



DIRECTORATE-GENERAL FOR INTERNAL POLICIES

POLICY DEPARTMENT **A** ECONOMIC AND SCIENTIFIC POLICY

Economic and Monetary Affairs

Employment and Social Affairs

Environment, Public Health and Food Safety

Industry, Research and Energy

**Internal Market and
Consumer Protection**

Scientific aspects underlying the regulatory framework in the area of fertilisers - state of play and future reforms

In-Depth Analysis for the IMCO Committee



DIRECTORATE GENERAL FOR INTERNAL POLICIES
POLICY DEPARTMENT A: ECONOMIC AND SCIENTIFIC POLICY

Scientific aspects underlying the regulatory framework in the area of fertilisers – state of play and future reforms

IN-DEPTH ANALYSIS

Abstract

This document was prepared by Policy Department A at the request of the Committee on the Internal Market and Consumer Protection (IMCO) on the proposed cadmium regulation for phosphate fertilisers. Cadmium is a metal that can have adverse health effects on the general population. The use of mineral phosphate fertilisers contributes to about 60% of current cadmium emissions to soil. The proposed regulation aims to reduce soil and crop cadmium concentrations on the long term in most European regions.

This document was requested by the European Parliament's Committee on the Internal Market and Consumer Protection (IMCO).

AUTHOR(S)

Prof. Dr Erik SMOLDERS (Katholieke Universiteit Leuven, Belgium)

RESPONSIBLE ADMINISTRATOR

Mariusz MACIEJEWSKI

EDITORIAL ASSISTANT

Andreea STOIAN

LINGUISTIC VERSIONS

Original: EN

ABOUT THE EDITOR

Policy departments provide in-house and external expertise to support EP committees and other parliamentary bodies in shaping legislation and exercising democratic scrutiny over EU internal policies.

To contact Policy Department A or to subscribe to its newsletter please write to:

Policy Department A: Economic and Scientific Policy

European Parliament

B-1047 Brussels

E-mail: Poldep-Economy-Science@ep.europa.eu

Manuscript completed in December 2016

© European Union, 2017

This document is available on the Internet at:

<http://www.europarl.europa.eu/studies>

DISCLAIMER

The opinions expressed in this document are the sole responsibility of the author and do not necessarily represent the official position of the European Parliament.

Reproduction and translation for non-commercial purposes are authorised, provided the source is acknowledged and the publisher is given prior notice and sent a copy.

CONTENTS

LIST OF ABBREVIATIONS	4
LIST OF FIGURES	4
LIST OF TABLES	5
EXECUTIVE SUMMARY	6
1. THE BACKGROUND OF THE CADMIUM REGULATION IN PHOSPHATE FERTILISERS	8
2. ENVIRONMENTAL CADMIUM: OCCURRENCE, EFFECTS AND RISK ASSESSMENTS IN EUROPE	10
2.1. Occurrence of cadmium in the environment	10
2.1.1. The geological (natural) contribution	10
2.1.2. The transfer of cadmium from soil to the food chain and human exposure to environmental cadmium	11
2.2. Adverse effects of cadmium in the general population	12
2.3. Risk assessment of environmental cadmium in the general population of Europe	12
3. TRENDS IN CADMIUM EXPOSURE VIA THE ENVIRONMENT AND THE ROLE OF PHOSPHATE FERTILISERS	16
3.1. Historical records	16
3.2. The future: current emissions from all sources, including fertilisers, and projections based on the mass balance	18
3.2.1. The concentration of cadmium in phosphate fertilisers on the EU market	18
3.2.2. The soil cadmium mass balance in Europe	19
4. FUTURE TRENDS IN SOIL CADMIUM AS AFFECTED BY CADMIUM CONCENTRATION IN PHOSPHATE FERTILISERS	22
4.1. A mass balance estimate of the trends as affected by the fertiliser cadmium concentrations	22
4.2. Fertiliser cadmium does not affect crop cadmium concentrations within the first years after application	24
5. RECOMMENDATIONS	25
REFERENCES	27

LIST OF ABBREVIATIONS

Cd	cadmium
ALDE	European Chemical Bureau
EFSA	European Food Safety Administration
P₂O₅	phosphorus pentoxide
P	phosphorus

LIST OF FIGURES

- Figure 1: Risk assessment of cadmium is made at various levels in the chain between the fertilisers and humans. The accuracy and precision of the risk assessment is highest based on body burden, i.e. using biomonitoring, and lowest when recalculated to emissions, i.e. fertilisers for the reasons shown 13
- Figure 2: The first experimental evidence reported that long-term use of phosphate fertilisers increase soil Cd. The soil Cd increases with cumulative application of P-fertilisers in experimental plots in Australia. The HCl soluble Cd increases from 0.03 to 0.06 mg/kg soil. Reprinted with permission [17] 17
- Figure 3: Soil Cd has increased during 120 years in untreated arable soils of a research station in Rothamsted, UK. Filled symbols denote observation in archived soils, open symbols are modelled values using a mass balance approach. These plots did not receive phosphate and the increase is ascribed to atmospheric deposition; The corresponding phosphate treated plots showed similar trends, i.e. the Cd input from phosphate may have been too small to detect statistically. In contrast, there was a clearly larger Cd accumulation in the phosphate treated permanent grassland than in the corresponding untreated plots. Reprinted with permission from [20]. 17
- Figure 4: The Cd concentrations in mineral fertilizers on a P₂O₅ basis (mg/kg P₂O₅) according to country of origin. The fertiliser had been sampled in 2014. Box plots show interquartiles, outliers are not shown (n=414). 18
- Figure 5: The soil cadmium mass balance in Europe. Table 4 lists the current (2016) estimated average values for EU28. 21
- Figure 6: The predicted change in soil Cd after 100 years of emissions using different Cd concentrations in phosphate fertilizers. Means are circles, the ranges is the 10-90th percentile of scenarios (soils) in Europe, the 90th percentiles were calculated at a realistic worst case of an almost threefold larger than average fertiliser use combined with higher use of sewage sludge in the same soils. 23

LIST OF TABLES

Table 1:	Occurrence of cadmium in the European environment.	11
Table 2:	Cadmium thresholds below used in various international risk assessments below which adverse health effects are not be expected. Values in bold are those that have been used in the two most recent European risk assessments.	14
Table 3:	Descriptive statistics of the Cd concentration in mineral fertilisers on a P ₂ O ₅ basis (mg/kg P ₂ O ₅) in current and a previous EU survey.	19
Table 4:	Fluxes of Cd considered in the mass balances of cadmium in the average of European agricultural (arable) soils. Adapted from [19] and refined in the final update in reactions to comments of the SCHER committee in Smolders, 2016.	21
Table 5:	Predicted change in soil Cd in the average EU scenario (top) and in worst case scenarios (bottom) according to the updated parameters set and requests by SCHER	23

EXECUTIVE SUMMARY

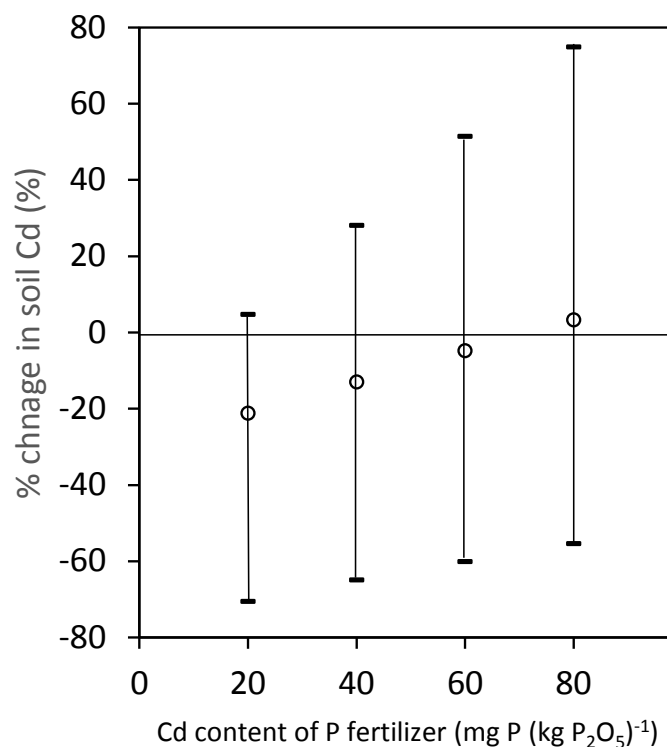
This document gives an in-depth analysis of the proposed cadmium regulation in phosphate fertilisers in Europe in the proposal for a new Fertilising Products Regulation No 1069/2009 and (EC) No 1107/2009; 17.3.2016; COM(2016) 157 final. Two extensive risk assessments in Europe have concluded that there is a need to limit risk of cadmium to the general population and that the current exposure to cadmium at the population level should be reduced. The cadmium regulation in fertilisers defines three concentrations (60, 40 and 20 mg Cd/kg P_2O_5) that are proposed to be adopted stepwise. These concentrations have been developed to limit further increasing cadmium exposure on the long term in Europe.

Cadmium is a naturally occurring element found in all soils, waters, crops, food items and humans, even in the most remote and pristine environments. Phosphate fertilisers contain cadmium because of its natural, but variable, occurrence in the rock phosphates, the ores used for production of the fertilisers. Rock phosphate sources originating from northern Africa have large cadmium concentrations contrasting the low cadmium rock phosphates from Kola (Russia). Both sources are used in Europe and explain the large regional variation in fertiliser cadmium concentrations across Europe. A recent fertiliser survey indicates that the fractions exceeding the proposed limits are 8% at the least stringent limit (60 mg Cd/kg P_2O_5) to 56% at the most stringent one (20 mg Cd/kg P_2O_5). The removal of cadmium from the fertilisers is economically not feasible at this stage.

The soil cadmium concentrations in European soils have increased by about 50% since phosphate fertilisers were ever adopted more than a century ago. Current cadmium emissions to the agricultural soils are strongly reduced from historic ones, but still about 60% of these emissions are derived from the application of mineral phosphate fertilisers. The current annual doses of cadmium via phosphate fertiliser are only about 1% of the total available cadmium in soil. These are small additions, however multiplied with several decades, the input is considerable.

Cadmium is a toxic metal that may cause a range of effects, the critical target organs for cadmium in the general population are the kidney and the bone. Epidemiological and experimental studies have led to the derivation of low effect levels of cadmium below which adverse health effects are not expected. Two extensive European risk assessments (2007 and 2009) have concluded that there is a significant fraction of the population for which the cadmium exposure exceeds these thresholds. At the same time, these assessments suggest that the adverse health effects are not large and more recent biomonitoring data confirm that only minor fractions of the non-smoking population exceed the exposure limits.

The cadmium regulation in phosphate fertilisers has been calculated to reduce cadmium exposure of the general population, it is not based on a risk assessment given the uncertainties surrounding the low level risks. The current *average* cadmium balance in European arable soils is a negative one, i.e. the average soil cadmium is predicted to decrease because the annual cadmium losses by natural leaching and uptake by the crop are now just larger than the small annual additions to the topsoil, obviously leaving some regions where the soil cadmium balance is still positive. The soil cadmium balance was calculated as a function of the fertiliser cadmium concentration, indicating the trends in soil cadmium, both at the average situation as for the regional variability, this is depicted in the figure below. This figure gives the predicted change in soil Cd after 100 years of emissions using four different Cd concentrations in phosphate fertilizers, the proposed limits in the fertiliser regulation are the three lower ones. Means are circles, the ranges is the 10-90th percentile of scenarios (soils) in Europe, the 90th percentiles were calculated at a realistic worst case of an almost threefold larger than average fertiliser use combined with higher use of sewage sludge in the same soils.



The three cadmium levels that are proposed in the fertiliser regulation are expected to all lead to a decrease in the EU *average* soil cadmium concentrations, only the most stringent one is expected to also ensure a depleting soil cadmium in about 90% of the European agricultural soils. The more conservative option is to directly select the most stringent, lower fertiliser threshold (20 mg Cd/kg P₂O₅) which leads to no further increase in soil cadmium in almost 90% of the EU soils and is associated with an overall negative EU wide cadmium balance. In contrast, the larger threshold is affected by lower conservatism based on a stand still principle in the average situation. The arguments for the latter option is the general knowledge that the lifetime cadmium intake is important for cadmium related health risks. Indeed, what almost all Europeans eat in their lifetime, is a mixture of food items that were grown in various places. When the average soil cadmium is in stand still, so is the expected human exposure in a completely mixed assumption. There is obviously no complete mixing of food items and, therefore, some conservatism is justified which, unfortunately, cannot be calculated because the extent of mixing cannot be estimated for the next 100 years to come. This means the three proposed limits (20-40-60 mg Cd/kg P₂O₅) bracket a spectrum of various degrees of conservatism within an uncertainty range, but all leading to a reduction of cadmium exposure in the future. With increasingly stringent limits, an increasingly larger fraction of EU soils will have a negative cadmium balance. The author of this document suggests that the deliberation of the thresholds should consider the cost-benefit balance, i.e. the % of the market affected by the limits versus the magnitude of the current human health risk of cadmium, the latter cannot be excluded but is a low one as indicated by the most recent high quality bio monitoring data of Europe and referred to in this document.

1. THE BACKGROUND OF THE CADMIUM REGULATION IN PHOSPHATE FERTILISERS

Key findings

- Two extensive risk assessments in Europe have concluded that there is a need to limit risk of cadmium to the general population [1] and that the current exposure to cadmium at the population level should be reduced [2].
- The proposed cadmium limits in phosphate fertilisers have been calculated to limit the exposure to cadmium on the long term. The regulation are only based on an exposure assessment, not on risk assessment.

This paper provides an in depth analysis of the scientific background of the proposed cadmium limits for CE marked phosphate fertilisers in the proposal for a new Fertilising Products Regulation No 1069/2009 and (EC) No 1107/2009; 17.3.2016; COM(2016) 157 final.

The proposed regulation on cadmium (Cd) stipulates the following quantities:

1. Where the CE marked fertilising product has a total phosphorus (P) content of less than 5 % phosphorus pentoxide (P_2O_5)-equivalent by mass: 3 mg Cd/kg dry matter, or
2. Where the CE marked fertilising product has a total phosphorus (P) content of 5 % phosphorus pentoxide (P_2O_5)-equivalent or more by mass ('phosphate fertiliser'):
 - a. As of the date of application of this Regulation: 60 mg/kg phosphorus pentoxide (P_2O_5)
 - b. As of three years after the date of application of this Regulation: 40 mg/kg phosphorus pentoxide (P_2O_5)
 - c. As of twelve years after the date of application of this Regulation: 20 mg/kg phosphorus pentoxide (P_2O_5)

Cadmium is a naturally occurring trace metal that causes human health effects at relatively low concentrations. Phosphate fertilisers have been a main source for widespread gradual enrichments of soils and food with trace levels of Cd in Europe. Several risk assessments on Cd in Europe have concluded that there is currently a risk of Cd in the general population of Europe. The main risk assessments are:

1. The European risk assessment of CdO and Cd metal in 2007 [1] performed under Council Regulation (EEC) 793/93 on the evaluation and control of risk of the existing substances. This was performed by the European Chemical Bureau (ECB, predecessor of the European Chemicals Agency, ECHA). The conclusion for risk to humans exposed via the environment was, "*Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.*" That conclusion was reached because "*at the mentioned exposure levels, health risks cannot be excluded upon environmental exposure (kidney and bone (all scenarios except adult non-smokers with sufficient iron stores) and lung (a specific scenario) repeated dose toxicity, carcinogenicity/genotoxicity for all scenarios)*". That risk assessment of 2007 was based on data collected until 2005.
2. In 2009, the European Food Safety Authority (EFSA) published the scientific opinion of the panel on contaminants in the food chain [2]. That panel evaluated the current

and tolerable Cd intake via the diet in Europe and concluded that *“although the risk for adverse effects on kidney function at an individual level at dietary exposures across Europe is very low, the Panel concluded that the current exposure to Cd at the population level should be reduced.”*

More details on the risk assessments are given below and will illustrate that risks are generally marginal or low and that there are diverging opinions when contrasted with international literature. It will be shown that the relationship between fertiliser Cd concentrations and the low level human health risk cannot be precisely calculated because of the variability in the exposure among soils, climates, crops, dietary habits and nutritional status of the consumers.

Because of this variability, the proposed regulations (i.e. concentrations) on Cd in fertiliser have been calculated to limit the long-term exposure, not on limiting the risk.

2. ENVIRONMENTAL CADMIUM: OCCURRENCE, EFFECTS AND RISK ASSESSMENTS IN EUROPE

Key findings

1. Cadmium is a naturally occurring element in the environment, it is persistent and not degradable.
2. Smoking is the single most important source of Cd in the general population.
3. For non-smokers, the diet and, mainly, grain products and potatoes, are major sources of Cd.
4. Cadmium related kidney and bone diseases has led to mortality in populations living near point sources of Cd outside Europe.
5. The most robust and recent human health risk assessment of Cd based on biomonitoring indicates that Cd exposure exceeds health limits in a small fraction of the general, non-smoking population.

2.1. Occurrence of cadmium in the environment

2.1.1. The geological (natural) contribution

Cadmium is a non-essential metal that is naturally present in the environment, even in the most pristine or remote ones. With current analytical facilities, it is well detectable in almost any environmental samples, e.g. rocks, soils, surface and rain water, plants and humans (Table 1). The concentrations of Cd are typically in the parts per billion or parts per trillion (mass based parts) concentration range, i.e. trace levels. Older environmental concentration data always need to be treated with caution but currently there are numerous data to reliably identify Cd occurrence in the environment. Cadmium also naturally occurs in rock phosphate, the primary material used to make mineral phosphate (P) fertilisers (section 3.4.1). The long-term use of mineral P fertilisers has enriched the agricultural soils with Cd as will be outlined below.

There are only a few specific Cd minerals that naturally occur in the environment whereas it more commonly occur in zinc (Zn) minerals. Soil Cd concentrations (Table 1) are typically found in the 0.1-1.0 mg/kg range and the average or median values of different large scale surveys range 0.1-0.2 mg Cd/kg (Table 1). European soil Cd concentrations are relatively well documented with EU-wide surveys that became available in the last decade (Table 1). The soil Cd concentrations vary largely with the underlying geology [3]. Soil Cd concentrations exceeding 1 mg Cd/kg may occur naturally, for example in soils developed on shales or in organic soils [4]. The natural variation of soil Cd (0.1-1.0 mg/kg) is larger than the long-term increase due to atmospheric deposition and phosphate ranging 0.1-0.3 mg Cd/kg, see chapter 3. This means that the natural variation is larger than the variation due to the enrichment from the historical emissions.

Table 1: Occurrence of cadmium in the European environment.

	descriptors\$	unit	reference
Rocks		mg Cd/kg	[5]
igneous		0.07-0.25	
sedimentary		0.01-2.60	
Soils		mg Cd/kg	
all land uses	min-mean-max	0.02-0.09-3.2	[6]
natural soils	P10-P50-P90	0.05-0.14-0.48	[7]
agricultural soils (arable)	P10-mean-P90	0.06-0.0.25-0.45	[8]
Water		µg/L	
streams	min-P50-P90	<0.002-0.01-0.053	[7]
bottled mineral water	P5-P50-P95	<0.003-0.003-0.037	[8]
Crops		µg/kg fresh product	
wheat grain	means/medians	22-80	[1]
potatoes	mean	23	[9]
	means/medians	15-32	[1]
Humans		µg/g creatinine	
urine: mothers	GM-P95	0.18-0.54	[10]

\$P50=50th percentile, P5= 5th percentile etc. ; GM=geometric mean

2.1.2. The transfer of cadmium from soil to the food chain and human exposure to environmental cadmium

Cadmium occurs in the environment as a divalent cation that can be taken up by biota. This means that this element can be transferred from soils and waters into the human food chains, with typically a larger importance of the terrestrial parts (food crops) than for the aquatic environment (fish) in the human diet. At identical total soil Cd concentrations, crop Cd varies typically >10-fold because the availability of this contaminant is strongly depending on soil, climate and crops [11]. Agricultural products that exceed food Cd limits are often durum wheat, sunflower kernels, cocoa products and peanuts. The evidence on long-term reactions of Cd in soil suggests that Cd remains available and is not irreversible bound. Isotope dilutions studies have shown that the indigenous soil Cd is, for most soils, equally available to plants as freshly added Cd [12]. This practically means that, unlike biodegradable organic contaminants, Cd remains as a persistent and available element in most soils. The evidence also means that increasing total soil Cd concentrations due to atmospheric deposition or use of phosphate fertilisers is associated with an almost proportional increase in crop Cd concentrations, all other factors being constant.

Smoking and dietary Cd are the main pathways of Cd exposure in uncontaminated areas. Tobacco plants naturally contain high Cd concentrations in leaves, the amount varying

considerably with the origin of the tobacco. This is readily detected in human bio monitoring for which smokers have markedly larger Cd body burden than non-smokers. Dietary intake is the major source of Cd exposure for the non-smoking general population. Levels of Cd in food items are typically high in offal, organs, equine products, shellfish, crustacean, cocoa, mushrooms and some leafy vegetables. The incidence of these products in the average dietary Cd intake is low because of their low average consumption. In contrast, grain and potato Cd concentrations have received considerable attention because of their larger impact on the dietary Cd intake by humans. For example, a daily consumption wheat grain products that only contain trace Cd concentrations contributes more to the Cd intake than a daily portion of leafy vegetables that have high Cd concentration because of the larger total intake by mass of the former. In addition, the chronic (lifetime) exposure matters far more than single high intake events because Cd has a long biological half-life of 15-20 years in humans and the effects manifest themselves mainly in older people (>50 years). This means that a rare consumption of a high Cd containing food item (e.g. spinach) grown in a local Cd contaminated soil has less effect than the lifetime consumption of moderately contaminated food. For these reasons, risk of point soil Cd contaminations may be low if there is sufficient 'dilution' with food items grown elsewhere.

2.2. Adverse effects of cadmium in the general population

The identification of the Cd toxicity to humans was already discovered before the end of the 19th century but it was not until the 1960s before risk of Cd in the environment became clear when an endemic bone disease in farmers of the Toyama prefecture (Japan) was related to Cd in soil [13]. The so-called *itai-itai* disease is a bone disease with fractures and severe pain that was identified in the early 1950s in that prefecture. Patients were mainly women who delivered several children and who consumed locally grown rice. The rice paddies (1-10 mg Cd/kg soil) had been irrigated with Cd contaminated water of a river that drained a metal mining area. The chronic Cd intoxication induced an accumulation of Cd in the kidneys and induced as disease (tubular dysfunction) leading to increased excretion of calcium and of low molecular weight proteins and finally to softening of the bones. In the Jinzu basin, 188 cases of this disease were officially recognized [13]. The current understanding of the *itai-itai* disease shows that the cumulative (life-time) exposure is the best index for estimating the effects and that the intake was obtained by a combination of a large daily rice consumption with highly contaminated rice.

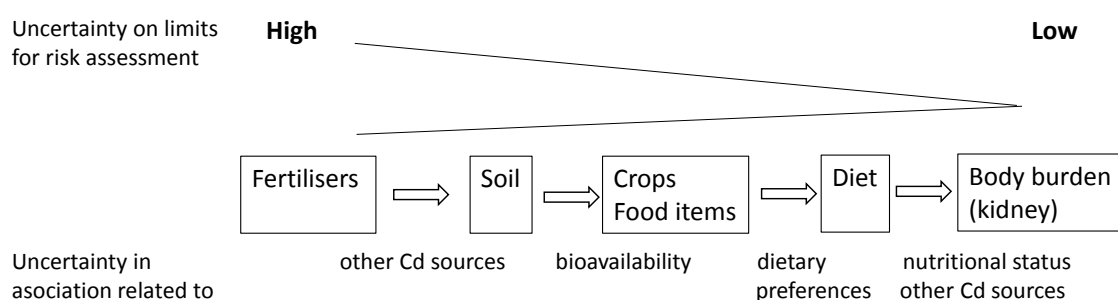
Cadmium is a toxic metal that may cause a range of effects. It is classified as a human carcinogen and there is some evidence for cardiovascular effects [14]. However, the weight of evidence suggest that the critical target organs for Cd in the general population are the kidney and the bone [1, 14]. Epidemiological and experimental studies have led to the derivation of low effect levels of Cd above which health effects start to occur. These effects levels are expressed using kidney cortex Cd or, now more generally, urinary Cd concentrations.

2.3. Risk assessment of environmental cadmium in the general population of Europe

There have been numerous risk assessments on Cd in the general population. Before illustrating the divergence in thresholds used, it is imperative to illustrate the complexity of environmental risk assessment in general and for Cd in particular. Risk assessments have

been made based on body-burden thresholds, dietary threshold, food standards, soil standards and finally standards for fertilisers and soil amendments. I postulate here (see Figure 1) that the uncertainty surrounding the accuracy of these standards to predict risk is smallest for the body burden (right hand side of Figure 1) and increasing towards the sources (left hand side). The main reason for this is that there are other Cd sources than indicated in the scheme and that there is variability in the transfers of environmental Cd between the compartments indicated by the boxes. This requires assumption for which safety factors are typically included. The accumulation of safety factors when going from the target organ (right) towards the environmental emissions (left), typically results in increased *theoretical* risk when confronted with the exposure data.

Figure 1: Risk assessment of cadmium is made at various levels in the chain between the fertilisers and humans. The accuracy and precision of the risk assessment is highest based on body burden, i.e. using biomonitoring, and lowest when recalculated to emissions, i.e. fertilisers for the reasons shown



The target organ in humans is the kidney where its accumulation typically peaks at about 50 years old. The adverse effects related to Cd are, therefore, more detectable in older than in younger individuals and these effects are the result of the lifetime exposure. The relationship between urinary Cd, as the biomarker of the lifetime exposure, and the microprotein concentration, as the biomarker of the lifetime effects in the kidney, leads to a dose-response relationship from which a threshold can be derived below which effects are not to be expected (further termed: no effect threshold). For the sake of illustration, we call this the **body-burden threshold**. Its value has been deliberated in several European risk assessments, leading to different values, probably due to the differences in acceptable margins of safety. The disparity in the no effect threshold between Europe and that of WHO/JECFA is markedly large, the latter is largely based on data of Asian populations which may be different than the Caucasians (Table 2).

The body burden of Cd is the result of exposure from the various sources listed in previous section. We recall that the diet is the main source of Cd. There is no unique relationship between daily dietary Cd intake and body burden. This is related to the nutritional status, mainly iron status, which largely affects the Cd uptake. An individual with low iron status has a larger absorption of Cd from the diet in the gastro-intestinal system than an individual with high iron status. For example, a controlled study with women consuming either high shellfish diet (and, hence, high Cd diet) or a mixed diet showed that blood Cd concentrations were unrelated to the dietary Cd intake but was well explained by the iron status of the individuals [15]. This means that the derivation of a **dietary threshold** which limits the exceedance of the body-burden threshold must account for two sources of variability, i.e. accounting for the other sources of Cd than diet only (inhalation, passive smoking, dust intake,...) and the variable nutritional status. It is clear that this has led to various assumption and safety factors to take into account that variability, thereby leading to variable proposed conversion from the body burden thresholds to tolerable dietary intake values.

Table 2: Cadmium thresholds below used in various international risk assessments below which adverse health effects are not be expected. Values in bold are those that have been used in the two most recent European risk assessments.

Compartment	
Body burden threshold (urine)	
ECB 2007 risk assessment, no effect on kidney	0.67 µg/g creatinine^{\$}
EFSA 2009 opinion: no effect on kidney	1.00 µg/g creatinine^{\$\$}
WHO/FAO (JECFA) 2010: effect on kidney	5.24 µg/g creatinine
Dietary thresholds	
EFSA opinion (2009)	25 µg/day (at 70 kg BW)
FAO/WHO (2011)	58 µg/day (at 70 kg BW)

^{\$}effect level of 2.00 µg/g divided by a margin of safety of 3; ^{\$\$} effect level of 3.9 µg/g divided by 3.9 accounting for inter-individual variation.

The risk assessment of the ECB (2007) concluded that there is a risk of Cd to the general population exposed via the environment because the margin of safety between exposure and effect level was borderline, but below an acceptable value (3) for non-smokers with depleted iron stores. The conclusion was mainly based on predicted exposure assessments since measured (bio monitoring) data were insufficiently available for the non-smoking general population at that time. The only cross sectional population bio monitoring data available at that time were that of Germany for which the margin of safety was below 3 (i.e. risk cannot be excluded) for between 5 and 10% of the population, including smokers.

The EFSA 2009 assessment used the estimated dietary intake values as the index of exposure and contrasted that with the tolerable intake as a threshold. The mean exposure for adults across Europe is close to, or slightly exceeding, that tolerable intake. Subgroups such as vegetarians, children, smokers and people living in highly contaminated areas may exceed the tolerable intake by about 2-fold. We stress, again, that a dietary threshold is inherently more uncertain for risk assessment than a body burden threshold for reasons given in Figure 1.

In both assessment, one can find a number of statements illustrating that the risk is considered small. For example, in the ECB 2007 risk assessment it is written that *"some scientists (including the rapporteurs of the present document) are convinced that clear adverse effects on the kidney with demonstrated clinical relevance occur only at levels 2.5-fold above the proposed threshold"*. This means that the clinical relevance of the threshold chosen is leading to diverging opinions. Since the margins of safety between actual exposure and effects are small, such factor 2.5 can make the difference between a conclusion of risk and no risk. Along the same lines, the EFSA 2009 reports stated that *"the risk for adverse effects on kidney function at an individual level at dietary exposures across Europe is very low"*.

Meanwhile, about 10 years after the last data search, more human biomonitoring data across Europe have become available that indicate limited reasons of concerns. For example, an EU-wide biomonitoring study, using harmonised sampling and analysis methods, with 1632 women (24–52 years of age) in 2011-2012 showed that the Cd exposure exceeded the no-effect threshold of 1 µg/g creatinine threshold (Table 2) in only 0.6% of the 1272 non-smoking women and that none of the mothers (including smokers) or children in that study exceeded the threshold 4 µg/g creatinine threshold above which there is an increased risk for adverse kidney effects [10]. Along the same lines, a whole population (cross sectional) biomonitoring in France in 2006-2007 showed that the no-effect threshold in both non-smokers as smokers was exceeded in 3.6% of the participants including smokers; only 1 of the 1939 participants (<0.1%) exceeded the effect threshold [16].

The above suggests that the health effects are likely small at population level. Because of the uncertainty typically surrounding low level risk, it was decided already in 2002 by the CSTEE that the fertiliser regulations would be calculated using a stand still principle, i.e. only considering the exposure, not the risk.

3. TRENDS IN CADMIUM EXPOSURE VIA THE ENVIRONMENT AND THE ROLE OF PHOSPHATE FERTILISERS

Key findings

1. Soil Cd has increased by about 50 % over >100 years in Europe and phosphate fertilisers have contributed to that increase
2. The long-term trends in crop Cd concentrations are inconsistent; the annual variability in these concentration likely obscures the trends of increased soil Cd
3. Current emissions of Cd to soil have decreased from historical ones and the losses of Cd by leaching and uptake via the crop now slightly exceed the annual emission to the soils

3.1. Historical records

Soil Cd is enriched by human activities via atmospheric deposition and through applications of phosphate fertilizers and sewage sludge. The Cd in the atmospheric deposition is, in turn, derived from industry, e.g. nonferrous metal smelters, but also from various unintentional sources such as burning of coal, steel production etc. The contribution of P-fertiliser to the accumulation of Cd in soil is well established, with earliest records in fertiliser experiment in Australia (Figure 2) where soil Cd of soil was found to increase with cumulative P applications to the fertiliser plots [17]. Numerous long-term studies in UK, Denmark, France and Australia have demonstrated the gradual build-up of Cd in soil with typical increases of 0.1-0.3 mg Cd/kg soil over >100 years. These increases, expressed as a factor increased of soil Cd concentrations, range 1.3-2.3, median 1.5 (or 50% increase) for the selected sets of archived soils of Europe, details are collated in the ECB 2007 risk assessment report. The probably oldest soil archive is that of the Rothamsted classic experiments where soil Cd concentration has increased markedly over the period 1850-1980 (Figure 3).

An important feature to remember from such data is that also the oldest archived soils contain more Cd than that which has been added to that, i.e. the natural background still dominates current soil Cd in most soils. While phosphate fertilisers are currently the main sources of *addition* of Cd in Europe, they are not the major source of the total stock on the root zone of the soils for which the geological natural background still dominates.

Long-term trends in crop Cd concentrations are much less consistent than those in soil Cd. In principle, crop Cd should increase proportionally to increasing soil Cd but the spatial and annual variability in soil Cd bioavailability obscure the trends. An overview of historic trends has been given in earlier studies [18] reporting increasing and decreasing historic trends. In the last three decades, decreasing trends in crop Cd, have been detected in some regions, e.g. in Sweden and Germany, probably reflecting the decrease in atmospheric deposition [19].

Figure 2: The first experimental evidence reported that long-term use of phosphate fertilisers increase soil Cd. The soil Cd increases with cumulative application of P-fertilisers in experimental plots in Australia. The HCl soluble Cd increases from 0.03 to 0.06 mg/kg soil. Reprinted with permission [17]

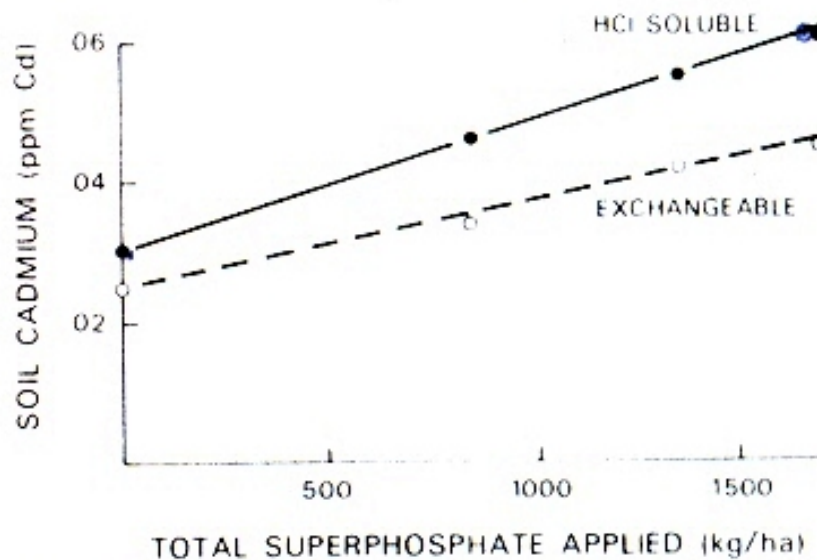
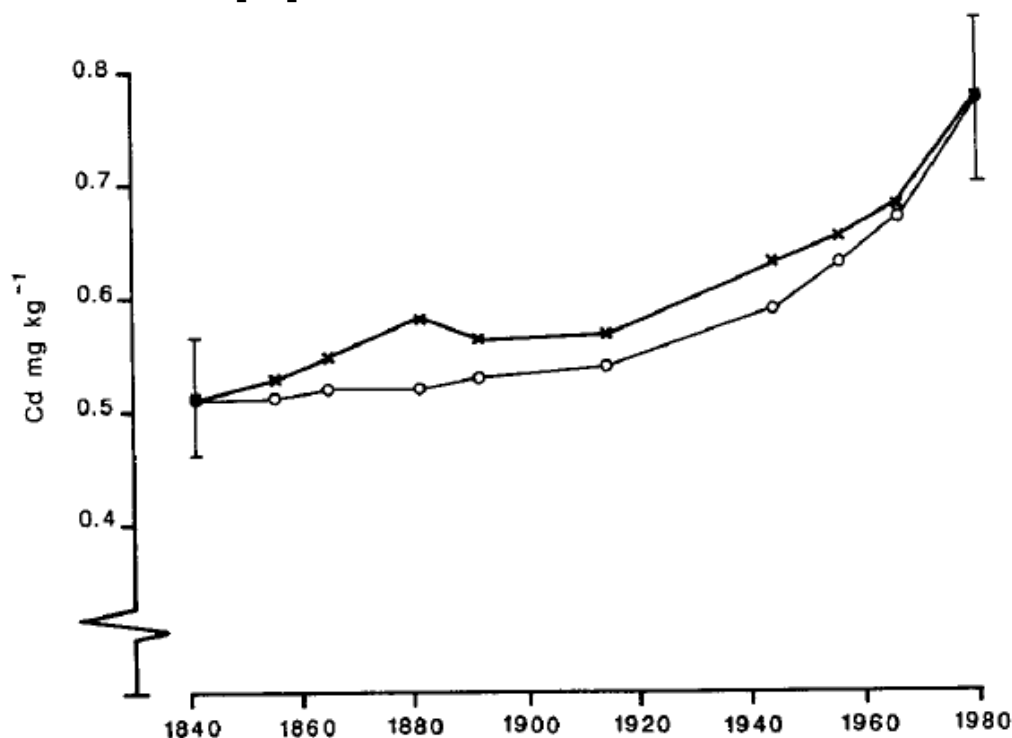


Figure 3: Soil Cd has increased during 120 years in untreated arable soils of a research station in Rothamsted, UK. Filled symbols denote observation in archived soils, open symbols are modelled values using a mass balance approach. These plots did not receive phosphate and the increase is ascribed to atmospheric deposition; the corresponding phosphate treated plots showed similar trends, i.e. the Cd input from phosphate may have been too small to detect statistically. In contrast, there was a clearly larger Cd accumulation in the phosphate treated permanent grassland than in the corresponding untreated plots. Reprinted with permission from [20].



3.2. The future: current emissions from all sources, including fertilisers, and projections based on the mass balance

3.2.1. The concentration of cadmium in phosphate fertilisers on the EU market

All fertilisers, mineral or organic, contain detectable traces of Cd. For phosphate fertilisers, this concentration is markedly large. This is due to its occurrence in the rock phosphate, the source of the phosphate mineral fertilisers. During the fertiliser production process, the Cd remains associated to the phosphate and the Cd/P ratio in the fertiliser product is similar as in the rock phosphate.

It is well established that there are two contrasting types of rock phosphates used on the EU market. Those derived from the Kola Peninsula (Russia) and surroundings contain <1 mg Cd/kg P_2O_5 whereas those of northern Africa (Morocco, Senegal, Togo,...) contain 38->200 mg Cd/kg P_2O_5 , the reason being different geological origins [21]. This is reflected in the very marked differences in Cd concentrations in phosphate fertilisers currently on the EU market (Figure 4). The weighed mean Cd concentrations for the entire EU is estimated at 32 mg Cd/kg P_2O_5 (Table 3).

Figure 4: The Cd concentrations in mineral fertilizers on a P_2O_5 basis (mg/kg P_2O_5) according to country of origin. The fertiliser had been sampled in 2014. Box plots show interquartiles, outliers are not shown (n=414).

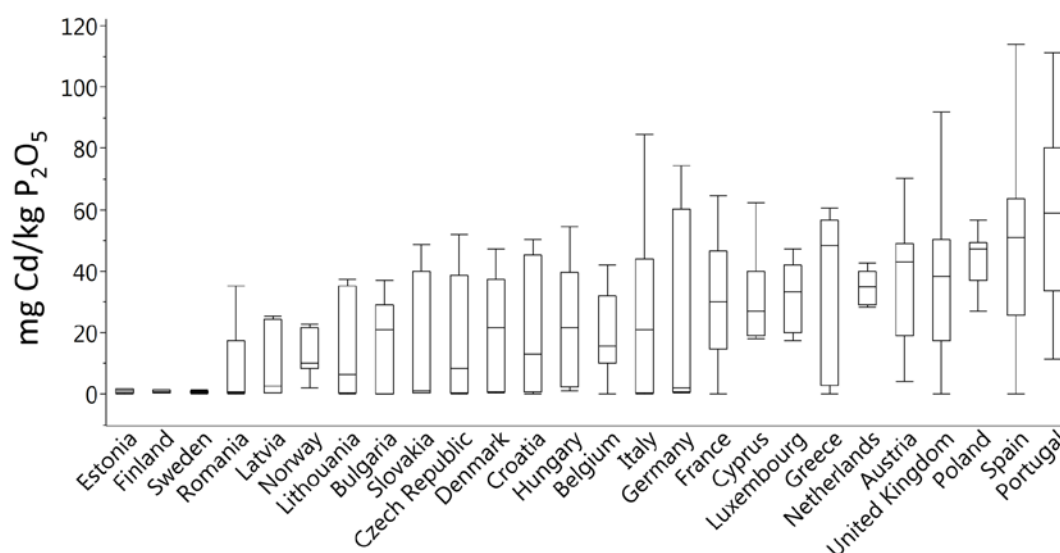


Table 3: Descriptive statistics of the Cd concentration in mineral fertilisers on a P₂O₅ basis (mg/kg P₂O₅) in current and a previous EU survey.

	Nziguheba et al. 2008 (EU15 data, [22])	Study in 2014 in EU27+Norway (Smolders and Salaets, unpublished)
n	196	397 (samples with >5%P ₂ O ₅)
samples collected in	2005	2014
mean Cd (mg/kg P ₂ O ₅)	36	27
weighted mean Cd (mg/kg P ₂ O ₅) *	no data	32
% samples exceeding		
60 mg Cd/kg P ₂ O ₅	21%	8%
40 mg Cd/kg P ₂ O ₅	47%	31%
20 mg Cd/kg P ₂ O ₅	80%	56%
3 mg Cd/kg fertilizer for samples <5%P ₂ O ₅ (n=17)		one in 17 samples (6%)

*weighted mean=country mean concentration multiplied with the corresponding fraction of total P use in that country relative to total tonnage used in EU.

3.2.2. The soil cadmium mass balance in Europe

The historical trends in soil Cd have been reconstructed by modelling, i.e. by an input-output balance (Figure 3). Such modelling has initiated a range of soil Cd balances, probably first described at the European scale in a consultancy report in 1982 [23]. This balance is very simple in principle, accounting for the various sources and the various fluxes in and out of soil (Figure 5). Important to mention is that the stock of Cd in the topsoil (about 750 g Cd/ha in an average EU soil) is more than 100-fold larger than the annual addition of Cd via various sources, i.e. what is added annually to a soil by fertilisers or atmospheric deposition is typically less than 1% of the stock. This also means that any emission control of Cd is only detectable on the very long run. Since the early mass balances were made in 1982, several updates have been made. Our research group has evaluated this balance once more in 2013 [19] and a more recent update was made in June 2016¹.

The technical details are described in our paper [19] but the main findings are summarised: (1) over the past 30 years, the use of P fertilizers in the EU-27+1 has decreased by about factor 4 due to the increased soil fertility and lower soil P requirements; (2) over the last 20

¹ A non-published paper by Erik Smolders of 15 June 2016 'Reply to the SCHER opinion document (May 31, 2015) about "new conclusions regarding future trends of cadmium accumulation in EU arable soils", hereinafter 'Smolders 2016'.

years, measured atmospheric deposition of Cd has decreased by about factor 4 due to stricter emission controls in industry, households etc. and because detection limits are currently lower, i.e. older data sometime overestimated true deposition; (3) the annual losses of Cd to the deeper soil layers, i.e. soil leaching, is a bit larger than estimated before because the soil acidity had been somewhat underestimated. Increasing soil acidity promotes the leaching of Cd and, therefore, increases the losses of Cd from soil. The analysis of 2013 concluded that the current average Cd balance in European arable soils is a negative one, i.e. soil Cd is predicted to decrease, albeit moderate (15% in 100 years) with decreasing trends in some scenarios being more prevalent than increasing trends in other scenarios. These Cd balances have reverted from the general positive balances estimated 10 or more years ago. The uncertainty analysis suggests that leaching is the most uncertain relative to other fluxes.

In 2015, the study was sent to the commission and an expert panel was invited to comment. That panel (SCHER) agreed with the general findings of these studies but has raised some requests for clarifications and for new calculations to account for some worst case scenarios. This was then addressed in the Smolders 2016 paper to SCHER. Most importantly, the question about the relevance of the different soil-fertilizer scenarios was addressed with a more accurate method to estimate the spatial variability in EU. A larger (realistic worst case) fertilizer application rate and national mean sludge application rate, both suggested by SCHER, was also included. The updated parameters and the new area based analysis did not largely change the predicted future *average* trends of soil Cd, this balance is given in Table 4. About 60% of the Cd emissions to EU agricultural soils are derived from phosphate fertilisers. The EU average soil Cd is predicted to decrease by 16 % in the next 100 years with current emissions. The predicted decrease is even >65% in a small fraction (10%) of EU arable soils, often the most acid ones as in Poland, while at the same time there can be >22% increase in 10% of the EU arable soils. These increases are predicted for southern Europe with a low natural background and limited leaching where any addition leads to a larger % increase relative to the background.

The leaching of Cd from soil brings Cd into groundwater and finally in surface water. The current leaching is estimated to be larger than earlier estimates because the agricultural soils are rather acid. The annual fluxes of Cd that reach groundwater are dominated by the geogenic (natural) Cd, not by the additions at the soil surface, simply because the soil stock is still dominated by the natural background (see above). The contribution of fertiliser derived Cd emissions (0.7 g Cd/ha annually) is not even 0.1% of the current stock and the same is true for its contribution to the leaching. The concentrations of Cd in leachates are not of immediate concern: surface water Cd is now rarely considered as a risk when confronted with the Environmental Quality Standard (EQS) of the Water Framework Directive (WFD) and the same is true for groundwater Cd. It can be shown that managing soil acidity by regular liming is by far a more important strategy to limit Cd leaching than managing the emissions to soil.

Figure 5: The soil cadmium mass balance in Europe. Table 4 lists the current (2016) estimated average values for EU28.

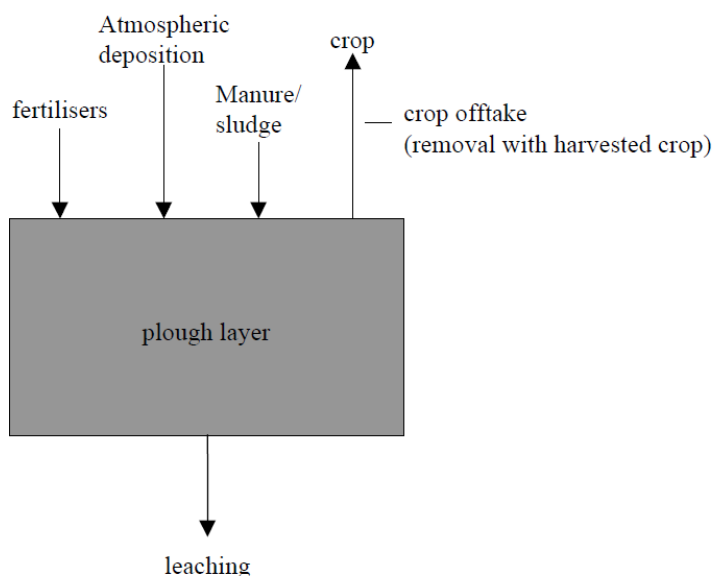


Table 4: Fluxes of Cd considered in the mass balances of cadmium in the average of European agricultural (arable) soils. Adapted from [19] and refined in the final update in reactions to comments of the SCHER committee in Smolders, 2016.

Balances	
Input	
atmospheric deposition	+0.3 g Cd/ha/y
phosphate fertilisers	+0.7 g Cd/ha/y
manure, sludge and lime	+0.2 g Cd/ha/y
Output	
leaching	-2.0 g Cd/ha/y
removal with harvested crop	-0.2 g Cd/ha/y
Net balance	-1.0 g Cd/ha/y
Current stock	750 g Cd/ha

4. FUTURE TRENDS IN SOIL CADMIUM AS AFFECTED BY CADMIUM CONCENTRATION IN PHOSPHATE FERTILISERS

Key findings

1. Future trends in soil Cd concentrations were calculated as a function of the fertiliser Cd in a business as usual assumption.
2. The three different fertiliser Cd levels in the different fertiliser regulation are all expected to lead to a reduction of the average soil Cd concentration.
3. Only the most stringent one is expected to also ensure a depleting soil Cd in about 90% of the European agricultural soils.

4.1. A mass balance estimate of the trends as affected by the fertiliser cadmium concentrations

The soil Cd mass balance of Europe has been used to calculate scenarios in which fertiliser Cd is regulated. This was first done in 2002 by the CSTE based on member state reports reflecting their scenarios of fertiliser use, soil types, crop rotations systems etc. Rather than using the actual fertiliser Cd concentrations in the member states, the fertiliser Cd concentrations were assumed at the three levels of investigation, i.e. 60, 40 and 20 mg Cd/kg P₂O₅. That same approach has been repeated in 2013 and in 2016, in each step taking into account most recent data and better tools to estimate the expected variability in the fluxes. That modelling calculates the future soil Cd after 100 years assuming business as usual in fertiliser practices and in soil properties affecting the Cd balance. The modelling was made for 2096 different soils, evenly spread over the EU agricultural land and including an average and realistic worst case scenario for high use of fertilisers and use of sewage sludge on the same soils.

Only the most recent (2016) calculation is given below which also includes a fourth potential Cd concentration (80 mg Cd/kg P₂O₅). The results are shown in Table 5 and Figure 6. The analysis showed that at a fertiliser Cd concentration of 73 mg Cd/kg P₂O₅, there is zero net accumulation (stand still) for the *average EU*. Note, however that such value should be treated with caution, i.e. it is an uncertain value not at least because no one can predict if the business as usual assumption is correct, i.e. if fertiliser practices, soil properties and climate, are constant over such a long period. Perhaps more helpful are the "error bars" in Figure 6: they show the predicted spatial variability (the 10-90% percentiles of the soil scenarios) that we can estimate, illustrating that a net zero balance is associated with a large fraction of soils with considerable depletion and a large fraction of soils where the soil Cd still increases.

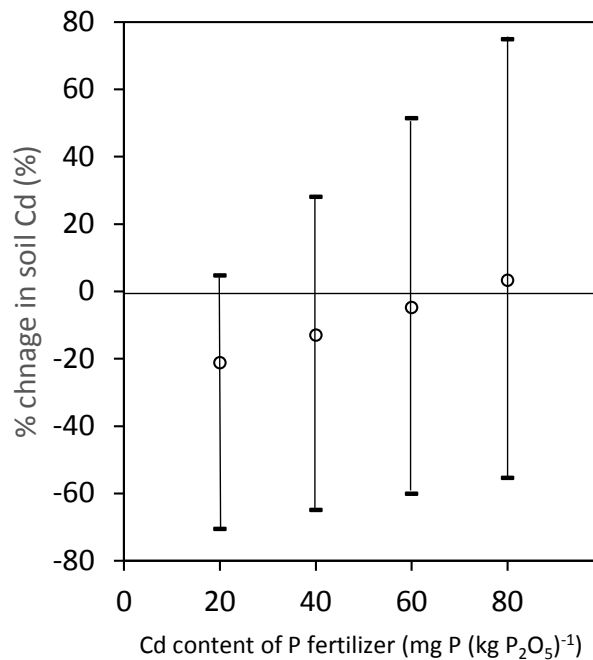
The Figure 6 illustrates the consequences of various options in the thresholds chosen. The more conservative option will select the lower fertiliser threshold (20 mg Cd/kg P₂O₅) which leads to no further increase in soil Cd in almost 90% of the EU soils and is associated with an overall negative EU wide Cd balance. In contrast, the other option is to advocate for a stand still principle in the average situation, i.e. at the higher fertiliser threshold. The arguments for the latter option are that the lifetime Cd intake matters for risk assessment

and that dietary Cd is not derived from one particular soil, even not from a particular region. What almost all Europeans eat in their lifetime, is a mixture of food items that were grown in various places. When the average balance is in stand still, so is the expected dietary Cd and human exposure in a completely mixed assumption. It can be argued, however, that locally grown food is often preferred by many Europeans, however we recall that the grain and potatoes are main sources of Cd and it is very unusual that an individual grows all these crops in a local soil for its entire lifetime. There is obviously no complete mixing of food items and, therefore, the true trends in the exposure will be affected by such mixing which cannot be estimated, certainly not over such a long future period.

Table 5: Predicted change in soil Cd in the average EU scenario (top) and in worst case scenarios (bottom) according to the updated parameters set and requests by SCHER

<i>Model parameters for mass balance</i>	<i>Update 2016 EU average</i>
<u>% change</u> in soil Cd after 100 years at actual EU average fertiliser Cd	-16%(=depletion)
20 mg Cd/kg P ₂ O ₅	-21%
40 mg Cd/kg P ₂ O ₅	-13%
60 mg Cd/kg P ₂ O ₅	- 5%
80 mg Cd/kg P ₂ O ₅	+3%

Figure 6: The predicted change in soil Cd after 100 years of emissions using different Cd concentrations in phosphate fertilizers. Means are circles, the ranges is the 10-90th percentile of scenarios (soils) in Europe, the 90th percentiles were calculated at a realistic worst case of an almost threefold larger than average fertiliser use combined with higher use of sewage sludge in the same soils.



4.2. Fertiliser cadmium does not affect crop cadmium concentrations within the first years after application

As a final note, the question is addressed how crop Cd responds to fertiliser Cd concentrations. There is a classic and persistent misconception that using low, rather than high Cd phosphate fertiliser will have an immediate effect on crop Cd in the subsequent years. That is not to be expected as has already been shown in numerous field results. In USA, field trials have suggested that the Cd concentrations in wheat products are not significantly changed by the phosphate fertilisation and production strategies. Small, but inconsistent, effects on grain Cd were found when changing fertiliser Cd in the range of 4-330 mg Cd/kg P₂O₅, i.e. in an extremely wide range in a two year field trial. The effects of lime and year were more important than that of the fertiliser Cd concentration [24]. The answer to explain that is fairly simple: the large soil stock is often >100-fold above the actual addition and the fertiliser Cd is well mixed with the soil. It has been shown in experimental studies with labelled Cd that "fresh" and "old" Cd in soil are equally available, i.e. what is added annually as a Cd dose to a soil is mixed with the existing stock of Cd in soil [12]. We also recall that the long-term effects of fertiliser on soil Cd are much stronger than comparative effects on crop Cd because of the inherently large variability of the soil Cd bioavailability (up to factor 5 between subsequent seasons) which obscures the effects of the increased total stock (<factor 2 over >100 years).

5. RECOMMENDATIONS

The main objective of the proposed regulation on the CE marked fertilizing products is to harmonize various standards for organic fertilizers and the cadmium levels for all fertilizers. This harmonization is expected to ease intra-EU trade in fertilizers and thus potentially reduce their prices. Currently, many Member States have detailed, national rules and standards in place for fertilisers, with environmental requirements such as the cadmium contaminant limits that differ among Member States.

This document has explained the background of proposed cadmium limits for CE marked phosphate fertilisers. The proposal is based on three limits that will be adopted stepwise and the most stringent one would only be in place in 12 years after adoption. It is well established that the regulation will affect the market, e.g. >50% of the current phosphate fertilisers on the EU market exceed the most stringent limit that would be adopted after 12 years. The so-called decadmiation of the fertiliser is not economically feasible at this stage.

Based on the scientific background on the issue, this author can give the following recommendations:

- This proposed cadmium regulation is a formal consequence of the two EU-wide risk assessments that concluded that there is a need to limit risk of cadmium to the general population [1] and that the current exposure to cadmium at the population level should be reduced [2]. Phosphate fertiliser use contributes about 60% of the net cadmium addition to soil in Europe (Table 4), therefore exposure can be decreased on the long-term by reducing this part of the net input. This means that a cadmium regulation in the fertilisers can be recommended. The highest proposed limit (60 mg Cd/kg P₂O₅) is currently exceeded by 8% of the fertilisers on the EU market. Hence, it is predictable that the adoption of that highest limit only would already decrease the average cadmium input.
- The risk assessments on cadmium in Europe had been based on very extensive analysis of the information and are supported by a qualified majority of EU representatives. Both indicated that the human health risk due to cadmium cannot be excluded. At the same time, one can find a number of statements in these documents illustrating that the risk is considered small. The more recent biomonitoring data that have come available since the two risk assessments (section 2.3 of this document), indicate that the risk is even lower than estimated earlier. The views on the levels of risk are inconsistent among scientists, even among different international health assessments as can be witnessed by the disparity between the EU proposed human health exposure limits for cadmium that are below those of the WHO (Table 2 of this document) which are less stringent. Because of the uncertainty typically surrounding low level risk, it was decided already in 2002 by the Commission that the fertiliser regulations would be calculated using a stand still principle, i.e. only considering the exposure, not the risk. However, when deliberating the range of proposed limits, it instructive to recall that the current health risk of cadmium are, in view of this author, low for the non-smoking, general population in Europe
- The stand-still principle has been calculated on the soil cadmium balance. Fertiliser cadmium concentrations were calculated that would not lead to a further accumulation in soil on the long term and that would, equally, not further increase food cadmium, dietary cadmium or human exposure to cadmium. The three cadmium levels that are proposed in the fertiliser regulation are expected to all lead to a decrease in the EU *average* soil cadmium concentrations, leaving some soils and regions with an accumulation balanced by regions where there is depletion. The more

conservative option will be to select the most stringent, lower fertiliser threshold (20 mg Cd/kg P₂O₅) which leads to no further increase in soil cadmium in almost 90% of the EU soils. It must be stressed that that this does *not* mean that there is still potential risk in 10% of the soils, in contrast, one can argue that the variability among EU soils does not affect the risk but that only a stand-still in the average EU soils is defensible, in which case a limit of 73 mg Cd/kg P₂O₅ holds. The arguments for the latter option is the general knowledge that the lifetime cadmium intake is important for cadmium related human health risk. Indeed, what almost all Europeans eat in their lifetime, is a mixture of food items that were grown in various places. When the average soil cadmium is in stand still, so is the expected human exposure in a completely mixed assumption. There is obviously no complete mixing of food items and, therefore, some conservatism is justified which, unfortunately, cannot be calculated, simply because the extent of mixing of foods within Europe cannot be estimated for the next decades to come. This means the three proposed limits (20-60 mg Cd/kg P₂O₅) bracket a spectrum of various degrees of conservatism within an uncertainty range, but all leading to a reduction of cadmium exposure in the future. With increasingly stringent limits, an increasingly larger fraction of EU soils will have a negative cadmium balance.

The author of this document suggests that the deliberation of the threshold should consider the cost-benefit balance, i.e. the % of the market affected by the limits versus the magnitude of the current human health risk of cadmium, the latter cannot be quantified but is a low one as indicated by the most recent high quality biomonitoring data of Europe and referred to in this document.

REFERENCES

1. ECB, *European Union Risk Assessment Report. Cadmium Metal. Part II Human Health* 2007, Office for Official Publications of the European Communities: Luxembourg. p. 704.
2. EFSA, *Scientific Opinion of the Panel on Contaminants in the Food Chain on a request from the European Commission on cadmium in food*. The EFSA Journal, 2009. 980: p. 1-139.
3. Baize, D., in *Teneurs totales en éléments traces métalliques dans le sols (France)* 1997, Institut National de la Recherche Agronomique: Paris, France.
4. Holmgren, G.G.S., et al., *Cadmium, lead, zinc, copper, and nickel in agricultural soils of the United States of America*. Journal of Environmental Quality, 1993. 22(2): p. 335-348.
5. Traina, S.J., *The Environmental Chemistry of Cadmium*, in *Cadmium in Soils and Plants*, M.J. McLaughlin and B.R. Singh, Editors. 1999, Kluwer Academic Publishers: Dordrecht/Boston/London. p. 11-37.
6. Toth, G., et al., *Maps of heavy metals in the soils of the European Union and proposed priority areas for detailed assessment*. Science of the Total Environment, 2016. 565: p. 1054-1062.
7. Salminen, R., ed. *Geochemical Atlas of Europe. Part 1: Background Information, Methodology and Maps*. 2005, Geological Survey of Finland: Espoo.
8. Reimann, C., et al., *Chemistry of Europe's Agricultural Soils. Part A. Methodology and Interpretation of the GEMAS Data Set*. Geol. Jb, 2014. B 1022014, Hannover: Bundesanstalt für Geowissenschaften und Rohstoffe (BGR). p.528.
9. Jansson, G., et al., *Teeltadvies voor de landbouw in kader van het Interreg project BeNeKampen. Final Report.* , 2007.
10. Berglund, M., et al., *Exposure determinants of cadmium in European mothers and their children*. Environmental Research, 2015. 141: p. 69-76.
11. Smolders, E. and J. Mertens, *Cadmium*, in *Heavy Metals in Soils*, B.J. Alloway, Editor 2013, Springer. p. 283-311.
12. Smolders, E., et al., *Cadmium fixation in soils measured by isotopic dilution*. Soil Science Society of America Journal, 1999. 63(1): p. 78-85.
13. Nordberg, G.F., *Historical perspectives on cadmium toxicology*. Toxicology and Applied Pharmacology, 2009. 238(3): p. 192-200.
14. Jarup, L. and A. Akesson, *Current status of cadmium as an environmental health problem*. Toxicology and Applied Pharmacology, 2009. 238: p. 201-208.
15. Vahter, M., et al., *Bioavailability of cadmium from shellfish and mixed diet in women*. Toxicology and applied Pharmacology, 1996. 136: p. 332-341.
16. Fréry, N., et al., *Exposition de la population française aux substances chimiques de l'environnement*, F. Institut de Veille Sanitaire, Editor 2011. p. 151.
17. Williams, C.H. and D.J. David, *The accumulation in soil of cadmium residues from phosphate fertilizers and their effects on the cadmium content of plants*. Soil Science, 1974. 121: p. 86-93.
18. ECB, *European Union Risk Assessment Report. Cadmium Metal. Part I Environment*, 2007, Office for Official Publications of the European Communities: Luxembourg: . p. 675.
19. Six, L. and E. Smolders, *Future trends in soil cadmium concentration under current cadmium fluxes to European agricultural soils*. Science of the Total Environment, 2014. 485: p. 319-328.
20. Jones, K.C., C.J. Symon, and A.E. Johnston, *Retrospective analysis of an archived soil collection II. Cadmium*. The Science of the Total Environment, 1987. 67: p. 75-89.
21. McLaughlin, M.J., et al., *Review: the behaviour and environmental impact of contaminants in fertilizers*. Australian Journal of Soil Research, 1996. 34: p. 1-54.
22. Nziguheba, G. and E. Smolders, *Inputs of trace elements in agricultural soils via phosphate fertilizers in European countries*. Science of the Total Environment, 2008. 390: p. 53-57.

23. Hutton, M., *Cadmium in the European Community: a prospective assessment of sources, human exposure and environmental impact*. 1982: p. 1-99.
24. Mortvedt, J.J., D.A. Mays, and G. Osborn, *Uptake by wheat of cadmium and other heavy metal contaminants in phosphate fertilizers*. *Journal of Environmental Quality*, 1981. 10: p. 193-197.

DIRECTORATE-GENERAL FOR INTERNAL POLICIES

POLICY DEPARTMENT ECONOMIC AND SCIENTIFIC POLICY **A**

Role

Policy departments are research units that provide specialised advice to committees, inter-parliamentary delegations and other parliamentary bodies.

Policy Areas

- Economic and Monetary Affairs
- Employment and Social Affairs
- Environment, Public Health and Food Safety
- Industry, Research and Energy
- Internal Market and Consumer Protection

Documents

Visit the European Parliament website:
<http://www.europarl.europa.eu/supporting-analyses>

PHOTO CREDIT:
iStockphoto.com; Shutterstock/beboy



ISBN 978-92-846-0636-8 (paper)
ISBN 978-92-846-0637-5 (pdf)

doi:10.2861/284502 (paper)
doi:10.2861/720 (pdf)

