase;
***) these critical levels often apply to groups and not directly to individuals, since the results have been gained by testing the significance of a difference between two or more groups with certain levels of urinary cadmium.**

In Finland, for the risk groups (reasonable worst cases) there is no margin of safety between the estimated urinary levels and the critical levels that have been associated with the health effects caused by cadmium. It is concluded that for these sub-groups of the population, risk reduction measures are necessary. It is estimated that the use of P-fertilizers, which contain 138 mg Cd/kg of P for 100 years would increase the average dietary intake of cadmium in Finland from 9.5 to 13.0 μg/day. Due to distribution of cadmium exposure in the population, this would cause that the size of population groups at risk will grow several times greater.

As compared to many other risk assessments, the present assessment is based on extensive human and epidemiological data and not only on toxicological experiments with animals. Therefore, the results can be considered to be very relevant in terms of human populations.

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