The Risks to Health and Environment by Cadmium used as a Colouring Agent or a Stabiliser in Polymers and for Metal Plating

Final Report

prepared for

The European Commission, DG Enterprise

by

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RPA REPORT - ASSURED QUALITY	
Project: Ref/Title	J316/Cadmium
Approach:	In accordance with the Contract, Scoping Meeting with the Commission and associated discussions
Report Status:	Final Report
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Approved for Issue by:	Dr Peter Floyd, Director
Date:	6 December, 2001

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Executive Summary

Background

Cadmium is a naturally occurring metallic element. Cadmium and cadmium compounds are always derived as by-products of the refining of both zinc and lead. Most of the resulting cadmium metal is shipped to Ni-Cd battery manufacturers for use in new batteries. About 1,000 tonnes of cadmium per annum is used in the EU for stabilisers, pigments and plating.

This report analyses the risks to human health and the environment arising from the use of cadmium in pigments, stabilisers and in metal plating. The work follows on from earlier studies undertaken by WS Atkins in 1998 and takes account of comments made by the Scientific Committee for Toxicity, Ecotoxicity and the Environment (CSTEE) on the WS Atkins work.

In preparing this report particular attention was given to a major EU-wide study being undertaken by the Belgian authorities in preparing risk assessment reports for cadmium and cadmium oxide under the Existing Substances Regulations. In the US, the Agency for Toxic Substances and Disease Registry has recently published a comprehensive review of cadmium toxicity which acted as a valuable reference source for this report.

Cadmium Usage in Stabilisers

The manufacture of polymers such as PVC requires stabilisers to prevent decomposition during both processing and use. The main use of cadmium as a PVC stabiliser is in window frames. As some countries (Sweden, Denmark, Austria, Netherlands) have already banned this use of cadmium and the industry has a voluntary agreement to phase out the use of new cadmium stabilisers, the overall use of cadmium in stabilisers has dropped from a level in 1997 of 270 tonnes per annum (tpa) to 30 tpa. However, in the future, it may be the case that while there is little 'new' cadmium being used in stabilisers, the cadmium from old PVC windows could be recycled and reused. For this reason, the analysis has considered options with and without recycling.

Cadmium Usage in Pigments

The usage of cadmium in pigments is about 830 tpa. Of these, about 90% are used in plastics, with only 4% in artists' colours and about 6% in colours for ceramics/glass. The prime use is in the manufacture of brilliant red, orange and yellow pigments that are used in a range of applications, particularly engineering plastics. Although there are restrictions on the use of cadmium pigments in some plastics, many plastics are exempt.

Cadmium Usage in Plating

About 195 tpa of cadmium is used in aerospace, marine and military applications to electroplate fasteners, electronic components, springs and general hardware. In broad terms, only those uses in which the properties of cadmium electroplated coatings are essential - such as in aircraft landing gear - are permitted.

Cadmium Emissions to the Environment

For each of the uses outlined above, it was possible to estimate the annual losses to the environment during product manufacture, product use and disposal. These emissions were then used as 'inputs' to the EUSES (European System for the Evaluation of Substances) computer model to predict the resultant environmental concentrations at continental, regional and local levels under both 'general' and 'acidic' environments, as well the resultant uptakes by mammals (secondary poisoning) and by humans.

Calculating Risks to the Environment

In order to evaluate the risk to the environment, it was first necessary to derive a set of predicted no-effect concentrations (PNECs). These were based on consideration of a range of experimental results into the effects of cadmium upon a wide range of species in the aquatic and terrestrial environments. The PNECs were then compared with the predicted environmental concentrations (PECs) to determine whether there would be any significant risks to the environment (as indicated by a PEC/PNEC ratio of greater than one).

Calculating Risks to Humans

The report provides a comprehensive review of cadmium toxicity with reference to both animal and epidemiological studies. Of particular interest is the no observed adverse effect level (NOAEL) which has been estimated to be of the order of 2 lg/kg/day (i.e. an intake of two micrograms of cadmium per kilogram of bodyweight per day) - which is twice the current 'tolerable daily intake' (TDI - as set by FAO/WHO). This 'threshold ' intake of 2 lg/kg/day will produce a 'critical' concentration of 200 lg Cd/g in the renal cortex after decades of exposure. However, in certain susceptible individuals, this critical concentration may be only 50 lg Cd/g. This, in turn, would suggest that the 'threshold' intake should be 0.5 lg/kg/day.

Although there are calls to set a lower limit of, say, 0.2 lg/kg/day, its imposition would result in about half the EU population being exposed to cadmium intakes above the perceived 'safe' level which may not be justifiable. A more pragmatic approach would be to adopt a reduced TDI which acknowledges that, in certain susceptible individuals, the 'critical' concentration in the renal cortex may be only 50 lg Cd/g rather than the 200 lg Cd/g associated with the 'general' NOAEL of 2 lg/kg/day. This, in turn, would suggest that the 'threshold' intake and TDI should be set at 0.5 lg/kg/day.

In addition, a NOAEL for worker exposure (via inhalation) was determined to be a 4 lg/m^3.

These values were then compared to those predicted (and, in some cases, measured) in the EU environment.

Key Findings

Cadmium usage in pigments, stabilisers and metal plating results in significant emissions of cadmium into the environment. At a regional level, the level of risk is small both in comparison to the natural background and to the 'safe' levels of cadmium.

At a local (site specific) level, there may be occupational risks (with particular reference to the onset of renal dysfunction) due to continued exposure (over decades) to concentrations of the order of 10 lg/m^3 or more. However, there is a broad drive to reduce occupational levels to 5 lg/m^3 or less (through regulation and best practice) which should practically eliminate such adverse effects (for which a NOAEL has been estimated at 4 lg/m^3).

Local emissions to the environment from the 11 identified pigment and stabiliser manufacturing sites were calculated for both 'general' and 'acidic' environments (to give 22 combinations). There would appear to be a slight risk to health and the environment associated with pigment and stabiliser manufacture - subject to further consideration of secondary poisoning via the terrestrial food chain.

Local emissions to the environment from artists' colours formulators and cadmium plating sites were similarly considered for both the 'general' and 'acidic' environments. There would appear to be a slight risk to the environment (but not health) associated with artists' colours formulators and cadmium plating sites - subject to further consideration of secondary poisoning via the terrestrial food chain.

Emissions to the environment during use of products containing cadmium stabilisers and pigments were determined and were found to be of negligible proportions. By contrast, the emissions from cadmium plated aircraft components accounted for most of the cadmium emitted to the environment associated with the uses under study. In summary, there would appear to be a significant risk to health and the environment associated with use of cadmium plated articles in aircraft. However, the analysis does carry a number of uncertainties and consideration should be given to a 'reality check' based on any cadmium monitoring data in the vicinity of major airports.

Although emissions from landfill were not determined (as they are outside the Technical Guidance Document), consideration was given to emissions from incinerators (both municipal solid waste - MSW - and sewage sludge). For the determination of the associated risks, it was assumed (as a worst case) that 10% of emissions could be assigned to a single incinerator. On this basis, there would appear to be a significant risk to health and the environment associated with MSW incineration and a lesser risk associated with sewage sludge incineration. However, the analysis does carry a number of uncertainties and there needs to be some caution in applying the results. For example, although it is highly unlikely that one incinerator (out of the 500+ MSW incinerators) would handle 10% of the cadmium being incinerated, it is possible that one 'rogue' incinerator could be responsible for a disproportionate amount of cadmium emissions to the atmosphere. Given the current interest of both the public and the regulators in incinerators, it is unlikely that such a situation would be tolerated for long (years).

Overall, using the approach prescribed by the Technical Guidance Note, it would appear that there are certain areas where the risks to health and the environment associated with

pigments, stabilisers and cadmium plating merit further consideration. However, it must be remembered that these 'risks' are based on assumptions which tend to:

- *maximise the emissions;*
- *maximise the exposure; and*
- *minimise the 'threshold' concentration for effects.*

Recommendations

Given the significance of the secondary poisoning results, further consideration should be given to whether assumptions used in both this and the ongoing study by the Belgian authorities are robust - with particular reference to the 'threshold' value for effects and the likely residence time of cadmium in the soil.

Given that much of the cadmium used in pigments, stabilisers and cadmium plating will ultimately end up in a landfill in one form or another, it would be of assistance if there was an agreed procedure within EUSES to address the associated risks.

Given the potential significance of cadmium losses from plated components on aircraft, it is recommended that further attention be given to cadmium levels around major EU airports.

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1. INTRODUCTION

1.1 Overview

This report presents the results of a study to assess the particular risks to human health and the environment arising from the use of cadmium in pigments, stabilisers and in metal plating. The full scope of work is presented in Annex 1 and follows a study by WS Atkins (1998a and 1998b). The focus of this study is to address issues raised by the Scientific Committee for Toxicity, Ecotoxicity and the Environment (CSTEE, 1999) with particular reference to:

- the evaluation of toxicity to humans should be expanded taking into consideration the extensive information available;
- the derivation of the Predicted Environmental Concentration (PEC) should be revised with regard to methodology and data;
- the estimation of daily intakes of cadmium and of Tolerable Daily Intake (TDI) should be reviewed especially taking the variation of measured values into account;
- the derivation of Predicted No Effect Concentrations (PNEC) should be revised especially with regard to data used, ensuring proper use of the probabilistic method;
- the risk characterisation should be revised and could be further improved by addressing the effects of environmental characteristics on cadmium toxicity and the inclusion of PNEC values adapted to the water/soil conditions using probabilistic approaches for European water/soil characteristics; and
- the study should also assess the relative risks from cadmium as a colouring agent, as a stabiliser in polymers and for metal plating compared with other sources of cadmium.

1.2 Background

Directive 91/338/EEC restricts the use of cadmium to colour, stabilise or plate specified products with certain exceptions (derogations) as detailed in Annex 2. A summary is presented in Box 1. It is the tenth amendment of Directive 79/769/EEC which sets out a framework for the approximation of the laws, regulations and administrative provisions of Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations.

The EC wishes to review the risks of cadmium in the light of more recent literature in order to assess whether Directive 91/338/EEC needs to be modified after the accession of Sweden, Finland and Austria to the EC. Currently, under Directive 1999/51/EC (adapting Directive 76/769/EEC to technical progress), provision is made for the restrictions in Austria and Sweden to continue to apply until the end of 2002 in the light of the cadmium risk assessment and development of knowledge and techniques in respect of substitutes (at which time the above provisions will be

reviewed). In Finland, provisions similar to those of 91/338/EEC are already enforced.

Box 1: Summary of Restrictions on Cadmium under Directive 91/388/EEC

As colourants in	As stabilisers in	Cadmium Plating of
Polyvinyl chloride	Packaging materials	Equipment and machinery for
Polyurethane	Office or school supplies	- food production
Low-density polyethylene	Fittings for furniture	- agriculture
Cellulose acetate	Articles of apparel and clothing accessories	- cooling and freezing
Cellulose acetate butyrate	Floor and wall coverings	- printing and book binding
Epoxy resins	Impregnated, coated, covered or laminated textiles	Equipment and machinery for the production of:
Melamine formaldehyde	Imitation leather	- household goods
Urea formaldehyde	Gramophone records	- furniture
Unsaturated polyesters	Tubes and pipes	- sanitary ware
Polyethylene terephthalate	Swing doors	- central heating and air conditioning plant
Polybutylene terephthalate	Vehicles for road transport	- paper and board
Polystyrene (transparent/general purpose)	Coating of steel sheet used in construction or in industry	- textiles and clothing
Acrylonitrile methylmethacrylate	Insulation for electrical wiring	- industrial handling equipment
Cross-linked polyethylene		- road and agricultural vehicles
High impact polystyrene		- rolling stock
Polypropylene		- vessels
Paints		
Derogation for products coloured for safety reasons	Derogation for products stabilised for safety reasons	Derogation for products and components used in the aeronautical, aerospace, mining, offshore and nuclear sectors where high safety standards are required and in safety devices in road and agricultural vehicles, rolling stock and vessels. Also for electrical contacts in any sector of use (for reliability reasons)

1.3 Approach

The study approach has been to take the WS Atkins report as a basis, and to determine whether there are any more recent (or more relevant) data available. This has involved extensive reviews of literature and other sources of information as well as consultation with a range of interested parties (and a full list of consultees is presented in Annex 3). Particular attention has been given to the risk assessments being prepared by the Belgian authorities (De Win M *et al*, 1999) on cadmium and cadmium oxide. In addition, a meeting was held with the International Cadmium Association, James M Brown Ltd. (a major pigments manufacturer) and the International Zinc Association on 17 May 2000 to facilitate the collection updated information on cadmium production and use in the EU.

In order to address the specific issues raised about the calculation of the Predicted Environmental Concentrations (PECs) and the Predicted No Effect Concentrations (PNECs), the existing methodologies and equations in the Technical Guidance Document (EC, 1996) and EUSES, the European System for the Evaluation of Substances[1] have been reviewed. In terms of human toxicity, a comprehensive review has been undertaken (see Section 6.3) which has drawn heavily upon a major recent US study (NTIS, 1999).

[1] EUSES is a decision support software package based on the Technical Guidance Document and is available from JRC Ispra. Further details (and demonstration) are available from http://ecb.ei.jrc.it/existing-chemicals

2. GENERAL SUBSTANCE INFORMATION

2.1 Physico-chemical Properties of Cadmium and Cadmium Compounds

Physico-chemical properties and classification numbers of cadmium compounds which may be used in pigments, stabilisers or in metal plating were summarised in the WS Atkins Report. These are presented in Table 2.1.

Table 2.1: Main physico-chemical properties and registration numbers of cadmium compounds

Substance	Formula	CAS No.	EINECS No.	Molecular Weight	Melting point ^{o}C	Boiling point ^{o}C	Solubility in water
Cadmium	Cd	7440-43-9	231-152-8	112	321	765	Insoluble
Cadmium oxide	CdO	1306-19-0	215-146-2	128	1500	NA	9.6 mgl^{-1}
Cadmium chloride	$CdCl_2$	10108-64-2	233-296-7	183	568	900	1400 gl^{-1}
Cadmium sulphide	CdS	1306-213-6	215-147-8	144	1750	NA	1.3 mgl^{-1}
Cadmium sulphate	$CdSO_4$	10124-36-4	233-331-6	208	1000	NA	755 gl^{-1}
Cadmium laurate	$CdC_{24}H_{46}O_4$	2605-44-9	NA	512	NA	NA	NA
Cadmium stearate	$CdC_{36}H_{70}O_4$	2223-93-0	NA	680	NA	NA	NA
Cadmium sulpho-selenide	$CdS_{(1-x)}Zn_xS$	8048-07-5	232-466-8	Variable	NA	NA	Insoluble
Cadmium zinc sulphide	$Cd_{(1-x)}Zn_xS$	8048-07-5	232-466-8	Variable	NA	NA	Insoluble
Cadmium mercury selenide	$Cd_{(1-x)}Hg_xS$	1345-09-1	215-717-6	Variable	NA	NA	Insoluble

Source: WS Atkins (1998a)

2.2 Environmental Classification and Labelling of Cadmium Compounds

There are currently no environmental classifications or labelling for cadmium and cadmium compounds.

2.3 Human Health Classification and Labelling of Cadmium Compounds

The cadmium compounds which are used in pigments, stabilisers or in metal plating have been given classifications under Directive 67/548/EEC (Dangerous Substances Directive) for human health and safety. These are summarised in Table 2.2.

Table 2.2: Classification of Cadmium Compounds for Human Health and Safety

Substance	Classification	Risk Phrases	Safety Phrases
Cadmium oxide	Carcinogenic Cat.2 Toxic (T) Harmful (Xn)	R 49 R 48/23/25 R 22	S 53; S 45
Cadmium chloride	Carcinogenic Cat.2 Toxic (T) *Mutagen Cat 2* *Toxic to Reproduction Cat 2*	R 45 R 48/23/25	S 53; S45
Cadmium sulphate	Carcinogenic Cat.2 Toxic (T) Harmful (Xn)	R 49/23/25 R 48/23/25 R 22	S 53; S45
Cadmium sulphide	Carcinogenic Cat.3 Toxic (T) Harmful (Xn)	R 40 R 48/23/25 R 22	S 1/2- 22-36/37-45

Sources: Agra Europe (1997)
Italicised entries above represent classifications proposed under the 23rd Amendment to the Directive 67/548/EEC

Risk Phrases:

R22 Harmful if swallowed;
R40 Possible risks of irreversible effects;
R45 May cause cancer;
R49 May cause cancer by inhalation;
R48/23/25 Toxic: danger of serious damage to health by prolonged exposure in contact with skin and if swallowed; and
R49/23/25 May cause cancer by prolonged exposure via inhalation and if swallowed.

Safety Phrases:

S 1/2 Keep locked up and out of reach of children;
S22 Do not breathe dust;
S36/37 Wear suitable protective clothing and gloves;
S45 In case of accident or if you feel unwell, seek medical advice immediately (show label where possible); and
S53 Avoid exposure - obtain special instructions before use.

3. GENERAL INFORMATION ON CADMIUM USAGE

3.1 Overview of Cadmium Production and Use

Cadmium is a naturally occurring metallic element, found in rocks, soils and sediments, which comprises part of the earth's crust at levels of approximately 0.2 ppb. Cadmium and cadmium compounds are always derived as by-products of the refining of both zinc and lead, because the ores of cadmium (sphalerite, CdS) and zinc (greenockite, ZnS) are geologically closely associated. An estimated 90-98% of cadmium present in zinc ores is recovered in the mining and extraction process. In cadmium recovery furnaces, cadmium is reduced using carbon, vaporised and then condensed. Most of the resulting cadmium metal is shipped to Ni-Cd battery manufacturers for use in new batteries (batteries have an average cadmium content of 12%-15% by weight). The cadmium is cast into small flattened discs (4mm to 6mm in diameter) to facilitate handling and reduce erratic rolling.

Total global production of cadmium was an estimated 19,000 tonnes in 1999 (USGS, 2000) as presented in Table 3.1. Estimated world *resources* were about 6 million tonnes based on zinc resources containing around 0.3% cadmium. World *reserves* (i.e. currently accessible) are estimated to be 600,000 tonnes (see Table 3.1).

Table 3.1: Cadmium World Refinery Production 1997-1999 (metric tonnes)

Country	1997	1998	1999*	Reserves
Australia	632	600	600	112,600
Belgium	1,600	1,320	1,300	-
Canada	2,380	2,310	2,300	55,000
China	1,600	2,000	2,100	13,000
Germany	1,140	1,150	1,100	6,000
Japan	2,460	2,340	2,300	10,000
Kazakhstan	1,200	900	1,000	25,000
Mexico	800	1,100	1,000	35,000
Russia	790	800	850	16,000
USA	2,060	1,880	1,800	90,000
Other	5,300	5,200	5,550	237,000
World Total (rounded)	**20,000**	**19,600**	**19,900**	**600,000**

* estimated

Source: USGS, 2000

Within the EU, Belgium and Germany account for nearly half of the EU cadmium metal production. The importance of other member states is set out in Table 3.2.

Table 3.2: EU Cadmium Metal Production	
Country	**% EU production (rounded figures for 1997/98)**
Belgium	29%
Germany	20%
Finland	14%
Italy	11%
Netherlands	9.6%
United Kingdom	8.4%
Spain	6.0%
France	2.5%
Source: World Bureau of Metal Statistics as quoted in WS Atkins (1998a - S3, Part II)	

Western world cadmium consumption totals approximately 15,500 tpa. Europe consumed around 6,400 tpa in 1984 which decreased to around 6,000 tpa in 1991 (OECD, 1994) and to 5,500 tpa by 1997/98 (WS Atkins, 1998a).

The consumption of cadmium within the product groups as set out in the WS Atkins report is summarised in Table 3.3. As indicated, these data have been up-dated through consultation with industry (as discussed in subsequent sections).

Table 3.3: EU Cadmium Consumption by Product Group			
Product	**EU 1997/98**		**EU 2000**
	tonnes/year	**% Use**	**tonnes/year**
Pigments	830	15%	830
Stabilisers	270	4.9%	30
Plating	195	3.5%	195
Others (mainly batteries)	4205	76%	
Total	5500	100%	
Sources: WS Atkins (1998a) and industry consultation			

3.2 Use of Cadmium in Stabilisers

3.2.1 Introduction

The manufacture of polymers such as PVC requires stabilisers to prevent decomposition during both processing and use. Stabilisers react with unsaturated sections of the polymer chain which occur roughly every 1,000 links, preventing further reductive dehydrochlorination. Stabilisers are also used to decrease deterioration through exposure to ultraviolet light and through weathering, and have an important influence on the physical properties and cost of a formulation.

The use of cadmium in stabilisers usually takes the form of a stearate or laurate, usually combined with a similar barium ester as well as a lead stabiliser. Finished PVC products usually contain no more than 0.2% cadmium. The cadmium is locked into the polymer matrix and has extremely low leachability.

3.2.2 Current Restrictions on Use

In 1988, a Council Resolution (88/C30/01) was made which restricts the use of cadmium in PVC products to those where satisfactory technical alternatives do not exist.

In 1991, the Resolution was followed by Directive 91/338/EEC, the 10th amendment of the Marketing and Use Directive (76/769/EEC). This limits the use of cadmium as a stabiliser to <0.01% in PVC in a range of products (as set out in Annex 2), except where stabilisers are used for safety reasons. The use of cadmium stabilisers in PVC windows is not included in the list of restricted products. Discussions concerning the revision of 91/338/EEC were held at the end of 1995 and the Directive is due to be reviewed again over the next couple of years (Donnelly, 1997).

With respect to individual member states, the use of cadmium in stabilisers has been banned in Sweden, Denmark and Austria for some time. Since June 1999, the use of cadmium as a stabiliser has also been banned in the Netherlands (VROM, 1999).

Industry has taken action concerning the use of cadmium in stabilisers and there is a voluntary agreement to phase out the use of new cadmium stabilisers.

3.2.3 Levels of Use

Information provided by stabiliser manufacturers in 1997 for the WS Atkins report indicated that 270 tonnes of cadmium were used by six facilities across the EU in the production of stabilisers each year. These solid stabilisers were used exclusively in unplasticised PVC window and door profiles.

Even for this remaining use, consumption is declining due to the introduction of other stabilisers and positive moves away from the use of cadmium. Thus in 1995, only 10% of the European window profile market incorporated materials based on cadmium stabilisers.

Consultation with the European Stabilisers Producers Association (ESPA) has indicated that cadmium usage has declined further in recent years as a result of the voluntary agreement. Thus, current production of cadmium-based stabilisers is of the order of 30 tpa (as compared to the 270 tpa at the time of the Atkins report), and production will be zero within a year.

The industry is currently investigating the possibility of recycling PVC window waste at the end of its life. There is currently little PVC window waste produced since most is still in use (only 1% of windows were over 25 years old in 1994 - Buehl, 1994). However, the availability of PVC window profiles for recycling is expected to increase over the next ten years or so. While dismantling old PVC windows into individual components is reported to be a time consuming and expensive process, Austria and Germany have established systems for returning old windows to manufacturers and two automatic recycling works for windows have been established in Germany (Buehl, 1994). TNO (1999) has estimated the PVC waste arising between 1998 and 2010 from window profiles as shown in Table 3.4.

Table 3.4: Estimated PVC Waste from Window Profiles by Year (in tonnes)

Year	1998	2000	2005	2010
PVC Waste from Window Profiles (t/yr)	24,550	30,600	51,850	84,250

Source: TNO (1999)

It is not clear at this time whether full scale recycling will be developed by the industry. The uncertainty is created by the council resolution which limits usage of cadmium stabilisers to areas where other products are not technically feasible. While this could be taken to mean that recycling of cadmium-stabilised windows is unacceptable (given the availability of alternative stabilisers), industry argues that it is better to re-use these materials than to dispose of them directly. It is reported that the industry is awaiting a horizontal communication from the commission concerning this issue.

In this regard it is of note that recycling of cadmium-containing products is permitted in the Netherlands provided a number of criteria are met, including those relating to the life time of the recycled product and end-of-life recovery rates (VROM, 1999).

To allow the risks associated with recycling to be considered, two scenarios have been developed:

- Scenario 1 is based on the current cadmium usage of 30 tpa with no recycling; and
- Scenario 2 assumes the current rate of cadmium usage continues, plus recycling at a rate which equates to a use of 270 tpa of cadmium in stabilisers.

3.2.4 Life Cycle of Cadmium in Stabilisers

Use in the Manufacture of Window Profiles

Heat stabilisers are sold either to compounders who mix them with PVC and other components or directly to the manufacturers of the end products who do their own mixing. In the case of window profiles, the PVC compound is then extruded and used to assemble a complete window unit or sold onto an assembler.

The PVC industry states that it is efficient at utilising waste PVC resulting from the fabrication of window frames. This is generally recovered, reground and used again in window profile applications (EVC, 1996). Consultation with the ESPA has indicated that 85% of PVC waste is recycled where this includes both PVC which does not meet the required specifications during extrusion and PVC which is sold to window manufacturers but is, for example, not quite of the correct size and shape.

Product Lifetimes

The guaranteed lifetime of PVC windows tends to be around 15 years, however, most are around 25 to 35 years and many are older. It is expected that window profiles will last at least 25 years, but given the costs of installation it may be reasonable to expect that windows may remain in buildings for longer. Therefore, it can be assumed that any window profiles fitted now will not be disposed of for 30 years (i.e. in 2030) (EVC, 1996).

End-of-Life Recycling

As indicated above, the stabiliser industry is looking at the possibility of recycling PVC windows at the end of their useful life. Old PVC windows can be granulated and fed straight into an extruder to produce new windows. However, what is required in material specifications for windows is a guarantee of light and colour stability. Therefore, the recycled PVC can only be used in the centre of the window profile. It would thus be co-extruded with virgin PVC for the outside which would use non-cadmium based stabilisers.

3.3 Use of Cadmium in Pigments

3.3.1 Levels of Use

The WS Atkins Report indicates that five pigment manufacturers are involved in the production of 830 tpa of cadmium pigments, typically containing 60% cadmium. Of these, about 90% are used in plastics, with only 4% in artists' colours and about 6% in colours for ceramics/glass.

Levels of usage given in the draft Belgian risk assessment for cadmium (DeWin *et al*, 1999) agree with this overall figure (831 tpa for use of cadmium in pigments).

For this current study consideration has been given to the extra-EU trade in pigments. Levels of trade are set out in Table 3.5 for "pigments and preparations based on cadmium compounds". This indicates that exports of cadmium-based pigments and preparations far exceed imports, with a positive trade balance (i.e. net export) of 583 tonnes per year (subject to uncertainties associated with data not provided), most of which is exported from the UK.

Table 3.5: 1998 Trade in Cadmium-based Pigments (tonnes)

Country	Extra-EU Imports	Extra-EU Exports	Trade Balance
Belgium	-	63	63
Denmark	6	-	-6
Germany	0.2	33.4	33.2
Greece	-	5.8	5.8
Spain	0.8	52.8	52.0
France	0.8	-	-0.8
Ireland	1.0	-	-1.0
Italy	1.2	7.9	6.7
Luxembourg	-	-	-
Netherlands*	0.6	1.0	0.4
Austria*	0.1	0.1	0
Portugal	-	-	-
Finland	3.2	16.2	13.0
Sweden	0	-	-
United Kingdom	5.7	422.4	416.7
Total	**19.6**	**602.6**	**583.0**

Source: Eurostat (1999): Data for 1998

*Notes: 1) * data are for 1997*

2) - data not given (but in most cases unlikely to be significant)

As indicated above, the pigments themselves contain around 60% cadmium, while in contrast marketed preparations contain only around 1% to 4% cadmium oxide (De Win *et al, 1999).* As the relative proportion of pigments to preparations is not known, it is not possible to say what quantity of cadmium is associated with these exports (this would be 350 tonnes if all were pigments, but only 6 tonnes if all were preparations containing 1% cadmium).

For the purposes of this assessment it has been assumed that the consumption of pigments in the EU equals the EU production rate (i.e. 830 tpa). Based on the above information it can be seen that this is a conservative assumption which, as it does not take into account exports, will over-estimate the risks to some extent.

3.3.2 Current Restrictions on Use

In 1991, the Resolution was followed by Directive 91/338/EEC, the 10th amendment of the Marketing and Use Directive (76/769/EEC). This limits the use of cadmium as a pigment to <0.01% in a range of plastics (including PVC) and in paints (as set out in Annex 2), except where products are coloured for safety reasons. There is also an exemption for low density polyethylene used for the production of coloured masterbatch on account of the reliability requirements of the apparatus. It should also be noted that the use of cadmium pigments in artists' colours, glass and ceramics is not covered by this amendment to the Directive[2].

In Sweden and Austria, although use of cadmium in pigments is strictly controlled, there are exemptions for its use in artists' paints, certain glass and ceramic products as well as some specialised plastics (De Win *et al,* 1999).

In the Netherlands, restrictions updated in 1999 permit cadmium to be used only in the production of two types of plastic (polyacrylate and polymethyl methacrylate) and then only until 1 January 2003. Use in other plastics is still permitted for 'safety applications', but with companies required to give satisfactory reasons as to why the cadmium is needed for safety purposes. With respect to use of cadmium-based pigments in artists' colours and ceramics, these uses are listed in an Annex to the Decree which sets out essential applications. Thus these uses can continue (VROM, 1999).

The International Cadmium Association has indicated there is a continuing trend away from the use of cadmium in pigments and the British Coatings Federation has indicated that:

- no cadmium-based materials are used in decorative coatings;
- no cadmium-based materials are used in printing inks; and
- to the best of their knowledge, most cadmium pigments have been phased out of industrial coatings, although some residual uses remain where high temperature resistance or bright colours are required.

The move away from the use of cadmium-based materials in printing inks is linked to an industry initiative in this area. In particular, the European Council of Paint, Printing Ink and Artists' Colours Industry (CEPE) has drawn up an exclusion list relating to printing inks and associated products (CEPE, 1999).

3.3.3 Pigmented Plastics

Cadmium is used in the manufacture of brilliant red, orange and yellow pigments that are used in a range of applications, particularly engineering plastics. Cadmium pigments are inorganic and the dispersion, non-migration and non-bleeding properties

[2] However, there are other legislative controls that may apply - for example, *Council Directive 84/500/EEC relating to ceramic articles intended to come into contact with foodstuffs* places limits on the amount of cadmium migrating from the article to an acetic acid (vinegar) solution.

of cadmium pigments make them useful in plastic applications where uniform colouring is important. Also, cadmium pigments retain their brightness and opacity under unfavourable conditions such as high temperatures (WS Atkins, 1998a; OECD, 1994; ICdA, 1999; etc.).

Typically, the concentration of cadmium pigments in plastics end applications will be 0.5% to 1% for highly coloured plastics, but much lower than 0.5% in other applications where cadmium pigments are used in conjunction with other pigments (especially white pigments) to give less highly coloured (pastel) plastic.

In its assessment of the impact of further marketing and use restrictions, WS Atkins (1998a) identifies a range of plastic products containing cadmium. This list was drawn up from Danish and Swedish experience of finding replacements for cadmium-coloured products. Comparison of these uses with the list of restricted uses in the Directive 91/338/EEC indicates that the following products could still be coloured with cadmium in the EU (i.e. the use of cadmium in these plastics is not restricted): high density polyethylene (HDPE) boxes, PEM pipes, PA plastic, ABS, ABA and acetal.

Information provided by the ICdA (1999) confirms that cadmium-based pigments could be used in ABS and also in the following materials: nylon, fluorocarbons and polycarbonates. These are plastics processed at high temperature where cadmium is used particularly for heat and light stability. The publication also indicates that cadmium is used in thermoplastics such as polystyrene and polypropylene. As polypropylene and high impact polystyrene are some of the restricted plastics under 91/338/EEC, it is assumed that cadmium is only used where products are coloured for safety reasons (if any is used at all).

Consultation with the International Cadmium Association has indicated that that use in PVC constitutes the major use of cadmium-based pigments, with cadmium being used for its weather resistant qualities. That said, cadmium pigments are no longer used in underground cables. With respect to other plastics, tends to be used only where ‘pillar-box red’ is required. For mixed colours (e.g. pinks, oranges, etc.) other non-cadmium based pigments are used. However, in Sweden (at least), this process of substitution has lead to some technical and financial difficulties (Őberg & Granath, 1997).

3.3.4 Pigments in Ceramics and Glass

Traditional ceramics include porcelain, glass, bricks and refractory materials, while industrial ceramics include, for example, silicon nitride, silicon carbine, alumina and zirconia. Ceramics are used because they have good corrosion resistance (including wear resistance in corrosive environment and at high temperatures) and low electrical and high thermal conductivity. Pigments containing cadmium, which are also heat and corrosive resistant, are therefore often required.

Traditional ceramic cadmium glazes contain cadmium sulphoselenide crystals incorporated as a pigment which are on the surface of the ceramic body. For

ceramic/glass applications, cadmium pigment will be used at about 10% concentration, and at 4% in vitreous enamels[3]. Within the pottery industry, cadmium pigments are used to obtain bright red, orange and yellow glazes (HMIP, 1993).

Coloured glass is available in a large variety of colours and textures with the pigments applied at various stages of the process, either between glass sheets, after or during manufacture, or then applied and stripped in places for effect.

3.3.5 Pigments in Artists Materials

The artists' colours industry report that cadmium pigments are used in specific colours for their unique properties of permanence, colour brightness and opacity. In this regard, cadmium pigments are heat and fade resistant and are helpful in art conservation. According to one US paints manufacturer, an estimated 5-7% of total cadmium pigment production is used in the manufacture of artists materials[4] (a figure which is comparable with the 4% estimated in WS Atkins, 1998a). The level of acid-extractable cadmium in pigments has steadily been reduced, by several orders of magnitude, making the pigment safer in use and final application.

Cadmium pigments are extremely insoluble and stable compounds of calcined cadmium zinc sulphide (green shade yellows to golden yellows) and cadmium sulphoselenide (yellow through oranges and reds to deep maroons). There is virtually no usage of cadmium pigments in conventional decorating paints. In artist's colours the concentration may be up to about 50%, but is typically lower at about 33%.

The WS Atkins report assumes that around 33tpa of cadmium is used in the production of artists' colours. Information provided by two thirds of the members of the Artists' Colours Group of CEPE reports a cadmium usage of 25.7 tpa amongst these members (with not all producers of artists' colours using cadmium). Thus this figure is consistent with the 33 tpa used by WS Atkins. With a cadmium content of around 33%, it can be estimated that around 100 tpa of cadmium-containing artists' colours are produced in the EU each year.

With respect to trade in artists' colours, exports were almost double imports in 1998 (Euro 50.4 million compared with Euro 27.4 million - GTIS, 2000). It is not known what proportion of these colours contain cadmium, but, on the overall trade balance it would appear that there is a net flow of cadmium-based artists' colours out of the EU.

For the purposes of this assessment it has been assumed that the consumption of artists' colours in the EU equals the EU production rate. Based on the above information it can be seen that this is a conservative assumption.

[3] Based on information supplied to RPA by James M Brown Ltd.

[4] See www.goldenpaint.com

3.4 Use of Cadmium for Metal Plating

3.4.1 Introduction

Electroplating is a coating technology used extensively in a large range of applications. It enhances mechanical and physical properties of substrates by depositing a metallic coating onto surfaces through an electric current applied to the electroplating bath. Electroplated cadmium is used in aerospace, marine and military applications to coat substrates such as fasteners, electronic components, springs and general hardware (see Box 2). Cadmium electroplated coatings are favoured in these applications due to their conductivity, solderability, and excellent self-lubricating properties. In addition, electroplated cadmium has unequalled corrosion resistance in aggressive, alkaline environments.

Box 2: Examples of use of Cadmium-plated components

- universal joints as used in controlling aircraft wing flaps
- aircraft landing gear
- aircraft propeller housing
- safety ladders
- bottom brackets on swing doors
- brackets for marine engines
- bearings and rollers
- fish hooks
- fittings for 'custom cars'
- fittings for turn/tilt window units
- heavy coil springs (on railway bogies, etc.)
- fasteners (nuts and bolts, etc.)
- car winches
- hinges
- brake (and other) hose couplings

Sources: various commercial UK websites

3.4.2 Current Restrictions on Use

In 1991, Directive 91/338/EEC (the 10th amendment of the Marketing and Use Directive (76/769/EEC)) banned the use of cadmium in a range of electroplated products such as cooling and freezing equipment and household goods (as set out in Annex 2). However, there were exemptions granted to products requiring high safety standards in the aeronautical, aerospace, mining, offshore and nuclear industries. Exemptions were also granted for safety devices in road and agricultural vehicles, rolling stock and vessels and electrical contacts in any sector of use. Broadly similar restrictions exist in Finland, Austria and Sweden.

The Decree issued in the Netherlands in 1999 list a number of essential uses for cadmium-plated products (VROM, 1999). These include applications where use of cadmium is prescribed by legislation (i.e. the Aviation Act) or in the safety specifications of the aviation, aerospace or offshore industries. Use is also permitted

in radio equipment for use by the shipping industry, in radar equipment and in electrical contact where cadmium is used to ensure the reliability.

3.4.3 Levels of Use

Information provided by the International Cadmium Association for the WS Atkins report indicated that 195 tonnes per annum of cadmium were used in metal plating each year (about 3.5% of the EU consumption).

At the time of the Atkins report, industry estimated that there were about 500 platers using cadmium in Europe. The majority of these companies were reported to be plating contractors or component manufacturers all plating a number of metals in addition to cadmium. Large engineering and aerospace companies were also involved. The plating industry was reported to be concentrated in those countries where the military is an important industry (such as the UK and France), with the UK alone accounting for 30% of cadmium usage in plating (WSA, 1998a).

In 1997, industry estimated that most cadmium was used in fasteners (53%) and structural components (30%). Other uses (17%) comprised electrical contacts and other safety applications for the vehicle industry. Overall, aerospace and defence applications were estimated to represent over 80% of the production of cadmium plated components. With respect to fasteners alone, 42.5% of these were assumed to be associated with offshore applications and 38% with defence applications (WSA, 1998a).

With respect to the current situation (see also Box 2), UK consultation has indicated that the main use for cadmium-plated metal is in aerospace where cadmium's "unique combination of high corrosion resistance, self repair properties, conductivity and lubricity remain properties unmatched by any other coating". A major use is reported to be components for aircraft, such as landing gear components (e.g. bearings) and certain pins which are subject to large changes in temperature[5].

Consultation indicates that the number of companies involved in cadmium plating has declined significantly in recent years due to requirements for licensing of facilities and installation of treatment works. This, combined with the marketing and use Directive (91/338/EEC) means that cadmium plating is only used for essential uses (i.e. those for which there are currently no effective alternatives). In order to move away from the use of cadmium, some in the aerospace and defence industries have, for some applications, moved away from cadmium-plated carbon steel to better grades of steel.

Many smaller plating companies are reported to have stopped cadmium plating, while several aerospace companies, which, in the past had in-house facilities now use outside specialists. This ties in with information from the ICdA which reports there to

[5] Of note is that the greatest concentration of aircraft steel is in landing gear where strength and reliability are of key importance. In addition, modern braking systems can generate temperatures of 550^{o}C in emergency conditions (such as aborted take-off) and structural integrity must be retained - hence the use of cadmium plated steels (*Source: British Steel Engineering Steels website)*.

be few plating facilities which carry out cadmium plating for customers, except in the USA.

There are reported to be 21 sites in the UK undertaking cadmium plating, with more in Italy and Spain but perhaps only one site in Germany (the German government recently re-authorised use of cadmium for this one application). Industry has estimated that the UK represents between 10% and 30% of cadmium usage and a similar percentage of plating shops. On this basis (taking the average) it can be estimated that there are around 100 sites involved in cadmium plating across the EU.

3.4.4 Recycling

In 1997 it was reported that cadmium plating facilities were increasingly geared towards re-fitting and repair (recycling), with an increasing amount of cadmium being recovered through recycling in Europe. Consultation for this study has confirmed that there is still an increasing trend towards the recycling of cadmium coatings through the remelting of cadmium-coated scrap steel. Large amounts of electric arc furnace dust (EAF) are now recycled rather than landfilled. Recent estimates in the US suggest a recycling rate of cadmium coatings of 35-50% which is expected to increase as EAF recycling becomes more widespread.

4. CADMIUM IN THE ENVIRONMENT

4.1 Environmental Exposure

4.1.1 Overview

Cadmium is a naturally occurring metal that persists in a number of environmental compartments, particularly soils. Through natural processes such as weathering and erosion, small amounts of cadmium are released naturally into the atmosphere and water systems. Table 4.1 shows the levels in each major environmental compartment.

Table 4.1: Natural Cadmium Levels in the Environment	
Compartment	**Levels**
Atmosphere	0.1-5 ng/m^3
Earth's Crust	0.1-0.5 mg/kg
Marine Sediment	~1 mg/kg
Sea Water	~0.1 g/l
Source: International Cadmium Association (1999), also available at www.jamesmbrown.co.uk/cd_pigments/cadmium.htm	

4.1.2 Releases of Cadmium into the Environment

There are a number of sources of anthropogenic cadmium emissions into the environment. Many of these are emitted as a result of activities not (directly) associated with the production and use of cadmium-containing products. These include:

- refining of non-ferrous metals - residual levels of cadmium in zinc and lead ores; copper refining. Although the level of emissions is low, large volumes are refined;
- production of iron and steel - emissions to air, water and landfill may result from several of the input materials and processes for converting iron ore, coke and limestone/fluorspar to iron; production of refined steel from pig steel; or for secondary production of steel from scrap steel. Emissions are likely due to the high volume of material being handled. Industrial emissions are now tightly controlled due to significant improvement in pollution control technology and to strict regulation and legislation - particularly in the metals industry;
- combustion of fossil fuels - cadmium is a natural component of fossil fuels. Electric power plants burning fossil fuels (coal and/or oil) release cadmium contained in the fuels which contain zinc sulphides. As much as 190 ppm of cadmium occurs in mid-continental coals in the US. Most cadmium will be captured in the fly ash and emission control devices; a small volume may be present in the slag/bottom ash which is landfilled or immobilised in cement or asphalt-type processes;

- production/use of phosphate fertiliser - cadmium is contained in the phosphate fertilisers from the original rock phosphate. It has been estimated that between 60 tonnes and 970 tonnes of cadmium is released into the soil each year in the EU through the use of fertilisers (EFMA, 1996 and Van Assche, 1998). Based on a recent report prepared for the Commission (Oosterhuis *et al*, 2000), a more precise estimate of 268 tonnes per annum can be derived. Emissions may also occur through losses to water during the manufacturing process and disposal of by-product gypsum to sea and disposal of gypsum to landfill. Minor emissions may take place to air through dust created during manufacture; and
- production of cement - cadmium is present as trace element in the raw materials of cement production. Emissions may result from the feed system, kiln system, clinker-cooling and handling system. Emissions to air as dust are likely, while direct emissions to water are not.

Cadmium also enters the environment through the disposal of solid and liquid wastes, some of which will be associated with the production and use of cadmium-containing products:

- application of sewage sludge - to soil through application to land as fertiliser. Almost all sewage treatment effluents contain cadmium as part of the sludge material;
- incineration of domestic waste - cadmium is also released as a result of incinerating cadmium stabilised PVC and other products containing cadmium pigments or cadmium compounds found in commercial and household waste; and
- leaching from landfill - cadmium concentration in leachates from controlled, state-of-the-art industrial or municipal landfills is often below the detection limit. By way of example, in a case study in Denmark on a landfill containing industrial/municipal waste, demolition waste, 3% hazardous waste - no elevated cadmium was observed and the concentration was below detection. However, leachates from older and uncontrolled landfills have a much higher cadmium content.

Table 4.2 shows the most significant sources of cadmium emissions in some European countries, taking into account overall use of cadmium.

Table 4.2: Top Sources of Cadmium Releases by Country (1990)			
Country	**Compartment**	**Source**	**Volume (tpa)**
Belgium	air	lead production	1.95
	water	production of iron and steel	3.5
	solid waste	production of copper	77
Denmark	air	waste products	2
	water	waste products	0.7
	soil	waste products	5.3
	waste deposits	waste products	30.6
	recycling/reuse	waste products	5.8
Finland (88/89 data)	air	production of zinc, copper and cadmium	4.85
	water	production of zinc, copper and cadmium	0.12
	solid waste	production of zinc, copper and cadmium	135.5
Netherlands	air	industry	1.5
	air	incineration of household waste	0.6
	water	industry	4
	soil (agricultural)	animal manure	4.5
Norway	air	smelters, incinerators, etc.	1.2
	water	not specified	1.5 (1992 data)
	solid waste	industry	20-30 (1989 data)
		household	5-6 (1989 data)
Sweden	air	primary metal works	1.3
	water	waste from mines	1
	solid waste	not specified	100-200
Source: OECD, 1994			

4.1.3 Emissions from Stabilisers

Information from WS Atkins Reports

The WS Atkins report made estimates of releases of cadmium from stabiliser production facilities and from the in-service use of window frames. Releases associated with end-of-life disposal to incinerator or landfill were also estimated. Cadmium losses from the production of PVC window frames were not quantified.

In this regard, the CSTEE (1999) Opinion states that "(i)a: the release estimation of cadmium from plastics and PVC does not properly take account of release during the entire period in which the material is used."

Stabiliser Manufacture

WS Atkins used site-specific data from all six stabiliser manufacturers in the EU to quantify losses of cadmium from stabiliser production. Site specific data were available for releases to all environmental compartments, save for losses to solid waste for half the sites. In the absence of these data it was assumed that these releases

would equal 1.8 tonnes of cadmium per annum - the largest amount released from any one of the other three sites.

As indicated in Section 3, current use of cadmium is 30 tpa, down from the 270 tpa at the time of the Atkins report. In the absence of information linking current production rates with releases from facilities, losses from stabiliser manufacturing facilities have been estimated by dividing the site-specific data in the Atkins report by a factor of 9. These losses occur under both Scenario 1 and 2 (without and with recycling).

Table 4.3 shows the resultant annual losses of cadmium during stabiliser manufacture (with more information provided on Figures 4.1 and 4.2 - presented at the end of Section 4). As shown by the percentage losses, losses to air, water and sewer are very low.

Table 4.3: Annual Losses of Cadmium associated with Stabiliser Manufacture (giving a Cadmium Usage of 30 tonnes per annum)

Units	Air	Watercourses	Sewer	Solid Waste
kg/yr	0.25	0.09	0.52	960
% of Use	0.0008%	0.0003%	0.0017%	3.2%

Source: Data in WS Atkins (1998a) divided by a factor of 9 to reflect the current situation.

PVC Product Manufacture

Cadmium stabilisers are added to PVC, along with other additives, and extruded to produce window profiles. Cadmium content of these is around 0.2% (Donnelly, 1995), but concentrations of up to 0.4% have also been measured (Argus, 2000).

WS Atkins (1998a) discounted emissions from PVC product manufacture on the basis that these would be negligible compared with a single stabiliser manufacturer. Data were provided from one window manufacturer who reported no losses to water and assumed no losses to air. Data on losses to solid waste were not available. Data were also provided by two stabiliser mixing facilities. No losses to water were reported and losses to air were reported as zero in the case of one facility or were assumed to be zero in the case of the other. Losses to solid waste were reported as 0.7 tpa and 0.2 kg/a

Information provided by the stabiliser production industry indicates that window manufacturer is a dry process, there not generally being any drains within the facility. Cadmium will be emitted to air during the process, dust being removed using a vacuum filter. Cadmium will be present in the contents of the filter. Residual losses to of cadmium to air will depend on engineering specification of the filter.

Information in the literature indicates that some steps have been taken to eliminate dust problems as much as possible in compounding PVC containing cadmium stabilisers. Products may be supplied as pellets or (as lots of a given size) in sealed bags. The advantage of the bags is that these dispense with weighing and can be

charged directly into the mixer. The material from which the bags are made is selected so that it is completely compounded into the PVC during mixing and subsequent extrusion (Bredereck, 1983). This practice is confirmed by a more recent publication (Akros, 1996). This indicates that in recent years there has been a trend to 'one-packs' where the stabiliser (Lead Barium Cadmium) is combined with lubricants (and other additives where appropriate) to give a low or non-dusting product. This takes the form of a powder mix in small bags for direct addition to the mixer, or granules, flakes or tablets.

It is also reported that the dust removed by the vacuum exhaust systems is collected and added to PVC compounds. In addition, waste obtained in machining and assembling sections is reground and recycled for the production of second-grade sections (Bredereck, 1983).

In the absence of site-specific emissions data, default values from the "UV and Other Weathering Stabilisers" section of the Use Category Document for Plastic Additives (BRE, 1998) have been used. The main losses are associated with raw materials handling, where these are 0.21% for powders of particle size >40 μm. Losses during compounding are 0.1%. While some losses would be to atmosphere, ultimately all losses will be to solid waste. These losses are believed to be worst case and where 'one-packs' are used will be over-estimates.

Under Scenario 1 (current situation, no recycling), cadmium inputs to window profile manufacturers account for 29 tpa (taking into account looses during stabiliser production). Losses during window manufacture account for 64 kg. Under Scenario 2 (future situation, with recycling), cadmium inputs to window profile manufacturers are 270 tpa. As indicated in Section 3, windows are recycled by granulating the PVC then co-extruding with new PVC which forms the outside of the window. As for the current situation losses of 0.22% are assumed[6]. (See spreadsheet in Figures 4.1 and 4.2 for more information).

PVC Product Use

In addition to emissions during manufacture, cadmium may also be released during in-service use, i.e. from windows when installed in houses and other buildings.

WS Atkins estimated total emissions of cadmium over a product's lifetime based on work by Bredereck (1983). Leaching experiments on cadmium-stabilised window frames were undertaken during which a 10kg window frame was exposed to 3% acetic acid for one year. Subsequent extraction measurements revealed that from 25mg cadmium contained in the sample, the total amount of cadmium leached was 1.2mg, which represented 0.0048% of the cadmium content. On the basis of 270 tpa of cadmium used in stabilisers this gave releases of 13kg which were assumed to be to soil.

[6] Note that the assessment is not sensitive to this assumption. This waste is assumed to go to solid waste which is either landfilled or incinerated in the ratio 80:20. If the window had not been taken out of the waste stream for recycling, it would also have been disposed of as solid waste to landfill or incineration in the ratio 90:10 (see Section 4.1.6 for explanation of these ratios).

Consultation with industry has suggested that using a figure of 0.005% for the amount of cadmium leached during in-service use may over-estimate emissions. When PVC windows are extruded the surface film contains relatively high levels of cadmium and it this cadmium which is extracted during leaching experiments such as that above. If the experiment were tried a second time very much less cadmium would be extracted as this film has been removed**.** Overall it has been suggested that the leaching of cadmium from such products is likely to be negligible since the cadmium is immobile.

Information provided in the use category document for plastic additives (BRE, 1998) puts forward a figure of 0.01% for leaching to the environment in outdoor service, which, on the basis of the above discussion, appears high. In line with WS Atkins a figure of 0.005% has been adopted for losses of cadmium from the in-service use of windows.

There may also be losses of cadmium occurring through cutting windows to size during installation. For the purpose of this assessment it is assumed that 0.05% of the window is lost through cutting it to shape (as dust).

The above figures for losses during installation and is-service use apply to both Scenarios 1 and 2 (as shown in Figures 4.1 and 4.2).

4.1.4 Emissions from Pigments

Information from WS Atkins Reports

The WS Atkins report made estimates of releases of cadmium from pigment production facilities and from the in-service use of pigmented plastics. Releases associated with end-of-life disposal to incinerator or landfill were also estimated. Cadmium losses from the production of pigmented plastics, artists' colours or ceramics were not quantified, neither were losses from the in-service use of artists' colours or ceramics.

In this regard, the CSTEE (1999) Opinion states that "(i)a: the release estimation of cadmium from plastics and PVC does not properly take account of release during the entire period in which the material is used."

Pigment Manufacture

WS Atkins (1998a) used site-specific data from all five pigment manufacturers in the EU to quantify losses of cadmium from pigments production. Site specific data were available for releases to all environmental compartments, save for losses to air for one of the sites and losses to solid waste for three of the sites. In the absence of these data losses were calculated based on the maximum losses for the facilities providing information, where these were scaled to take account of differences in production.

For this current study up-to-date information from four of the five pigment manufacturers has been gathered via the ICdA. Of these, sites C and D have provided information for 1999, with site A providing a copy of the 1996 data supplied to Atkins. One further company has indicated that they do not have any further data. Thus, Table A.1 in the Atkins report has been updated as in Table 4.4:

Table 4.4: Data from Pigment Preparation (updated Appendix 1 from WS Atkins Report)

Facility	Production	Loss to Air	Loss to Water	Loss to Solid Waste
A	230 d/yr	1.15 kg/yr	0.60 kg/yr 0.020 mg/l 131 m^3/d Rec. water flow: 6 Ml/d	180 kg/yr
B	231 d/yr	1.66 kg/yr	4.02 kg/yr 2.2 mg m^{-3} 213 m^3/hr Rec. water flow: 58.4 m^3 s^{-1}	49 kg/yr **
C	**276 d/yr**	**6.4 kg/yr**	**4.8 kg/yr** **0.06 mg/l** **2.3 l s^{-1}** **Rec. water flow: 135 Ml/d**	**39.6 kg/yr**
D	**230 d/yr**	**5.3 kg/yr ****	**2.6 kg/yr** **0.008 mg/l** **326,080 m^3/yr** **Dilution factor: 250**	**35 kg/yr ****
E	145 d/yr	0.2 kg/yr	13.4 kg/yr 0.044 mg/l 307,500 m^3/yr Rec. water flow: 1,040 m^3 s^{-1}	102 kg/yr **

** Calculated using maximum value of ratio of losses and production for pigment manufacturers

Bold entries indicate revised data

Source: Data in WS Atkins (1998a) amended with available information from industry.

One company reports a continual decline in emissions of cadmium since the 1960s and that emissions will approach zero in ten years time, when cadmium will be almost totally recycled.

The Atkins additional assessment (WS Atkins, 1998b) estimated that there were unacceptable local risks for pigment production facility D, where these arose through the atmosphere. Losses to air have been estimated as for the Atkins assessment (see above). (While this value has not changed, some of the values for site D have changed quite significantly, with greater losses to water but lower concentrations and also lower quantities emitted to solid waste).

On the basis of the above data, total losses associated with pigment manufacture are as indicated in Table 4.5.

Table 4.5: Annual Losses During Pigment Manufacture (830 tonnes of Cadmium)				
	Air	**Watercourse**	**Sewer**	**Solid Waste**
Atkins (kg/yr)	11.9	24.8	0	554
Revised (kg/yr)	14.7	25.4	0	406
Revised % of Use	0.002%	0.003%	0%	0.05%
Source: Data in WS Atkins (1998a) amended with available information from industry.				

Polymer Manufacture

Cadmium pigments may be incorporated into plastics by three routes:

- dry colouring, involving the mixing of pigment powder with polymer nibs, granules or powder. This mix is then injection moulded. This process is increasingly rare within the EU;
- a pre-dispersion of pigment in polymer compound, at a concentration required for the end use. It is estimated that between 5-10% of Cd pigments are used in this way, and typically this method is used for the high melting point engineering polymers such as polyamide and nylon); and
- a pre-dispersion pigment at a concentration higher than that for the end-use ('masterbatch' - typically about 25% by weight). Most usage of cadmium pigments in the EU is via masterbatch. The master batcher then supplies masterbatch to the moulder, who will add additional unpigmented polymer to achieve the required concentration in the end application.

There may be several further stages before the end product containing cadmium pigment reaches the public, but essentially the cadmium pigment is encapsulated in plastic once in the form of masterbatch, compound or moulded article.

As with stabilisers in PVC, WS Atkins (1998a) discounted emissions from plastic manufacture on the basis that these would be negligible compared with a single stabiliser manufacturer.

In the absence of site-specific emissions data, default values from the "Colourants" section of the Use Category Document for Plastic Additives (BRE, 1998) have been used. The main losses are associated with raw materials handling, where these are 1.6% for powders of particle size <40 μm. Losses during compounding are 0.05% and those during conversion 0.01%. Total losses are thus 1.66%, or 12.4 tonnes. While some losses would be to atmosphere, ultimately all losses will be to solid waste - as shown in Figure 4.3.

In-Service Use of Plastics

In addition to emissions during manufacture, cadmium may also be released during in-service use of pigmented plastics.

WS Atkins estimated total emissions of cadmium over a product's lifetime to be 0.0048% based on work by Bredereck (1983) on cadmium-stabilised window frames (see section 4.1.3). On the basis of 830 tpa of cadmium used in stabilisers this gave a release of 40kg which was assumed to be to soil. As indicated above, consultation with industry suggested that using a figure of 0.005% for the amount of cadmium leached during in-service use may over-estimate emissions (see Section 4.1.3). It is reported that once encapsulated, cadmium pigments are immobile and do not migrate through the polymer. Information provided in the use category document for plastic additives (BRE, 1998) puts forward a figure of 0.01% for leaching of pigments over the service life. In line with WS Atkins a figure of 0.005% has been adopted for losses of cadmium from the in-service use of pigmented plastics giving losses of about 35kg.

As for stabilisers, there may also be losses of cadmium occurring during installation. For the purpose of this assessment (and probably overly pessimistic) it is assumed that 0.05% of the pigment is lost during installation (as dust).

Formulation of Artists' Colours

Cadmium will be lost from the formulation of artists' colours. Losses at this stage of the life cycle were not considered in the Atkins assessment.

Information on losses for this report have been collated though consultation with the Artists' Colours Group of CEPE (the European Council of Paint, Printing Ink and Artists' Colours Industry). CEPE questioned the 18 members of the Group concerning emissions with responses being received from 12 of these (i.e. two thirds). A further two or three companies may use cadmium (though not all producers of artists' colours use cadmium). Those companies reported use of 25.6 tpa (to be compared with the overall value of 33 tpa used in this assessment). Thus, the amounts of paints used will be around 150 tpa.

Overall, releases are reported to be of the order of 1% of usage (and within limits set in EU and national legislation) and less in the case of manufacturing sites equipped with sewage treatment equipment which is generally the case.

Information provided by one manufacturer indicates that losses can be considerably higher in the case of solid waste. The company uses 2 tpa of cadmium metal is used, with losses of 6.4% (0.128 tpa). The majority of this goes to landfill as special waste (99.7% of losses), with only 0.037 kg going to sewer (0.03% of losses).

For the purposes of the assessment it has been assumed that 5% of the cadmium used in the production of artists' colours is lost to solid waste with negligible amounts to sewer (see Figure 4.3).

Use of Artists' Colours

Cadmium will be lost from artists' colours during use, for example through brush cleaning. Losses at this stage of the life cycle were not considered in the Atkins assessment.

For use of paints, CEPE states that cadmium colours are very expensive and the artist is likely to carefully control the amounts used and avoid wastage. The greatest part of the paint on a dirty brush is removed with rags; the tiny residual paint is removed either with solvents or with soap and water. While water-borne colours may disappear in drains as the wash-up of brushes with soap and water, the majority of cadmium containing colours is oil based and these would be less likely to be disposed of via drains. With respect to used tubes or containers, these are solid waste.

For the purposes of this assessment it has been assumed that 5% of cadmium used by artists finds its way into the sewer as a result of brush washing, etc. (i.e.1.58 tpa). It is further assumed that 20% of cadmium is disposed as solid waste on wipes (i.e. 6.3 tpa).

In relation to disposal, it has been assumed that 50% of paintings are retained while 50% are disposed of as solid waste.

Production and Use of Ceramics

Small amounts of cadmium will be lost from the production and use of ceramics. Losses at this stage of the life cycle were not considered in the Atkins assessment.

It is reported that cadmium-based glazes are applied as suspensions, with any excess being removed before the pottery passes to the furnace for firing. Releases to air will take place during firing. With respect to releases to water, systems are available whereby the area can be segregated and all wash or cleaning waters collected. Solids are separated out and the water recycled. This process then achieves a zero discharge to water and all the separated solids are returned for cadmium recovery. In terms of releases to solid waste, these will be associated with damaged pottery and other contaminated wastes (HMIP, 1993).

In the absence of any additional information the losses associated with ceramics production and use are currently assumed to be the same as for the production and use of pigmented plastics. While it may be argued that 'installation' losses are not so significant for ceramics, any reduction in losses (to solid waste) at this stage would be immediately countered by a corresponding increase in losses (to solid waste) at the product disposal stage.

4.1.5 Emissions from Metal Plating

Information from WS Atkins Reports

The WS Atkins report made estimates of releases of cadmium from cadmium plating facilities and from the in-service use of plated materials. Releases associated with end-of-life disposal to incinerator or landfill were also estimated.

One of the concerns highlighted by the CSTEE was that "the emissions from cadmium plated material are averaged out over the environment, both aquatic and terrestrial. They do not take account of the specific uses of the plated materials and emissions arising from these uses".

Cadmium Plating Facilities

Tables 4.6 summarises data from the WS Atkins report for emissions during metal plating production. The estimate of water-based releases was obtained from OECD (1994) and it was assumed that all water-based releases are to sewer. Emissions to solid waste included residual sludge and sludge generated in the treatment of plating solutions. Estimates of release levels were again taken from OECD (1994) with the assumption (from ICdA) that 30% of solid waste is recycled.

Table 4.6: Annual Losses of Cadmium from Plating Processes

Units	Air	Watercourses	Sewer	Solid Waste
kg/yr	Negligible	0	250	3,360
% of Use	Negligible	0%	0.13%	1.72%

Source: WS Atkins (1998a)

The part of the plating process with the potential to contribute most to releases to water is rinsing in the drag-out tank after plating to remove residual plating solution. To minimise carry over to the plating bath, some operators spray rinse and air blow components over the plating bath. Several methods exist to treat cadmium-containing water to such a degree that the water can be recycled as process water – essentially giving a 'zero emission to water' process. In such cases the only releases to water are from the regeneration or cleaning of ion exchange resin or membranes and such waste streams are themselves treated prior to release (HMIP, 1993).

With respect to releases to air, these may arise from adjustment of the plating baths which is carried out only occasionally. With respect to release to solid waste, these are minimised through the recycling of both cadmium scrap and cadmium-containing sludges from effluent treatment. In some cases it may not be possible to treat spent plating solutions on-site in which case these will be disposed of through authorised disposal sites (HMIP, 1993).

Consultation in the UK has confirmed that (excluding emissions of solid waste), emissions are exclusively to sewer, below the acceptable level in potable water and probably <50 kg/yr. The UK Environment Agency's Pollution Inventory gives the

values presented in Table 4.7 for emissions to sewer from seven companies which appear to be involved in cadmium plating

Table 4.7: Sample Emissions to Sewer from UK Plating Facilities	
Site	**Emissions (kg/yr)**
A	16 kg
B	2.5 kg
C	2.3 kg
D	1.76 kg
E	1.1 kg
F	1.012 kg
G	0.329 kg
Total	**25.0 kg**
Source: UK Environment Agency - 1998 Data	

On the basis of the above data it seems reasonable to assume losses of 50 kg emissions per year to sewer from the 21 sites in the UK and thus around 250 kg from all sites in the EU (i.e. the data in the WS Atkins report have been confirmed).

Consideration was also given to whether estimates of losses from the production of cadmium anodes should be included as part of the assessment. Consultation indicates that cadmium anodes are produced by cadmium producers and are not a separate stage in the life-cycle. As already pointed out by WS Atkins (1998a), levels of cadmium production would be the same through continued production of zinc, whether or not there was continued demand for cadmium, because it is a natural by-product. It is, therefore, considered justifiable to exclude emissions for the production of cadmium from the total emissions resulting from the production and loss through the specific use of cadmium compounds in pigments, stabilisers and in metal plating. As the production of cadmium metal is excluded from consideration in this assessment, estimates of losses from the production of cadmium anodes are not made.

Product Use

WS Atkins made an estimate of cadmium emissions over the lifetime of plated products based on work by Morrow (1996). 50% of cadmium is assumed to be lost from plated materials in abrasive conditions, while products in non-abrasive conditions are assumed to lose almost no cadmium. It was therefore assumed that 25% of the cadmium coating would be lost over the lifetime of the products, giving 49 tpa for the 195 tpa of cadmium used in the EU. This cadmium was assumed to be distributed over the EU to both the marine and non-marine environment, in the ratio of those two areas. Thus, 26 tpa were distributed to the marine environment (which, given the EU's risk assessment methodology, did not appear again in the assessment). Of the 23 tpa in the non-marine environment, 3% was assumed the be in watercourses and 97% to soil.

Since many of the components in question are used in aircraft, it seems a reasonable assumption that the cadmium lost would be evenly distributed across the EU.

However, since many of the components are used in landing gear, the quantities lost around airports may be relatively greater. As a worst case, it is assumed that all losses will be local to airports, using the same assumptions as Atkins for the relative areas of soil and water. This approach means that marine uses are excluded from consideration (but as indicated above, the European risk assessment methodology ignores such losses in any event). With respect to the relative proportions of abrasive versus non-abrasive environments, in the absence of additional information it is assumed that half are abrasive and half are non-abrasive (which equates to a 25% loss rate overall).

4.1.6 Solid Waste Disposal

Overview

Solid waste is produced during production, formulation and use, and products become solid waste at the end of their useful lives (unless re-used or recycled). Information on the level of releases from production and formulation activities was set out in previous sections. In this regard it should be noted that:

- solid waste arising from the production of stabilisers, pigments, artists' colours and ceramics and from metal plating activities are to controlled landfill; and
- of the cadmium emitted to sewer, 90% will be retained in the sludge of sewage treatment works, with 41% of this being landfilled, 11% incinerated and 47% spread onto agricultural soil.

All of the solid wastes arising from the following activities are assumed to end up in the municipal waste stream:

- cadmium-stabilised PVC product manufacture;
- installation losses associated with cadmium-stabilised windows;
- the production of cadmium-pigmented plastics and ceramics; and
- the in-use disposal of artists' materials (e.g. from wipes).

The WS Atkins report assumes that approximately 20% of municipal waste in the EU is incinerated and 80% disposed to landfill. This is confirmed by a recent study which indicates that the ratio between incineration and landfilling of mixed MSW within the European Union is 21 to 79 (Argus, 2000). On this basis the assumptions used in the Atkins assessment have been used in this report.

With respect to the final disposal of products, the following assumptions are made:

- stabilisers: under scenario 1 (current situation, no recycling) all windows become solid waste (assumed to contain 270 tpa of cadmium), while under scenario 2 (future situation, with recycling) 240 tpa of cadmium the cadmium in windows is recycled, with 30 tpa becoming solid waste;
- pigmented plastics and ceramics: all plastics enter the solid waste stream at the end of their useful lives;

- artists' materials: 50% of paints are displayed/stored on paintings while 50% are disposed of to solid waste; and
- cadmium-plated metals: 30% of cadmium is recycled, with 70% entering the solid waste stream.

With respect to the destination of these wastes, it is assumed that the 80:20 ratio for landfill to incineration will hold true for pigmented plastics and artists' materials[7]. It is however assumed that all ceramics and cadmium-plated materials will be landfilled (i.e. landfill is 100%).

With respect to cadmium-stabilised windows, it is assumed that most of these will become builders' wastes. On the basis that between 91% and 100% of building plastic waste entered landfill in 1995 (APME, 1997), it is assumed that the ratio of landfill to incineration is 90:10. (In the Atkins report, PVC waste resulting from construction and demolition activities was not considered separately, but it was assumed that the non-recyclable fractions were disposed of in the same proportions for municipal waste.)

Incineration

In terms of emissions of cadmium from incineration, WS Atkins (1998a) based their estimates of losses to air and landfill assuming a 95% retention of cadmium in ash (using an average of six EU Member States from a 1996 study). Based on information from the International Ash Working Group (IAWG, 1994) it was assumed that 15% of cadmium introduced into the incinerator would be found in the bottom ash and 80% in the fly ash (with 5% to air as above). With respect to ash disposal, fly ash was assumed to be disposed of in controlled landfills and bottom ash within a monofill compartment of a municipal landfill.

A more recent study has examined issues surrounding the cadmium content of incinerator ashes (Bertin Technologies, 2000). This reports that mixed solid waste may yield between 0.006 and 0.011 kg cadmium per tonne in the bottom ash of an incinerator, with bottom ash accounting for 300 kg/tonne of municipal solid waste and fly ash for 25 kg/tonne. Cadmium is reported to be almost always present in the fly ash. When fly ash is mixed with neutralisation products (dry and semi-dry systems with lime) heavy metals and trace elements, including cadmium, are almost fully recovered in the solid residue. When the wet process is used, with electrostatic precipitation yielding 96-99% filtration yield, the filter cake contains a large part of the cadmium produced by incineration. Filter cake is usually disposed of via landfill.

This new information does not indicate the need to adopt different assumptions from those used by WS Atkins.

[7] Consultation with the artists' colours industry has indicated that in some European areas municipalities collect 'chemical wastes' and that recommendations to dispose of colour waste through these channels usually appear in the technical information given by the artists' colours manufacturers.

Landfill

The WS Atkins report discussed a range of issues associated with the leaching of cadmium from landfill sites including leaching from pigmented plastics, cadmium plated materials, sewage sludge and incinerator ashes, and emissions from both municipal landfills and those accepting only controlled wastes.

One study has since reported concerning the leaching of a number of PVC compounds, including barium/cadmium-stabilised window frames (Argus, 2000). Experiments included a leaching test, lysimeter studies and an analysis of PVC within an existing landfill. While it was possible to conclude that PVC additives such as stabilisers will not be released completely after more than 20 years in landfill conditions, no clear interpretation of the behaviour of stabilisers in PVC under landfill simulating conditions was possible. No conclusions could be drawn regarding the source of cadmium found in the leachate or the possible contribution of the PVC to cadmium levels. The authors concluded that further research was necessary to explain stabilisers' behaviour and share of landfill emissions.

One further study has also reported concerning the leachability of incineration residues in landfill (Bertin Technologies, 2000). No direct relationship between the total content of cadmium and its leachability has been observed. Leachability depends on the chemical nature of the cadmium and the leaching conditions, for example it is possible that an increased level of chloride from PVC may cause an increase in leaching. It is reported that most leaching of cadmium from landfilled residues will occur during the initial stage when the chloride content is highest, because of the formation of soluble chloride/cadmium complexes.

As landfills are not addressed in the technical guidance, to determine the impact of cadmium present in landfills, Atkins considered data for cadmium concentrations in landfill leachate and compared these with background soil concentrations for example. In this regard, it was not possible to proportion the cadmium present in a municipal solid waste landfill (or in the leachate) between cadmium arising from the three applications of concern (i.e. stabiliser, pigments and plated products) and cadmium arising from other sources.

4.2 Human Exposure

4.2.1 Overview

Human exposure to cadmium is primarily influenced by dietary intake and, for occupational exposure, by the presence of cadmium fume/dust in the workplace. In broad terms, a 'tolerable' daily intake is of the order of 1 μg/kg of bodyweight (about 70 μg/day for an average adult).

4.2.2 Non-Occupational Exposure

Typical figures for cadmium intake by members of the public within the EU are summarised in Table 4.8.

Table 4.8: Cadmium Intake for Members of the Public	
Source	**Intake (μg/day)**
Air in urban areas	0.04
Drinking water (2 l/day)	1.0
Smoking (20/day)	1 - 2
'Typical' Diet (in EU countries)	10 - 20
Total	**10 or more**
Source: OECD, 1994	

As can be seen, the cadmium intake is dominated by dietary intake and some foodstuffs are cadmium 'rich' - such as shellfish, root vegetables and cereals - leading to certain population groups being at a higher risk. In areas of contamination, levels of cadmium may be much higher leading to daily intakes of >100μg (Järup, *et al*, 1998). Although cadmium use in stabilisers, pigments and plating will make a contribution to these figures, other sources may be more significant.

4.2.3 Occupational Exposure

Apart from the types of exposure outlined above, workers involved with the handling and use of cadmium may be exposed to cadmium dust and/or fume. The primary route for human uptake is via inhalation of workplace air. By way of example, air concentrations in the workplace of the order of 10-50 $\mu g/m^3$ would lead to a daily uptake of 25-125 μg (OECD, 1994).

WS Atkins (1998a) provided details on workplace exposures at stabiliser and pigment preparation facilities, stabiliser mixing facilities and plating facilities. Of those few facilities that provided information on air concentrations, it was found that the levels were consistent with those given above (i.e. 10-50 $\mu g/m^3$) - although those in plating facilities were much lower at around 1 $\mu g/m^3$. Overall (although no data from plating facilities were provided) it was found that cadmium levels in workers' blood were less than 5 μg/l - a value to be compared with a typical value of 1 μg/l amongst the general population (levels for smokers are about twice those for non-smokers).

Figure 4.1: Cadmium Losses from Stabilisers - Scenario 1: No Recycling

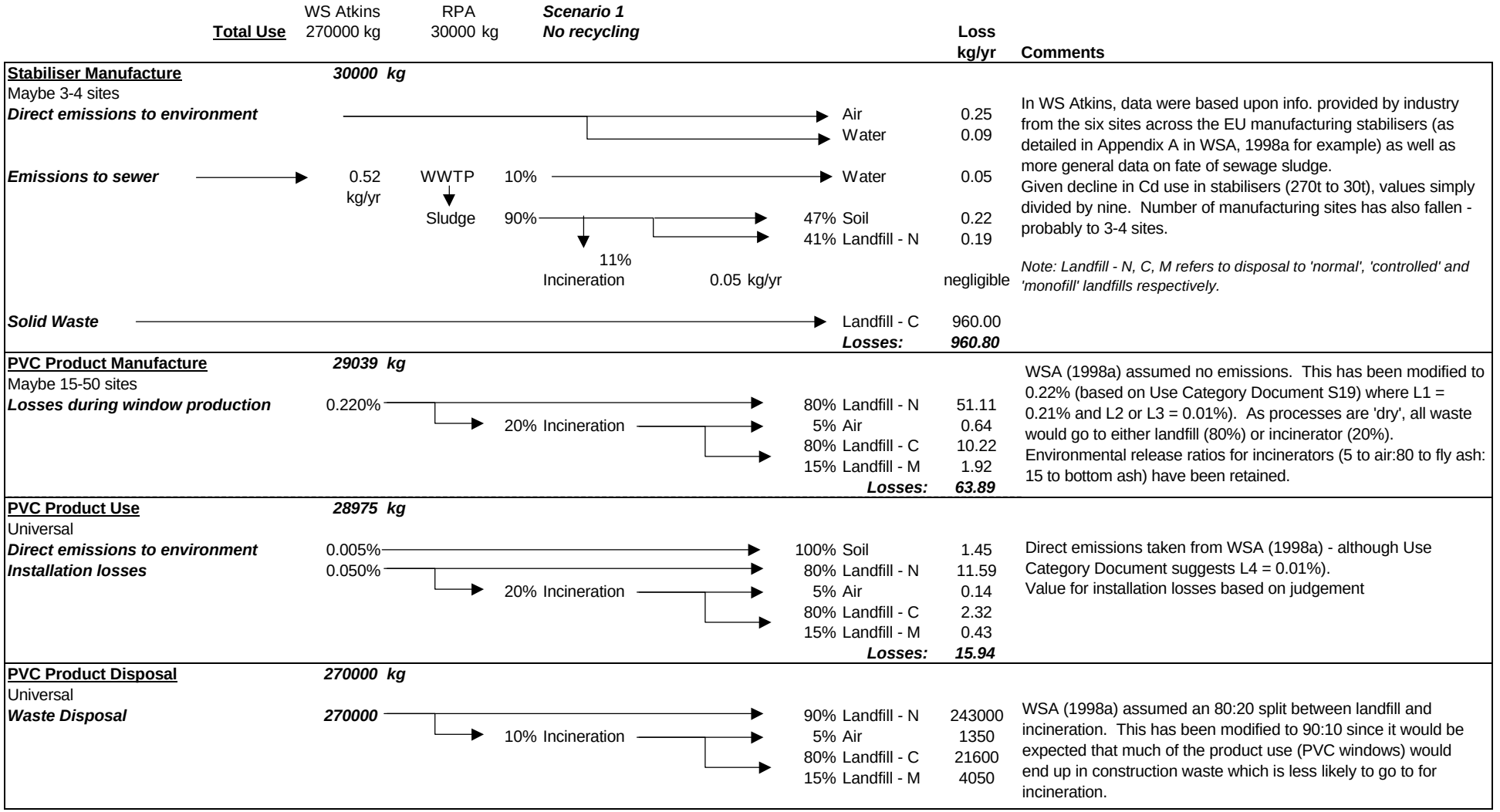


Figure 4.2: Cadmium Losses from Stabilisers - Scenario 2: Much Recycling

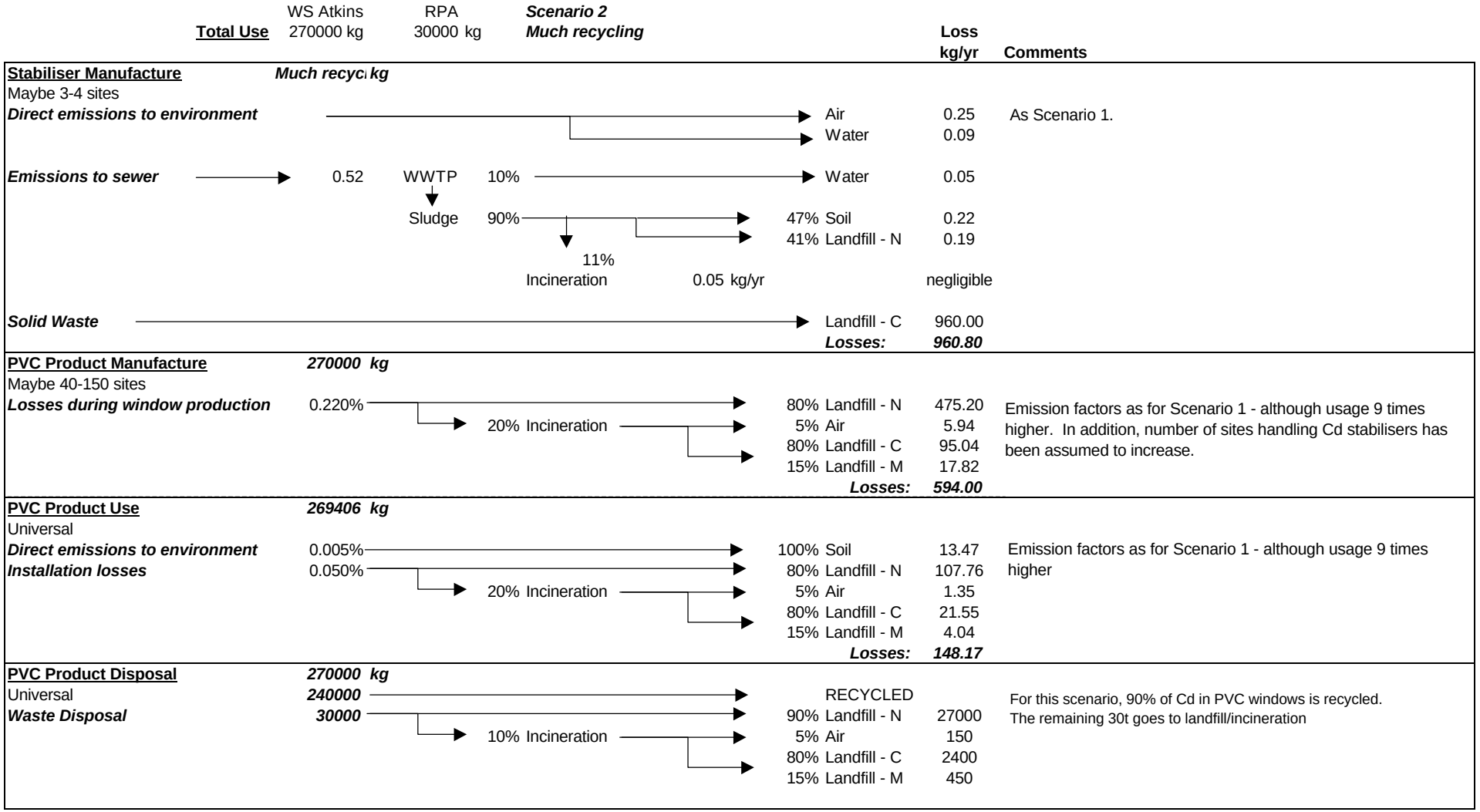


Figure 4.3: Cadmium Losses from Pigments

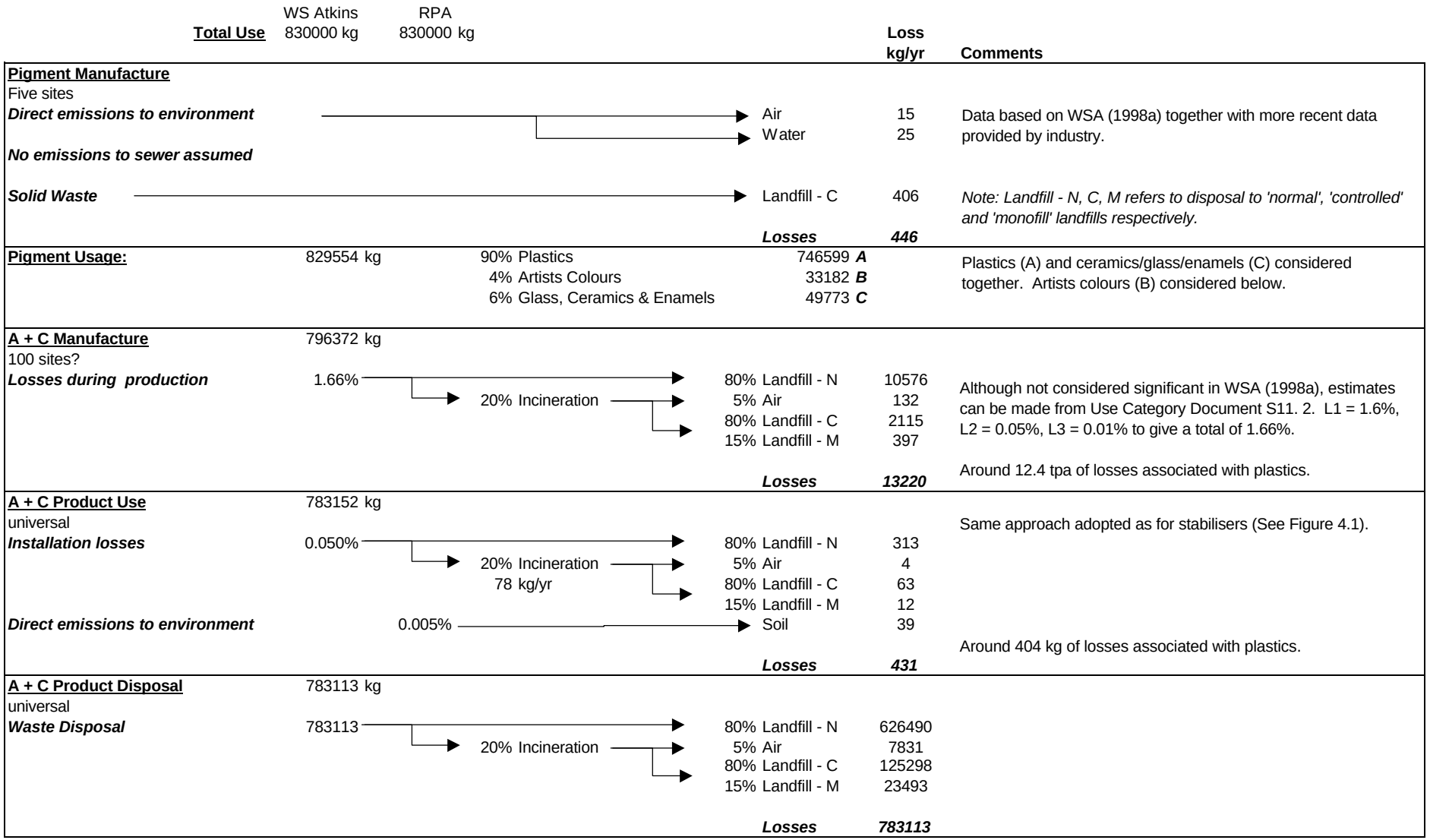


Figure 4.3 (cont.): Cadmium Losses from Pigments - Artists' Colours

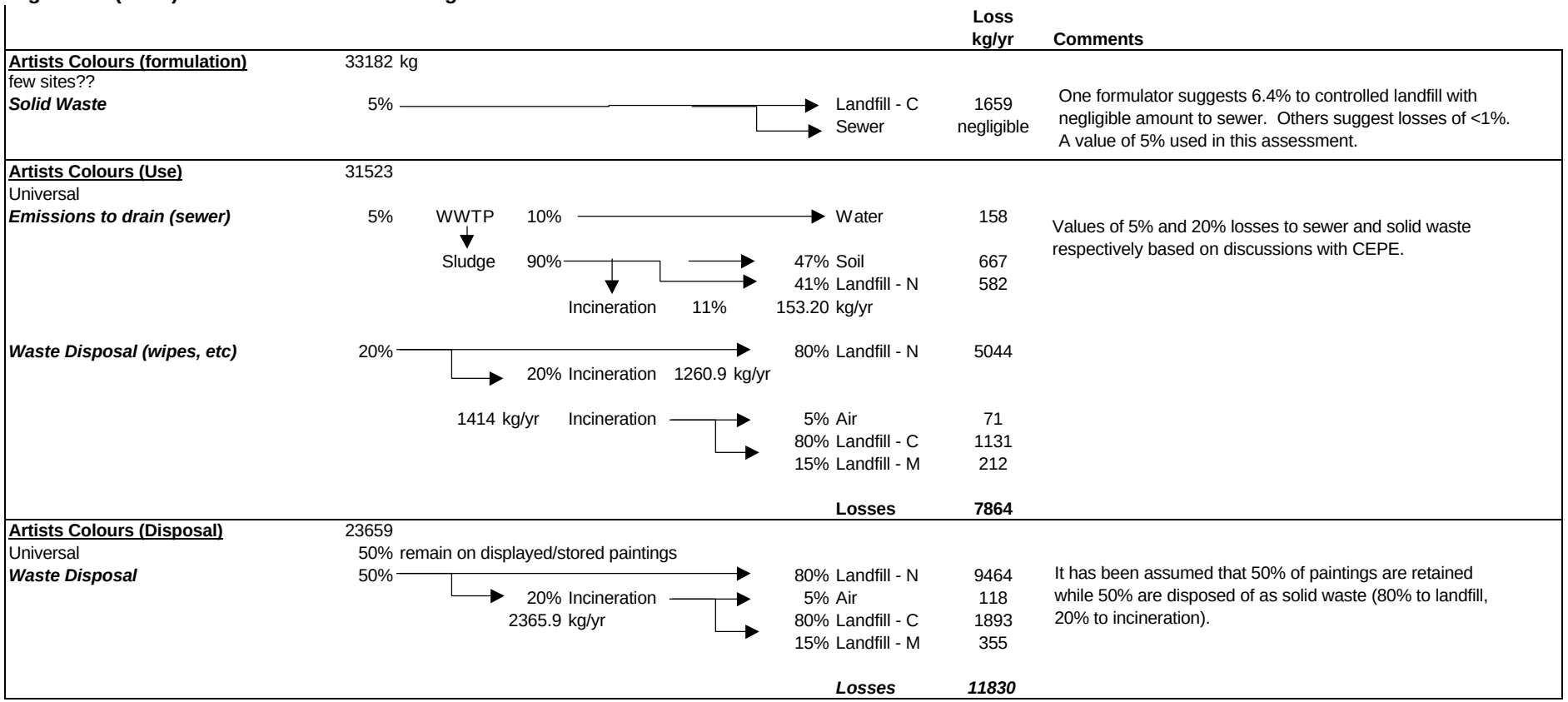
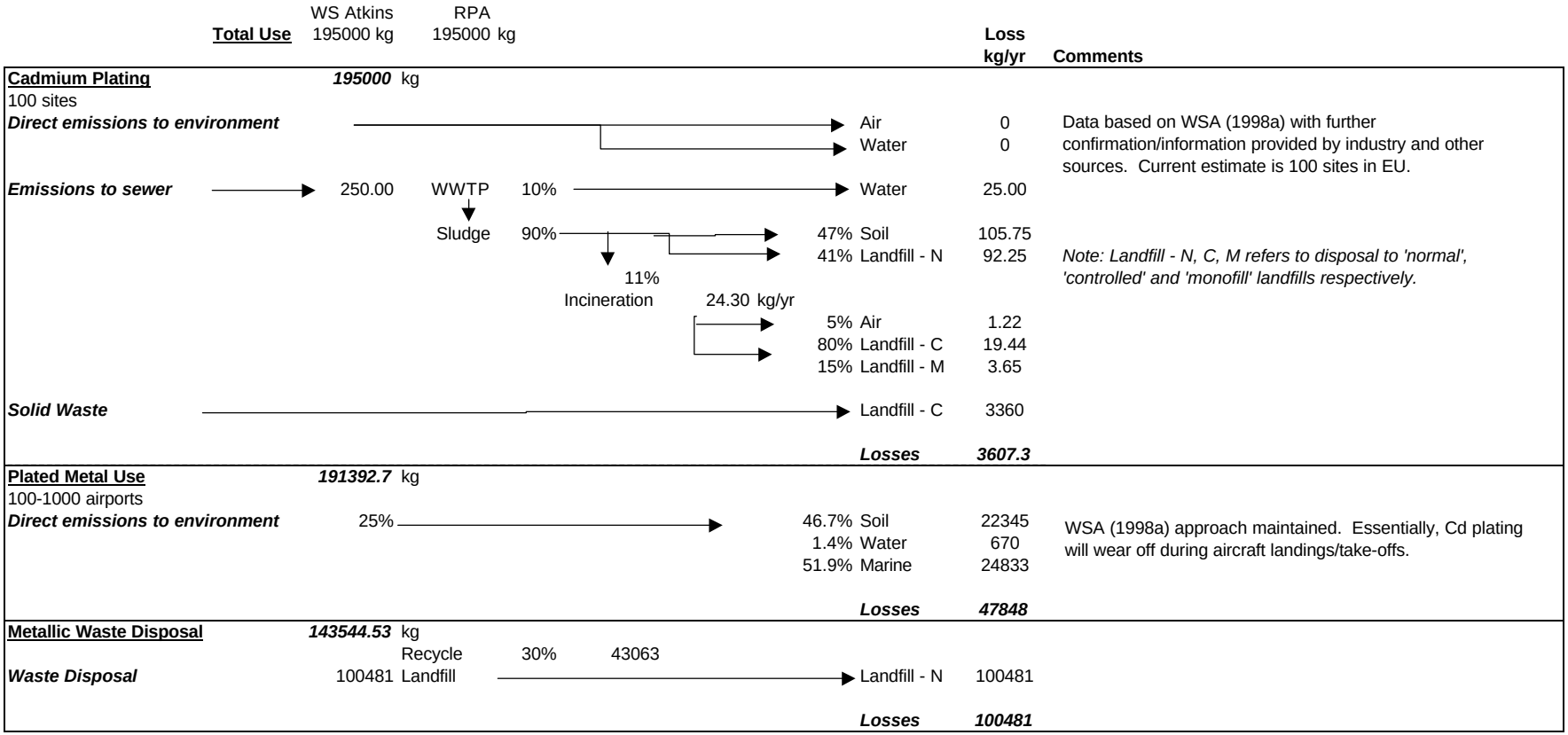


Figure 4.4: Cadmium Losses from Metal Plating



5. CALCULATION OF PREDICTED ENVIRONMENTAL CONCENTRATIONS (PECS)

5.1 Overview

Consideration has been given to the calculation of the Predicted Environmental Concentrations (PECs). Metals have a long residence time and complicate the way that existing models, including EUSES, can be used to predict or assess levels in different environmental compartments. There are three components to the total concentration of cadmium in the environment:

- local emissions (sites where releases of cadmium take place);
- anthropogenic background (historical background levels of cadmium resulting from national or regional anthropogenic activities); and
- natural background (natural sources of cadmium in the environment).

The background and existing concentrations of cadmium have been summarised by WS Atkins (Tables 3.1 and 3.2, WS Atkins, 1998a). WS Atkins estimated PECs for various compartments (WS Atkins, 1998a and 1998b) using methods similar to those outlined in the EC's Technical Guidance Document (EC, 1996). In this section, the data used for some of the important modelling parameters are discussed and suitable values are selected for use in the EUSES modelling program. Where appropriate, reference has been made to the data review presented in the Draft Risk Assessment Report for Cadmium, being produced by the Belgian Competent Authority under the EU Existing Substances Regulation (De Win *et al*, 1999).

The methods outlined in the TGD/EUSES have been used, with suitable modifications where appropriate, to estimate cadmium concentrations in the environment at both the local and regional scales from the sources identified in this report. As the behaviour of cadmium in the environment is dependent on properties such as pH, amongst others, two different environmental conditions are considered in the calculations. These are considered to be an acidic environment, where the availability and mobility of cadmium is maximised, and the 'general' environment (pH>7).

5.2 Partition Coefficients

5.2.1 Overview

WS Atkins (1998a) did not appear to define the partition coefficients to be used in the PEC calculations directly. Instead, residence times of cadmium in various soil and sediment systems were estimated, which deviated somewhat from the approach laid down in the TGD. Here, a different approach is taken in that the TGD/EUSES is used to define the residence time of cadmium in the system, once appropriate partition coefficients etc. are known. Thus the approach taken here is in line with that recommended in the TGD.

The methods for estimating concentrations in the TGD (as implemented in the EUSES program) require knowledge of certain partition coefficients. Normally these are estimated from the log Kow of a substance, but for cadmium such methods are not applicable and so actual or more realistic values have to be obtained in order to carry out the environmental modelling. The relevant values are discussed below.

5.2.2 Suspended Sediment-water Partition Coefficient (Kp_{susp})

Table 5.1 contains the values that were reported in the Draft EU Risk Assessment Report (RAR) for cadmium. A value of 130,000 l/kg was used in the RAR, being the approximate mean value for the results obtained in EU waters. This appears to be a reasonable value. Factors that were reported to be important in influencing the actual value for the Kp_{susp} are the pH, total metal concentration, the water hardness and the presence of complexing agents. In order to take this into account, the lower end of the range e.g. 7,900 l/kg will also be considered to apply to some situations, for example acidic waters where the availability of cadmium may be increased.

Table 5.1: Suspended Sediment-Water Partition Coefficients

$Kp_{(susp)}$ (l/kg)	**Location**	**Reference**
Mean 170,000 Range 28,000-280,000	Flanders	VMM, 1997
Mean 129,000	Four locations in the Netherlands, 1983-1986	Crommentuijn et al, 1997
Mean 151,000	Four locations in the Netherlands, 1988-1992	
Mean 224,000	Four locations in the Netherlands, 1992-1994	
Range 30,000-300,000	Rhine, Meuse and Schelde rivers, the Netherlands	Ros and Slooff, 1990
Mean 100,000 Range 7,900-794,000	St. Lawrence River basin, 1991-1992	Quemerais and Lum, 1997

Information reported in WS Atkins (1998a) indicated that 10-40% of cadmium present in surface water is in solution, with the remainder being associated with particulate or colloidal matter. In addition, it was also reported that the fraction of cadmium in the dissolved phase may increase with acidity, with 90% being associated with the dissolved phase in one report (Lithner and Borg, 1996), although the basis behind this value appeared uncertain.

Using the methodologies given in the TGD a Kp_{susp} of 130,000 l/kg implies that around 67% of the cadmium in water will be associated with the suspended sediment phase, with around 33% in the dissolved (solution) phase. Similarly, a Kp_{susp} of 7,900 l/kg implies around 11% will be associated with the suspended sediment phase and 89% will be in the dissolved (solution) phase. These values are in agreement with the information reported by WS Atkins and indicate that the values for Kp_{susp} chosen here are appropriate for use in the EUSES modelling for cadmium.

5.2.3 Sediment-water Partition Coefficient (Kp_{sed}).

Crommentuijn et al (1997) gives mean values for Kp_{sed} for the Netherlands as 85,100 l/kg (estimated from suspended matter partition coefficients) and 79,430 l/kg (based on measurements of sediments at 3 locations in the Netherlands). The value for Kp_{sed} of 85,100 l/kg will be used in the EUSES modelling for cadmium in the general environment. For the acidic environment, the value of Kp_{sed} will be taken to be around 7,900 l/kg as above.

5.2.4 Soil-water Partition Coefficient (Kp_{soil})

The cadmium RAR (De Win, 1999) gives several equations relating the soil-water partition coefficient to various properties of the soil. These are reproduced in Table 5.2.

Table 5.2: Soil-Water Partition Coefficients

Relationship		Comments	Reference
$\log Kp_{soil} = -1.00 + 0.51pH + 0.51\log(\%OM)$	A	n=63 – Danish agricultural soils, including subsoils	Christensen, 1989
$\log Kp_{soil} = 0.89 + 0.52pH + \log(\%OM/100)$	B	n=15 – Soils from New Jersey	Lee et al, 1996
$\log Kp_{soil} = -1.8 + 0.59pH + \log(\%OM)$	C	n=33 – Dutch, French and British soils	Gerritse and Van Driel, 1984
$\log Kp_{soil} = -1.16 + 0.56pH$	D	n=100 – Agricultural and forest soils from the Netherlands	Römkens and Salomons, 1998
$\log Kp_{soil} = -1.34 + 0.64pH$	E	n=28 – Grassland soils from Belgium (in situ) measurements	De Win *et al* (1999)
$\log Kp_{soil} = -2.02 + 0.60pH + 0.96\log(\%OC)$	F	n=58 – Grassland soils from Belgium	De Win *et al* (1999)
Note: OM = organic matter content *OC = organic carbon content*			

From the Technical Guidance Document, the default values are organic matter content of 3.4% or an organic carbon content of 2%. Using these values in the above regression equations, Kp_{soil} values can be estimated for soils of differing pH. These are shown in the Table 5.3.

As can be seen from these values, the Kp_{soil} decreases with increasing acidity. This means that cadmium is more mobile in acidic soils than neutral and alkaline soils.

For the EUSES modelling, a value for Kp_{soil} of 3,029 l/kg will be used for the general environment and a value for Kp_{soil} of 59 will be used for the acidic environment.

Table 5.3: Variation of Estimated Kp_{soil} with pH				
Equation (from Table 5.2)	**Estimated Kp_{soil} (l/kg)**			
	pH 5	**pH 6**	**pH 7**	**pH 8**
A	66.2	214	694	2,244
B	105	348	1,152	3,815
C	48.0	187	727	2,828
D	43.7	158	575	2,089
E	72.4	316	1,380	6,026
F	18.6	74.0	294	1,172
Mean value	**59**	**216**	**804**	**3,029**

5.3 Behaviour during Biological Waste Water Treatment

The Simpletreat waste water treatment plant model used in the TGD and also the EUSES program requires estimates for various suspended matter-water partition coefficients. These partition coefficients are usually similar to, but higher than, the suspended sediment-water partition coefficient used in the assessment, due to the higher organic matter content of the suspended matter in the waste water treatment plant. Since equivalent measured values are not available for cadmium, the Simpletreat model was run using the value of 130,000 l/kg for these partition coefficients. This gave the following behaviour in the waste water treatment plant: 90% to sludge and 10% to water.

Information reported in WS Atkins (1998a) is in good agreement with the Simpletreat estimates above, and so these values will be used in the PEC calculations where appropriate (and, indeed, were used in deriving Figures 4.1 to 4.4)

5.4 Uptake by Biota

5.4.1 Overview

In order to use the TGD approach to risk assessment, bioconcentration factors or transfer factors are needed for various parts of the environmental and human food chain. The most important of these factors concern the uptake by fish from water, uptake by earthworms from soil, uptake by plants (grass, leaf crops, root crops) from soil, uptake by plants (grass, leaf crops, root crops) from air, and transfer from grass/food to cattle (meat and milk). These are discussed in the following sub-sections.

5.4.2 Uptake by Fish from Water (BCF_{fish})

WS Atkins (1998a) used a fish BCF of 38 l/kg (on a fresh weight basis) as a representative value for the waters of around pH 7.7, as would be typical for the general EU environment. This was the geometric mean value from a review of data

for freshwater and marine fish carried out by Van der Plassche *et al* (1994). In addition to this, WS Atkins (1998a) considered a higher value for BCF_{fish} of 3,000 l/kg (on a dry weight basis) as being a worst case value for acidic waters, but did not give the source of this information. This BCF_{fish} was estimated to be around 750 l/kg (on a fresh weight basis) by using a dry weight to fresh weight conversion factor of 4.

Many studies have investigated the bioconcentration of cadmium in freshwater fish and other freshwater aquatic organisms. These are reviewed in the draft RAR for cadmium. For fish, the whole fish BCF values determined on wet fish weight basis ranged between 0.51 l/kg and 511 l/kg, with a median value of around 15 l/kg. The whole fish BCF values determined on a dry fish weight basis were in the range 5 to 1,385 l/kg, with a median value of 80. For algae, the BCFs determined were in the range 1,640-23,100 l/kg (median value 7,500 l/kg) for the wet weight values and 2,200-310,000 l/kg (median value 115,100 l/kg) for the dry weight values. Finally, the BCF values for invertebrates were in the range 396-17,560 l/kg (median 994) for the wet weight values and 546-33,300 l/kg (median 5,000 l/kg) for the dry weight values. There was some evidence of decreasing uptake with increasing concentration of cadmium in the water phase (above around 10 µg/l), which resulted in generally lower BCF values at higher cadmium exposure concentrations.

Taylor (1983) presents results from over 40 laboratory investigations (and displayed the range of results found for freshwater organisms and marine organisms in graphical form). The results were split between four main groups: algae, vertebrates (i.e. fish), mollusca and crustacea. The following ranges of BCF values for the groups can be read from the graphs. All the BCF values used in the study were wet organism weight values, and are whole organism values or, in a small number of cases, edible tissue values.

	Fresh water organisms	*Marine organisms*
Alga	*~10-10,000 l/kg wet wt.*	*~70-800 l/kg wet wt.*
Mollusca		*~4-5,000 l/kg wet wt.*
Crustacea	*~10-1,500 l/kg wet wt.*	*~2-1,000 l/kg wet wt.*
Fish	*~1-3,000 l/kg wet wt.*	*~1-4 l/kg wet wt.*

From these data, it appears that this review may have been the source of the BCF of 3,000 l/kg reported in the WS Atkins (1998a) report, although it is clearly a wet fish weight value. The Taylor (1983) review concluded that although values as high as 10,000 l/kg wet wt. have been found in some algal experiments, the vast majority of BCF values are <1,000 l/kg wet wt. They calculated the median BCF for all the organisms included as 90 l/kg wet wt. for freshwater species and 40 for marine species. When the fresh water fish data were considered, although BCF values up to 3,000 l/kg wet wt. had been measured, around 60% of the measured BCF values were <20 l/kg wet wt. The data set was much smaller for marine fish, but BCF values found were all <5 l/kg wet wt.

Factors that may affect the uptake of cadmium by organisms from water include pH and water hardness (and other factors which affect the availability of cadmium in solution). However such factors may have differing effects depending on the species in question. For example, the high BCF found in some experiments with algae could be as a result of adsorption of cadmium onto the cell walls, rather than uptake into the

cells. The ability for cadmium to adsorb onto cell walls could vary with these factors in a different manner to the cadmium that is taken into the cells, or is taken up by other organism (e.g. uptake via the gills in fish). For example, the draft Cadmium RAR gives examples of increasing accumulation of cadmium by willow moss (*Fontinalis antipyretica*) with increasing pH (Lithner *et al*, 1995). Increasing pH would be expected to reduce the bioavailability of cadmium. Similarly, the draft Cadmium RAR gives examples of lower rates of uptake of cadmium by *Daphnia magna* from water with a hardness of 30 mg $CaCO_3$/l compared to water with a hardness of 6 mg $CaCO_3$/l (Penttinen et al, 1995), whereas for an alga (*Selenastrum capricornutum*), no significant change in bioconcentration of cadmium was seen over the hardness range 57 to 230 mg $CaCO_3$/l.

For the EUSES modelling, a fish BCF of 38 l/kg wet wt. will be used for the general environment and a higher value of 3,000 l/kg wet wt. will be used for the acidic environment as a worst case.

5.4.3 Uptake by Earthworms from Soil

This did not appear to have been considered in the WS Atkins (1998a) report.

The draft Cadmium RAR reviews a large amount of data relating the concentrations in worms (all worms in the studies had their guts voided prior to analysis) to the concentrations found in soil. Most of these data are from field studies. The accumulation factors derived in the draft RAR were calculated on either a wet earthworm weight or dry earthworm weight basis as follows (the soil concentrations used were always dry soil weight values):

$$\mathrm{AF} = \frac{[\text{concentration in earthworms (mg/kg wet weight or mg/kg dry weight)}]}{[\text{concentration in soil (mg/kg dry weight)}]}$$

For the data determined from the wet earthworm weight data (17 data points), the range of accumulation factors was between 4 and 32.4, with a median value of 14.9. For the data determined from the dry earthworm weight data (79 data points), the range of accumulation factors was between 1.6 and 151.4, with a median value of 15.5.

The TGD uses a factor of 0.16 to convert data from a dry earthworm weight basis to a wet earthworm weight basis (i.e. the earthworm is 84% water). Thus the maximum of the dry earthworm weight accumulation factor from above (151.4) would be equivalent to a wet earthworm weight value of around 24, which is similar to the upper end of the range found with the wet weight data.

Generally, it was found that the accumulation factor decreased with increasing cadmium concentration. The accumulation factor was also dependent to some extent on the soil properties, generally increasing with decreasing pH, cation exchange capacity and organic carbon content.

For the EUSES modelling, the highest measured accumulation factor of 32.4 kg/kg will be used for the acidic environment. A slightly lower value of 15 kg/kg will be

used for the general environment to reflect the fact that the uptake appears to decrease with increasing pH, amongst other factors. The measured values above are based on dry soil weights. As the TGD default conversion factor between dry soil weights and wet soil weights is relatively small (and certainly much smaller than the uncertainties inherent in choosing a representative value for the accumulation factor), it will be assumed that the chosen factors will apply equally well to wet soil concentrations.

5.4.4 Uptake by Plants from Soil

WS Atkins (1998a) used a soil-grass transfer factor of 0.7 (on a dry weight plant/wet weight soil basis). This was derived from a four year field study looking at the transfer of metals from soil contaminated with smelter flue dust (cadmium assumed to be present as cadmium oxide). A conversion factor of 4 (for the dry weight to fresh weight conversion for grass) was used to give a value of 0.18 on a fresh weight plant/fresh weight soil) basis. This was reported to be in good agreement with the transfer factor for leafy green vegetables derived by Dorgelo (1998) used in the Netherlands to determine an intervention level for soil remediation. This was used as the transfer factor for grass and crops in the generalised (pH 7.7) environment.

For acidic soils, the WS Atkins report used a value of 0.88 of a fresh weight plant/wet weight soil basis. This was based on the observations of Andersson & Nilsson (1974), who found a five times increase in transfer of cadmium from soil to fodder rape grown in pots using sludge amended soil, when the soil pH was decreased from 7.2 to 4.8.

WS Atkins did not specifically consider the uptake of cadmium by root crops from soil. The draft RAR for Cadmium reviewed a large body of information on the levels of cadmium in plants compared to the levels in soil. The data was generated from the average concentrations found in crops/average concentration found in soil. The soil-plant transfer factors determined in that draft RAR are summarised in Table 5.4. All values are reported on a fresh plant weight basis (it is not clear if the soils are on a wet or dry weight basis, but, as explained earlier, this correction would make only a small difference to the values reported).

Table 5.4: Soil-Plant Transfer Factors	
Plant	**Soil-plant transfer factor (on a fresh plant weight basis)**
Wheat (grain)	0.055-0.20
Potato tuber	0.07-0.19
Carrot	0.08-0.11
Lettuce	0.10
Spinach	0.15
Leek	0.28 (pH<5) 0.032 (pH>5.5)
Cabbage	0.01
Cauliflower	0.15
Tomato	0.025
Onion	0.032

The draft RAR for Cadmium also reported several regression equations relating the soil-plant transfer factor to various soil properties. These are shown in Table 5.5

Table 5.5: Regression Equations for Soil-Plant Transfer Factors.		
Plant	**Soil-plant transfer factor (TF) (on a fresh plant weight basis)**	**Reference**
Wheat (grain)	$TF = \frac{(92 - 10.3 \times pH + 0.10 \times [Cdsoil] - 0.26 \times [Znsoil])}{290}$	Eriksson et al, 1996
	$TF = \frac{(181 - 27 \times pH)}{100}$ for pH 5-6.2 $TF = 0.1$ for pH >6	Gavi et al, 1997
Potatoes	$TF = \frac{(193 - 24 \times pH - 0.94(\%OM) + 0.039 \times [Cdsoil])}{270}$	Eriksson et al, 1996
Carrots	$TF = \frac{(3.39 - 0.29 \times pH - 0.01(\%C) + 3.5 \times 10^{-4} \times [Cdsoil])}{300}$	Jansson and Öborn, 1997
Note: *[Cdsoil] = cadmium soil concentration in µg/kg* *[Znsoil] = zinc concentration in mg/kg = average value from study 42 mg/kg* *%OM = organic matter content of the soil* *% C = organic carbon content of the soil*		

Using these equations, the variation of the value of the soil-plant transfer factor with pH can be investigated. This is shown in Table 5.6, assuming a [Cdsoil] of around 500 µg/kg and the TGD default organic carbon and organic matter contents (2% and 3.4% respectively). The estimates obtained from the equation reported in the draft RAR for carrots (Jansson & Öborn, 1997) appear to be out of line with the other estimates. The original paper has been obtained and the equation given in that paper relates the cadmium concentrations in carrots (in mg/kg on a dry weight basis) to the cadmium concentration in soil (in mg/kg on a dry weight basis) in the following way:

$$\log[\text{carrot concentration}] = 4.35 - 0.29 \times \text{pH} - 0.01 \times (\%\text{C}) + 0.35 \times [\text{Cdsoil}]$$

However, there is some uncertainty over this equation as the data presented in the paper do not appear to be consistent (e.g. the cadmium concentrations in carrots were reported to be in the range 0.062-0.87 mg/kg, yet the graphs showing the correlation with pH etc. indicate that the log [carrot concentration] were in the range 1.5-3, which would imply that the concentration units used in the regression were not mg/kg as stated in the paper but µg/kg). Therefore this equation is not considered further.

Table 5.6: Variation of Soil-Plant Transfer Factor with pH.

Equation	Soil-plant transfer factor (on a fresh plant weight basis)			
	pH 5	pH 6	pH 7	pH 8
Wheat; Eriksson *et al*, 1996	0.27	0.23	0.20	0.17
Wheat; Gavi *et al*, 1997	0.46	0.19	0.1	0.1
Potatoes; Eriksson *et al*, 1996	0.33	0.24	0.15	0.06

The available measured soil-plant transfer factors are not in a form that can be readily incorporated into the EUSES model. However, the factors can still be used to estimate concentrations resulting in crops from uptake via soil in a similar way as recommended in the TGD. These calculations are shown in the Annex 4. The following soil-plant transfer factors are used in these calculations: soil-plant transfer factor for root crops = 0.15 kg/kg for the general environment and 0.3 for the acidic environment; soil-plant transfer factor for leaf crops and grass = 0.1 kg/kg for the general environment and 0.2 for the acidic environment. These are based on the data reported in Tables 5.4 and 5.6, and are again assumed to be applicable to wet weight soil concentrations.

5.4.5 Uptake by Plants from Air

This did not appear to have been considered by WS Atkins (1998a) nor were actual air-plant transfer coefficients reported in the draft RAR for Cadmium, and it is not possible to estimate these using the methods outlined in the TGD (which rely on the log Kow value).

The majority of the data on soil-plant transfer has been generated from field studies, where air exposure (transfer) would also be occurring. Therefore, in most cases the soil-plant transfer factor will also include the atmospheric contribution. The exception to this may be where high air levels are found and are dominant over the uptake from soil. However, in terms of the procedures outlined in the TGD, this situation is unlikely to occur as the local assessment assumes that the atmospheric cadmium also contributes to the local soil concentration by atmospheric deposition. Thus, if air concentrations are predicted to be high, the soil concentrations will also be elevated due to this deposition. Therefore, it appears to be sensible to base the estimated concentrations in plants on the soil-plant transfer factors only.

5.4.6 Uptake by Cattle from Food (BTF_{meat}) and Transfer to Milk (BTF_{milk})

WS Atkins (1998a) used transfer factors of 2×10^{-2} d/kg and 3×10^{-4} d/kg for the transfer factors to meat (BTF_{meat}) and milk (BTF_{milk}) respectively. These were the values recommended in a review undertaken by Morgan (1991). As no further data were reported in the draft risk assessment report on Cadmium, these values will be used in the EUSES calculations. As these factors represent transfer from diet to meat and milk, they will be the same for both the acidic and general environment.

5.5 Calculation of PECs

5.5.1 Inputs to EUSES

PECs for the various endpoints considered have been estimated using the methodology outlined in the TGD, as implemented in the EUSES program. In order to run the EUSES program in a meaningful way for cadmium, representative values have to be chosen for many of the partition coefficients and rate constants within the program as some of the estimation methods used within EUSES are not relevant for cadmium. Table 5.7 outlines the values chosen for the assessment, based on the information reported above.

Table 5.7: EUSES Model Parameters used for Cadmium

Input to EUSES program	Value chosen for cadmium	
	General environment	Acid environment
Physico-chemical properties		
Molecular weight (g/mole)	112.41	112.41
Melting point (^oC)	320.9	320.9
Boiling point (^oC)	765	765
Vapour pressure at 25^oC (Pa)	1×10^{-6} (in terms of the model, Henry's law constant is more important than this value)	1×10^{-6} (in terms of the model, Henry's law constant is more important than this value)
Octanol-water partition coefficient (log value)	-1 (value not important as all derived partition coefficients are overwritten)	-1 (value not important as all derived partition coefficients are overwritten)
Water solubility (mg/l)	9.6	9.6
Solids-water partition coefficient		
Organic carbon-water partition coefficient	not used	not used
Solids-water partition coefficient in soil (Kpsoil; l/kg)	3,029	59
Solids-water partition coefficient in sediment (Kpsed; l/kg)	85,100	7,900 (a)
Solids-water partition coefficient in suspended sediment (Kpsusp; l/kg)	1.3×10^5	7,900
Suspended matter-water partition coefficient (Ksusp-water; m^3/m^3)	3.25×10^4 (estimated by EUSES from Kpsusp)	1.98×10^3 (estimated by EUSES from Kpsusp)
Soil-water partition coefficient (Ksoil-water; m^3/m^3)	4.54×10^3 (estimated by EUSES from Kpsoil)	88.3 (estimated by EUSES from Kpsusp)

Table 5.7: EUSES Model Parameters used for Cadmium		
Input to EUSES program	**Value chosen for cadmium**	
	General environment	**Acid environment**
Sediment-water partition coefficient (Ksed-water; m^3/m^3)	4.26×10^4 (estimated by EUSES from Kpsediment)	3.95×10^3 (estimated by EUSES from Kpsusp)
Solids-water partition coefficients in raw sewage sludge, settled sewage sludge, activated sewage sludge and effluent sewage sludge	not used – behaviour during waste water treatment overwritten	not used – behaviour during waste water treatment overwritten
Air partitioning coefficients		
Sub-cooled liquid vapour pressure	not used – Henry's law constant overwritten	not used – Henry's law constant overwritten
Fraction of chemical associated with aerosol particles	1	1
Henry's law constant (Pa m^3/mole)	1×10^{-9} – very low value entered as little potential for volatilisation	1×10^{-9} – very low value entered as little potential for volatilisation
Air-water partition coefficient (m^3/m^3; dimensionless form of Henrys Law constant)	4.22×10^{-13}	4.22×10^{-13}
Degradation		
Characterisation of biodegradability	Assumed no degradation – used default rate constants for non-degradable substances	
Rate constant for degradation in air (day^{-1})	0	0
Total rate constant for degradation in surface water (day^{-1})	1.39×10^{-6} (default value – estimated by EUSES)	1.39×10^{-6} (default value – estimated by EUSES)
Total rate constant for degradation in bulk sediment (day^{-1})	6.93×10^{-8} (default value – estimated by EUSES)	6.93×10^{-8} (default value – estimated by EUSES)
Total rate constant for degradation in bulk soil (day^{-1})	6.93×10^{-7} (default value – estimated by EUSES)	6.93×10^{-7} (default value – estimated by EUSES)
Rate constant for volatilisation from agricultural soil (day^{-1})	5.58×10^{-14} (estimated by EUSES from Henry's law constant and Ksoil-water)	2.86×10^{-12} (estimated by EUSES from Henry's law constant and Ksoil-water)
Rate constant for volatilisation from grassland (day^{-1})	1.12×10^{-13} (estimated by EUSES from Henry's law constant and Ksoil-water)	5.71×10^{-12} (estimated by EUSES from Henry's law constant and Ksoil-water)
Rate constant for leaching from agricultural soil (day^{-1})	5.28×10^{-7} (estimated by EUSES from Ksoil-water)	2.70×10^{-5} (estimated by EUSES from Ksoil-water)
Rate constant for leaching from grassland (day^{-1})	1.06×10^{-6} (estimated by EUSES from Ksoil-water)	5.41×10^{-5} (estimated by EUSES from Ksoil-water)
Total rate constant for removal from agricultural top soil (day^{-1})	1.22×10^{-6} (estimated by EUSES from above data)	2.77×10^{-5} (estimated by EUSES from above data)
Total rate constant for removal from grassland top soil (day^{-1})	1.75×10^{-6} (estimated by EUSES from above data)	5.47×10^{-5} (estimated by EUSES from above data)
Behaviour during waste water treatment		
Fraction of emission directed to air	0	0
Fraction of emission directed to water	0.1	0.1
Fraction of emission directed to sludge	0.9	0.9
Fraction of emission degraded	0	0

Table 5.7: EUSES Model Parameters used for Cadmium		
Input to EUSES program	**Value chosen for cadmium**	
	General environment	**Acid environment**
Bioconcentration factors		
Partition coefficient worm-porewater (l/kg)	not used	not used
Bioconcentration factor for earthworms (kg/kg)	15	32.4
Bioconcentration factor for fish	38	3,000
Partition coefficient between plant tissue and water (m^3/m^3)	not used – data available but in a different form to that required by EUSES – calculations carried out by hand	not used – data available but in a different form to that required by EUSES – calculations carried out by hand
Partition coefficient between leaves and air (m^3/m^3)		
Transpiration-stream concentration factor		
Bioaccumulation factor for meat (d/kg)	0.02	0.02
Bioaccumulation factor for milk (d/kg)	0.0003	0.0003
Purification factor for surface water	1	1
Note: *a) value not crucial to the calculations – assumed to be similar to the value for suspended sediment as a worst case.*		

5.5.2 Estimation of Regional and Continental PECs

The regional and continental PECs have been estimated using the EUSES program. The total estimates for the emissions to the environment from the applications considered are shown in Table 5.8.

To put these emissions into context, the draft RAR for cadmium estimated that the total EU emissions of cadmium to agricultural soil from the use of phosphate fertilisers was around 270 tonnes/year (see also Section 4.1.2)

The approach taken in the modelling assumes that the cadmium released behaves in the same way independently of the form in which it was released. Thus, for example, the calculations assume that the cadmium released as stabilisers behaves in an identical manner in the environment to that released from plated metal.

The resulting Regional PECs calculated by EUSES using the above input data are shown in Table 5.9. Sample EUSES printout is presented in Annex 5 to this report.

Table 5.8: Summary of emission estimates for regional and continental model					
Use/lifecycle step	**Total EU emission (kg/year)**	**EUSES default fate of sewage sludge[c]**		**Assuming 11% of sewage sludge is incinerated**	
		Regional emission (kg/year)	**Continental emission (kg/year)[d]**	**Regional emission (kg/year)**	**Continental emission (kg/year)**
Stabilisers – manufacture	0.25 to air 0.09 to surface water 0.52 to waste water[c] 960.19 to landfill	0.004 to air[e] 0.45 to waste water[c, e] 180 to landfill[e]	0.246 to air 0.09 to surface water 0.07 to waste water[c] 780.19 to landfill	0.004 to air[e] 0.045 to surface water 0.190 to agricultural soil via sewage sludge 0.166 to landfill via sewage sludge 0.002 to air from incineration of sewage sludge 0.042 to landfill from incineration of sewage sludge 180 to landfill[e] as solid waste	0.246 to air 0.09 to surface water negligible amount to agricultural soil, landfill and air via sewage sludge and sewage sludge incineration 780.19 to landfill as solid waste
Stabilisers – PVC product manufacture	0.64[a] or 5.94[b] to air 63.25[a] or 588.06[b] to landfill	0.064[a, e] or 0.594[b, e] to air 6.325[a, e] or 58.806[b, e] to landfill	0.576[a] or 5.346[b] to air 56.925[a] or 529.25[b] to landfill	0.064[a, e] or 0.594[b, e] to air 6.325[a, e] or 58.806[b, e] to landfill	0.576[a] or 5.346[b] to air 56.925[a] or 529.25[b] to landfill
Stabilisers – PVC product use	0.14[a] or 1.35[b] to air 1.45[a] or 13.47[b] to industrial/urban soil 14.34[a] or 133.35[b] to landfill	0.014[a] or 0.135[b] to air 0.145[a] or 1.347[b] to industrial/urban soil 1.434[a] or 13.335[b] to landfill	0.126[a] or 1.215[b] to air 1.305[a] or 12.123[b] to industrial/urban soil 12.91[a] or 120.02[b] to landfill	0.014[a] or 0.135[b] to air 0.145[a] or 1.347[b] to industrial/urban soil 1.434[a] or 13.335[b] to landfill	0.126[a] or 1.215[b] to air 1.305[a] or 12.123[b] to industrial/urban soil 12.91[a] or 120.02[b] to landfill
Stabilisers – PVC product disposal	1,350[a] or 150[b] to air 268,650[a] or 29,850[b] to landfill	135[a] or 15[b] to air 26,865[a] or 2,985[b] to landfill	1,215[a] or 135[b] to air 241,785[a] or 26,865[b] to landfill	135[a] or 15[b] to air 26,865[a] or 2,985[b] to landfill	1,215[a] or 135[b] to air 241,785[a] or 26,865[b] to landfill
Pigments – manufacture	14.7 to air 25.4 to surface water 406 to landfill	6.4 to air 4.8 to surface water 35 to landfill	8.3 to air 20.6 to surface water 371 to landfill	6.4 to air 4.8 to surface water 35 to landfill	8.3 to air 20.6 to surface water 371 to landfill
Pigments – A and C manufacture	132 to air 13,086 to landfill	13.2 to air 1,308.6 to landfill	118.8 to air 11,777.4 to landfill	13.2 to air 1,308.6 to landfill	118.8 to air 11,777.4 to landfill
Pigments – A and C product use	4 to air 39 to industrial/urban soil 388 to landfill	0.4 to air 3.9 to industrial/urban soil 38.8 to landfill	3.6 to air 35.1 to industrial/urban soil 349.2 to landfill	0.4 to air 3.9 to industrial/urban soil 38.8 to landfill	3.6 to air 35.1 to industrial/urban soil 349.2 to landfill
Pigments – A and C product disposal	7,830 to air 775,171 to landfill	783 to air 77,517.1 to landfill	7,047 to air 697,653.9 to landfill	783 to air 77,517.1 to landfill	7,047 to air 697,653.9 to landfill

Table 5.8: Summary of emission estimates for regional and continental model					
Use/lifecycle step	**Total EU emission (kg/year)**	**EUSES default fate of sewage sludge[c]**		**Assuming 11% of sewage sludge is incinerated**	
		Regional emission (kg/year)	**Continental emission (kg/year)[d]**	**Regional emission (kg/year)**	**Continental emission (kg/year)**
Pigments – Artists colours – formulation	1,659 to landfill	165.9 to landfill	1,493.1 to landfill	165.9 to landfill	1,493.1 to landfill
Pigments – Artists colours – use	1,576 to waste water[c]	157.6 to waste water[c]	1,418.4 to waste water[c]	15.76 to surface water 66.7 to agricultural soil via sewage sludge 58.2 to landfill via sewage sludge 0.78 to air from incineration of sewage sludge 14.8 to landfill from incineration of sewage sludge	141.84 to surface water 600.3 to agricultural soil via sewage sludge 523.8 to landfill via sewage sludge 7.02 to air from incineration of sewage sludge 133.2 to landfill from incineration of sewage sludge
Pigments – Artists colours – waste disposal (wipes etc)	63 to air 1,198 to landfill	6.3 to air 119.8 to landfill	56.7 to air 1,078.2 to landfill	6.3 to air 119.8 to landfill	56.7 to air 1,078.2 to landfill
Pigments – Artists colours – waste disposal	118 to air 11,709 to landfill	11.8 to air 1,170.9 to landfill	106.2 to air 10,538.1 to landfill	11.8 to air 1,170.9 to landfill	106.2 to air 10,538.1 to landfill
Plating – plating sites	250 to waste water[c] 3,360 to landfill	25 to waste water[c] 336 to landfill	225 to waste water[c] 3,024 to landfill	2.5 to surface water 10.575 to agricultural soil via sewage sludge 9.225 to landfill via sewage sludge 0.122 to air from incineration of sewage sludge 2.31 to landfill from incineration of sewage sludge 336 to landfill as solid waste	22.5 to surface water 95.175 to agricultural soil via sewage sludge 83.025 to landfill via sewage sludge 1.098 to air from incineration of sewage sludge 20.79 to landfill from incineration of sewage sludge 3,024 to landfill as solid waste

Table 5.8: Summary of emission estimates for regional and continental model

Use/lifecycle step	Total EU emission (kg/year)	EUSES default fate of sewage sludge[c]		Assuming 11% of sewage sludge is incinerated	
		Regional emission (kg/year)	Continental emission (kg/year)[d]	Regional emission (kg/year)	Continental emission (kg/year)
Plating – plated metal use	1,435 to surface water 46,413 to urban/industrial soil	143.5 to surface water 4,641.3 to urban/industrial soil	1,291.5 to surface water 41,771.7 to urban/industrial soil	143.5 to surface water 4,641.3 to urban/industrial soil	1,291.5 to surface water 41,771.7 to urban/industrial soil
Plating – metallic waste disposal	100,481 to landfill	10,048.1 to landfill	90,432.9 to landfill	10,048.1 to landfill	90,432.9 to landfill
Total[g]		956.2[a] or 836.8[b] to air 148.3 to surface water 183.1 to waste water[f] 4,645[a] or 4,647[b] to urban/industrial soil 117,793[a] or 93,977[b] to landfill	8,556.5[a] or 7,482[b] to air 1,312.2 to surface water 1,643.5 to waste water[f] 41,808[a] or 41,819[b] to urban/industrial soil 1,059,353[a] or 845,013[b] to landfill	957.1[a] or 837.7[b] to air 166.6 to surface water 4,645[a] or 4,647[b] to urban/industrial soil 77.4 to agricultural soil 117,861[a] or 94,045[b] to landfill	8,564.7[a] or 7,490.6[b] to air 1,476.5 to surface water 41,808[a] or 41,819[b] to urban/industrial soil 695.2 to agricultural soil 1,019,959[a] or 845,619[b] to landfill

Note: a) scenario 1 – assumes no recycling
b) scenario 2 – assumes much recycling
c) distribution during waste water treatment is 90% to sludge (which is then applied to soil) and 10% to surface water. Normally, in the EUSES model, the sewage sludge containing the substance is assumed to be applied to land. However, in the EU sludge is also incinerated and this could lead to emissions to air, as cadmium is not completely destroyed during the process. Thus, as well as considering the normal default fate of sewage sludge within the EUSES model, the model will also be run, assuming that 11% of the sewage sludge is incinerated, with 41% of the sewage sludge going to landfill and 47% being applied to agricultural soil. During incineration of sewage sludge it is assumed that 5% of the cadmium is released to air, with 80% going to controlled landfill and 15% going to monofill landfill.
d) in the EUSES model continental emissions = total emissions-regional emissions, where the regional emissions are taken as 10% of the total emissions
e) site with highest release to surface water/waste water assumed to occur in the region.
f) within the EUSES model a 70% connection rate to sewage treatment plants is assumed for releases to waste water.
g) emissions identified as to landfill are not included in the releases to the environment in the EUSES modelling.

Table 5.9: Estimated Regional Concentrations								
Compartment	**$PEC_{regional}$**							
	General environment				**Acid environment**			
	a	b	c	d	a	b	c	d
Air (mg/m^3)	4.26×10^{-8}	3.73×10^{-8}	4.26×10^{-8}	3.73×10^{-8}	4.26×10^{-8}	3.73×10^{-8}	4.26×10^{-8}	3.73×10^{-8}
Surface water (µg/l)	9.48×10^{-3}	9.42×10^{-3}	9.28×10^{-3}	9.22×10^{-3}	0.0612	0.0607	0.0597	0.0592
Sediment (mg/kg wet wt.)	0.474	0.471	0.464	0.461	0.186	0.184	0.181	0.180
Agricultural soil (mg/kg wet wt.)	0.0671	0.0637	0.0540	0.0506	2.63×10^{-3}	2.49×10^{-3}	2.11×10^{-3}	1.98×10^{-3}
Pore water of agricultural soil (µg/l)	0.0251	0.0238	0.0202	0.0189	0.0503	0.0478	0.0405	0.0380
Natural soil (mg/kg wet wt.)	0.0359	0.0314	0.0359	0.0314	1.08×10^{-3}	9.42×10^{-4}	1.08×10^{-3}	9.43×10^{-4}
Industrial/ urban soil (mg/kg wet wt.)	5.74	5.74	5.74	5.74	0.172	0.172	0.172	0.172
Note: *a) No recycling of PVC products and no incineration of sewage sludge in the regional model.* *b) Recycling of PVC products but no incineration of sewage sludge in the regional model.* *c) No recycling of PVC products but incineration of sewage sludge in the regional model.* *d) Recycling of PVC products and incineration of sewage sludge in the regional model.*								

As can be seen from the results presented in Table 5.9, the regional concentrations are relatively insensitive to the different emission scenario estimates, but do vary depending on whether the properties of the 'general' or 'acidic' environment are chosen. The values reported here will be used as the regional background concentrations for the local concentrations estimated in the following Sections.

The draft cadmium RAR summarised the available monitoring data for cadmium in the EU for surface water, sediment, soil and air. The report concluded that the natural background level of cadmium in surface water was around 0.21 µg/l, the background cadmium concentrations in sediment were around 1-10 mg/kg (and may be dominated by historic contamination), the natural background cadmium concentrations in soil were around 0.3 mg/kg and the background cadmium concentrations in air were around $<1\text{-}5\times10^{-6}$ mg/m^3 in rural locations, $5\text{-}15\times10^{-6}$ mg/m^3 in urban locations and 15-50 mg/m^3 in industrial locations. Similar measured background concentrations were reported by WS Atkins (1998a). As these are based on measured levels, they necessarily include contributions from all sources of cadmium (including natural sources) and not just those considered in this report. They do, however, indicate that in most cases the regional concentrations arising from the cadmium sources considered in this report, contribute only a small proportion of the total background cadmium concentration.

5.5.3 Estimation of Local PECs

Overview

Local PECs are estimated for the point source releases of cadmium identified in this report. The methods used are those given in the Technical Guidance. The EUSES model has been used to carry out many of the calculations, but some example calculations have also been presented below to allow transparency in the methods used. Sample EUSES printout has been attached as Annex 5 (note: there are some very small differences between the results calculated here and those presented in the EUSES printout – these are mainly as a result of rounding errors).

Aquatic Compartment (Surface Water and Sediment)

The local PECs can be estimated using the methodology outlined in the EU Technical Guidance Document which is also implemented in EUSES. The following values are used.

$$\text{Clocal}_{\text{inf}} = \frac{\text{Elocal}_{\text{water}} \times 10^3}{\text{EFFLUENT}_{\text{stp}}}$$

where $\text{Elocal}_{\text{water}}$ = local emission rate to waste water treatment plant (kg/day)
$\text{EFFLUENT}_{\text{stp}}$ = effluent discharge rate of waste water treatment plant (m^3/day) – default value is 2,000 m^3/day
$\text{Clocal}_{\text{inf}}$ = concentration in influent to waste water treatment plant (mg/l)

$$\text{Clocal}_{\text{eff}} = \text{Clocal}_{\text{inf}} \times \text{Fstp}_{\text{water}}$$

where $\text{Fstp}_{\text{water}}$ = fraction of emission directed to water in the waste water treatment plant. For cadmium this is taken as 0.1 (i.e. 90% of the cadmium is removed during biological waste water treatment by adsorption onto sewage sludge, with 10% going to the effluent).

$$\text{Clocal}_{\text{water}} = \frac{\text{Clocal}_{\text{eff}}}{(1 + \text{Kp}_{\text{susp}} \times \text{SUSP}_{\text{water}} \times 10^{-6}) \times \text{DILUTION}}$$

where Kp_{susp} = solids-water partition coefficient for suspended matter = 130,000 l/kg for the general situation or 7,900 l/kg for acidic environments
$\text{SUSP}_{\text{water}}$ = concentration of suspended matter in river (mg/l) – default value = 15 mg/l
DILUTION = dilution factor for effluent in receiving water – default value = 10
$\text{Clocal}_{\text{water}}$ = local concentration in surface water during emission episode.

$$\text{Clocal}_{\text{water,ann}} = \text{Clocal}_{\text{water}} \times \frac{\text{Temission}}{365}$$

where Temission = number of days per year that emission takes place
$Clocal_{water,ann}$ = annual average local water concentration

$$PEClocal_{water} = Clocal_{water} + PEC_{regional}$$

and $$PEClocal_{water,ann} = Clocal_{water,ann} + PEC_{regional}$$

where $PEC_{regional}$ = 9.22×10^{-3} – 9.48×10^{-3} µg/l for the general environment and 0.0592-0.0612 µg/l for the acidic environment.

$$PEClocal_{sed} = \frac{K_{susp-water}}{RHO_{susp}} \times PEClocal_{water} \times 1000$$

where $K_{susp-water}$ = suspended matter-water partitioning coefficient (m^3/m^3) = 3.24×10^4 m^3/m^3 for the general environment and 1.98×10^3 m^3/m^3 for the acidic environment.
RHO_{susp} = bulk density of suspended sediment = 1,150 kg/m^3.
$PEClocal_{sed}$ = predicted local concentration in sediment.

Pigment Manufacturing Sites

Site specific information on the releases to surface water from the five pigment manufacturing sites in the EU were reported by WS Atkins (1998a). More up-to-date information has been obtained for two of these sites, and the data are shown in Table 5.10.

Table 5.10: Emissions to Surface Water from Pigment Manufacturing Sites in the EU

Parameter	Pigment Manufacturing Site				
	A	**B**	**C**	**D**	**E**
Use Pattern identity in EUSES printout	1 Formul-ation	1 Processing	1 Private use	1 Recovery	2 Formul-ation
Losses of Cd to surface water[a] (kg/year)	0.60	4.02	4.8	2.6	13.4
Number of days of manufacture	230	231	276	230	145
Loss to surface water/day (kg/day)	0.003	0.0174	0.0174	0.0113	0.0924
Volume of effluent (m^3/day)	131	5,112	199	1,418	2,121
Total Cd concentration in effluent (µg/l)	22.9	3.4	87	8.0	43.5
Flow rate of receiving water (m^3/day)	6,000	5,045,760	135,000	-(354,500[c])	89,856,000
Dilution factor[b]	45.8	987	678	250	42,365
$Clocal_{water}$ (µg/l) – general environment	0.17	0.0012	0.043	0.011	0.00035

Table 5.10: Emissions to Surface Water from Pigment Manufacturing Sites in the EU					
Parameter	**Pigment Manufacturing Site**				
	A	**B**	**C**	**D**	**E**
$Clocal_{water}$ (µg/l) – acidic environment	0.45	0.0031	0.11	0.029	0.00092
$Clocal_{water, ann}$ (µg/l) – general environment	0.11	0.00076	0.033	0.0069	0.00014
$Clocal_{water, ann}$ (µg/l) – acidic environment	0.28	0.0020	0.083	0.018	0.00037
$PEClocal_{water}$ (µg/l) – general environment	0.179	0.0104-0.0107	0.052	0.0202-0.0205	0.0096-0.0098
$PEClocal_{water}$ (µg/l) – acidic environment	0.509-0.511	0.0623-0.0643	0.169-0.171	0.088-0.090	0.060-0.062
$PEClocal_{sediment}$ (mg/kg wet wt.) – general environment	5.05	0.293-0.301	1.47-1.48	0.570-0.577	0.270-0.277
$PEClocal_{sediment}$ (mg/kg wet wt.) – acidic environment	0.876-0.880	0.107-0.111	0.291-0.295	0.151-0.155	0.104-0.107
Note: a) losses are to surface water after on-site treatment *b) dilution factor = daily effluent flow/daily flow of receiving water* *c) flow rate not given – estimated from known dilution*					

Stabiliser Manufacturing Sites

Site specific information on the releases from stabiliser manufacturing sites in the EU were presented by WS Atkins (1998a). No further site specific information was obtained as part of this update and so the original data are shown in Table 5.11. There is a voluntary agreement to phase out the use of cadmium stabilisers and the current production is of the order of 30 tonnes/year compared with the 270 tonnes/year reported by WS Atkins. The production will reportedly fall to zero within a year. No specific information has been obtained as to which sites are still producing cadmium stabilisers. Therefore, calculations will be undertaken for all sites using the WS Atkins emission data, but scaled down according to the reduced level of production (i.e. it will be assumed that both the amounts produced (and hence total emissions) and the number of days of production at the sites will be around 9 times less than considered in the WS Atkins report. This approach gives the same daily emission as before, but assumes that the emission occurs over fewer days, and so is consistent with the reduced production of these substances.

Table 5.11: Site Specific Emissions to Waste Water and Surface Water from Stabiliser Manufacturing Sites in the EU (Modified) from WS Atkins (1998a)						
Parameter	**Stabiliser manufacturing site**					
	F	**G**	**H**	**I**	**J**	**K**
Use pattern identity in EUSES printout	3 Formulation	3 Processing	3 Private use	3 Recovery	4 Formulation	4 Processing
{Emission to sewer (waste water treatment	-	{0.5}	-	{0.1}	-	{4.1}

Table 5.11: Site Specific Emissions to Waste Water and Surface Water from Stabiliser Manufacturing Sites in the EU (Modified) from WS Atkins (1998a)

Parameter	Stabiliser manufacturing site					
	F	G	H	I	J	K
plant) (kg/year) – from WS Atkins report}						
Emissions to sewer (waste water treatment plant) (kg/year) – assumed here[e]		0.0556		0.0111		0.456
Size of WWTP $EFFLUENT_{stp}$ (m^3/day)	-	2,000[d]	-	2,000[d]	-	2,000[d]
{Losses of Cd to surface water[a] (kg/year) – from WS Atkins report}	{0.03}	-	{0.78}	-	{0}	-
Losses of Cd to surface water[a] (kg/year) – assumed here[e]	3.33×10^{-3}		0.0867		0	
{Number of days of manufacture – from WS Atkins report}	{20}	{48}	{60}	{unknown}	{13}	{12}
Number of days of manufacture – assumed here[e]	2.22	5.33	6.67	unknown – assume 5	1.44	1.33
Daily emission to waste water treatment plant ($Elocal_{water}$) (kg/day)	-	0.0104	-	0.0011	-	0.342
$Clocal_{inf}$ (mg/l)	-	0.0052	-	0.00055	-	0.171
Daily loss to surface water[a] (kg/day)	0.0015	-	0.013	-	0	-
Volume of effluent (m^3/day)	1,490	-	2,571	-	unknown	-
Total Cd concentration in effluent ($Clocal_{eff}$) (µg/l)	1.01	0.52	5.06	0.11	0	17.1
Flow rate of receiving water (m^3/day)	725,760	-	259,200	-	unknown	-
Dilution factor[b]	487	10[d]	101	10[d]	10[d]	10[d]
$Clocal_{water}$ (µg/l) – general environment	0.0007	0.018	0.017	0.0038	0	0.58
$Clocal_{water}$ (µg/l) – acidic environment	0.00185	0.0465	0.0448	0.0098	0	1.53
$Clocal_{water, ann}$ (µg/l) – general environment	4.3×10^{-6}	0.00026	0.00031	5.2×10^{-5}	0	0.0021
$Clocal_{water, ann}$ (µg/l) – acidic environment	1.1×10^{-5}	0.00068	0.00082	0.00013	0	0.0056
$PEClocal_{water}$ (µg/l) – general environment	0.0099-0.010	0.0272-0.0275	0.0262-0.0265	0.0130-0.0133	0	0.59

Table 5.11: Site Specific Emissions to Waste Water and Surface Water from Stabiliser Manufacturing Sites in the EU (Modified) from WS Atkins (1998a)

Parameter	Stabiliser manufacturing site					
	F	G	H	I	J	K
$PEClocal_{water}$ (µg/l) – acidic environment	0.061-0.063	0.106-0.108	0.104-0.106	0.069-0.071	0	1.59
$PEClocal_{sediment}$ (mg/kg wet wt.) – general environment	0.279-0.287	0.767-0.774	0.739-0.746	0.367-0.374	-	16.6
$PEClocal_{sediment}$ (mg/kg wet wt.) – acidic environment	0.105-0.109	0.182-0.185	0.179-0.183	0.119-0.122	-	2.74

Note: *a) losses are to surface water after on-site treatment*
b) dilution factor = daily effluent flow/daily flow of receiving water
c) dilution factor not given – estimated from known dilution
d) TGD default value
e) emissions and number of days reduced by 9 to take account the reduction in production since the original data were obtained (see main text)

Stabilisers – PVC Product Manufacturing Sites

This relates to the use of cadmium stabilisers in window profiles. No site specific release information is available for this use. The process is, however, a dry process and there will not generally be any drains within the facility. The only source of emissions will be to air. These are considered later. As there are negligible emissions to water or waste water from this use it is not appropriate to calculate a PEC_{local} for soil or sediment.

Pigments – "A and C" Manufacturing Sites

This relates to the use of cadmium pigments in plastics ("A") and glass/ceramics/enamels ("C"). No site specific release information is available for this use. The major source of emissions are likely to be to air. These are considered later. As there are negligible emissions to water or waste water from this use it is not appropriate to calculate a PEC_{local} for soil or sediment.

Artists Colours (Formulation)

The major source of emissions from this process appear to be to landfill, with negligible amounts going to waste water. There are thought to be around 18 companies carrying this operation in the EU, with a total of around 33.2 tonnes/year of cadmium being used in the process. Information has been received from one company that used 2 tonnes/year of cadmium which indicated that around 0.037 kg/year was emitted to waste water. No indication on the number of days on which formulation would be carried out was provided. The default value from the TGD for this type of process is usually 300 days. In this case, since the actual quantity of cadmium involved is relatively small, this is likely to be an overestimate and so a

smaller number of days (20) will be assumed in this calculation. Based on this information, the following scenario can be developed.

Table 5.12: Emissions to Waste Water and Surface Water from Artists Colours Formulation	
Parameter	**Artists Colours Formulation Site**
Use pattern identity in EUSES printout	5 Formulation
Emissions to sewer (waste water treatment plant) (kg/year)	0.037
Size of WWTP EFFLUENT$_{stp}$ (m^3/day)	2000
Number of days of formulation	20
Daily emission to waste water treatment plant (Elocal$_{water}$) (kg/day)	0.00185
Clocal$_{inf}$ (mg/l)	9.3×10^{-4}
Removal during WWTP	90% to sludge
Total Cd concentration in effluent (Clocal$_{eff}$) (µg/l)	0.093
Dilution factor[b]	10
Clocal$_{water}$ (µg/l) – general environment	0.0031
Clocal$_{water}$ (µg/l) – acidic environment	0.0083
Clocal$_{water, ann}$ (µg/l) – general environment	0.00017
Clocal$_{water, ann}$ (µg/l) – acidic environment	0.00045
PEClocal$_{water}$ (µg/l) – general environment	0.0123-0.0126
PEClocal$_{water}$ (µg/l) – acidic environment	0.0675-0.0695
PEClocal$_{sediment}$ (mg/kg wet wt.) – general environment	0.347-0.354
PEClocal$_{sediment}$ (mg/kg wet wt.) – acidic environment	0.116-0.120

Cadmium Plating Sites

There are reported to be 21 sites in the UK and more in Italy and Spain, with only one site in Germany. Overall it is estimated that there are around 100 sites in the whole of the EU. It has been estimated that <50 kg/year are emitted to sewer (waste water) from the sites in the United Kingdom, and around 250 kg for all sites in the EU. The highest emission from a single site is 16 kg/year to waste water and this will be used to estimate the worst case PEC_{local}. Again, no information was provided as to the number of days of operation of this process, but in this case a figure of around 300 days will be assumed. This is based on information reported in the Use Category Document on the metal finishing industry (BRE, 1997). This indicated that at the time around 80 tonnes/year of cadmium were used in the UK for plating, the typical surface area produced was 11.6 m^2 per kg of cadmium used, and the typical rate of production was 12 m^2/hour. Assuming 21 sites in the UK, the average surface area produced per site would be 44,190 m^2/year, and it would take around 3,683 hours to produce that area. Assuming a 12 hour working day, this equates to around 307 days.

Table 5.13: Emissions to Waste Water and Surface Water from Cadmium Plating Sites	
Parameter	**Cadmium Plating Sites**
Use pattern identity in EUSES printout	6 Processing
Emissions to sewer (waste water treatment plant) (kg/year)	16
Size of WWTP EFFLUENT$_{stp}$ (m^3/day)	2000
Number of days of plating	300
Daily emission to waste water treatment plant (Elocal$_{water}$) (kg/day)	0.0533
Clocal$_{inf}$ (mg/l)	0.027
Removal during WWTP	90% to sludge
Total Cd concentration in effluent (Clocal$_{eff}$) (µg/l)	2.7
Dilution factor[b]	10
Clocal$_{water}$ (µg/l) – general environment	0.090
Clocal$_{water}$ (µg/l) – acidic environment	0.24
Clocal$_{water, ann}$ (µg/l) – general environment	0.074
Clocal$_{water, ann}$ (µg/l) – acidic environment	0.197
PEClocal$_{water}$ (µg/l) – general environment	0.099
PEClocal$_{water}$ (µg/l) – acidic environment	0.299-0.301
PEClocal$_{sediment}$ (mg/kg wet wt.) – general environment	2.80
PEClocal$_{sediment}$ (mg/kg wet wt.) – acidic environment	0.515-0.518

Plated Metal Use

It has been confirmed that the major use of cadmium-plated metal is in components for aircraft, such as landing gear components (e.g. bearings) and certain pins which are subject to large changes in temperature. It is also used in propeller housings. A total loss the environment of 47,848 kg/year in the EU was estimated. In the Atkins report, this loss was essentially assumed to be evenly distributed across the whole of the EU, resulting in 46.7% being lost to soil, 51.9% to the marine environment and 1.6% to surface water. However, since some of the components in question are used in landing gear, there may be an relatively greater loss in the vicinity of airports. In order to take this into account, as a worst case assumption it will be assumed that of the total losses, 97% are to soil and 3% are to surface water in the vicinity of airports (these percentages represent the relative areas of soil and surface water in the EUSES model, and hence the EU). Thus the total loss to surface water in the EU is estimated as 1,435 kg/year.

There is no methodology available in the Technical Guidance Document for assessing the loss from this type of application. For the local assessment, a further worst case assumption that could be made is that 10% of this loss will occur at one very busy airport. This would lead to an estimated 143.5 kg/year of cadmium being lost to surface water. There are several, large, uncertainties in this assumption since aircraft parts produced in the EU will be used on aircraft that fly to- and from the EU, and so some of this loss will be outside the EU. Conversely, aircraft produced and based in countries other than the EU will fly into the EU, adding to the EU emissions. Thus, it is impossible to reliably quantify these local emissions and hence the resulting PEC is open to a great deal of uncertainty.

It has been estimated that there are around 100-1,000 airports in the EU. The average amount of cadmium lost to surface water at an airport would be 1.4-14 kg/year. Thus the average PEC estimated for this use would be 10-100 times lower than the worst case value estimated in Table 5.14.

Table 5.14: Emissions to Surface Water from Plated Metal Use	
Parameter	**Sites**
Use pattern identity in EUSES printout	7 Processing
Emissions to surface water (kg/year)	143.5
Number of days of operation	365
Daily emission to surface water ($Elocal_{water}$) (kg/day)	0.39
Flow of receiving water[a]	20,000 m^3/day
$Clocal_{water}$ (µg/l) – general environment	6.6
$Clocal_{water}$ (µg/l) – acidic environment	17.4
$Clocal_{water, ann}$ (µg/l) – general environment	6.6
$Clocal_{water, ann}$ (µg/l) – acidic environment	17.4
$PEClocal_{water}$ (µg/l) – general environment	6.6
$PEClocal_{water}$ (µg/l) – acidic environment	17.4
$PEClocal_{sediment}$ (mg/kg wet wt.) – general environment	186
$PEClocal_{sediment}$ (mg/kg wet wt.) – acidic environment	30
Note: a) TGD default	

It should be noted that these calculations assume no waste water treatment of the water prior to discharge to the environment. In reality, run-off water from airports may be directed to waste water treatment plants. If this is taken into account in the calculations, the resulting PECs for sediment and water would be approximately a factor of 10 lower (as 90% of the cadmium would be removed in the waste water treatment plant). The calculation also assumes that the loss occurs to a river of the default size for a local assessment as given in the Technical Guidance Document. It is not known if this assumption is valid for a very large airport.

Given the many assumptions in these calculations, it should be stressed that the resulting PECs are subject to a great deal of uncertainty.

Waste Incinerators

Emissions of cadmium from incineration of waste and/or sewage sludge can occur to air (see later) or landfill. Emissions to waste water or surface water are likely to be negligible and so local PECs are not calculated for water or sediment.

Air

The following methodology is used in the TGD to estimate air concentrations:

$$Clocal_{air} = Elocal_{air} \times Cstd_{air}$$

$$Clocal_{air,ann} = Clocal_{air} \times \frac{Temission}{365}$$

$$PEClocal_{air,ann} = Clocal_{air,ann} + PECregional_{air}$$

where: $Elocal_{air}$ = emission rate to air during an emission episode (kg/day).
$Cstd_{air}$ = concentration in air at source strength of 1 kg/day = 2.78×10^{-4} mg/m^3.
Temission = number of days per year on which emission occurs
$Clocal_{air}$ = local concentration in air during emission episode
$Clocal_{air, ann}$ = annual average concentration in air 100 m from source.
$PECregional_{air}$ = regional air concentration = 3.73×10^{-8} – 4.26×10^{-8} mg/m^3

The TGD also gives equations for estimating the aerial deposition flux of aerosol-bound and gaseous compounds. For cadmium, it will be assumed that it is all associated with the aerosol/particulate phase, with the amount being present in the free gaseous phase being negligible compared to this. The equations for estimating the deposition flux then become:

$$DEPtotal = Elocal_{air} \times DEPstd_{aer}$$

$$DEPtotal_{ann} = DEPtotal \times \frac{Temission}{365}$$

where: DEPtotal = total deposition flux during an emission episode
$DEPtotal_{ann}$ = annual average total deposition flux
$DEPstd_{aer}$ = standard deposition flux of aerosol-bound compounds at a source strength of 1 kg/day = 1×10^{-2} mg/m^2/day

In the calculations, it is assumed that there are no air emissions of cadmium from waste water treatment plants.

Pigment Manufacturing Sites

Site specific information on the releases to surface water from the five pigment manufacturing sites in the EU were given in the WS Atkins report. More up-to-date information has been obtained for two of these sites, and the data are shown in Table 5.15.

Table 5.15: Atmospheric Emissions from Pigment Manufacturing Sites					
Parameter	**Pigment Manufacturing Site**				
	A	**B**	**C**	**D**	**E**
Use Pattern identity in EUSES printout	1 Formul-ation	1 Processing	1 Private use	1 Recovery	2 Formul-ation
Emission of Cd to air (kg/year)	1.15	1.66	6.4	5.3	0.2
Number of days of manufacture – Temission	230	231	276	230	145
Emission of Cd to air/day - $Elocal_{air}$ (kg/day)	0.0050	0.0072	0.0232	0.0230	0.0014
DEPtotal ($mg/m^2/day$)	5.0×10^{-5}	7.2×10^{-5}	2.32×10^{-4}	2.3×10^{-4}	1.4×10^{-5}
$DEPtotal_{ann}$ ($mg/m^2/day$)	3.2×10^{-5}	4.6×10^{-5}	2.4×10^{-4}	1.4×10^{-4}	5.6×10^{-6}
$Clocal_{air}$ (mg/m^3)	1.39×10^{-6}	2.00×10^{-6}	6.45×10^{-6}	6.39×10^{-6}	3.89×10^{-7}
$Clocal_{air, ann}$ (mg/m^3)	8.76×10^{-7}	1.27×10^{-6}	4.88×10^{-6}	4.03×10^{-6}	1.55×10^{-7}
$PEClocal_{air}$ (mg/m^3)	1.43×10^{-6}	2.03×10^{-6}- 2.04×10^{-6}	6.49×10^{-6}	6.43×10^{-6}	3.93×10^{-7}- 4.32×10^{-7}
$PEClocal_{air, ann}$ (mg/m^3)	9.13×10^{-7}- 9.19×10^{-7}	1.31×10^{-6}	4.92×10^{-6}	4.07×10^{-6}	1.92×10^{-7}- 1.98×10^{-7}

Stabiliser Manufacturing Sites

Site specific information on the releases from stabiliser manufacturing sites in the EU were presented by WS Atkins (1998a). As discussed earlier, no further site specific information have been obtained but the total emissions and the number of days of emission have been reduced by a factor of 9 to take into account the recent reduction in use in this application - as shown in Table 5.16 (opposite).

Stabilisers – PVC Product Manufacturing Sites

This relates to the use of cadmium stabilisers in window profiles. No site specific release information is available for this use area. Air emissions as dust have been estimated for this use, but it is assumed that these would eventually end up in landfill (80% of the total) or incinerated (20% of the total). The emissions from incinerators are considered separately later. Therefore, no PEC_{local} has been estimated for air at PVC product manufacturing sites.

Pigments – "A and C" Manufacturing Sites

This relates to the use of cadmium pigments in plastics and glass/ceramics/enamels. No site specific release information is available for this use. The major source of emissions are likely to be to air. Similar to the case with stabilisers at PVC product manufacturing sites, these are thought to eventually end up in landfill (80% of total) or incinerated (20% of total). The emissions from incinerators are considered separately later. Therefore, no PEC_{local} has been estimated from air at "A and C" manufacturing sites.

Table 5.16: Atmospheric Emissions from Stabiliser Manufacturing Sites (modified from WS Atkins, 1998a)						
Parameter	**Stabiliser manufacturing site**					
	F	**G**	**H**	**I**	**J**	**K**
Use pattern identity in EUSES printout	3 Formulation	3 Processing	3 Private use	3 Recovery	4 Formulation	4 Processing
{Emission of Cd to air (kg/year) – from WS Atkins report}	{0.09}	{0.8}	{0.5}	{0.1}	{0.7}	{0.04}
Emission of Cd to air (kg/year) – assumed here	0.01	0.0889	0.0556	0.0111	0.0778	0.00444
{Number of days of manufacture – Temission – from WS Atkins report}	{20}	{48}	{60}	unknown	{13}	{12}
Number of days of manufacture – Temission – assumed here	2.22	5.33	6.67	unknown – assume 5	1.44	1.33
Emission of Cd to air/day - $Elocal_{air}$ (kg/day)	0.0045	0.0167	0.00833	0.00222	0.0539	0.0033
DEPtotal ($mg/m^2/day$)	4.5×10^{-5}	1.7×10^{-4}	8.3×10^{-5}	2.2×10^{-5}	5.4×10^{-4}	3.3×10^{-5}
$DEPtotal_{ann}$ ($mg/m^2/day$)	2.7×10^{-7}	2.5×10^{-6}	1.5×10^{-6}	3.0×10^{-7}	2.1×10^{-6}	1.2×10^{-7}
$Clocal_{air}$ (mg/m^3)	1.25×10^{-6}	4.64×10^{-6}	2.32×10^{-6}	6.17×10^{-7}	1.50×10^{-5}	9.17×10^{-7}
$Clocal_{air, ann}$ (mg/m^3)	7.6×10^{-9}	6.8×10^{-8}	4.2×10^{-8}	8.5×10^{-9}	5.9×10^{-8}	3.3×10^{-9}
$PEClocal_{air}$ (mg/m^3)	1.29×10^{-6}	4.68×10^{-6}	2.36×10^{-6}	6.5×10^{-7}-6.6×10^{-7}	1.5×10^{-5}	9.5×10^{-7}-9.6×10^{-7}
$PEClocal_{air, ann}$ (mg/m^3)	4.5×10^{-8}-$5.\times10^{-8}$	1.1×10^{-7}	7.9×10^{-8}-8.5×10^{-8}	4.6×10^{-8} – 5.1×10^{-8}	9.6×10^{-8}-1.0×10^{-7}	4.1×10^{-8}-4.6×10^{-8}

Artists Colours (Formulation)

The major emissions from this process appear to be to landfill, with negligible amounts going to air. Therefore, no PEC_{local} is estimated for this use.

Cadmium Plating Sites

The major emissions from this process appear to be to waste water, with negligible amounts going to air. Therefore, no PEC_{local} is estimated for this use.

Metal Plating Use

The emissions from this application are likely to be directly to surface water or soil. Therefore, no PEC_{local} is estimated for this use.

Waste Incinerators

Emissions of cadmium from incineration of waste and/or sewage sludge can occur to air or landfill. From Table 5.8, the total emissions to air from incinerators have been estimated as 9,498 kg/year from waste incineration and 9 kg/year from sewage sludge incineration in scenario (a) that assumes no recycling of PVC containing cadmium stabilisers, and 8,304 kg/year from waste incineration and 9 kg year from sewage sludge incineration in the scenario (b) that assumes PVC containing cadmium stabilisers is recycled. As a worst case approach it will be assumed that 10% of the total EU emissions from incinerators occurs at one site over 300 days. The resulting PECs are shown in Table 5.17.

Table 5.17: Atmospheric Emissions from Incinerators

Parameter	Waste incinerator – Scenario A	Waste incinerator – Scenario B	Sewage sludge incinerator
Use Pattern identity in EUSES printout	8 Recovery	8 Recovery	9 Recovery
Emission of Cd to air (kg/year)	950	830	0.9
Number of days of manufacture – Temission	365	365	365
Emission of Cd to air/day - $Elocal_{air}$ (kg/day)	2.60	2.27	2.47×10^{-3}
DEPtotal ($mg/m^2/day$)	0.026	0.0227	2.47×10^{-5}
$DEPtotal_{ann}$ ($mg/m^2/day$)	0.026	0.0227	2.47×10^{-5}
$Clocal_{air}$ (mg/m^3)	7.23×10^{-4}	6.31×10^{-4}	6.87×10^{-7}
$Clocal_{air, ann}$ (mg/m^3)	7.23×10^{-4}	6.31×10^{-4}	6.87×10^{-7}
$PEClocal_{air}$ (mg/m^3)	7.23×10^{-4}	6.31×10^{-4}	7.24×10^{-8}-7.29×10^{-8}
$PEClocal_{air, ann}$ (mg/m^3)	7.23×10^{-4}	6.31×10^{-4}	7.24×10^{-8}-7.29×10^{-8}

As for airports, considerable caution should be applied to the figures derived above on two counts:

- according to the European Environment Agency (1999), there are 533 municipal solid waste (MSW) and 239 hazardous waste incinerators in the EU. Although data are not complete, assigning 10% emissions to one incinerator could be overly pessimistic; and
- historically, incinerators have been identified as a significant source of cadmium emissions to the atmosphere and, as a result, there are now very tight limits on cadmium emissions. Against a limit of 0.1 mg/m^3, cadmium emissions would be less than 50 kg/year for a 60,000 tpa MSW incinerator.

Terrestrial (Soil) Compartment

Agricultural Soil and Grassland

The TGD provides a method for estimating the concentrations in soil, taking into account both aerial deposition of a substance and application of sewage sludge containing the substance. The method is implemented in the EUSES model and this has been used to estimate the resulting concentrations in soil here. The local emissions to air and waste water are as outlined previously.

In order to use the model appropriately for cadmium the following data were used as input. From the total rate constants estimated for soil (see Table 5.7), the residence times (1/rate constant) in agricultural soil and grass land can be estimated. These are 2,245 years for agricultural soil and 1,565 years for grassland in the general environment and 99 years for agricultural soil and 50 years for grassland in the acidic environment. These values are longer than used by WS Atkins (1998a), which were a residence time of 100 years for the general environment and 10 years for the acidic environment, but are within the upper limits of the residence times (1,000 years and greater; approximately and order of magnitude lower in acidic environments) given by WS Atkins (1998a). The resulting local PECs for the various endpoints are shown in Table 5.18.

For plated metal use, it a local release to urban/industrial soil could occur near to sites of use, which in this case can be considered as airports. There is no scenario available in the TGD or EUSES for dealing with local releases to urban/industrial soil (the compartment is only considered in the regional model). In the absence of a suitable methodology, a crude example calculation is outlined below assuming that 10% of the total release to urban/industrial soil occurs at the local site. This equates to a cadmium emission of 4,641.3 kg/year (see Table 5.8). This is assumed to be a major airport with two runways, and that the release occurs within a 1 km "band" each side of each runway, to take into account that release could occur during take-off and landing, and during taxiing. The length of the runways will be assumed to be 3,900 m (approximate length of the longest runway at Heathrow Airport). This gives a total area for release of $2\times3{,}900\times2\times1000 = 1.56\times10^{7}$ m^2. Assuming the mixing depth of the soil is 5 cm (i.e. the cadmium released only enters the top 5 cm of soil) and the TGD bulk density of soil of 1,700 kg/m^3, this surface area corresponds to a volume of 7.8×10^{5} m^3 and a wet soil mass of 1.326×10^{9} kg. Thus the yearly soil concentration is $4{,}641.3/1.326\times10^{9}$ = 3.5 mg/kg wet wt. This represents the concentration after one years emissions. As the removal of cadmium from soil may be slow, the concentration may build up further over subsequent years. The TGD methodology usually considers the local concentration in soil after ten years emissions, which in this case would lead to a concentration of around 35 mg/kg wet wt., assuming no significant removal. As discussed earlier, there are considerable uncertainties inherent in this emission estimate, and also in the methodology used to estimate the concentrations.

Table 5.18: Estimated Concentrations in Soil									
Endpoint	**Use pattern identity in EUSES printout**	**Local PEC in agricultural soil, averaged over 30 days (mg/kg wet wt.)**		**Local PEC in agricultural soil, averaged over 180 days (mg/kg wet wt.)**		**Local PEC in grassland, averaged over 180 days (mg/kg wet wt.)**		**Local PEC in groundwater under agricultural soil (mg/l)**	
		General	**Acidic**	**General**	**Acidic**	**General**	**Acidic**	**General**	**Acidic**
Pigment manufacturing site A	1 Formulation	0.0317-0.0363	0.00127-0.00140	0.0317-0.0363	0.00127-0.00141	0.0321-0.0366	0.00157-0.00170	1.19×10^{-5}-1.36×10^{-5}	2.44×10^{-5}-2.70×10^{-5}
Pigment manufacturing site B	1 Processing	0.0319-0.0364	0.00141-0.00154	0.0319-0.0364	0.00142-0.00155	0.0324-0.0369	0.00185-0.00198	1.19×10^{-5}-1.36×10^{-5}	2.72×10^{-5}-2.98×10^{-5}
Pigment manufacturing site C	1 Private use	0.0333-0.0378	0.00274-0.00288	0.0333-0.0378	0.00278-0.00291	0.0352-0.0398	0.00443-0.00457	1.25×10^{-5}-1.42×10^{-5}	5.32×10^{-5}-5.58×10^{-5}
Pigment manufacturing site D	1 Recovery	0.0330-0.0375	0.00243-0.00257	0.0330-0.0375	0.00246-0.00259	0.0346-0.0391	0.00383-0.00397	1.23×10^{-5}-1.40×10^{-5}	4.71×10^{-5}-4.97×10^{-5}
Pigment manufacturing site E	2 Formulation	0.0315-0.0360	9.99×10^{-4}-0.00113	0.0315-0.0360	0.00100-0.00114	0.0315-0.0360	0.00105-0.00119	1.18×10^{-5}-1.35×10^{-5}	1.92×10^{-5}-2.18×10^{-5}
Stabiliser manufacturing site F	3 Formulation	0.0314-0.0359	9.45×10^{-4}-0.00108	0.0314-0.0359	9.45×10^{-4}-0.00108	0.0314-0.0359	9.48×10^{-4}-0.00108	1.18×10^{-5}-1.34×10^{-5}	1.81×10^{-5}-2.07×10^{-5}
Stabiliser manufacturing site G	3 Processing	0.0314-0.0360	9.72×10^{-4}-0.00111	0.0314-0.0360	9.72×10^{-4}-0.00111	0.0315-0.0360	9.92×10^{-4}-0.00113	1.18×10^{-5}-1.35×10^{-5}	1.86×10^{-5}-2.12×10^{-5}
Stabiliser manufacturing site H	3 Private use	0.0314-0.0359	9.58×10^{-4}-0.00109	0.0314-0.0359	9.58×10^{-4}-0.00109	0.0314-0.0360	9.73×10^{-4}-0.00111	1.18×10^{-5}-1.34×10^{-5}	1.84×10^{-5}-2.10×10^{-5}
Stabiliser manufacturing site I	3 Recovery	0.0314-0.0359	9.46×10^{-4}-0.00108	0.0314-0.0359	9.46×10^{-4}-0.00108	0.0314-0.0359	9.49×10^{-4}-0.00108	1.18×10^{-5}-1.34×10^{-5}	1.81×10^{-5}-2.07×10^{-5}
Stabiliser manufacturing site J	4 Formulation	0.0314-0.0359	9.64×10^{-4}-0.00110	0.0314-0.0359	9.64×10^{-4}-0.00110	0.0314-0.0360	9.85×10^{-4}-0.00112	1.18×10^{-5}-1.35×10^{-5}	1.85×10^{-5}-2.11×10^{-5}
Stabiliser manufacturing site K	4 Processing	0.0316-0.0361	0.00109-0.00123	0.0316-0.0361	0.00109-0.00123	0.0315-0.0360	0.00100-0.00114	1.18×10^{-5}-1.35×10^{-5}	2.09×10^{-5}-2.35×10^{-5}
Artists colours formulation	5 Formulation	0.0314-0.0359	9.43×10^{-4}-0.00108	0.0314-0.0359	9.43×10^{-4}-0.00108	0.0314-0.0359	9.43×10^{-4}-0.00108	1.18×10^{-5}-1.34×10^{-5}	1.81×10^{-5}-2.07×10^{-5}

Table 5.18: Estimated Concentrations in Soil									
Endpoint	**Use pattern identity in EUSES printout**	**Local PEC in agricultural soil, averaged over 30 days (mg/kg wet wt.)**		**Local PEC in agricultural soil, averaged over 180 days (mg/kg wet wt.)**		**Local PEC in grassland, averaged over 180 days (mg/kg wet wt.)**		**Local PEC in groundwater under agricultural soil (mg/l)**	
		General	**Acidic**	**General**	**Acidic**	**General**	**Acidic**	**General**	**Acidic**
Cadmium plating	6 Processing	0.0314-0.0359	9.65×10^{-4}-0.00110	0.0314-0.0359	9.65×10^{-4}-0.00110	0.0314-0.0359	9.51×10^{-4}-0.00109	1.18×10^{-5}-1.35×10^{-5}	1.85×10^{-5}-2.11×10^{-5}
Plated metal use	7 Processing	0.0314-0.0359	9.42×10^{-4}-0.00108	0.0314-0.0359	9.42×10^{-4}-0.00108	0.0314-0.0359	9.42×10^{-4}-0.00108	1.18×10^{-5}-1.34×10^{-5}	1.81×10^{-5}-2.07×10^{-5}
Endpoint	Use pattern identity in EUSES printout	Local PEC in agricultural soil, averaged over 30 days (mg/kg wet wt.)		Local PEC in agricultural soil, averaged over 180 days (mg/kg wet wt.)		Local PEC in grassland, averaged over 180 days (mg/kg wet wt.)		Local PEC in groundwater under agricultural soil (mg/l)	
Waste incineration	8 Recovery	0.276-0.316	0.234-0.268	0.280-0.321	0.238-0.273	0.529-0.606	0.452-0.518	1.05×10^{-4}-1.20×10^{-4}	4.56×10^{-3}-5.23×10^{-3}
Sewage sludge incineration	9 Recovery	0.0317-0.0362	0.00120-0.00133	0.0317-0.0362	0.00120-0.00134	0.0319-0.0365	0.00143-0.00157	1.19×10^{-5}-1.35×10^{-5}	2.3×10^{-5}-2.56×10^{-5}

Landfills

Waste containing cadmium is disposed of to landfill. These have been quantified for the various sources considered in this report (see Table 5.8). However, the TGD does not provide a methodology for estimating concentrations or carrying out a risk characterisation for releases to landfills and so it is not possible to estimate PECs for this endpoint. In terms of the behaviour of cadmium, significant volatilisation from landfills is unlikely as cadmium has a negligible vapour pressure. Leaching from landfill is theoretically possible, particularly under acidic conditions, but in many instances the cadmium-containing waste will be disposed of in controlled sites, which further minimises the potential for leaching.

Non-compartment Specific Exposure Relevant for the Food Chain

In the TGD, two aspects are considered. Firstly exposure of higher mammals and birds via their food chain (secondary poisoning) and secondly the exposure of humans through environmental routes.

Secondary Poisoning

The EUSES model has been used to estimate the concentrations in the food chain for higher mammals and birds, using the release estimates and partition coefficients outlined earlier. Sample EUSES printout is attached as Annex 5. The results are shown in Table 5.19.

Table 5.19: Estimated Concentrations for Secondary Poisoning

Endpoint	Use Pattern Identity in EUSES Printout	Concentration in Fish (mg/kg wet wt.)		Concentration in Earthworms (mg/kg wet wt.)	
		General	Acidic	General	Acidic
Pigment manufacturing site A	1 Formulation	0.00238-0.00239	0.600-0.606	0.618-0.775	0.0527-0.0653
Pigment manufacturing site B	1 Processing	3.64×10^{-4}-3.74×10^{-4}	0.181-0.186	0.619-0.776	0.0551-0.0677
Pigment manufacturing site C	1 Private use	9.75×10^{-4}-9.85×10^{-4}	0.308-0.314	0.630-0.787	0.0771-0.0897
Pigment manufacturing site D	1 Recovery	4.79×10^{-4}-4.89×10^{-4}	0.204-0.210	0.627-0.784	0.0719-0.0846
Pigment manufacturing site E	2 Formulation	3.53×10^{-4}-3.63×10^{-4}	0.178-0.184	0.616-0.773	0.0483-0.0609
Stabiliser manufacturing site F	3 Formulation	3.50×10^{-4}-3.60×10^{-4}	0.178-0.184	0.615-0.772	0.0474-0.0600
Stabiliser manufacturing site G	3 Processing	3.55×10^{-4}-3.65×10^{-4}	0.179-0.185	0.616-0.773	0.0479-0.0605
Stabiliser manufacturing site H	3 Private use	3.56×10^{-4}-3.66×10^{-4}	0.179-0.185	0.615-0.772	0.0476-0.0603
Stabiliser manufacturing site I	3 Recovery	3.51×10^{-4}-3.61×10^{-4}	0.178-0.184	0.615-0.772	0.0474-0.0601

Table 5.19: Estimated Concentrations for Secondary Poisoning					
Endpoint	**Use Pattern Identity in EUSES Printout**	**Concentration in Fish (mg/kg wet wt.)**		**Concentration in Earthworms (mg/kg wet wt.)**	
		General	**Acidic**	**General**	**Acidic**
Stabiliser manufacturing site J	4 Formulation	3.50×10^{-4}-3.60×10^{-4}	0.178-0.184	0.615-0.772	0.0477-0.0604
Stabiliser manufacturing site K	4 Processing	3.90×10^{-4}-4.01×10^{-4}	0.186-0.192	0.616-0.773	0.0498-0.0624
Artists colours formulation	5 Formulation	3.54×10^{-4}-3.64×10^{-4}	0.178-0.184	0.615-0.772	0.0474-0.0600
Cadmium plating	6 Processing	0.00176-0.00177	0.471-0.477	0.616-0.773	0.0477-0.0604
Plated metal use	7 Processing	0.126[a]	26.3[a]	0.615-0.772	0.0474-0.060
Waste incineration	8 Recovery	3.50×10^{-4}-3.60×10^{-4}	0.178-0.184	2.48-2.91	3.89-4.46
Sewage sludge incineration	9 Recovery	3.50×10^{-4}-3.60×10^{-4}	0.178-0.184	0.617-0.774	0.0516-0.0642
Note: a) these concentrations depend on the predicted concentrations in surface water. As mentioned earlier, these are highly uncertain for this application.					

Exposure of Man via the Environment

Estimation of exposure of man via the environment has been carried out using the methods outlined in the TGD based on the partition coefficients and release estimates outlined earlier. Some of the partition coefficients available for cadmium are not in a form that can readily be entered in the EUSES program and so some of the calculations have been carried out by spreadsheet (Annex 4), which also highlights the intermediate concentrations in food. This relates particularly to the concentrations in plants. The resulting daily human intake figures that are required for the risk characterisation are outlined in Table 5.20 (opposite). The basic assumption in the local calculations is that humans take all their intake from air, drinking water, and food (fish, meat, milk, roots crops and leaf crops) from areas close to the source/site of release (and hence reflect the local concentrations in air, soil and surface water) and so represent a worst case that may not occur in reality. The calculations also assume no removal of cadmium during purification of drinking water.

For the general environment, the calculations indicated that uptake from soil into food was a significant source of exposure in the general environment but when the acidic environment was considered uptake into fish was a significant contributor to the total daily dose.

Table 5.20: Estimated Daily Human Intake Figures		
Endpoint	**Total Daily Human Intake from Environmental Sources (mg/kg bw/day)**	
	General Environment	**Acidic Environment**
Pigment manufacturing site A	1.12×10^{-4}-1.27×10^{-4}	1.69×10^{-3}-1.70×10^{-3}
Pigment manufacturing site B	1.03×10^{-4}-1.17×10^{-4}	3.13×10^{-4}-3.24×10^{-4}
Pigment manufacturing site C	1.12×10^{-4}-1.26×10^{-4}	7.25×10^{-4}-7.36×10^{-4}
Pigment manufacturing site D	1.08×10^{-4}-1.23×10^{-4}	4.01×10^{-4}-4.12×10^{-4}
Pigment manufacturing site E	1.01×10^{-4}-1.15×10^{-4}	3.02×10^{-4}-3.11×10^{-4}
Stabiliser manufacturing site F	1.01×10^{-4}-1.15×10^{-4}	3.00×10^{-4}-3.11×10^{-4}
Stabiliser manufacturing site G	1.01×10^{-4}-1.15×10^{-4}	3.03×10^{-4}-3.14×10^{-4}
Stabiliser manufacturing site H	1.01×10^{-4}-1.15×10^{-4}	3.04×10^{-4}-3.15×10^{-4}
Stabiliser manufacturing site I	1.01×10^{-4}-1.15×10^{-4}	3.00×10^{-4}-3.11×10^{-4}
Stabiliser manufacturing site J	1.01×10^{-4}-1.15×10^{-4}	3.00×10^{-4}-3.11×10^{-4}
Stabiliser manufacturing site K	1.01×10^{-4}-1.16×10^{-4}	3.28×10^{-4}-3.39×10^{-4}
Artists colours formulation	1.01×10^{-4}-1.15×10^{-4}	3.02×10^{-4}-3.12×10^{-4}
Cadmium plating	1.08×10^{-4}-1.22×10^{-4}	1.41×10^{-4}-1.42×10^{-4}
Plated metal use	7.32×10^{-4}-7.47×10^{-4} [a]	0.086[a]
Waste incineration	1.19×10^{-3}-1.37×10^{-3}	2.35×10^{-3}-2.66×10^{-3}
Sewage sludge incineration	1.02×10^{-4}-1.16×10^{-4}	3.02×10^{-4}-3.12×10^{-4}
Regional sources	1.50×10^{-4}-1.95×10^{-4}	3.05×10^{-4}-3.18×10^{-4}
Note: a) the uptake from fish makes an important contribution to these levels for this application (56-57% of the total for the general environment and >99% for the acidic environment). As mentioned earlier, these calculations are highly uncertain for this use.		

6. EFFECTS OF CADMIUM

6.1 Overview

Cadmium is a non-essential element and, as such, is not required for any biological process. There had been extensive research into the effects of cadmium upon both humans and the environment.

WS Atkins presented a considerable amount of data on environmental toxicity (see Appendix D of WS Atkins, 1998a). In this report, reliance has been placed on the recent comprehensive data set compiled for the Belgian authorities (De Win *et al*, 1999). Although some data on human toxicity were presented by WS Atkins, it was considered insufficient by CSTEE. For this reason, we present the findings of a comprehensive review which draws heavily on information compiled in the US (NTIS, 1999).

6.2 Environmental Toxicity

6.2.1 Data Sources

There is a significant body of literature on the ecotoxicity of cadmium. A comprehensive database has been established on toxicity of cadmium in the environment, with the co-operation of the International Cadmium Association, for the purposes of the risk assessment on cadmium oxide prepared by the Belgian authorities under the Existing Substances Regulation (De Win, 1999). This is considered to be the most up to date source of data (as confirmed by the International Cadmium Association at the meeting on 17 May 2000). The Belgian authorities have given their permission for this data set to be used in this work. The data in this set were screened by the Belgian rapporteur using a set of criteria, and the same criteria have been applied here. The full data set is attached as Annex 6; a summary of the selected data is included in this part of the report. Environmental factors which may affect the toxicity of cadmium, such as pH, hardness in water, clay content in soils, will be discussed in the relevant section.

6.2.2 Aquatic toxicity

The test results in the data set were assigned a Reliability Index (RI) according to the following criteria (De Win, 1999):

RI 1: standard test (OECD approved tests) and performed according to the standard procedures;

RI 2: no standard test but complete background information is given, i.e. the following information is present:

a) water hardness (either measured or calculated from Ca and Mg concentrations);
b) pH;

c) measured Cd concentrations in test systems for all data < 1 μg l^{-1};
d) measured Cd concentrations or indications that nominal concentrations are close to measured concentrations;
e) information that actual Cd concentrations were maintained during the test;
f) statistical analysis of the dose-response relationship;
g) no varying metal contamination along with increasing Cd application;
h) the control must be tested along with at least two Cd concentrations above the control;
i) information about the origin of the test organisms; and
j) information on the test concentration range.

RI 3: no standard test and one or more of the following information from the above-mentioned list is missing as background information: b), d), e), f), i), or j). All other information from that list is present; and

RI 4: no standard test and one or more of the following information from the above-mentioned list is missing as background information: a), c), g) or h). The requirement c) is critical since some tests have reported toxic effects below 1 μg l^{-1} nominal Cd concentrations. Background Cd concentrations in filtered water typically range between 0.05 and 0.2 μg l^{-1} and the lack of reporting the background concentration may underestimate the Cd concentration at which the first toxic effects are found. Some tests were included that did not show Cd toxicity up to the highest Cd concentration tested. These tests cannot be used for risk assessment (no NOEC can be found) and were considered unreliable (RI4) but were quoted in the tables for illustration.

The data selected in this way in the draft RAR (De Win, 1999) includes NOEC, LOEC and EC values. For the purpose of this work only the NOEC values have been used. The resulting values are in Table 6.1.

A number of factors are considered to affect the toxicity of metals in water, the primary ones being hardness, pH and dissolved organic carbon. Very few of the test reports in the database include the dissolved organic carbon content, but a significant proportion report the hardness and pH. To investigate the significance of these parameters for cadmium, the NOEC values were plotted against hardness or pH. For data with a Reliability Index of 1 or 2, there are no obvious trends with either parameter (see Figures 6.1 and 6.2). When data with RI 3 are added (Figures 6.3 and 6.4), the lowest values do occur at the lowest hardness values, but there are also low NOEC values from tests in high hardness waters. Considering the data set in two groups, those tested at hardness levels <50 mg $CaCO_3$/l and those above, there is little difference between the lowest values found in each group: 0.47 and 0.6 μg/l for the low hardness group and 0.8 and 0.85 μg/l for the high hardness group.

On the basis of this analysis it is concluded that the data cannot be separated by hardness and so all of the values will be used in deriving the PNEC irrespective of the hardness or other properties of the water used. The set of data with RI 1-2 contains 17 values. There are two values for the same endpoint for *Daphnia magna* (reproduction) in waters of different hardness; the higher value has been excluded so that only one value for each species is included.

Table 6.1: Aquatic Toxicity Data for Cadmium

Species	Group	Hardness (mg $CaCO_3$/l)	pH	NOEC (µg/l)	Endpoint	RI
Selenastrum capricornutum	A	49	9.05	2.5	cell number	1
Coelastrum proboscideum	A	32	5.3	6.3	Biomass	2
Asterionella formosa	A	121	8	0.85	growth rate	2
Chlamydomonas reinhardii	A	42	6.7	7.5	steady state cell number	3
Scenedesmus quadricauda	A	55		31	Biomass	3
Lemna paucicostata	A	120	6	5	number of fronds	3
Lemna paucicostata	A	120	5.1	10	number of fronds	3
Lemna paucicostata	A		5.1	10	number of fronds	3
Ankistrodesmus falcatus	A	34		500	cell number	3
Daphnia magna	I	11	8	0.6	Reproduction	2
Daphnia magna	I	300	8	0.8	Reproduction	2
Aplexa hypnorum	I	45.3	7.45	4.41	Growth	2
Physa integra	I	46	7.4	8.3	Mortality	2
Daphnia magna	I	240	8	2.5	reproductive impairment	3
Daphnia pulex	I	240	8	7.5	reproductive impairment	3
Daphnia magna	I	45	7.7	1	weight/animal	3
Daphnia pulex	I	65	7.7	1	Longevity	3
Crustacean plankton communities	I	120		2.5	crustacean density	3
Daphnia magna	I	90		2	Reproduction	3
Daphnia magna	I	200	8.4	1	Mortality	3
Daphnia magna	I	224	8.1	3.2	intrinsic rate of natural increase	3
Daphnia galeata mendotae	I	120		2	number of individuals	3
Daphnia magna	I	150	8.4	2.5	biomass production/female	3
Ceriodaphnia reticulata	I	67	7.5	3.4	Reproduction	3
Salmo salar	V	23.5	6.9	0.47	total biomass	2
Catostomus commersoni	V	45	7.6	4.2	Biomass	2
Esox lucius	V	45	7.6	4.2		2
Oncorhyncus kisutch	V	45	7.6	1.3	biomass (sac fry)	2
Salvelinus namaycush	V	45	7.6	4.4		2
Salvelinus fontinalis	V	45	7.6	1.1		2
Salmo trutta	V	45	7.6	1.1		2
Salvelinus fontinalis	V	44.5	7.5	0.9	weight of young/female in second generation	2
Jordanella floridae	V	44	7.45	4.1	reproduction, growth	2
Salmo gairdneri	V	382.5	8.3	12	Mortality	2
Brachydanio rerio	V	100	7.2	1	Reproduction	3
Oryzias latipes	V	200		6	mortality, behaviour	3
Oryzias latipes	V	100		3	mortality, behaviour	3
Xenopus laevis	V	170		9	inhibition of larvae	3
Salmo gairdneri	V	100	8.3	4	median survival time	3
Pimephales promelas	V	202.5	7.65	13	Reproduction	3
Pimephales promelas	V	202.5	7.65	14	Reproduction	3

Notes: Group: A = algae; I = invertebrate; V = vertebrate; and RI = Reliability Index

The 16 remaining values cover a range of species. Algae are represented by both chlorophyta and diatoms; invertebrates include cladocerans and snails; the fish include species which feed on other fish as well as those feeding on insects. It is considered appropriate to use the statistical extrapolation method on these data, in order to make use of all the values rather than base the PNEC on a single result. The method has been applied assuming a log-logistic distribution of NOEC values. The fit of the data set to this distribution was tested using the Kolmogorov-Smirnov goodness-of-fit test. This did not reject the hypothesis that the data came from such a distribution, at a significance level of 0.05. A plot of expected and observed cumulative frequencies is shown in Figure 6.5.

The method of Aldenberg and Slob was used (as in the risk assessment report on cadmium oxide, De Win et al 1999) to obtain the HC_5 value from the distribution, using the 50% confidence limit on this value. This value was taken as the PNEC for surface water, giving a value of 0.44 μg/l.

6.2.3 Sediment

The available database on toxicity to sediment organisms is included in Annex 6. The range of species included in the set with results of Reliability Index 1-3 is limited to four. Although there are 14 test results, many of these involve the same organisms tested with different composition sediments. The available results come from tests with relatively short exposure times (up to 10 days), and are only concerned with mortality rather than endpoints such as reproduction. They could therefore be considered to be extended acute studies. As a result, they are not considered appropriate to use in a statistical extrapolation.

Instead the equilibrium partitioning method will be used based on the PNEC for the aquatic compartment. It is recognised that the partitioning behaviour of metals is potentially more complex than the situation with organic substances. However, some of this can be considered to be included by using measured partition coefficients between suspended matter and water. Values for the suspended matter-water partition coefficient for cadmium were selected in the section on distribution in this report: 1.3 x 10^5 l/kg dw for the general environment, and 7,900 l/kg dw for the acidic environment. These values are used here.

Properties which may affect the toxicity in sediments include the hardness and pH of the interstitial and superficial water as well as the composition of the sediment itself. One specific aspect for sediments is the presence of acid volatile sulphide (AVS). In anaerobic sediments, metals may form poorly soluble metal-sulphide complexes with AVS which can precipitate the metals and make them unavailable. Carlson *et al* (1991) and Di Toro *et al* (1992) demonstrated that little or no toxicity was observed in sediments when the molar concentration ratio between cadmium and AVS was less than 1. For ratios above this, mortality increased significantly. It is difficult to apply this concept to general situations, for a number of reasons. The levels of AVS can vary between sediments. AVS act on a range of heavy metals, so that the 'spare' capacity will depend on the amounts of other metals present as well as cadmium. AVS are also found in deeper anaerobic sediments; the surface layers are more

aerobic and AVS are virtually absent. Therefore for the purpose of this assessment the influence of AVS will be neglected. In effect this is a worst case assumption that there are no AVS present or that they are already saturated.

Applying the partition coefficient for suspended matter - water from above for the general environment to the PNEC for water of 0.44 µg/l gives a PNEC sediment of 57 mg/kg wet weight, or 12.4 mg/kg dry weight (assuming the standard sediment composition from the Technical Guidance Document). A similar calculation for the acidic environment gives a PNEC of 0.76 mg/kg wet weight.

6.2.4 Soil

The tests on soil organisms in the data set were assigned a Reliability Index (RI) according to the following criteria (De Win et al, 1999):

RI 1: standard test. Two such test included are the OECD 207 acute toxicity test with *Eisenia fetida* in OECD-soil and the ISO 1994: soil quality effects of soil pollutants on Collembolla (*Folsomia candida*): method for the determination of effects on reproduction;

RI 2: no standard test but complete background information is given, i.e. the following information is present:

a) soil pH
b) soil organic matter or carbon content
c) texture (class or texture fractions)
d) total Cd content of the soil at zero Cd application if the NOEC or LOEC value is below 2µg g^{-1}
e) equilibration time after soil contamination and prior to the test
f) statistical analysis of the dose-response relationship
g) no varying metal contamination along with increasing Cd application
h) the control soil must be tested along with at least two Cd concentrations above the background concentration
i) the soil must be homogeneously mixed with the metal prior to the test;

RI 3: no standard test and one or more of the following information from the above-mentioned list is missing as background information: b), c), e) or f). All other information from that list is present; and

RI 4: no standard test and one or more of the following information from the above-mentioned list is missing as background information: a), d), g), h) or i). The requirement d) is critical since some tests reporting LOEC values < 2 µg g^{-1} are considered unreliable. Background Cd concentrations in soil typically range between 0.1 and 0.5 µg g^{-1} and the lack of reporting the background concentration may underestimate the total Cd concentration in soil at which the first toxic effects are found. Some tests were included that did not show Cd toxicity up to the highest Cd concentration tested. These tests cannot be used for risk assessment (no NOEC can be found) and were considered unreliable (RI4) but were quoted in the tables for illustration.

The data set of values with RI 1 or 2 has been used in the derivation of the PNEC for soil. This set includes a number of results for the same species and the same endpoint in different soils. In these cases the lowest value for each species for that endpoint has been used, and the others removed from the dataset. The resulting values are summarised in Table 6.2.

Table 6.2: Toxicity Data for Soil Organisms

Species	Endpoint	NOEC (mg/kg)	Group	RI
native soil microflora	substrate induced respiration rate	3.6	sm	2
native soil microflora	substrate induced respiration rate	3.6	sm	2
native soil microflora	NO3 prod rate, +NH4 substrate	10	sm	2
native soil microflora	substrate induced respiration rate	14.3	sm	2
native soil microflora	24h respiration	14.3	sm	2
native soil microflora	NO3 prod rate, -NH4 substrate	50	sm	2
native soil microflora	NO3 prod rate, -NH4 substrate	50	sm	2
native soil microflora	glutamic acid decomposition time	55	sm	2
native soil microflora	NO3 prod rate, +NH4 substrate	100	sm	2
native soil microflora	glutamic acid decomposition time	150	sm	2
native soil microflora	respiration	150	sm	2
native soil microflora	respiration	150	sm	2
native soil microflora	respiration	200	sm	2
native soil microflora	respiration	200	sm	2
native soil microflora	respiration	500	sm	2
Dendrobaena rubida	cocoon production	10	sf	2
Dendrobaena rubida	hatching success	10	sf	2
Folsomia candida	fresh weight at 45% MC	80	sf	2
Caernorhabditis elegans	mortality	112	sf	2
Folsomia candida	number of offspring	148	sf	2
Lumbriculus rubellus	weight	150	sf	2
Picea sitchensis	root length	1.8	hp	2
Triticum aestivum	shoot dry weight	7.1	hp	2

Notes: Group: sm = soil microflora; sf = soil fauna; hp = higher plants; and MC = moisture content

Soil properties can have an effect on the toxicity of cadmium. In general terms, factors which increase the mobility of cadmium (for example decreasing soil pH, decreasing organic matter content) tend to increase the toxicity. However there are examples in the data base of exceptions to these general effects. In the risk assessment report on cadmium oxide (De Win et al 1999) the data with RI 1-3 were used to test the relationship between toxicity and the pH and clay content of soils. For pH, no clear relationship was found; although there appears to be a trend of increasing NOEC values (ie decreasing toxicity) with pH up to pH values of 5-6, the data are very scattered especially beyond pH 6. Considering the data in two groups, pH $\leq$ 6.0 and pH > 6.0, these gave almost identical results for extrapolated PNEC values. For clay content, again there was no clear relation between % clay and log NOEC. When the data were considered in two groups, with clay content $\leq$10% and >10%, the extrapolated PNEC value for the higher clay content was double that for the lower clay content. The authors commented that it is not clear if this is a real relationship.

Values for LOECs and EC_xs were also compared to the total soil cadmium and to the soluble cadmium concentration. Conversion to the soluble cadmium concentration did not lead to any significant relationships. It was concluded that at present it is not justifiable to normalise cadmium soil toxicity data on the basis of dissolved cadmium or any other soil parameters. Therefore in this work the data values have been used as they were determined, without any adjustment for soil properties.

The three types of organisms represented in the soil toxicity database are soil fauna, higher plants and soil microflora. The last of these can be considered to be different from the others in that they are not single species but are communities of mixed species. As such it is possible that although a metal may have a deleterious effect on certain parts of the community, other parts may be able to take advantage of this, with the net effect being that the function of the system is maintained. This could mean that effects on overall function are not seen at concentrations which are in fact harmful to components of the system. Figure 6.6 compares the data for these three types of organism, using the full data set with RI 1-3 (so that there are duplicate entries for some species). This shows that there is not a great deal of difference between the values for soil microflora and the other organisms. The lowest NOEC values are obtained for higher plants, but these are only a factor of 2 below the lowest values for soil microflora (note that the plot is of log (NOEC)).

Despite this apparent lack of difference in sensitivity it was decided to consider the soil toxicity data in two parts - soil microflora in one part, and fauna and plants in the other. The statistical extrapolation method was used as there are considered to be sufficient data in the two groups (15 for the microflora, 8 for the fauna and plants) with RI 1 or 2. The method was also applied to the whole data set of 23 values. For the two groups and the whole data set the fit of the data to a log-logistic distribution was tested with the Kolmogorov-Smirnov test. In all three cases the test did not reject the hypothesis that the data came from such a distribution, at a significance level of 0.05. The results of the extrapolation procedure are in Table 6.3.

Table 6.3: PNEC values derived for soil organisms

Data set	Number of values	PNEC (mg/kg)
Soil microflora	15	3.65
Soil fauna and plants	9	1.37
Combined data	24	2.64

Plots of the observed and expected cumulative frequencies in each of these groups are shown in Figures 6.7 to 6.9 and a plot of the expected relative frequencies in each group is shown in Figure 6.10. As a worst case estimate the PNEC for soil fauna and plants is taken as the PNEC for soil, i.e. 1.37 mg/kg. It is not clear from the source report (De Win *et al*, 1999) whether the soil effect concentrations are on a wet weight or dry weight basis, but the PNEC values are described as being on a dry weight basis. Considering the value derived here to be on a dry weight basis, and using the default water content for soil from the Technical Guidance Document (20%) the PNEC for soil on a wet weight basis is 1.21mg Cd/kg wet soil. (The concentrations in soil estimated earlier in this report are on a wet weight basis.)

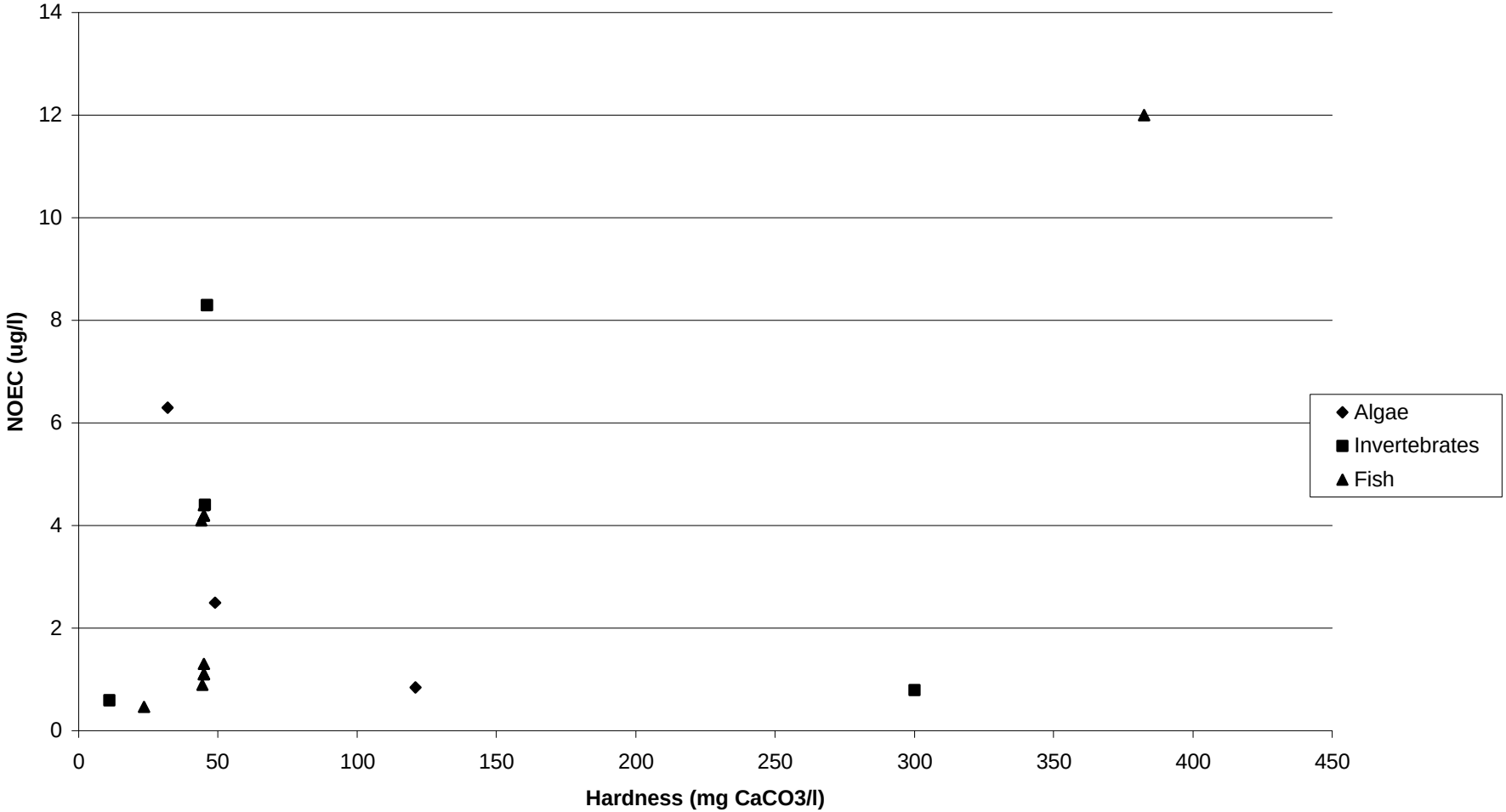


Figure 6.1: Cadmium Toxicity and Hardness (RI 1-2 data)

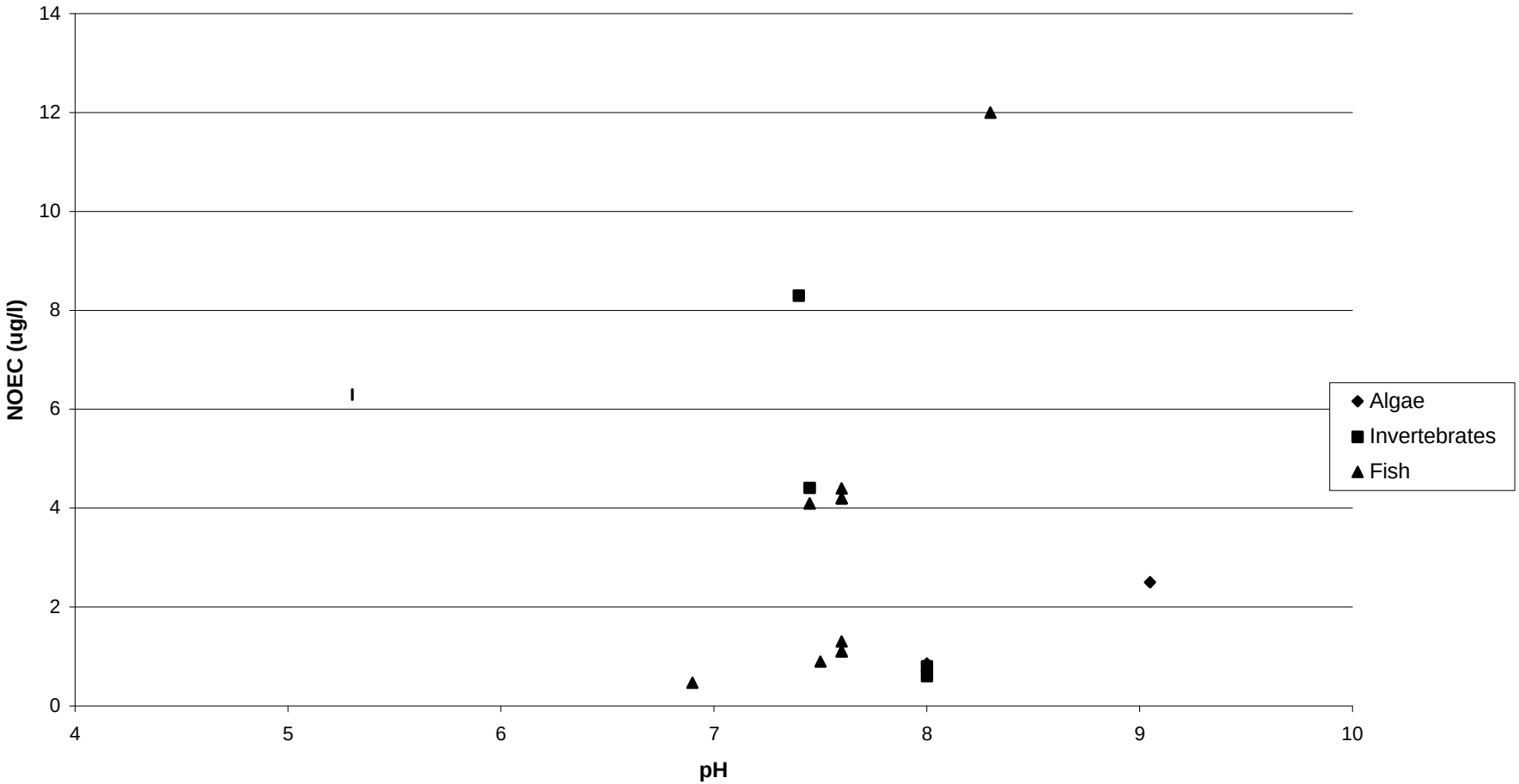


Figure 6.2: Cadmium Toxicity and pH (RI 1-2 data)

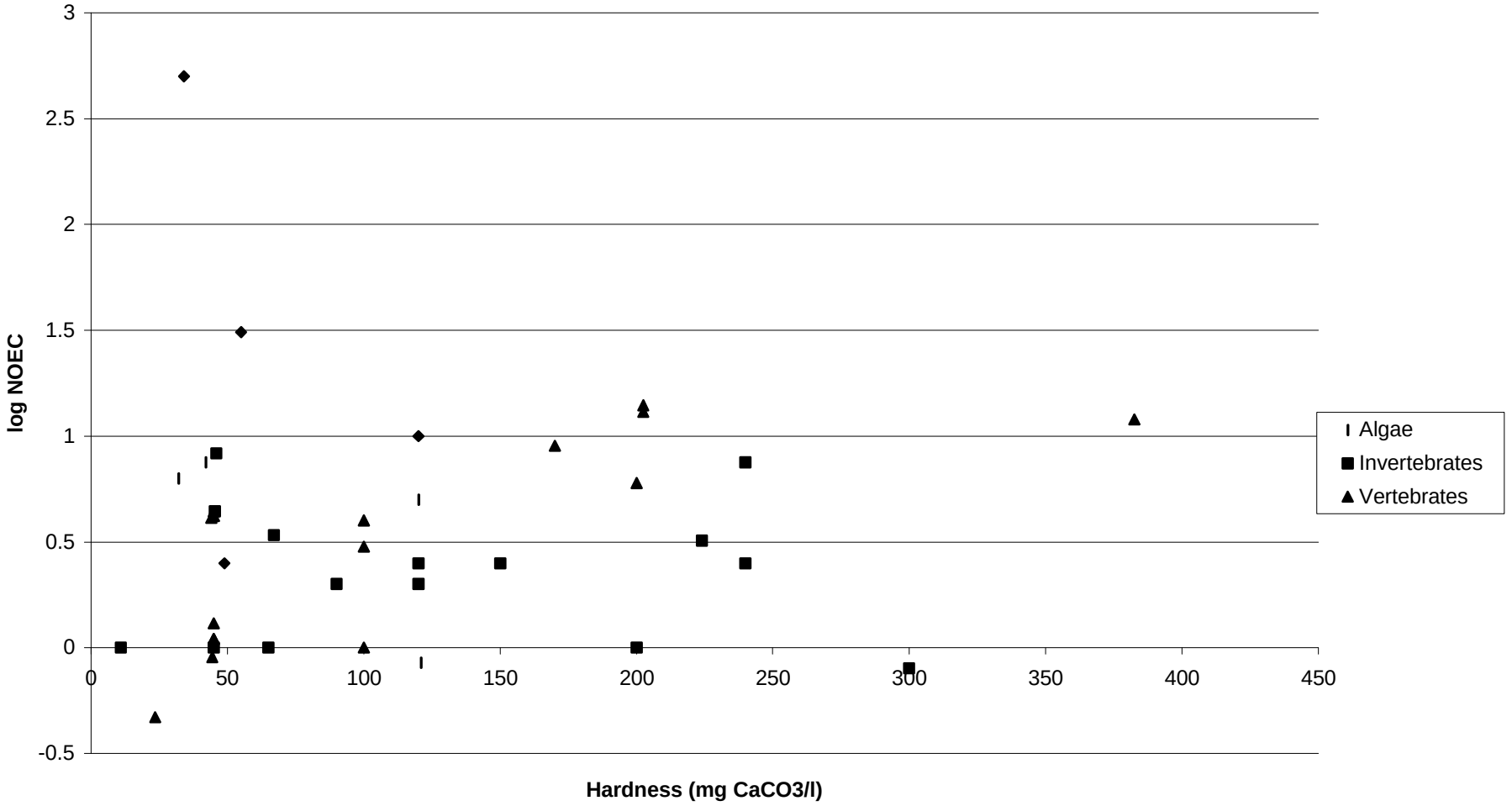


Figure 6.3: Cadmium Toxicity and Hardness (RI 1-3 data)

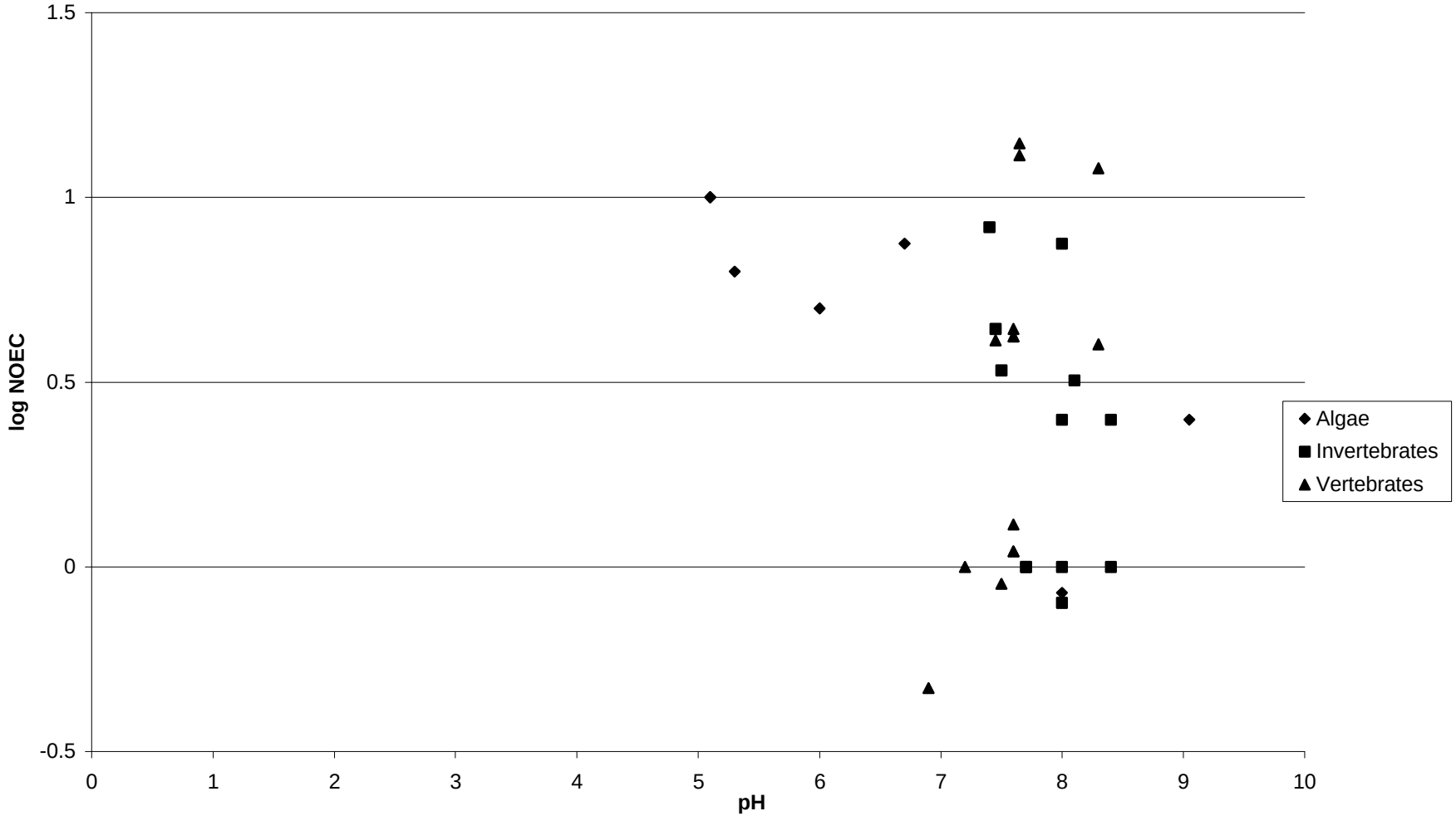


Figure 6.4: Cadmium Toxicity and pH (RI 1-3 data)

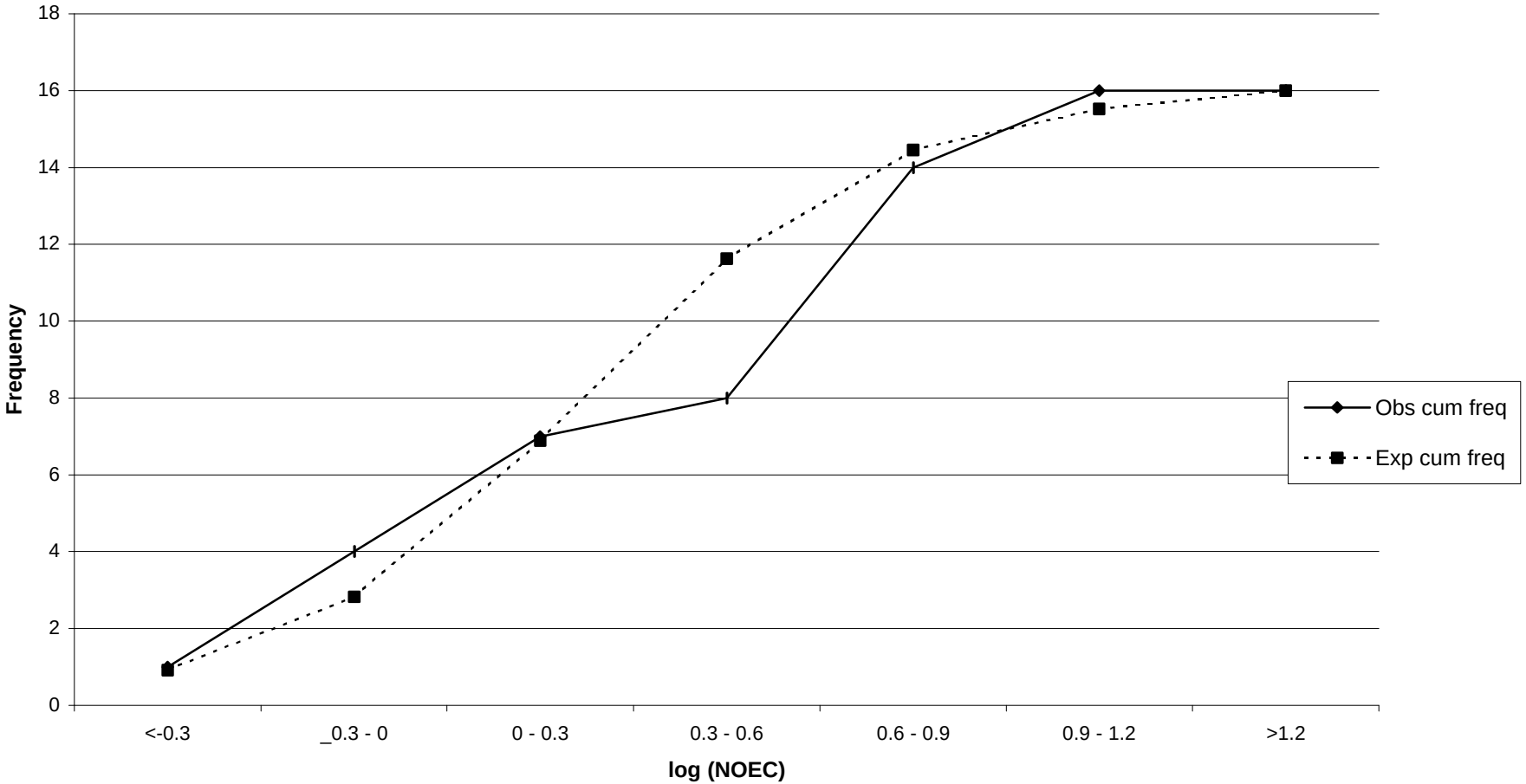


Figure 6.5: Aquatic Toxicity - Expected and Observed Cumulative Frequencies

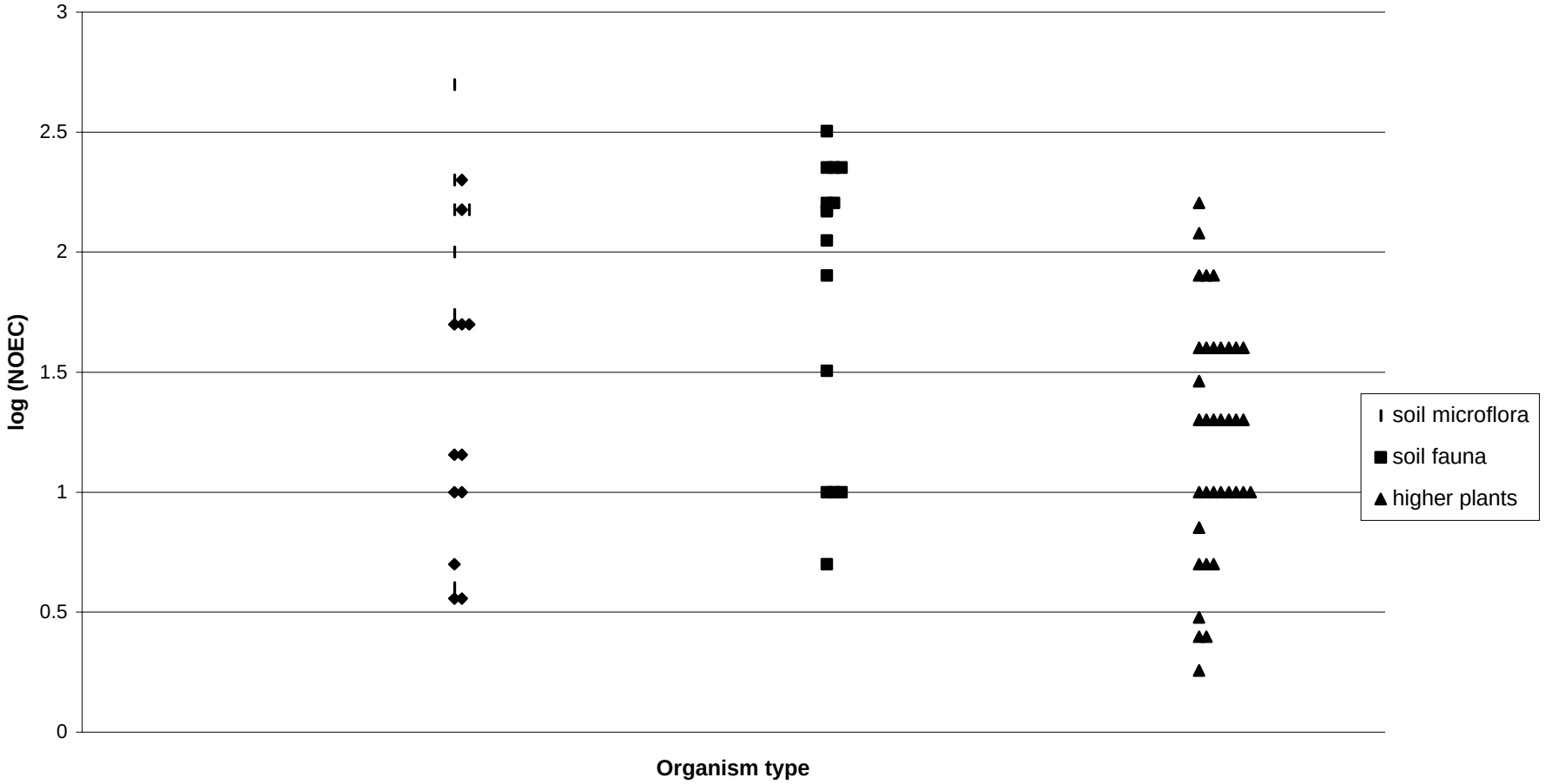


Figure 6.6: Soil Toxicity by Organism Type (RI 1-3 Data)

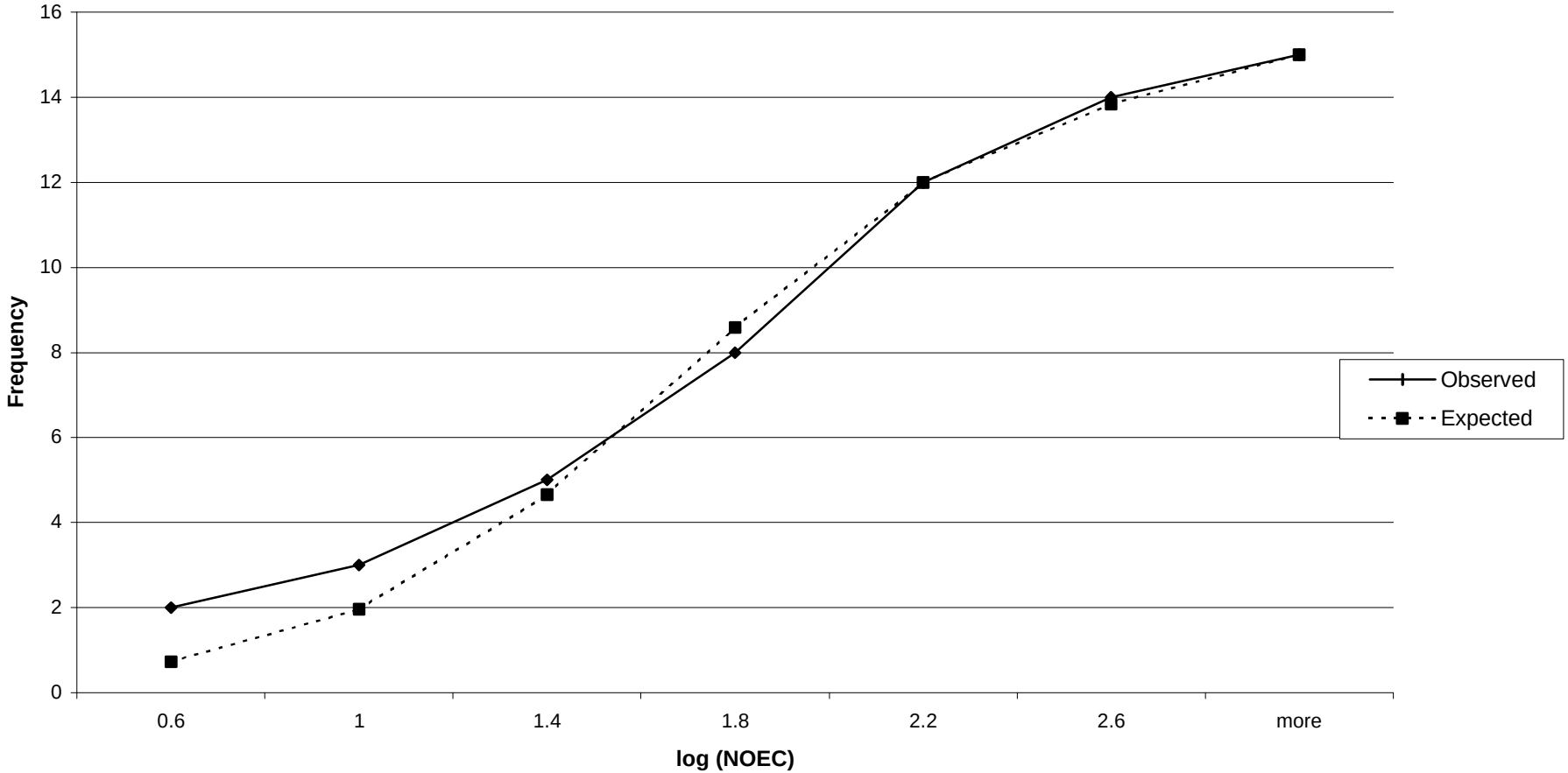


Figure 6.7: Soil Microflora - Expected and Observed Cumulative Frequencies

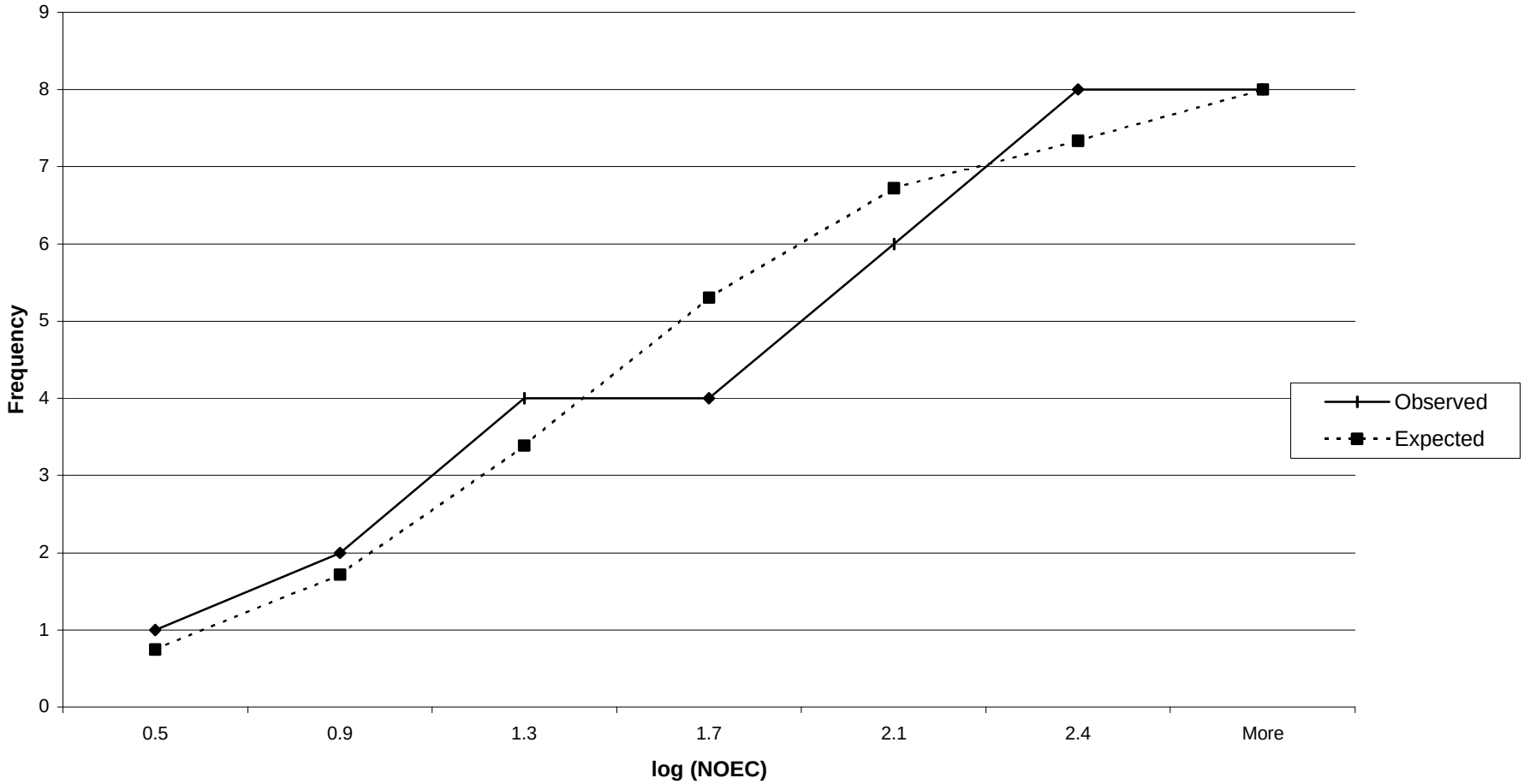


Figure 6.8: Soil Fauna & Plants - Expected and Observed Cumulative Frequencies

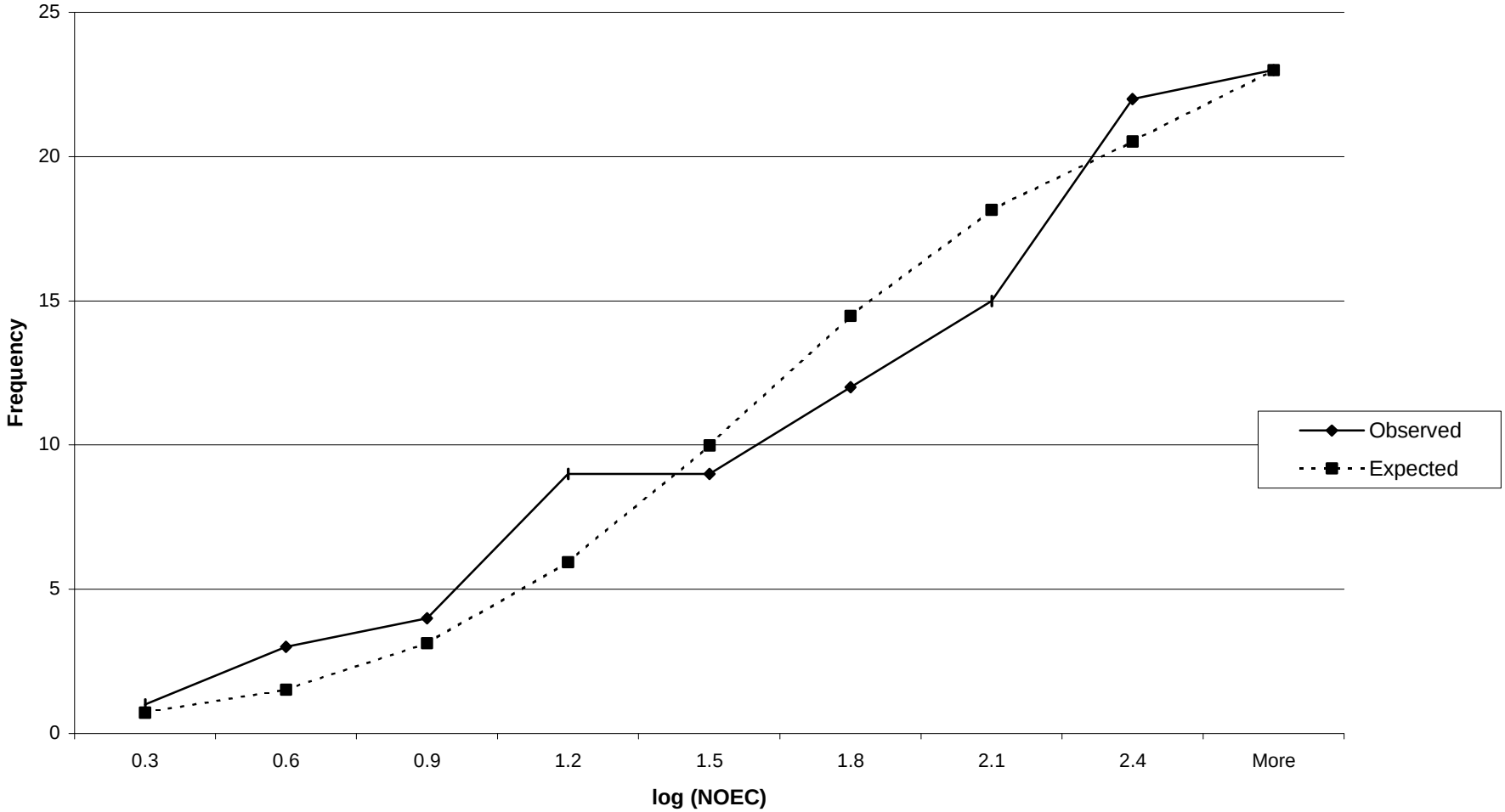


Figure 6.9: Soil Combined Data - Expected and Observed Cumulative Frequencies

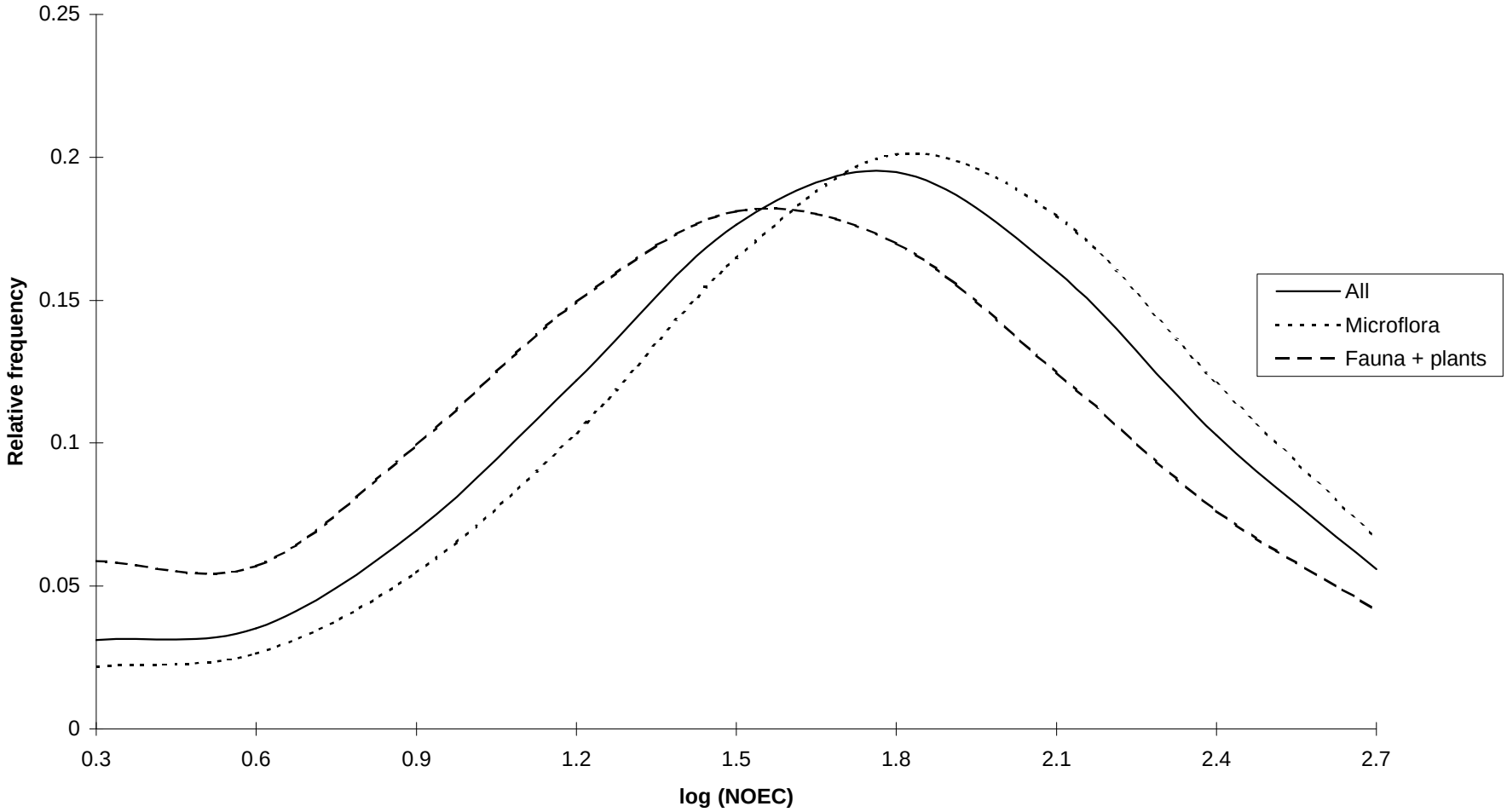


Figure 6.10: Expected Relative Frequencies for Soil Toxicity Data

6.3 Human Toxicity

6.3.1 Overview

Cadmium is toxic to humans. It is not required for any biological process. It is a non-essential metal and therefore unlike the trace elements such as, potassium and zinc, it cannot be present in concentrations that are too low. Cadmium is therefore assessed in this review in terms of concentrations that are high enough to result in toxic effects.

Occupational exposure to cadmium and other cadmium compounds may occur in industrial plants where cadmium is produced, used or extracted.

In the general population, both non-smokers and smokers, i.e. those not working in industries using or producing cadmium, intake of cadmium occurs primarily during ingestion of food or drinking water contaminated by cadmium and/or cadmium compounds. Exposure may also occur by inhalation of air containing cadmium and cadmium products. These exposures are the result of the release/presence of significant quantities of cadmium and cadmium compounds in the environment and its transfer/presence in soil, water and air.

Consumers may also be exposed to cadmium and cadmium products through using products, which may be cadmium compounds or preparations or items containing cadmium compounds.

The effects, which have occurred in humans and animals, vary depending on the route and duration of exposure. For each cadmium compound under consideration, the routes of exposure, in order of importance, have therefore been considered in terms of the effects on health, including, death, systemic, immunological, neurological, reproductive, developmental, genotoxic and carcinogenic effects. These data have then been discussed in terms of duration of exposure, i.e. acute (14 days or less), intermediate (15 - 364 days) and chronic (365 days or more).

6.3.2 Exposure by Inhalation

Overview

The information on the health effects of inhalation exposure to cadmium in humans is derived from studies of workers exposed to cadmium fumes or dusts in pigment production, metal plating, stabiliser production and use and battery manufacture. Occupational exposure occurs primarily by inhalation of fumes and/or dust. Some GI tract exposure may also occur when dust is removed from the lungs by mucocilliary clearance and subsequently swallowed or by ingestion of dust on hands, cigarettes or food (Adamsson *et al*, 1979). Similarly in toxicity studies with animals, some ingestion may also occur after exposure by the inhalation route due to mucocilliary clearance or from grooming. Cadmium oxide is the form of cadmium most commonly found in occupational exposure. The effects of cadmium oxide, cadmium chloride, cadmium sulphate and cadmium sulphide have been investigated in toxicity

studies in laboratory animals which have been exposed to the compounds by the inhalation route. In general, the different cadmium compounds have similar toxicological effects when exposure occurs by the inhalation route, although quantitative differences may be the result of different absorption and distribution characteristics, particularly for the less soluble cadmium pigments (e.g. cadmium sulphide and cadmium selenium sulphide) (NTIS, 1999).

Death - Acute Exposure (< 15 days)

Studies in Humans

Fatal acute exposures by inhalation have occurred in occupational accidents. During and immediately afterwards the symptoms are relatively mild. However within a few days severe pulmonary oedema and pneumonitis develop leading to death as a result of respiratory failure (Beton *et al*, 1966; Lucas *et al*, 1980; Patwardhan & Finckh, 1976; and Seidal *et al*, 1993). Elinder (1986) estimated that an exposure to 1-5 mg/m^3 for 8 hours would be immediately dangerous (cited in NTIS, 1999).

Studies in Animals

A single exposure to cadmium oxide has caused death in rats, mice, rabbits, guinea pigs, dogs and monkeys. The incidence of death was apparently related to the product of the duration of exposure and the concentration of cadmium in the atmosphere by Barrett *et al* (1947). The LC_{50} 7 days after exposure was 7.5 hr-mg Cd/m^3 for rats, based on a 15-minute exposure to 30 mg Cd/m^3 (Barrett *et al*, 1947 – cited in NTIS, 1999). Rusch *et al* (1986) produced a high incidence of mortality (25/32 within 7 days) among Sprague-Dawley rats using a 2-hour exposure period to cadmium fumes at 112 mg Cd/m^3. A two hour exposure to cadmium carbonate, at 132 mg Cd/m^3 resulted in a lower incidence of deaths (3/22 by day 30). A 2-hour exposure to 99 mg Cd/m^3 of cadmium sulphide or 97 mg Cd/m^3 cadmium selenium sulphide (cadmium red pigment) did not cause death (Rusch *et al*, 1986 - cited in NTIS, 1999). Two of 36 rats died after a two hour, nose only exposure to 0.45 mg Cd/m^3 of cadmium oxide dust (Grose *et al*, 1987 – cited in NTIS, 1999). Exposure to cadmium chloride aerosol at a concentration of 61 mg Cd/m^3 for one hour per day, for three days resulted in 17/18 deaths (Snider *et al*, 1973, - cited in NTIS, 1999). In another study no deaths were reported in rats exposed to 6.29 mg Cd/m^3 cadmium yellow (CdS) pigment for 10 days, 6 hours/day (Klimisch, 1993 – cited in NTIS, 1999). It appears from the above data that the relatively more soluble cadmium chloride, cadmium oxide fumes, and cadmium carbonate compounds are more toxic than the relatively less soluble sulphide compounds following acute exposure. However, Glaser *et al* (1986 as cited in NTIS, 1999) demonstrated that there is not a strict correlation between toxicity and solubility and that the solubility of cadmium oxide, for example, was greater in biological fluids than in water.

These and further data are summarised in Table 6.4.

Table 6.4: Summary of Animal Studies for Death following Acute Exposure				
Dose (hr-mg/m3)	**Exposure**	**Target**	**Effect**	**Author**
0.9 Cd	CdO dust (2 hrs @0.45 mg/m^3)	Rats (nose only)	2/36	Grose *et al*, 1987
7.5 Cd	CdO (15 mins @30 mg/m^3)	Rats	7d LC50	Barrett *et al*, 1947
12* Cd	CdO (15 mins @46.7 mg/m^3)	Mice	7d? LC50	Barrett *et al*, 1947
51* Cd	CdO (15 mins @204 mg/m^3)	Guinea pigs	7d? LC50	Barrett *et al*, 1947
57* Cd	CdO (15 mins @230 mg/m^3)	Dogs	7d? LC50	Barrett *et al*, 1947
183 Cd	$CdCl_2$ (1 hr @61 mg/m^3 for 3d)	Rats	17/18	Snider *et al*, 1973
194 Cd	CdSeS (2hrs @97 mg/m^3)	Rats?	No deaths	Rusch *et al*, 1986
198 Cd	CdS (2hrs @99 mg/m^3)	Rats?	No deaths	Rusch *et al*, 1986
219* Cd	CdO (15 mins @940 mg/m^3)	Monkeys	7d? LC50	Barrett *et al*, 1947
224 Cd	Cd fume (2hrs @112 mg/m^3)	Sprague-Dawley rats	25/32 within 7d	Rusch *et al*, 1986
264 Cd	$CdCO_3$ (2hrs @132 mg/m^3)	Rats	3/22 within 30d	Rusch *et al*, 1986
377 Cd	CdS (6hrs @6.3 mg/m^3 for 10d)	Rats	No deaths	Klimisch, 1993
** The authors considered these values to be approximations because of inadequacies in the data and/or the small number of animals used.*				

Death - Intermediate Exposure (15 - 364 days)

Studies in Humans: No studies on deaths in humans from intermediate term exposures were found.

Studies in Animals

Various studies have been undertaken into the effects of repeated exposures to cadmium compounds over a period of weeks. Table 6.5 summarises some of the studies cited in NTIS (1999).

Death - Chronic Exposure (1 year or more)

Studies in Humans

One study (Friberg, 1950) attributes the deaths of two workers (aged 57 and 60) to exposure to an average of 6.8 mg Cd/m^3 (range 3-15 mg/m^3) cadmium dust for 14 or 25 years.

Table 6.5: Summary of Animal Studies for Death following Intermediate Exposure				
Dose (hr-mg/m3)	**Exposure**	**Target**	**Effect**	**Author**
145 Cd	CdO dust (22 hrs/d for 63d @0.105 mg/m^3)	Rats (female)	5/12	Oldiges & Glaser, 1986
159 Cd	CdO dust (24 hrs/d for 63d @0.105 mg/m^3)	Rats (female)	5/12	Prigge, 1978a
394 Cd	$CdCl_2$ (6 hrs/d for 62d @1.06 mg/m^3)	Rats	5/54	Kutzman *et al*, 1986
500 Cd	CdO dust (5 hrs/d for 100d @1mg/m^3)	Rats (female)	100% dead	Baranski & Sitarek, 1987
Source: References cited in NTIS, 1999.				

A detailed post mortem of the elder worker revealed the presence of emphysema and hyaline casts in renal tubules and slight nephrotic changes. Pneumonia, an acute complication of chronic bronchitis and pulmonary emphysema was the cause of death. The exposure was estimated from 6 samples taken in 1946, and though conditions were thought to be similar in earlier years this exposure value must be considered as an approximation/guide to exposure spanning 34 years.

Studies in Animals

Takenaka *et al* (1983, cited in NTIS, 1999) reported that 5/40 rats died as a result of exposure to cadmium chloride at a concentration of 0.0508 mg Cd/m^3 for 23 hours/day, 7 days/week for 18 months. However, the most comprehensive study on chronic exposure was undertaken by Oldiges *et al* (1989, cited in NTIS, 1999) which evaluated the long term effects of inhaling cadmium as cadmium oxide, cadmium chloride, cadmium sulphate or cadmium sulphide. Rats were exposed to aerosols 22 hours/day, 7 days/week, for 18 months and observed during a 12 month post-treatment period. If mortality reached 25% in the test animals during the exposure period or reached 75% of the animals during the treatment-free period which followed the period was terminated. In these studies the order of toxicity (most to least) was found to be cadmium chloride > cadmium sulphate = cadmium oxide dust > cadmium sulphide. All the cadmium compounds studied caused death. Of note is that Oldiges & Glaser (1986, cited in NTIS, 1999) reported that at the concentrations tested in their chronic studies, cadmium toxicity appeared to be more related to the long-term lung retention of the bioavailable cadmium than to the solubility of the various compounds in water. The associated results are given in Table 6.6.

Respiratory Effects - Acute Exposure (< 15 days)

Studies in Humans

In humans, inhalation exposure to high levels of cadmium oxide fumes or dust is intensely irritating to respiratory tissue, but symptoms can be delayed. Precise estimates of cadmium concentrations leading to acute respiratory effects in humans are not currently available.

Table 6.6: Summary of Animal Studies for Death following Chronic Exposure				
Dose (hr-mg/m3)	**Exposure**	**Target**	**Effect**	**Author**
241 Cd	$CdCl_2$ (22 hrs/d for 12m @0.03 mg/m^3)	Rats (m & f)	75% dead	Oldiges *et al*, 1989
460 Cd	CdO dust (22 hrs/d for 7m @0.09 mg/m^3)	Rats (male)	>25% dead	Oldiges *et al*, 1989
640 Cd	$CdCl_2$ (23 hrs/d for 18m @0.0508 mg/m^3)	Rats	5/40	Takenaka *et al*, 1983
722 Cd	CdO dust (22 hrs/d for 11m @0.09 mg/m^3)	Rats (female)	>25% dead	Oldiges *et al*, 1989
1182 Cd	$CdSO_4$ (22 hrs/d for 18m @0.09 mg/m^3)	Rats (male) Rats (female)	>25% dead 18m >75% dead 30m	Oldiges *et al*, 1989
1182 Cd	CdS (22 hrs/d for 18m @0.09 mg/m^3)	Rats (m & f)	no deaths in 18m >75% dead 30m	Oldiges *et al*, 1989
Source: References cited in NTIS, 1999.				

During and immediately after (up to 2 hours) an acute exposure for 5 hours of 8.63 mg/m^3, Beton *et al* (1966) reported that the symptoms of toxicity were limited to coughing and slight irritation of the throat and mucosa. Four to 10 hours post-exposure, influenza-like symptoms began to appear, including cough, tight chest, pain in chest on coughing, dyspnea, malaise, ache, chilling, sweating, shivering, and aching pain in back and limbs. Eight hours to seven days post-exposure, the pulmonary response included severe dyspnea and wheezing, chest pain and precordial constriction, persistent cough, weakness and malaise, anorexia, nausea, diarrhoea, nocturne, abdominal pain, hemoptysis, and prostration.

Acute, high-level exposures can be fatal, and those who survive may have impaired lung function for years after a single acute exposure. Barnhart & Rosenstock (1984) report that a 34-year-old worker exposed to cadmium fume from soldering for 1 hour (dose not determined) had persistent impaired lung function when examined 4 years following the exposure. Initial symptoms were dyspnea, cough, myalgia, and fever. An initial chest X-ray revealed infiltrates. Townshend (1982) reported the case of a male welder who developed acute cadmium pneumonitis from a single exposure (dose not determined). Nine years after the exposure, this worker continued to show signs of progressive pulmonary fibrosis and had no improvement in respiratory function.

Studies in Animals

In rats, a single 1-5 hour exposure to a concentration of 5-10 mg/m^3 cadmium oxide dust, cadmium oxide fumes or cadmium chloride has been found to cause moderate – severe changes in the lungs, including increased weight, interstititial pneumomitis, diffuse alveolitis with haemorrhage, inhibition of macrophages, focal interstitial thickening, oedema and necrosis of alveolar type 1 cells leading to type 2 cell hyperplasia and fibroblasts (Boudreau *et al*, 1989; Buckley & Basset, 1987; Bus *et al*, 1978; Grose *et al*, 1987; Hart *et al*, 1989a; and Palmer *et al*, 1986).

These and some further results are summarised in Table 6.7.

Table 6.7: Summary of Animal Studies for Respiratory Effects following Acute Exposure

Dose (hr-mg/m^3)	Exposure	Target	Effect	Author
5 Cd	$CdCl_2$ (30 mins @10 mg/m^3)	Hamsters	severe pneumonitis	Henderson *et al*, 1979
9 Cd	CdO dust (2 hrs @4.5 mg/m^3)	Rabbits	severe pneumonitis	Grose *et al*, 1987
10 Cd	$CdCl_2$ (6 hrs/d for 10d @0.17 mg/m^3)	Rats	absolute lung weight +16%	Klimisch, 1993
5-50 Cd	CdO & $CdCl_2$ (1-5hrs @5-10 mg/m^3)	Rats	severe changes	as given in text above
30-91 Cd	$CdCl_2$ (1 hr/d for 5,10 & 15 @0.61 mg/m^3 & 1 hr/d for 3d @61 mg/m^3	Rats	emphysema and pulmonary haemorrhage respectively	Snider *et al*,, 1973
224 Cd	CdO fumes (2 hrs @112 mg/m^3)	Rats	rales, laboured breathing and discolouration of the lungs	Rusch *et al*, 1986
264 Cd	$CdCO_3$ (2 hrs @132 mg/m^3)	Rats	rales, rapid breathing and 2-3-fold increases in lung weight	Rusch *et al*, 1986

Some cadmium compounds are more toxic than others. Cadmium acetate is similar to cadmium chloride in that exposure to 1-5 mg/m^3 resulted in severe respiratory effects. In addition a single intra-tracheal instillation of 0.5 mg/kg body weight (estimated to be 2.4 mg/m^3) resulted in changes in the levels of lung enzymes, e.g. depressed levels of catalase and superoxide dismutase, increases in non protein sulfhydryl, glucose-6-phosphate dehydrogenase and glutathione peroxidase in lung tissue and increases in lactic dehydrogenase and protein in bronchoalveolar lavage fluid (Salovsky *et al*, 1992). Exposure to cadmium sulphide at a concentration of 6.29 mg/m^3 for 6 hours daily for 10 days caused an 8% increase in absolute lung weight compared to the 16% increase seen above with cadmium chloride (Klimisch, 1993). Neither 99 mg/m^3 cadmium sulphide nor 97 mg/m3 cadmium selenium sulphide had any effect on the respiratory tract after a 2 hour exposure (Rusch *et al*, 1986).

Persistent damage following a single exposure has been reported. Fibrosis caused by acute exposure was observed up to 12 months post-exposure (Dervan & Hayes, 1979). Driscoll *et al* (1992) also reported that in rats exposed once, via intratracheal instillation, to cadmium chloride at levels of 25, 100 or 400 μg/kg significant increases in BALF, LDH, total protein and NAG levels were present 3 and 7 (except LDH and NAG at the low dose) days post-exposure and that total protein was still elevated in the high level group 28 days post exposure. Neutrophil and lymphocyte numbers were increased initially but returned to control levels by days 14 and 28 respectively. Alveolar macrophage numbers increased after day 7 and remained elevated. Macrophage fibronectin also increased (in a dose related manner) and remained high 28 days post-exposure. Cell viability was not affected.

Hydroxyproline levels increased at the 2 higher levels. Histopathological changes in the lung consisted of chronic interstitial inflammation characterised by increase thickening of the alveolar wall, increased numbers of mononuclear cells, type 2 cell hyperplasia, and at times brown pigment-laden macrophages. Alveolar spaces were variably collapsed with dilatation of some terminal bronchioles, alveolar ducts and adjacent alveoli. The overall histopathological response was more severe 90 days post-exposure than 28 days post-exposure. Fibrosis, indicated by minimal to moderate prominence of collagen, was also more severe after 90 than 28 days.

In other studies similar transient increase in BALF enzymes or other indicators of pulmonary pneumonitis were seen, though histopathological changes were also found to be transient. For example in a study by Hart (1986) rats, exposed to 1.6 mg/m^3 of cadmium oxide for 3 hours, daily for 5 days/week for 1-6 weeks, developed interstitial pulmonary pneumonitis (indicated by changes in the levels LDH, ALP, acid phosphatase, protein and polymorphonuclear leucocytes in the airways) in the 1^{st} 2 weeks. The levels of these biochemical indicators and cytological indicators of toxicity and the associated histopathological alterations returned to expected levels in the following 3 weeks despite the fact that cadmium continued to accumulate in the lung. The author suggested that the adaptive synthesis of Cd-binding protein (presumptive metallothioneins) in the lung served to sequester cadmium and protect the tissue from further toxicity. Palmer *et al* (1986) in a study which, evaluated the role of thyroid hormones in the pulmonary repair process in rats exposed to 10 mg/m^3 cadmium chloride for 2 hours, suggested that the persistence of lung damage or the development of further damage for a given level of acute exposure is probably related to the capacity of the pulmonary repair mechanisms and to adaptive responses such as the production of metal binding proteins to sequester free cadmium away from target sites.

Respiratory Effects - Intermediate Exposure (15 - 364 days)

Studies in Humans

The initial symptoms of respiratory distress observed in the acute exposures to high concentrations of cadmium do not occur following lower-level, longer-term inhalation exposures (Friberg, 1950). Longer-term occupational exposure to levels of cadmium below those causing lung inflammation, however, have been reported to cause emphysema and dyspnea in humans (Bonnell, 1955; Friberg 1950; Lane & Campbell, 1954; and Smith *et al*, 1960). Kjellstrom *et al* (1979) reported a significant increase in deaths due to respiratory diseases in cadmium-exposed battery factory workers exposed for longer than 5 years.

A significant, dose-dependent excess in the ratio of observed to expected deaths from bronchitis (i.e. Standardised Mortality Ratio = 434) but not emphysema was found among 6,995 men occupationally exposed to cadmium for an average of 11 years (Armstrong & Kazantzis, 1983). Dose level was not determined and there was no control for the health effects of cigarette smoking. There is some evidence that cadmium may accelerate the development of emphysema in smokers. Leduc *et al* (1993) reported the case history of a 59-year-old male worker who smoked a pack of

cigarettes per day since age 16, but had no prior history of respiratory disease in 1975 until developing emphysema in 1979 after inhaling various concentrations of cadmium (range of 0.0164 - 1.192 mg/m^3, mean of 0.446 mg/m^3, about nine times the threshold value of 0.050 mg/m^3) for 4 years as a furnace operator. Very high levels of cadmium in air samples at the workplace and in the patient's blood, urine, and lung tissue confirmed massive exposures. Lung-function tests declined rapidly, with a faster than usual onset of emphysema compared to other smokers. The mean concentration of cadmium in a removed section of lung was 580 μg/g dry tissue, compared to 14 μg/g in three unexposed controls matched for age, sex, and smoking habit who had also under-one resection of a bronchial carcinoma. The authors stated that this case supports the hypothesis for an etiological role of cadmium fume inhalation in the development of emphysema.

More recent studies that controlled for smoking report lung impairment in cadmium-exposed workers (Chan *et al*, 1988; Cortona *et al*, 1992; Davison *et al*, 1988; and Smith *et al*, 1976). Cortona *et al* (1992) measured respiratory function parameters in 69 smoking and non-smoking male subjects (average age 45) who were exposed to concentrations of 0.008 - 1.53 mg/m^3 of cadmium fumes over a period of several years in a factory that produced cadmium alloys (silver-cadmium-copper). There was a significant increase in 'residual volume' (but NOT of other lung functions) of more than 8% in exposed workers; this effect was notably greater in those with higher cumulative exposures to cadmium (> 10%). It is uncertain how much of a factor on the increased RV was due to the tendency of smokers to develop an initial emphsematous alteration in lung tissue due to smoking.

Studies in Animals

Exposure levels in the concentration range 0.4 - 4 mg Cd/m^3 generally result in serious damage to the lungs. Some examples are presented in Table 6.8.

Table 6.8: Summary of Animal Studies for Respiratory Effects following Intermediate Exposure

Dose (hr-mg/m3)	Exposure	Target	Effect	Author
48-72 Cd	$CdCl_2$ (6 hrs/d, 5d/wk, 4-6wks @0.4 mg/m^3)	Rabbits	type 2 cell hyperplasia and interstitial inflammation	Johansson *et al*, 1984
120-144 Cd	CdO (3 hrs/d for 25-30d @1.6 mg/m^3	Rats	type 2 cell hyperplasia and dry weight of the lungs +41%	Hart *et al*, 1989a
293 Cd	$CdCl_2$ (24 hrs/d for 21d @0.581 mg/m^3)	Pregnant rats	emphysema and bronchiolitis	Prigge, 1978b
394 Cd	$CdCl_2$ (6 hrs/d for 62d @0.106 mg/m^3)	Rats	fibrosis with a significant increase in collagen	Kutzman *et al*, 1986
2268 & 2704 Cd	Cd dust (3 hrs/d, 21 & 23 d/m, 9 & 7m @4 & 5.6 mg/m^3)	Rabbits	chronic pneumonia and emphysema	Friberg, 1950

As the exposure period lengthens increasingly lower doses result in respiratory toxicity. Cadmium oxide dust for example, has been shown to induce emphysema and histiocytic cell granulomas in rats when administered continuously at a concentration of 0.105 mg Cd/m3 for 62 days (Prigge, 1978a). The continuous administration of cadmium oxide at a lower dose of 0.025 mg Cd/m^3 for 90 days produced less severe toxicity, as evidenced by hypercellularity in the bronchoalveolar region and an increase in the relative weight of the lungs. Glaser *et al* (1986) also found bronchoalveolar hypercellularity when 0.098 mg Cd/m^3 cadmium oxide dust, 0.105 mg Cd/m^3 cadmium chloride and 1.034 mg Cd/m^3 cadmium sulphide were administered for 22 hours daily for 30 days. Cadmium sulphide appears to be less toxic than cadmium oxide or cadmium chloride when administered as a single or repeated dose.

A degree of tolerance may develop as the duration of the dose/exposure period increases so that lung lesions that developed after a few weeks of exposure are not seen to progress and sometimes regress after longer exposures (Hart, 1986; Hart *et al*, 1989a). These authors believe multiple mechanisms including the synthesis of lung metallothionein and an increase in type 2 cells are involved in this apparent tolerance. Oberdorster *et al* (1994) compared the responses of rats and mice to long term exposure to cadmium chloride at 100 μg Cd/m^3, 6 hrs/d, 5 d/wk for 4 wks. The parameters monitored included metallothionein, levels of cadmium in the lung, BALF neutrophil counts, β-glucuronidase and LDH, cell proliferation, measured by bromodeoxyuridine (BrdU) incorporation into lung tissue, lung morphology (using histochemical staining), and induction of metallothionein concentration in lung tissue. Mice were found to be more susceptible to exposure to cadmium chloride than rats under these conditions. There was a greater response in the following parameters: inflammation in the lungs, cell proliferation, the baseline metallothionein level was higher, lung metallothionein was more inducible and the lung burden of cadmium metallothionein was twice as high as that in the rats. Due to the higher respiratory rate in mice the higher burdens were not unexpected. In addition to the increase in cell proliferation, the mice also responded with a significant induction of metallothionein in the epithelial cells of the conducting airways and alveolar region. No such response was seen in the rats. The authors suggested that the enhanced responses in mice may contribute to the lack of pulmonary carcinogenicity found in mice as increased metallothionein levels may provide more protection against the development of lung tumours in proliferating cells. The authors also noted that an increase in the rate of cell proliferation does not necessarily lead to an increased risk of tumour development as the rat in which, the proliferative response is lower, is more prone to lung tumours from inhaled cadmium.

Respiratory Effects - Chronic Exposure (1 year or more)

Studies in Humans

Davison *et al* (1988) evaluated lung function in 101 men who had manufactured copper-cadmium alloy in a plant in England for one or more years since 1926. The exposed men were compared to controls from the factory's other seven divisions matched for age and employment status. The number of smokers among exposed and

control men was similar. Between 1951 and 1983, 933 measurements of airborne cadmium had been made, 697 with static samplers and 236 with personal samplers. The various sampling methods used before 1964 are no longer considered to be reliable, so estimates of air concentrations were made based on changes in production techniques, ventilation, levels of production, and discussions with occupational health physicians, industrial hygienist, the management, and the workers. Cadmium concentrations in air from 1926 to 1972 were determined to have declined from 0.6 to 0.156 mg/m^3. In 1973, concentrations were 0.085 mg/m^3, then from 1974 to 1983 concentrations ranged from 0.034 - 0.058 mg/m^3. The lung function of 77 of the men occupationally exposed to cadmium was significantly impaired compared to the unexposed controls, with the greatest abnormalities in the highest-dose group. Regression of the lung transfer coefficient versus cadmium exposure indicated a linear relationship with no apparent threshold.

Smith *et al* (1976) studied the pulmonary function of 17 high-exposure workers, 12 low-exposure workers, and 17 controls. Cadmium air concentrations where high-exposure subjects worked were >0.2 mg/m^3. High-exposure subjects had worked at the plant a median of 26.4 years, with a maximum of 40.2 years, and low-exposure subjects had worked a median of 27.1 years, with a maximum of 34.8 years. Workers with high exposure to cadmium had significantly decreased forced volume capacity (FVC) compared to low-exposure workers and controls. Chest X-rays indicated mild or moderate interstitial fibrosis in 29% of high exposure workers. A dose-response relationship was found between forced vital capacity and urinary cadmium, and with months of exposure to cadmium fume but not cadmium sulphate aerosol. In an analysis of the smoking habits, there was no significant difference between the two cadmium-exposed groups with respect to the proportion of present or past cigarette smokers, the intensity or duration of cigarette smoking, or cigar or pipe smoking habits. The control subjects, however, had a significantly ($p<0.05$) "higher" exposure to cigarette smoke than the cadmium exposed workers with substantially greater numbers of pack-years, cigarettes smoked per day, and years smoked. A step-down and multiple regression analyses with a dependent variable of FVC (as percent of predicted), and the independent variables, age-height, cigarette pack-years, and urinary cadmium, resulted in no indication that an interaction between the independent variables led to the observed relationship between FVC and cadmium excretion.

Other studies, however, have not shown a cadmium-related increase in impaired respiratory function. Edling *et al* (1986) studied Swedish workers occupationally exposed to cadmium oxide (CdO) fume from cadmium-containing solders. Cadmium-containing solder had been used at the plant from 1955 to 1978. The results from the lung-function analysis showed no significant difference in symptoms or lung function between the Cd-exposed and the reference group. The exposed and the reference groups were similar with respect to sex, age, and height. There was a higher percentage of smokers in the reference group (52%) than in the exposed group (42%), but the difference was not statistically significant. The authors could not explain why significant differences in effects were not seen in these workers since other studies have shown significant effects at comparable cadmium exposure levels. The authors suggest that a possible bias could have been introduced if people who had

worked for more than 5 years in the plant had changed their occupation because of lung disease, so that only "healthy" workers remained. Significant effects may also have been found if the reference group included workers other than those who worked with solder, but the purpose of the study was to resolve the effects of cadmium exposure among workers with similar occupations. An analysis that factored out smoking by evaluating the data from smokers and non-smokers separately also showed no significant impairment function between smoking exposed and smoking unexposed or non-smoking exposed and non-smoking unexposed. The lung impairment due to smoking was observed in that smokers in both the exposed and unexposed groups had a somewhat deteriorated closing volume and other lung function indicators in accordance with previous studies on the effects of smoking. These results support the hypothesis that the response to occupational dust exposure differs from the response to tobacco smoking.

Another possible reason for differing results is that lung injury caused by high-level cadmium exposure may be partially reversible (Bonnell, 1955 and Chan *et al*, 1988), with a return towards normal several years after exposures have been significantly reduced. Chan *et al* (1988) studied a cohort of 36 female and 8 male workers at a Singapore cadmium battery factory exposed to cadmium oxide dust. Cadmium concentrations in air were 0.03-0.09 mg/ m^3 (geometric means). Lung function was measured using spirometry, helium dilution, tidal sampling, X-ray, and respiratory symptoms. The recovery of lung function after reduction or cessation of occupational exposure to cadmium dusts was assessed. Total lung capacity increased following reduction of exposure and, following cessation of exposure, vital capacity, FEV, and prevalence of respiratory symptoms all improved. Blood and urine cadmium concentrations were considerably lower with the reduction or cessation of exposure and were consistent with a decrease in the cadmium air levels.

Additional respiratory symptoms less frequently reported in workers occupationally exposed to cadmium are chronic rhinitis and impairment or loss of the sense of smell (Adams *et al*, 1969; Bonnell, 1955; Friberg, 1950; and Rose *et al*, 1992). The cause of these effects may be chronic irritation or necrosis of the nasal membranes, as these findings are generally found only in individuals who have been exposed to cadmium at a high concentration.

Studies in Animals

There are few chronic inhalation exposure studies in animals that specifically address respiratory effects. Oldiges & Glaser (1986) reported an unspecified increase in lung weights of rats exposed to cadmium sulphate at a concentration of 0.092 mg Cd/m^3 or cadmium sulphide at a concentration of 0.254mg Cd/m^3 for 22 hours daily for 413-455 days. Takenaka *et al* (1983) observed hyperplasia in the bronchoalveolar region in rats exposed to cadmium chloride at 0.0134 mg Cd/m^3 for 23 hours daily for 18 months.

Cardiovascular Effects

Studies in Humans

Evidence of toxicity which could be related to cadmium has not been found in the majority of occupationally exposed workers.

In some studies, the mortality from cardiovascular disease was lower in the cadmium-exposed population. Armstrong & Kazantzis (1983) reported that a cohort of 6,995 British men occupationally exposed to cadmium for an average duration of 11 years had a significantly lower mortality from vascular disease.

Fifty-three male workers exposed to cadmium and lead and 52 male controls were examined for correlations in urine levels and blood pressure. The average duration of exposure was 12.5 years. Correlations between blood pressure and urinary cadmium in exposed workers were not significant after controlling for age or age and heart rate. Exposure to lead was a significant confounding factor (de Kort *et al*, 1987).

Friberg (1950) investigated the health of workers in a manufacturing plant that made cadmium-containing electrodes. Fifty-eight workers (30-50 years of age) were divided into 2 groups based on number of years at the plant**.** Workers were clinically examined for subjective symptoms and corresponding morphological or functional changes of the respiratory, cardiovascular, and excretory systems. The cardiovascular exam was largely unremarkable. Only a slight rise in blood pressure in a few cases was observed in Group 1. Electrocardiograms (ECG) were not significantly different from a matched control group in Group 1. Group 2 had neither increased blood pressure nor altered ECGs.

Kazantzis *et al* (1988) studied mortality in a cohort of 6,958 cadmium-exposed male workers with average occupational exposures of 12 years. This was a follow-up study to the work of Armstrong & Kazantzis (1983). There was a significant deficit in deaths from cerebrovascular disease among men occupationally exposed to cadmium. There was no significant excess risk from hypertensive or renal disease.

Smith *et al* (1980) studied 16 male high-exposure production workers and 11 male low-exposure office and supervisory workers for renal function. Average duration of exposure was 25 years. High-exposure workers were exposed to CdO concentrations of 0.23 - 45.2 mg/m^3 and CdS concentrations of 0.04 - 1.27 mg/m^3. No difference was found in hypertension between high- and low-exposure workers, adjusted for age and weight or cigarette smoking.

Sorahan & Waterhouse (1983) examined mortality rates in a cohort of 3,205 nickel-cadmium battery workers (2,559 males and 466 females). Cadmium levels in air ranged from 0.05 to 2.8 mg/m3, primarily as CdO. Duration of exposure ranged from 1 year to more than 6 years. No increase in mortality from diseases of the circulatory system (e.g., hypertension) were seen in cadmium-exposed workers.

Staessen & Lowerys (1993), in a study known as the Cadmibel Study (a cross-sectional population study), evaluated 2,327 people from a random sample of the population of four Belgian districts chosen to provide a wide range of environmental exposure to cadmium. Participants completed a questionnaire regarding their medical history, current and past occupations, smoking habits, alcohol consumption, and intake of medications. Urine and blood samples were taken, and pulse rate, blood pressure, height, and weight were recorded. Exposure to cadmium was considered to be by both the oral and inhalation routes. Cadmium levels in blood and urine were significantly increased in the high-exposure areas compared to the low-exposure areas ($p<0.001$). Blood pressure was not correlated with the urine or blood cadmium levels. The prevalence of hypertension or other cardiovascular diseases was similar in all four districts, and was not correlated with urine or blood cadmium levels. These results do not support a hypothesis that cadmium increases blood pressure, prevalence of hypertension', or other cardiovascular diseases.

One study found a statistically significant increase in blood pressure in exposed workers compared to controls (Thun *et al*, 1989), but mortality in this cohort was lower than expected (Thun *et al*, 1985).

Exposure to cadmium by the inhalation route does not appear to have significant effects on the cardiovascular system.

Studies in Animals

Only one study was found regarding effects in animals after exposure to cadmium via the inhalation route. In this study Kutzman *et al* (1986) reported an increase in the relative weight of the heart in rats exposed to 1.06 mg Cd/m^3 cadmium chloride for 6 hrs/d, 5 d/wk for 62 days. The body weights of these animals were also significantly reduced after this exposure and in the absence of absolute weights of the heart the toxicological significance of this observation is equivocal.

Gastrointestinal Effects

Studies in Humans

Friberg (1950), found no relationship between the exposure of workers to cadmium by the inhalation route, and gastrointestinal toxicity. Symptoms that had been reported in case histories from the 1920s included pain or tenderness at the epigastrum, and nausea and constipation. None of the other human studies reported any cadmium related GI toxicity following exposure by inhalation.

Studies in Animals

Rusch *et al* (1986), in the only animal study found, observed erosion in the stomach of rats following whole-body exposure to cadmium carbonate at a concentration of 132 mg Cd/m^3 for 2 hours when the animals were examined at post mortem 1, 3, 7, and 30 days post-exposure. After the exposure the rats were vacuumed to remove any cadmium carbonate dust adhering to the fur. The dose used was relatively high and

3/10 rats died during the exposure period. The significance of the change in the stomach is uncertain.

Haematological Effects

Studies in Humans

The evidence concerning the toxicological effects of cadmium on haematological parameters and systems is conflicting. Lowered haemoglobin concentrations and decreased packed cell volumes have been observed in some workers exposed occupationally to cadmium (Bernard *et al*, 1979; Friberg, 1950; and Kagamimori *et al*, 1986), but not in others (Bonnell, 1955; Chan *et al*, 1988; and Davison *et al*, 1988). The observed changes were often not statistically significant (Bernard *et al*, 1979; and Friberg, 1950), and examination of the bone marrow smears of some workers with low haemoglobin levels detected no abnormalities (Friberg, 1950).

Studies in Animals

Cadmium has produced conflicting results on haematological parameters as illustrated in Table 6.9.

Table 6.9: Summary of Animal Studies for Haematological Effects

Dose (hr-mg/m3)	Exposure	Target	Effect	Author
112 Cd	CdO dust (24 hrs/d for 90d @0.052 mg/m^3	Rats	increase in levels of haemoglobin and haematocrit	Prigge, 1978a
103-293 Cd	$CdCl_2$ (24 hrs/d for 21d @0.204, 0.394 and 0.581 mg/m^3)	Rats	increase in levels of haemoglobin and haematocrit	Prigge, 1978b
471 Cd	Cd (24 hrs/d for 218d @0.09mg/m^3)	Rats	No effect	Oldiges & Glaser, 1986
744 Cd	CdS (24 hrs/d for 30d @1.034 mg/m^3)	Rats	No effect	Glaser *et al*, 1986
2268 Cd	CdO dust (3 hrs/d, 21 d/m, 9m @4 mg/m^3)	Rabbits	eosinophilia and a slightly lower haemoglobin level	Friberg 1950

A possible, if partial, explanation for these conflicting results may be that the primary cause of cadmium induced anaemia is impaired absorption of iron from the diet following GI exposure to cadmium and the GI exposure following cadmium inhalation is variable depending on the form and dose level.

Musculoskeletal Effects

Studies in Humans

Calcium deficiency, osteoporosis, or osteomalacia was observed in some workers after long term occupational exposure to high levels of cadmium (Adams *et al*, 1969; Blainey *et al*, 1980; Bonnell, 1955; Kazantis, 1979; and Scott *et al*, 1980). Generally effects on bone appear after kidney damage has been detected and they are likely therefore to be secondary to the associated changes in calcium, phosphorus and vitamin D metabolism (Blainey *et al*, 1980).

However, Staessen *et al* (1999) concluded that skeletal demineralisation, resulting in an increase in bone fragility and an increase in the risk of fractures, was probably promoted by a low level of environmental exposure to cadmium. Similarly, Alfven *et al* (2000) concluded that environmental and or occupational exposure to low levels of cadmium is associated with an increase in the risk of osteoporosis.

Studies in Animals: No studies of muscoskeletal effects in animals were found.

Hepatic Effects

Studies in Humans

Liver effects are not usually associated with exposure to cadmium via the inhalation route. Non-specific signs of liver disease were observed in some workers, exposed to cadmium for 20 years, in their occupation Friberg (1950). The tests, indicators of cirrhosis or hepatitis, performed are generally not in current use, The significance of the findings in relation to cadmium exposure is questionable as subsequent studies on workers exposed to cadmium in the air have not detected toxic effects on the liver (Adams *et al*, 1969; and Bonnell, 1955).

Table 6.10: Summary of Animal Studies for Hepatic Effects

Dose (hr-mg/m3)	Exposure	Target	Effect	Author
72 Cd	Cd (24 hrs/d for 30d @0.1 mg/m^3)	Rats	transient (2m post-exposure) increase in serum ALT, indicative of liver damage	Glaser *et al*, 1986
159 Cd	CdO dust (24 hrs/d for 63d @0.105 mg/m^3	Rats	No effect	Prigge, 1978a
177 Cd	$CdCl_2$ (24 hrs/d for 255d @0.029mg/m^3)	Rats	No effect	Oldiges & Glaser, 1986
293 Cd	$CdCl_2$ (24 hrs/d for 21d @0.581 mg/m^3)	Rats	No effect	Prigge, 1978b
394 Cd	$CdCl_2$ (6 hrs/d for 62d @1.06 mg/m^3)	Rats	an increase in relative weights of the liver	Kutzman *et al*, 1986
471 Cd	CdO (24 hrs/d for 218d @0.09mg/m^3)	Rats	No effect	Oldiges & Glaser, 1986

Studies in Animals

Effects on the liver have occasionally been found in the liver as illustrated in Table 6.10. Cadmium accumulates in the liver as well as the kidney, the main target organ for cadmium toxicity. The resistance of the liver may be related to a higher capacity to produce metallothionein that would bind to cadmium and thus lower the concentrations of free cadmium ions.

Renal Effects

Studies in Humans

There is very strong evidence that the kidney is the main target organ of cadmium toxicity following extended inhalation exposure to cadmium. The sensitivity of the kidney to cadmium was recognised in an early investigation of workers exposed to cadmium oxide dust and cadmium fumes in a factory producing nickel-cadmium batteries (Friberg, 1950). These workers suffered from a high incidence of abnormal renal function, indicated by proteinuria and a decrease in glomerular filtration rate. Similar signs of renal damage have been observed in many other studies of workers occupationally exposed to cadmium (Adams *et al*, 1969; Beton *et al*, 1966; Bernard *et al*, 1979; Bonnell, 1955; Bustueva *et al*, 1994; Chia *et al*, 1989; Elinder *et al*, 1985a, 1985b; Falck *et al*, 1983; Gompertz *et al*, 1983; Iwata *et al*, 1993; Jakubowski *et al*, 1987; Jarup & Elinder, 1993; Jarup *et al*, 1988; Kjellstrom *et al*, 1977; Mason *et al*, 1988; Piscator, 1966; Roels *et al*, 1981b; Rose *et al*, 1992; Smith *et al*, 1980; and Thun *et al*, 1989).

The proteinuria caused by cadmium exposure is characterised by the presence of a number of low-molecular-weight proteins in urine, including β2-microglobulin, lysozyme, ribonuclease, immunoglobulin light chains, and retinol-binding protein (Piscator, 1966). These low-molecular-weight proteins are all readily filtered by the glomerulus and are normally reabsorbed in the proximal tubules of the kidney. Elevated urinary excretion of these proteins is indicative of proximal tubular damage. Urinary excretion of high-molecular-weight proteins such as albumin has also been reported in occupationally exposed workers (Bernard *et al*, 1979; Elinder *et al*, 1985b; Mason *et al*, 1988; Roels *et al*, 1989; Thun *et al*, 1989), but there is some debate as to whether this represents glomerular damage (Bernard *et al*, 1979; Roels *et al*, 1989) or severe tubular damage (Elinder *et al*, 1985a; Mason *et al*, 1988; Piscator, 1984).

The tubular proteinuria caused by cadmium exposure may be accompanied by depressed tubular resorption of other solutes such as enzymes, amino acids, glucose, calcium, copper, and inorganic phosphate (Elinder *et al*, 1985a, 1985b; Falck *et al*, 1983; Gompertz *et al*, 1983; Mason *et al*, 1988). It has been suggested that the urinary concentrations of some of these solutes, particularly renal enzymes, are more sensitive than low-molecular-weight proteins for detecting tubular dysfunction in exposed humans.

An additional effect on the kidney seen in workers after high levels of inhalation exposure to cadmium is an increased frequency of kidney stone formation (Elinder *et al*, 1985a; Falck *et al*, 1983; Kazantzis, 1979; Scott *et al*, 1978; and Thun *et al*, 1989). This effect is likely to be secondary to disruption of calcium metabolism due to kidney damage.

Tubular dysfunction generally develops only after cadmium reaches a minimum threshold in the renal cortex. This threshold is often referred to as the 'critical concentration'. Care must be taken in its interpretation because it is not invariant, but depends on a number of variables (Foulkes, 1990). The critical concentration of cadmium in the renal cortex associated with increased incidence of renal dysfunction in an adult human population chronically exposed to cadmium has been estimated to be about 200 μg/g wet weight by several investigators (Friberg *et al*, 1974; Kjellstrom *et al*, 1977a, 1984; and Roels *et al*, 1983).

Several quantitative evaluations of kidney toxicity have been performed using cumulative dose (exposure duration times cadmium concentration) as the independent variable. An early study found a 10% prevalence of proteinuria at an average 30-year exposure to cadmium oxide dust of 0.017 mg Cd/m^3 (Kjellstrom *et al*, 1977a), but a subsequent follow-up study found only a 4% prevalence at this level of exposure (Jarup *et al*, 1998). The definition of proteinuria used in these studies is an excretion exceeding the 95th percentile of a normal population. Thus, a prevalence of 5% or less was considered to be unrelated to cadmium exposure. Among the workers in the follow-up study, the prevalence of proteinuria was 1.1 % in the lowest exposure group with a 30-year exposure to 0.00437 mg Cd/m^3 and 9% at 0.023 mg/m^3 (Jarup *et al*, 1998). Logistic regression generated a prevalence of 4% at a 30-year exposure to 0.017 mg/m^3, which was considered to be the NOAEL for this cohort. Other recent analyses have found 30-year thresholds for proteinuria of 0.027 mg/m^3 (Thun *et al*, 1989), 0.033 mg/m^3 (Elinder *et al*, 1985b), or 0.0367 mg/m^3 (Mason *et al*, 1988). In another cohort, with an average 30-year exposure to cadmium fume of 0.026 mg/m^3, the average exposures of workers with and without proteinuria were 0.038 and 0.015 mg/m^3, respectively (Falck *et al*, 1983).

Cessation of cadmium exposure generally does not lead to a decrease in proteinuria in occupationally exposed workers (Elinder *et al*, 1985b; Mason *et al*, 1988; Piscator, 1984; Thun *et al*, 1989), possibly because the kidney cadmium level declines very slowly after cessation of exposure. Kidney damage may continue to worsen after exposure ceases. A progressive reduction of the glomerular filtration rate in excess of the usual age-related decline was found in 23 workers 5 years after they were removed from cadmium exposure because of proteinuria and/or albuminuria (Roels *et al*, 1989). End-stage renal disease is not a common cause of death among workers occupationally exposed to cadmium, but it is significantly elevated over expected values in some occupational cohorts (Elinder *et al*, 1985c; Kazantzis *et al*, 1988).

To further evaluate the reversibility of proteinuria, Roels *et al* (1997) studied the progression of Cd-induced renal tubular dysfunction in cadmium workers according, to the severity of the microproteinuria at the time the exposure was substantially decreased. A total of 32 cadmium male workers were divided into two groups on the

basis of historical records of urinary cadmium concentration (CdU) covering, the period until 1984. The workers with CdU values of > 10 μg Cd/g creatinine were subdivided further on the basis of the urinary concentration of β2-microglobulin (β2-MG-U) measured during the first observation period (1980-1984). In each group, the tubular microproteinuria as reflected by β2-MG-U and the concentration of retinol-binding protein in urine, as well as the internal cadmium dose as reflected by the concentration of cadmium in blood and urine, were compared between the first and second (1990-1992) observation periods. Increased microproteinuria was often diagnosed in cases with CdU values of >10 μg Cd/g creatinine. The progression of tubular renal function was found to depend on the extent of the body burden of cadmium (as reflected by CdU) and the severity of the initial microproteinuria at the time high cadmium exposure was reduced or ceased. When cadmium exposure was reduced and β2-MG-U did not exceed the upper reference limit of 300 μg/g creatinine, the risk of developing tubular dysfunction at a later stage was likely to be low, even in cases with historical CdU values occasionally >10 but always < 20 μg/g creatinine. When the microproteinuria was mild (β2-MG-U >300 and ≤ 1,500 μg/g creatinine) at the time exposure was reduced, and the historical CdU values had never exceeded 20 μg/g creatinine, there was indication of a reversible tubulotoxic effect of cadmium. When severe microproteinuria (β2-MG-U > 1,500 μg/g creatinine) was diagnosed in combination with historical CdU values exceeding 20 μg/g creatinine, cadmium-induced tubular dysfunction was progressive in spite of reduction or cessation of cadmium exposure.

Studies in Animals

Renal damage has been found in animal studies following exposure to cadmium via the inhalation route. Rabbits exposed to cadmium metal dust at 4 mg/m^3 for 3 hours/day, 21 days/month for 4 months, developed mild proteinuria. Histological lesions were found after an additional 3-4 months of exposure (Friberg 1950). Proteinuria has not been found in most subsequent studies, because the levels of exposure (1-5 mg/m^3 in intermediate exposure and 0.2-2 mg/m^3 in chronic exposure) and the duration of the follow up periods which produce serious respiratory effects are not high enough to produce a toxicologically important concentration in the kidney (Glaser *et al*, 1986; Kutzman *et al*, 1986; and Prigge 1978a, 1978b).

Dermal Effects

Studies in Humans

Dermal toxicity is not a significant effect of inhalation exposure to cadmium. Studies of workers occupationally exposed to cadmium have not reported dermal effects following acute or chronic exposure (Barnhart & Rosenstock, 1984; Bonnell, 1955; and Friberg, 1950).

Studies in animals: No studies were found that specifically investigated dermal toxicity in animals following inhalation exposure to cadmium.

Ocular Effects

Studies in Humans

Ocular toxicity is not a significant effect of inhalation exposure to cadmium. Studies of workers occupationally exposed to cadmium have not reported ocular effects following acute or chronic exposure (Barnhart & Rosenstock, 1984; Bonnell, 1955; and Friberg, 1950).

Studies in Animals

In rats exposed to approximately 100 mg Cd/m^3, as cadmium pigments, in a single 2 hour exposure period excessive lacrimation was observed 4 hours after exposure (Rusch *et al*, 1986); this effect is probably due to a local contact irritation of the eyes rather than a systemic effect.

Body Weight Effects

Studies in Humans: No data have been found regarding the effects of inhaled cadmium on body weight in humans.

Studies in Animals

Cadmium has been shown to significantly reduce body weights in animals as illustrated in Table 6.11.

Table 6.11: Summary of Animal Studies for Body Weight Effects

Dose (hr-mg/m3)	Exposure	Target	Effect	Author
6.5 Cd	$CdCl_2$ (1 hr @6.5 mg/m^3)	Rats (male)	significant reduction in body weight	Bus *et al*, 1978
9 Cd	$CdCl_2$ (2 hrs @4.5 mg/m^3)	Rats (male)	significant reduction in body weight	Grose *et al*, 1987
13.8 Cd	CdO dust (3 hrs @4.6 mg/m^3)	Rats (male)	significant reduction in body weight	Buckley & Bassett, 1987
224 Cd	CdO fumes (2 hrs @112 mg/m^3)	Rats (male)	significant reduction in body weight	Rusch *et al*, 1986
264 Cd	$CdCO_3$ (2 hrs @132 mg/m^3)	Rats	weight gain reduced	Rusch *et al*, 1986

A range of NOAEL (no observable adverse effect level) values have also been derived as summarised in Table 6.12.

Table 6.12: Summary of Animal Studies for NOAEL (Body Weight Effects)				
Dose (hr-mg/m3)	**Exposure**	**Target**	**Effect**	**Author**
0.9 Cd	$CdCl_2$ (2 hrs @0.45 mg/m^3)	Rats	NOAEL	Grose *et al*, 1987
10.2 Cd	$CdCl_2$ (6 hrs/d for 10d @0.17 mg/m^3)	Rats	NOAEL	Klimisch, 1993
12 Cd	$CdCl_2$ (2 hrs @6 mg/m^3)	Rats	NOAEL	Palmer *et al*, 1986
>58 Cd	CdO dust (>15d @0.16 mg/m^3)	Rats (female)	NOAEL	Baranski & Sitarek, 1987
>119 Cd	$CdCl_2$ (>15d @0.33 mg/m^3)	Rats	NOAEL	Kutzman *et al*, 1986
>142 Cd	$CdCl_2$ (>15d @0.394 mg/m^3)	Rats (non-pregnant f)	NOAEL	Prigge, 1978a
>162 Cd	CdO dust (>15d @0.45 mg/m^3)	Rabbits (male)	NOAEL	Grose *et al*, 1987
>183 Cd	$CdCl_2$ (>15d @0.508 mg/m^3)	Rats (male)	NOAEL	Takenaka *et al*, 1983
198/194 Cd	CdS/CdSeS (2 hrs @99/97 mg/m^3 resp.)	Rats	NOAEL	Rusch *et al*, 1986
>372 Cd	CdS (>15d @1.034 mg/m^3)	Rats (male)	NOAEL	Glaser *et al*, 1986
>8322 Cd	$CdSO_4$ (>365d @0.95 mg/m^3)	Rats	NOAEL	Oldiges & Glaser, 1986

Other Systemic Effects

Studies in Humans

Yellow discolouration of the teeth has been occasionally observed in workers who have been exposed to high levels of cadmium in the work place (Friberg, 1950). No data has been found that indicates this finding was due to any functional impairment (NTIS, 1999)

Studies in Animals: No studies were found that specifically investigated teeth abnormalities in animals following inhalation exposure to cadmium.

Immunological and Lymphoreticular Effects

Studies in Humans

There is limited evidence that inhalation exposure to cadmium has effects on the immune system. Leucocytes in the blood of workers exposed to cadmium for 1-14 years had a slight, statistically significant decrease in ability to generate reactive oxygen species when compared to unexposed controls (Guillard & Lauwerys, 1989). The toxicological significance of this effect is unknown.

Serum IgG, IgM and IgA, as well as the cadmium concentrations of blood and urine, were measured in a group of 37 males employed in zinc/cadmium smelters and a small Cd-electroplating plant. Cadmium concentrations in blood (5.55 versus 2.01 μg/g creatinine, $p<0.05$) and urine (2.39 versus 0.69 μg/100 ml) were significantly higher in exposed workers compared to controls. There were no differences between the concentrations of IgG, IgM and IgA in the serum of the exposed and non-exposed groups. With the possible exception of monocyte counts, which were significantly increased, there were no changes in the differential white cell counts in either group.

Studies in Animals

Cadmium has been found to have effects on the immune system in some animal experiments as shown in Table 6.13.

Table 6.13: Summary of Animal Studies for Immunological and Lymphoreticular Effects

Dose (hr-mg/m3)	Exposure	Target	Effect	Author
0.22 Cd	$CdCl_2$ (2 hr @0.11 mg/m^3)	Mice	NOAEL	Graham *et al*, 1978
0.38 Cd	$CdCl_2$ (2 hr @0.19 mg/m^3)	Mice	suppression of the primary humoral immune response	Graham *et al*, 1978
199 Cd	$CdCl_2$ (24 hrs/d for 21d @0.394 mg/m^3)	Rats	increase in relative weight of spleen	Prigge, 1978b
394 Cd	$CdCl_2$ (6 hrs/d for 62d @1.06 mg/m^3)	Rats	increase in relative weights of spleen and lymphoid hyperplasia	Kutzman *et al*, 1986

Other studies such as that conducted by Daniels *et al* (1987) have found no effects on the activity of natural killer cells or viral induction of interferon in exposed mice. Evidence of the effects of exposure to cadmium on resistance to infection is conflicting in the opinion of Bouley *et al* (1982) because the same exposure decreases resistance to bacterial infection while at the same time increasing resistance to viral infection.

Neurological Effects

Studies in Humans

Neurotoxicity is not generally associated with exposure to cadmium though a few studies have specifically looked for neurological effects in humans. In a group of 31 men occupationally exposed to cadmium for an average of 14.5 years, Hart *et al* (1989b) reported there was some correlation between cadmium exposure and decreased performance in neuropsychologic tests to measure attention, psychomotor speed and memory. The small group size made it difficult to evaluate the significance of these findings.

Studies in Animals

Cadmium has been found to produce limited neurological effects in some animal experiments as shown in Table 6.14.

Table 6.14: Summary of Animal Studies for Neurological Effects

Dose (hr-mg/m3)	Exposure	Target	Effect	Author
71 Cd	Cd fume (30d @0.098 mg/m^3)	Rats	no neurological effect	Glaser *et al*, 1986
76 Cd	$CdCl_2$ (30d @0.105 mg/m^3)	Rats	no neurological effect	Glaser *et al*, 1986
123 Cd	$CdCl_2$ (6hr/d for 62d @0.33 mg/m^3)	Rats	no neurological effect	Kutzman *et al*, 1986
224 Cd	Cd fume (2 hr @112 mg/m^3)	Rats	reduced activity	Rusch *et al*, 1986
264 Cd	$CdCO_3$ (2 hrs @132 mg/m^3)	Rats	tremors	Rusch *et al*, 1986
385 Cd	$CdCl_2$ (6hr/d for 62d @1.034 mg/m^3)	Rats	increase in relative weight of brain	Kutzman *et al*, 1986
744 Cd	CdS (30d @1.034 mg/m^3)	Rats	no neurological effect	Glaser *et al*, 1986

Reproductive Effects

Studies in Humans

There is insufficient evidence to determine whether exposure to cadmium via the inhalation route is associated with reproductive effects.

Gennart *et al* (1992) studied male reproductive effects of cadmium in 83 occupationally exposed blue-collar Belgian workers in 2 smelting operations. The workers were exposed to cadmium in dust and fumes. Information was recorded on age, residence, education, occupational and health history, actual and previous occupations, smoking habits, and coffee and alcohol consumption. Fertility parameters included dates of birth of wife and husband, date of marriage, and the number of children born alive and their dates of birth. Blood and urine samples were also collected from each worker. Some cadmium workers had been excessively exposed; 25% of them already had signs of kidney dysfunction as evidenced by microproteinuria and/or a serum creatinine level above 13 mg/l. No effects were observed on male fertility as evidenced by no significant influence of cadmium on the probability of a live birth. The limitation of this study, as described by the authors, included the fact that the wives were not interviewed and, therefore, factors that could have influenced their reproductive ability were not considered.

Men occupationally exposed to cadmium at levels causing renal damage had no change in testicular endocrine function, as measured by serum levels of testosterone, luteinizing hormone, and follicle stimulating hormone (Mason, 1990).

Noack-Fller *et al* (1993) measured concentrations of cadmium, lead, selenium, and zinc in whole semen and seminal fluid of 22 unexposed men (13 were smokers) to evaluate intra-individual variability and to examine the statistical association between element concentrations and semen characteristics and sperm motion parameters. None of the men had any known occupational exposure to cadmium.

Concentrations of cadmium were similar in semen and seminal plasma (0.40!0.23 and 0.34±0.19 µg/l, respectively). Sperm motility ($p<0.02$), linear velocity ($p<0.001$), and curvilinear velocity (CV) ($p<0.002$) were significantly correlated with semen cadmium levels. Intra-individual coefficients of variation for sperm count (CV=46±4%) and sperm concentration (CV=37±6%) showed the highest variability. No positive correlation was found between cadmium concentration in semen and sperm density. The smokers had slightly elevated levels of cadmium. The concentrations of cadmium in semen of these volunteers was very low. Additional studies are needed (preferably with larger sample sizes) to evaluate the robustness of this association between cadmium (at the low levels detected) and sperm motion parameters. Saaranen *et al* (1989) measured cadmium, selenium, and zinc in seminal fluid and serum in 64 men, half of whom were smokers. Smokers had significantly higher serum cadmium concentration than non-smokers. Seminal fluid cadmium was also elevated in smokers, and was higher than serum cadmium in smokers consuming more than 20 cigarettes daily. Semen quality was measured for volume, sperm density, morphology, motility, and number of immature germ cells. No differences were found in semen quality or fertility between smokers and non-smokers. There was no significant correlation between seminal fluid cadmium levels and semen quality or fertility.

Xu *et al* (1993a) measured trace elements in blood and seminal plasma and their relationship to sperm quality in 221 Singapore men (age range 24-54; mean 34.8) who were undergoing initial screening. for infertility. Men with significant past medical history and those who had been occupationally exposed were excluded. Parameters monitored included semen volume and sperm density, motility, morphology, and viability. Graphite furnace atomic absorption was used to determine cadmium concentration in blood and semen. No differences were observed in sperm quality (density, motility, morphology, volume, and viability) of the 221 men compared to a cohort of 38 fertility proven men (wives had recently conceived). Cadmium levels in blood did have a significant inverse relationship with sperm density ($r=-0.15$, $p<0,05$) in oligospermic men (sperm density below 20 million/ml), but not in normospermic men. There was a significant reduction in sperm count in men with blood cadmium of >1.5 µg/l. Also, there was a weak negative correlation between defective sperm and concentration of cadmium in semen ($r=-0.21$, $p<0.05$). The volume of semen was inversely proportional to the cadmium concentration in semen ($r=-0.29$, $p<0.05$). These findings suggest that cadmium may have an effect on the male reproductive system. Limitations of the study include lack of control for potential confounding factors such as the lower levels of zinc in seminal plasma, and the validity of using

infertile men as the study group (i.e., again because of confounding factors that may be affecting both cadmium levels and sperm levels).

A post-mortem study of men occupationally exposed to cadmium who died from emphysema found high levels of cadmium in their testes, but no histological lesions other than those attributable to terminal illness (Smith *et al*, 1960).

Russian women occupationally exposed to cadmium concentrations up to 35 mg/m^3 had no irregularities in their menstrual cycles (Tsvetkova, 1970). Fertility and other indices of reproductive function were not measured. No studies were located that showed reproductive effects in women following inhalation exposure to cadmium.

Studies in Animals

Some effects have been observed in animal studies as summarised in Table 6.15.

Table 6.15: Summary of Animal Studies for Reproductive Effects

Dose (hr-mg/m3)	Exposure	Target	Effect	Author
372 Cd	Cd (6hr/d for 62d @1.0 mg/m^3)	Rats (m & f)	no loss of reproductive performance but relative weight of the testes was increased	Kutzman *et al*, 1986
500 Cd	CdO dust (5hr/d for 100d @1 mg/m^3)	Rats (female)	longer oestrus cycle	Baranski & Sitarek, 1987
>4000 Cd	$CdSO_4$ (60 d @2.8 mg/m^3)	Rats (female)	longer oestrus cycle (in 50% of animals)	Tsvetkova, 1970

Developmental Effects

Studies in Humans

Russian women occupationally exposed to cadmium at concentrations ranging from 0.02 to 35 mg/m^3 had offspring with decreased birth weights compared to unexposed controls, but without congenital malformations (Tsvetkova, 1970). No association was found between birth weights of offspring and length of maternal cadmium exposure. Moreover, no control was made for parity, maternal weight, gestational age, or other factors known to influence birth weight (Tsvetkova, 1970). A nonsignificant decrease in birth weight was found in offspring of women with some occupational exposure to cadmium in France; however, no adverse effects were documented in these newborns (Huel *et al*, 1984). Huel *et al* (1984) used hair samples to estimate exposure, and this method is limited without controls to distinguish between erogenous and endogenous sources. No other studies were located regarding developmental effects in humans after inhalation exposure to cadmium.

Studies in Animals

Developmental toxicity, as evidenced by delayed ossification, decreased locomotor activity, and impaired reflexes in the offspring), was found in the offspring of female rats exposed to cadmium oxide at a concentration of 0.02 mg Cd/m^3 5 hours/day, 5 days/week, for 4-5 months prior to mating and during the first 20 days of gestation. At a higher concentration, 0.16 mg/m^3 decreases in body weight gain, osteogenesis and viability were noted (Baranski, 1985).

In rats exposed to cadmium chloride aerosols, at concentrations of 0.204, 0.394, or 0.581 mg/m^3, during gestation, maternal weight gain and foetal weight were reduced (Prigge, 1978b).

Genotoxic Effects

Studies in Humans

Examination of lymphocytes from workers occupationally exposed to both cadmium and lead have shown statistically significant increases in chromosomal aberrations (Bauchinger *et al*, 1976, Deknudt & Leonard 1975, Deknudt *et al*, 1973), though not in men exposed primarily to Cadmium (Bui *et al*, 1975, O'Riordan *et al*, 1978).

Studies in Animals: No studies have been found on the genotoxic effects of exposure to cadmium by the inhalation route.

Cancer

Studies in Humans

The relationship between occupational exposure to cadmium and increased risk of cancer, particularly lung and prostate cancer has been investigated in a number of epidemiological studies. The data and some of the analyses for lung cancer are conflicting. Controls for confounding factors such as co-exposure with other metal carcinogens and smoking have been included in only a few studies (NTIS, 1999). Overall, the results provide little evidence of an increased risk of lung cancer in humans following long term exposure to cadmium by inhalation. The early studies indicated that the incidence of prostate cancer was increased in men occupationally exposed to cancer (Kipling & Waterhouse, 1967; Kjellstrom *et al*, 1979; and Lemen, 1976). Later investigations however found slight, nonsignificant increase or no increases (Elinder *et al*, 1985; Kazantis *et al*, 1988, 1992; Sorahan, 1987; and Thun *et al*, 1985).

Some increases in lung cancer have been found in occupationally exposed cohorts in UK, and Sweden (Ades & Kazantizis, 1988; Elinder, 1985c; Kazantzis, 1988; and Sorahan 1987, 1995). Sorahan (1995), in a recent study on mortality rates from lung cancer and non-malignant respiratory diseases amongst 347 copper cadmium alloy workers in the UK concluded that exposure to Cadmium oxide fumes increases the

risk of mortality from non-malignant diseases respiratory system but does not increase the risk from lung cancer.

An increased risk of lung cancer was reported in studies on the only US cohort, a group of workers in a cadmium recovery plant in Colorado (Thun *et al*, 1985; Stayner, 1992). More recent studies have attributed the increase in risk to exposure to arsenic and/or smoking (Lamm *et al*, 1992, 1994; and Sorahan *et al*, 1997).

The US EPA has classified cadmium as a probable human carcinogen when administered via the inhalation route (Group B1), based on limited evidence of an increase in lung cancer in humans (Thun *et al*, 1985) and sufficient evidence of lung cancer in rats (IRIS, 1996; and Takenaka *et al*, 1983). US EPA has calculated an inhalation unit risk (the risk corresponding to lifetime exposure to 1 $\mu g/m^3$) of 1.8 x 10^{-3} (IRIS, 1996). The National Toxicology program (NTP) has classified cadmium and certain cadmium compounds as substances that are reasonably anticipated to be carcinogens (NTP, 1994). The IARC on the other hand has classified cadmium as carcinogenic in humans (Group1), based on sufficient evidence for carcinogenicity in both human and animal studies (IARC, 1993).

Studies in Animals

Studies in rats, mice, and Syrian golden hamsters provide evidence of the carcinogenic potential of chronically inhaled cadmium to the lung.

Oldiges *et al* (1989) found a definite dose related increase in the incidence of lung tumours in rats following an 18-month continuous exposure to cadmium compounds. Increased incidence of lung tumours were observed for cadmium chloride and oxide at concentration of 0.03 mg/m^3 and for cadmium sulphate and sulphite at concentrations of 0.09 mg/m^3.

A study by Takenaka *et al* (1983) confirmed that cadmium chloride aerosols containing 0.0134, 0.0257 and 0.0508 $mgCd/m^3$, when administered to male rats for 18 months, was carcinogenic.

Heinrich *et al* (1989) in a study similar to that conducted by Oldiges (1989) did not observe an increase in lung tumours in male or female Syrian golden hamsters following chronic exposure to cadmium oxide dust or fumes, cadmium chloride, cadmium sulphate, or cadmium sulphide. According to the same author, in female mice the incidence of lung tumours increased at all levels. There was a high incidence in the controls and the cadmium induced increases were not statistically significant. With the exception of cadmium oxide fumes (weak increase) the incidence of lung tumours in the cadmium treated mice did not increase in a dose related manner.

6.3.3 Inhalation Toxicity

Cadmium chloride and cadmium oxide tend to be most toxic compounds while the effects of cadmium sulphide and cadmium selenium sulphide (red pigment) are much less severe (by 1 or 2 orders of magnitude). The findings of the review presented above are summarised in Table 6.16.

Table 6.16: Summary of Cadmium Toxicity due to Inhalation

Effect		Acute (< 15 days)	Intermediate (15 - 364 days)	Chronic (>1 year)
Death	H	Occupational accidents have led to deaths	No data	Unreliable data only
	A	Rats: LOAEL < 0.9 CdO (to nose)	Rats: LOAEL <145 CdO	Rats: LOAEL <241 $CdCl_2$
Respiratory	H	LOAEL < 40 Cd	Limited data	Conflicting data
	A	Hamsters: LOAEL < 5 $CdCl_2$	Rabbits: LOAEL < 48 $CdCl_2$	Rats: LOAEL <169 $CdCl_2$
Cardiovascular	H	No significant effect in humans		
	A	No data	Rats: LOAEL < 394 $CdCl_2$	No data
Gastrointestinal		No significant effect in humans/animals		
Haemotological		Conflicting evidence suggests no significant effects in humans/animals		
Musculoskeletal		Some effects observed in workers after high occupational exposure		
Hepatic	H	No effects in human observed		
	A	No data	Rats: NOAEL 471 CdO *but* Rats: LOAEL <72 Cd	No data
Renal	H	No data	No data	NOAEL/LOAEL: 30 years @0.004 mg/m^3
	A	Very high doses required to effect kidneys in mammals		
Dermal		No significant effect in humans/animals		
Ocular		No significant effect in humans/animals		
Body weight	H	No effects in humans observed		
	A	Rats: LOAEL <6.5 $CdCl_2$ *but* NOAEL 12 $CdCl_2$	Rats: NOAEL >183 $CdCl_2$	No data
Other Systemic		Some teeth discolouration in workers but no relevant animal studies		
Immune System	H	Limited effects in humans observed		
	A	Mice: NOAEL 0.22 $CdCl_2$	Rats: LOAEL <199 $CdCl_2$	No data
Neurological	H	No significant effects in humans observed		
	A	Rats: LOAEL < 224 Cd	Rats: NOAEL >123 $CdCl_2$ Rats: LOAEL <385 $CdCl_2$	No data
Reproductive	H	No significant effects in humans observed		
	A	No data	Rats: LOAEL <372 Cd	No data
Development	H	No significant effects in humans observed		
	A	No data	Rats: LOAEL <9 Cd	No data
Genotoxic		Conflicting evidence on significant effects in humans - but no animal data		
Cancer	H	Conflicting evidence on human carcinogenicity		
	A	No data	No data	Rats: LOAEL < 176 $CdCl_2$

Note: H relates to human effects and A to animal effects, units are hr-mg/m^3

6.3.4 Exposure by the Oral Route

Overview

Information on health effects of oral exposure to cadmium in humans is derived mainly from studies of residents of areas polluted with cadmium. The exposure of these populations is usually estimated by measuring the levels of cadmium in blood and/or urine. Exposure is generally via the diet, though smokers are also exposed to cadmium by inhalation. Smoking by itself, however is not a route of exposure but a variable, because of the large number of other toxic compounds present in cigarette smoke, and the primary objective is to study the effects of cadmium and cadmium compounds (NTIS, 1999). In water, cadmium is more often found in the free ionic form, while in food the cadmium generally exists as a complex with ligands, including proteins such as metallothionein (Crews *et al*, 1989; Groten *et al*, 1990; and Nordberg *et al*, 1986 - cited in NTIS, 1999).

The soluble cadmium salts (particularly cadmium chloride) have generally been used in toxicology studies in animals to investigate the effect of exposure to cadmium in food and water and oral administration (gavage).

Death

Studies in Humans

Doses estimated to be 25 and 1,840 mg/kg of cadmium iodide and cadmium chloride respectively have been ingested to commit suicide (Buckler *et al*, 1986; and Wisniewska-Knypl *et al*, 1971). Death, due to fluid loss, oedema and organ damage occurred within 7 days and 33 hours of ingestion.

Studies in Animals

In rats and mice, acute oral LD_{50} values for cadmium range from 100-300 mg/kg/day Baer & Benson, 1987; Bassinger *et al*, 1988; Kostial *et al*, 1978; Kotsonis & Klaassen, 1978; and Shimizu & Morita 1990). The lowest dose causing death (2 of 20 animals) was 15.3 mg/kg in Sprague-Dawley rats (Borzelleca *et al*, 1989). Very young animals have lower LD_{50} values than adults (Kostial *et al*, 1978, 1989).

Deaths related to medium term exposure to cadmium have been observed in the only 2 studies found. In both studies, cadmium chloride was administered. Baranski & Sitarek (1987) used Wistar rats, and administered the compound by gavage at 40 mg Cd/kg, 5 days/week for up to 14 weeks. Four of 13 rats died by week 8. In female albino Swiss mice, used by Blakley (1986), to study the effect of the compound on chemical and viral induced tumour production, the cadmium chloride was administered in the drinking water for 280 days at doses of 0, 5, 10, or 50 ppm. These mice have a high incidence of spontaneous lypmhocytic leukemia of thymic origin. A significant 33% increase ($p=0.0228$, chi-square analysis) in deaths from virally induced leukaemia was observed from exposure to 1.9 ppm, or 9.5 mg Cd/kg/day.

The author attributed the deaths to the impairment of the immunosurveillance mechanisms that control expression of the murine lymphocytic leukaemia virus.

Respiratory Effects

Studies in Humans: No studies of the effects of exposure to cadmium by the oral route on the respiratory system were found.

Studies in Animals

Various studies on animals provide information on respiratory effects as summarised in Table 6.17.

Table 6.17: Summary of Animal Studies for Respiratory Effects

Intake (mg/kg/day)	Exposure	Target	Effect	Author
1.2	Cd 200d in water	Sprague-Dawley rats (male)	reduced static compliance and unspecified lung lesions	Petering *et al*, 1979
2.4	$CdCl_2$ 6-16 wks	Rats	Fibrosis	Miller *et al*, 1974b
3.62	Cd 120d in water	Sprague-Dawley rats (male)	emphysema	Petering *et al*, 1979
4	$CdCl_2$ 9y via diet	Rhesus monkeys	No respiratory effect	Masaoka *et al*, 1994
8	Cd in water	Sprague-Dawley rats (male)	no histopathological lesions	Kotsonis and Klaassen, 1978
16	Cd 90d in water	Wistar rats	no effects on lung weight	Prigge, 1978a

Cardiovascular Effects

Studies in Humans

Studies investigating cardiovascular effects in humans after oral exposure to cadmium have concentrated on the relationship between blood pressure and biomarkers of cadmium exposure such as cadmium levels in the blood, urine or other tissues (NTIS, 1999). Smoking is an important confounding factor as smokers generally have higher levels of cadmium in blood, urine and tissues (NTIS, 1999). When smoking has been adequately controlled generally no association between dietary exposure and hypertension in case-control and cohort epidemiological studies (Beevers *et al*, 1980, Cummins *et al*, 1980, Ewers *et al*, 1985, Lazebnik *et al*, 1989, Shiwen *et al*, 1990). Conflicting results such as positive or negative correlations have also been reported by Geiger *et al* (1989), Tulley and Lehmann (1982) and Kagamimori *et al* (1986) and Stassen *et al* (1984) respectively. Contradictory results have also been reported in

the analysis of death rates from cardiovascular disease among populations exposed to cadmium in the diet. Disorders of the cardiac conduction system, lower blood pressure and decreased frequency of ischemic changes were found in elderly women with a previous history of dietary exposure to cadmium (Kagamimori *et al*, 1986). Ventricular fibrillation and other rythmic disturbances were seen in an individual who had ingested 25 mg/kg of cadmium iodide (Wisniewska-Knypl *et al*, 1971).

Studies in Animals

In some studies oral exposure of rats, rabbits and monkeys to cadmium for intermediate and chronic periods has been found to increase blood pressure (Akahori *et al*, 1994; Boscolo & Carmignani, 1986; Carmignani & Boscolo, 1984; Kopp *et al*, 1982; Perry *et al*, 1989; and Tomera and Harakal 1988), but not in others (Fingerle *et al*, 1982; Kotsonis & Klaassen, 1978; Loeser & Lorke, 1977a, 1977b; Mangler *et al* 1988; and Willis *et al*, 1981). In general, the effects were small and the control groups had lower blood pressure in studies where an effect was found than the controls in studies in which no effect was seen (NTIS, 1999). Some examples of studies on animals are summarised in Table 6.18.

Table 6.18: Summary of Some Animal Studies for Cardiovascular Effects

Intake (mg/kg/day)	Exposure	Target	Effect	Author
0.35	Cd 3 y	Rats	Enlarged and arteriosclerotic hearts	Schroeder *et al*, 1965
2.5	Cd in diet 7wk	Rats	congestion and separation of muscle fibres at the histopathological examination	Jamall *et al*, 1989
2.79	Cd 100d	Rats	Enlarged and arteriosclerotic hearts	Wilson *et al*, 1941
150	Cd (single dose)	Sprague-Dawley rats	no effect on blood pressure	Kotsonis and Klaassen, 1978

Overall there is evidence that oral exposure to cadmium causes slight cardiovascular toxicity (NTIS, 1999).

Gastrointestinal Effects

Studies in Humans

Oral exposure to high concentrations of cadmium has been found to cause severe irritation to the epithelium of the GI tract (Andersen *et al*, 1988; and Frant & Kleeman, 1941). The symptoms most commonly observed after ingesting food or drink containing high concentrations of cadmium include nausea, vomiting, salivation, abdominal pain, cramp and diarrhoea (Baker & Hafner, 1961; Buckler *et*

al, 1986; Frant & Kleeman, 1941; Nordberg *et al*, 1973; Shipman, 1986; and Wisniewska-Knypl *et al*, 1971). Although exact doses were not determined GI symptoms have been seen in children by 13-16 mg/l. Assuming an intake of 0.15 l and a body weight of 35 kg Nordberg *et al* (1973) have calculated that the emetic dose is 0.07 mg/kg.

Studies in Animals

In rats and mice, histopathological lesions including severe necrosis, haemorrhage and ulcers, have been observed in the epithelium of the GI tract after acute oral , gavage, exposure to cadmium at doses of greater than 30 mg/kg/day (Andersen *et al*, 1988; Basinger *et al*, 1988; and Machemer & Lorke, 1981), but not after 8 mg/kg/day in the drinking water for 24 weeks (Kotsonis and Klaassen 1978).

Haematological Effects

Studies in Humans

Oral exposure to cadmium reduces the gastrointestinal uptake of iron, and anaemia can develop if the content of iron in the diet is low (NTIS, 1999). Kagamimori *et al* (1986) found anaemia among humans with chronic exposure to cadmium in the their diet. Roels *et al* (1981a) and Shiwen *et al* (1990), found no relationship between exposure to cadmium in the diet and anaemia. Other findings were hypoproteinaemia and hypoalbuminaemia in a male who had ingested 25 mg/kg of cadmium iodide (Wisniewska-Knypl *et al*, 1971).

Studies in Animals

Anaemia, which can be prevented by the use of additional iron, has been observed in laboratory animals after oral exposure to cadmium in a number of studies (Decker *et al*, 1958; Groten *et al*, 1990; Hays & Margaretten, 1985; Itokowa *et al*, 1974; Kawamura *et al*, 1978; Kelman *et al*, 1978; Kozolowska *et al*, 1993; Ogoshi *et al*, 1989; Pleasants *et al*, 1992, 1993; Pond & Walker, 1975; Sakata *et al*, 1988; Sorell & Graziano, 1990; Stowe *et al*, 1972; Watanabe *et al*, 1986; Webster, 1978; and Wilson *et al*, 1941). These typically involved oral intake by rats of 2-14 mg/kg/day for an intermediate duration. There were some exceptions principally in rat studies using similar dose levels, which were performed by Kotsonis and Klaassen (1978), Loeser and Lorke (1977a), Petering *et al* (1979) and Prigge (1978a).

The results of studies on animals are summarised in Table 6.19.

Table 6.19: Summary of Animal Studies for Haemotological Effects				
Intake (mg/kg/day)	**Exposure**	**Target**	**Effect**	**Author**
0.75	Cd intermediate	Mice Rabbits Dogs	Anaemia Anaemia No anaemia	Webster, 1978 Stowe *et al*, 1972 Loeser & Lorke, 1977b
0.79	Cd in water 12m	Rats	No anaemia	Decker *et al*, 1958
2-14	Cd intermediate	Rats	Anaemia and no anaemia	various
4	Cd in diet 90d	Monkeys	Some signs of anaemia	Masoka *et al*, 1994
57	Cd in water 12m	Mice	Decrease in no. of erythroid progenitor cells (bone marrow)	Hays and Margaretten, 1985
65.6	Cd 10d	Rats (male)	Slight increases in haemoglobin, haematocrit, and erythrocytes	Borzelleca *et al*, 1989
150	Cd (single dose)	Sprague-Dawley rats	Not anaemic after 14d	Kotsonis & Klaassen, 1977

Musculoskeletal Effects

Studies in Humans

In some humans exposed to cadmium in food bone disorders including ostomalacia, osteoporosis and spontaneous, and painful bone fractures (Itai-Itai disease) have been observed. This disease and osteomalacia have been found to frequently affect women with poor nutrition and frequent pregnancies living in a cadmium contaminated area in Japan (Shigematsu, 1984). Kido *et al* (1989b) found an increased incidence of osteoporosis and osteomalacia in other populations of Japanese women (and men) exposed to cadmium in their diet. The degree of loss of bone density is correlated with urinary excretion of β_2-microglobulin, the index of renal injury (see below) (Kido *et al*, 1990a). There were 56 cases of Ita-Itai disease, 26 of which were associated with ostomalacia and 26 cases of Itai-Itai disease alone, in a population of elderly Japanese women who were exposed by ingesting drinking water, rice and fish contaminated with cadmium during World Wars 1 and 2 and continued exposure (low-grade) through ingestion of agricultural produce (Kagamimori *et al*, 1986).

As already noted, it has been reported that low levels of cadmium exposure may lead to skeletal demineralisation (Staessen *et al*, 1999) and osteoporosis (Alfven *et al*, 2000).

Studies in Animals

In rats, exposure to cadmium has been shown to affect the skeleton. A decrease in the calcium content of bone and an increase in the excretion of calcium in the urine are common findings in studies of exposure to 2-8 mg Cd/kg/day for periods of intermediate and chronic duration (Kawamura *et al*, 1978; Nogawa *et al*, 1981; Pleasants *et al*, 1992; and Watanabe *et al*, 1986).

Kotsonis and Klaassen (1978) and Kelman *et al* (1978) found no changes in bone calcification after rats were exposed in drinking water to 8 mg/kg/day for 24 weeks and female rats exposed to 3.8 mg/kg/day in drinking water for 22 days during gestation respectively.

Adverse effects on bone are exacerbated by a calcium deficient diet (Itokawa *et al*, 1974; Kimura *et al*, 1974; Larssen & Piscator, 1971; Wang & Bhattacharyya, 1993; and Wang *et al*, 1994), by exposure at an age when the bones are growing (Ogoshi *et al*, 1989), by ovarectomy (Bhattacharyya *et al*, 1988c), and repeated gestation and lactation periods (Bhattacharyya 1988b).

Ogoshi (1989) assessed the strength of the femurs of 19-22 young (21 days old), 18-25 adult (24 weeks old) and 25-27 old (1.5 years old) female rats following 4 weeks exposure to cadmium chloride at various dose levels in drinking water. Femur compression and bending strengths, and the cadmium and zinc content of bone were determined. In the young rats 5 and 10 ppm (Note: 1 ppm = 1 mg/l) reduced bone strength, while in the adult and old rats there was no change up to doses of 160 ppm. Bone strength was found to correlate well with the cadmium content of bone, but not with that of liver or kidney. The accumulation of cadmium in the bones was greatest in young rats (100 and 150 ng/g dry weight at 5 and 10 ppm respectively In the adult and old rats accumulation was 65 ng/g at 160 ppm.

In non pregnant mice given a calcium deficient diet the addition of cadmium at concentrations of up to 25 ppm significantly increased bone resorption immediately, according to the levels in serum and faeces(Wang and Bhattacharyya 1993).

In pregnant mice fed a calcium deficient diet exposure to cadmium in the diet induced an Itai-Itai like syndrome. Almost all of the cadmium lost from the dams appeared in the pups, 20% during gestation and 80% via the dam's milk during lactation (Wang *et al*, 1994). In mice some investigators have detected effects in bone prior to the development of proteinuria or histopathological kidney damage was detectable (Bhattacharyya *et al*, 1988a,1988b, Ogoshi 1989, Watanabe *et al*, 1986). These results indicate that disturbances in calcium metabolism may occur prior to proteinuria following long term/chronic exposure to cadmium.

Hepatic Effects

Studies in Humans

Liver damage is not usually associated with exposure to cadmium via the oral route, unless the levels are very high. In humans a fatal dose of cadmim iodide or chloride can cause marked liver damage (Buckler *et al*, 1986, Wisniewska-Knypl *et al*, 1971). Increased serum concentration of the urea-cycle amino acids at levels which reflect liver damage, among individuals exposed to cadmium in the diet have been reported (Nishino *et al*, 1980).

Studies in Animals

Exposure to cadmium via the oral route has caused effects in the liver of mice, rats and rabbits as summarised in Table 6.20.

Table 6.20: Summary of Animal Studies for Hepatic Effects

Intake (mg/kg/day)	Exposure	Target	Effect	Author
0.75	Cd 3m	Dogs	No effects on liver	Loeser & Lorke, 1977b
0.05-10	Cd intermediate/ chronic	Rats	decreased cytochrome c oxidase in mitochondria and increased ALT and AST activity	Groten *et al*, 1990; Muller & Stacey, 1988; and others
1.6-15	Cd intermediate/ chronic	Rats	Necrosis	Cha, 1987; Gill *et al*, 1989; and others
3	Cd in diet intermediate /chronic	Rats	No effect on liver	Loeser and Lorke 1977a
5.95	Cd in diet 6wk	Rats (male)	Decreased liver weight	Kozloska *et al*, 1993
8	Cd in water 24wk	Rats	No effect on liver	Kotsonis & Klaassen, 1978
13.9	Cd in water 10d	Rats	No effect on liver	Borzellaca *et al*, 1989
14	Cd in water 6wk	Rats	No effect on liver	Hopf *et al*, 1990
30-138	Cd (gavage) acute	Rats	Necrosis	Andersen *et al*, 1988; Basinger *et al*, 1988; Borzelleca *et al*, 1989; Shimizu and Morita, 1990
150	Cd (single dose)	Sprague-Dawley rats	No histopathological evidence of damage to the liver	Kotsonis & Klaassen, 1977

Renal Effects

Studies in Humans

The kidney is identified as the main target organ of cadmium toxicity following long term exposure in numerous studies. The effects are similar to those seen following exposure by the inhalation route (see above). Increased incidences of tubular proteinuria have been found in many epidemiological studies of populations residing in the cadmium-polluted areas of Japan (Nogawa *et al*, 1980, 1989), Belgium (Buchet *et al*, 1990; Roels *et al*, 1981a; and others) and China (Shiwen *et al*, 1990).

A recent study of Belgians (aged 20-80 years) from polluted and nonpolluted urban and rural areas found abnormal rates of urinary excretion of β_2-microglobulin, retinol binding protein, N-acetyl-β-glucosaminidase, amino acids, and calcium in individuals with cadmium excretion rates >2 lg/day (Buchet *et al*, 1990). The cadmium excretion rate of 2 lg/day was estimated to correspond to a cadmium level of 50 lg/g wet weight in the renal cortex (Buchet *et al*, 1990). This study suggests that the critical concentration may be lower in members of the general population than in workers (Buchet *et al*, 1990). However, data from Japanese residents of cadmium-polluted areas support a critical concentration of 200 lg/g wet weight, as found in occupationally exposed workers (Roels *et al*. 1983). Quantitative analysis of the prevalence of elevated urinary β_2-microglobulin as a function of cadmium ingestion indicates that after a total intake of approximately 2,000 mg cadmium (for a 53-kg person), renal damage will occur (Nogawa *et al*, 1989). This intake corresponds to a 50-year dose of approximately 0.0021 mg/kg/day. A kinetic model of cadmium metabolism predicts that this intake will produce elevated β_2-microglobulin levels in about 5% of a non-smoking European population (body weight=70 kg) and about twice that rate in a Japanese population (body weight=53 kg), assuming a log-normal distribution in critical concentrations with 10% of the population having a critical concentration of 180 lg/g or less and 50% having a critical concentration at 250 lg/g or less (Kjellstrom, 1986). This kinetic model also assumes that kidney concentrations will be log-normally distributed in a population with a given intake (Kjellstrom, 1986); it has been suggested that the standard deviation for this distribution is 1.75 rather than 2, and that intakes to produce a given probability of renal effects are 50% higher than predicted by the model (Piscator, 1985). Possible reasons for the discrepancy of the model with the Buchet *et al* (1990) study, but agreement with the Nogawa *et al*. (1989) study, include differences in the cutoffs for elevated β_2-microglobulinuria and differences in estimation of dietary cadmium absorption.

Proteinuria does not decrease when oral exposure to cadmium stops. Renal tubular dysfunction and reduced glomerular filtration increase in severity after cessation of environmental exposure (Iwata *et al*, 1993; Kido *et al*, 1990b). Although kidney failure is not the primary cause of death among populations environmentally exposed to cadmium, increased rates of mortality from renal disease have been observed in populations of Belgium (Lauwerys and De Wals, 1981), England (Inskip and Beral

1982) (although not significant), and Japan (Nakagawa *et al*, 1987) (significant for females and not significant for males). The increased calcium excretion associated with cadmium-induced renal damage may also increase the risk for osteoporosis, particularly in post-menopausal women (Buchet *et al*, 1990).

Studies in Animals

Studies by Andersen *et al* (1988), Bernard *et al* (1980, 1988, 1992), Bomhard *et al*, 1984 and others in mice rats and rabbits have found that oral exposure to cadmium causes proteinuria and tubular damage. Histopathological findings include focal necrosis of proximal tubular epithelial cells and cloudy swelling in renal tubules (Cha, 1987).

Other studies (Basinger *et al*, 1988; Borzelleca *et al*, 1989; Boscolo & Carmignani, 1986; Groten *et al*, 1990; Jamall *et al*, 1989; and Loeser & Lorke 1997a, 1977b) have shown that exposure to cadmium via the oral route has no effect on renal function.

Proteinuria (Bernard *et al*, 1988a; Cardenas *et al*, 1992a, 1992b; Kotsonis & Klaassen, 1978; and others) and histopathological lesions (Gatta *et al*, 1989; Itokawa *et al*, 1974; Kotsonis & Klaassen, 1978; and Wilson *et al*, 1941) have often been found in intermediate duration oral exposure studies in rats given cadmium at dose levels of 2-30 mg/kg/day. At these levels decreased renal clearance (Kawamura *et al*, 1978) and increases and decreases in the relative weights of the kidney have been reported by Pleasants *et al* (1992, 1993) and Kozlowska *et al* (1993).

Other relevant studies include:

- in dogs, cadmium at a dose level of 0.75 mg/kg/day for 3 months had no effect on the kidney (Loeser and Lorke 1977b);
- in rabbits, a dose level of 14.9 mg/kg/day cadmium for 200 days caused interstitial fibrosis (Stowe *et al*, 1972);
- in rhesus monkeys exposed to cadmium for 9 years renal dysfunction was observed at a dose level of 1.2 mg/kg/day though not at 0.4 mg/kg/day (Masaoka *et al*, 1994); and
- in rats following chronic-duration exposure to cadmium at dose levels ranging from 1.8 –12.5 mg/kg /day, via the oral route proteinuria (Bernard *et al*, 1992, Bomhard *et al*, 1984) and histopathological damage (Fingerle *et al*, 1982, Mangler 1988) are common findings.

The hypothesis that a concentration of approximately 200 lg/g in the renal cortex is required to produce proteinuria is generally supported by the animal data (Bhattachayya *et al*, 1988c; Kotsonis & Klaassen, 1978; Mangler *et al*, 1988; Shaikh *et al*, 1989; and Viau *et al*, 1984 – cited in NTIS 1999).

Endocrine Effects

Studies in Humans: No studies of the effects, of previous exposure to cadmium by the oral route, on the endocrine organs in humans were found.

Studies in Animals

The evidence of the effect(s) of oral exposure to cadmium is based on histopathological examination of the endocrine organs. Examples of animal studies include:

- in rats given 2.79 mg/kg/day in the diet for 100 days, atrophy of the pancreas and pancreatitis were observed (Wilson *et al*, 1941);
- in Wistar rats given 3 mg/kg/day in the diet for 3 months there was no effect on the adrenal glands, the pituitary gland, thyroid glands and the thymus (Loeser and Lorke, 1977a);
- in female Wistar and male Sprague-Dawley rats exposed to 8 mg Cd/kg/day via the drinking water for 90 days and 24 weeks respectively, there was no adverse effect on the parathyroid gland (Kawamura *et al*, 1978) and adrenal gland (Kotsonis and Klaassen, 1977); and
- in rabbits exposed to 14.9 mg Cd/kg/day via the drinking water for 200 days moderate concentrations of cadmium were found in the pancreas but there were no pathological changes observed (Stowe *et al*, 1972).

Dermal Effects

Studies in Humans: No studies of the effects, of previous exposure to cadmium by the oral route, on the skin of humans were found.

Studies in Animals: No dermal effects after oral exposure to cadmium, which were relevant to humans were found.

Ocular Effects

No studies investigating the ocular effects in humans or animals following oral exposure to cadmium were found.

Body Weight Effects

Studies in Humans: No data have been found regarding the effects of previous oral exposure to cadmium on body weight in humans.

Studies in Animals

Lower body weight and reduced rates of growth are common observations in animals exposed to cadmium by the oral route as indicated in Table 6.21.

Metabolic Effects

Studies in Humans

Hypothermia and metabolic acidosis were reported in a human male who had ingested 25 mg/kg cadmium in the form of cadmium iodide (Wisniewska-Knypl *et al*, 1971).

Studies in Animals: No studies of metabolic effects in animals after oral exposure to cadmium were found.

Table 6.21: Summary of Animal Studies for Bodyweight Effects

Intake (mg/kg/day)	Exposure	Target	Effect	Author
0.4	Cd 9y	Rhesus monkeys	No effect	Masaoka *et al*, 1994
1.2			Decrease in growth rate	
3-232	Cd intermediate	Rats	Decrease in bodyweight	Pleasants *et al*, 1992,1993; Carmignani and Boscolo, 1984; Jamall *et al*, 1989; and others
14.9	Cd 200d	Rabbits	Small decrease in bodyweight	Stowe *et al*, 1972
< 15	Cd intermediate	Rats	No decrease in bodyweight	Kostial *et al*, 1993; Prigge, 1978a; Viau *et al*, 1984; and others
15.3	Cd 10d	(m) Sprague-Dawley rats	79% decrease in bodyweight gain	Borzellaca *et al*, 1989
57	Cd in water 12m	Mice	Decrease in bodyweight	Hays and Margaretten, 1985
100	Cd (single dose)	Sprague-Dawley rats	No decrease in bodyweight	Kotsonis & Klaassen, 1977
150	Cd (single dose)		12% decrease in bodyweight	

Immunological and Lymphoreticular Effects

Studies in Humans: No studies of immunological effects in humans after oral exposure to cadmium were found.

Studies in Animals

Cadmium has affected the immune system in studies in mice, rats and monkeys, however the clinical significance of these effects is equivocal as the effects are often

contradictory and the models used inappropriate indicators of the immune response in the general human population.

Neurological Effects

Studies in Humans

An association between environmental cadmium exposure and neurological function has been reported in a few studies. The parameters affected included verbal IQ, acting out short attention span and disruptive behaviour. The value of these data is equivocal because of the possibility of simultaneous lead exposure and other variables, which were not adequately controlled.

Studies in Animals

Cadmium induced neurotoxicity has been observed in acute and intermediate duration animal studies.

Reproductive Effects

Studies in Humans: No studies of reproductive effects in men or women exposed to cadmium were found.

Studies in Animals

Exposure to cadmium has been shown to have adverse effects on the reproductive systems of male and female animals. Specific examples include:

- acute exposure to near lethal doses (60-100 mg/kg) have been reported to cause testicular atrophy (Andersen *et al*, 1988; Bomhard *et al*, 1987; Borzelleca 1989) and consequently a decrease in fertility (Kotsonis and Klaassen 1978) in male mice and rats;
- intermediate periods (10-117 weeks) of exposure to lower doses (5.8-12.9 mg/kg/day) have caused an increase in the absolute and relative weight of the testes, necrosis, atrophy of the seminiferous tubule epithelium, seminiferous tubule damage, decreased sperm count, and decreased motility (Cha, 1987; Pleasants *et al*, 1992; Saxena *et al*, 1989);
- exposure to higher levels of cadmium was necessary to elicit reproductive toxicity in female rats. In a study by Borzelleca *et al*, 1989 a dose of 65.6 mg Cd/kg/day, administered for 10 days, caused testicular atrophy and loss of spermatogenic element in male rats but 138 mg Cd/kg/day had no effect on their female counterparts; and
- in female rats exposed to 61.32 mg Cd/kg/day, but not 18.39 or lower, for days 6-15 of gestation there was a decrease in the % of females which successfully mated and which subsequently became pregnant (Machemer and Lorke 1981).

Developmental Effects

Studies in Humans

Data on the effects of cadmium on development in humans is limited. The concentration of cadmium in the urine of women 3 days after giving birth was measured by Cresta *et al* (1989). The smoking habits and the birth weights of the offspring were compared. In non-smoking women cadmium levels, expressed as lg/l, were higher, though, when expressed as lg/g creatinine they were lower in women with offspring of lower than normal birth weight. In women in the smoking group cadmium levels, expressed as lg/l or lg/g creatinine, were lower in women with offspring of lower than normal birth weight.

Studies in Animals

In rats and mice oral exposure to cadmium prior to gestation and during gestation has resulted in fetotoxicity. This is generally manifested as reduced fetal or pup weight (Ali *et al*, 1986; Baranski, 1987; Gupta *et al*, 1993; Petering *et al*, 1979; Pond and Walker, 1975; Sorell and Graziano, 1990; and others). At dose levels in the 1-20 mg/kg/day range, malformations, particularly of the skeleton or skeletal effects including sireomelia, amelia, and delayed ossification of the sternum and ribs (Baranski 1985), dysplasia of facial bones and rear limbs, oedema, extenteration, cryptorchism and palatoschisis (Machemer and Lorke 1981) and sharp angulation of the tail (Schroeder and Michener 1971) have been reported.

The most sensitive indicator of the developmental toxicity of cadmium appears to be neurobehavioural development. Offspring of female rats exposed to 0.04 mg/kg/day via the oral route prior to and during gestation, had reduced locomotor activity and reduced performance on the rota rod at 2 months of age (Baranski *et al*, 1983). Pups from dams exposed 0.7 mg/kg/day during gestation also had neurobehavioural problems (learning difficulties and increased locomotor activity).

Behavioural and functional neurotoxicological changes have been reported in a three generation study of reproduction in rats (Nagymajtenyi *et al* (1997). Cadmium chloride at dose levels of 3.5, 7.0, or 14.0 mg Cd/kg was administered orally by gavage to three consecutive generations of Wistar rats over the period of pregnancy, lactation and up to 8 weeks after weaning. Behavioural (open field) and electrophysiological (spontaneous and evoked cortical activity, etc.) parameters of the male rats of each generation were investigated when they were 12 weeks of age. The principal behavioural findings were increased rearing (vertical exploration activity)and increased exploration of an open-field centre. There were dose and generation dependent changes in spontaneous and evoked electrophysiological parameters (represented by increases in the frequencies in the EEG, lengthened latency and duration of evoked potentials etc.), indicating a change in neural function. The results indicate that the exposure of 3 generations of rats to low-levels of cadmium affected nervous function.

Cadmium associated changes in behaviour and neurological function in rats were further investigated by Desi *et al* (1998). Female Wistar rats were given the same doses as in the previous study at 3 different intervals: days 5-15 of pregnancy, days 5-15 of pregnancy and + 4 weeks of lactation, and days 5-15 of pregnancy + 4 weeks of lactation followed by the same oral treatment of male rats of the F1 generation for 8 weeks. Behavioural (open field) and electrophysiological (spontaneous and evoked cortical activity, etc.) parameters of the F1 male rats were investigated when they were 12 weeks of age. The results confirmed those of the previous study that cadmium altered the spontaneous and evoked electrophysiological function in a dose-related and treatment time dependent manner. The results indicate that pre- and postnatal exposure to low levels of cadmium affects the electrophysiological and higher order functions of the nervous system (NTIS, 1999).

The concentrations of zinc and cadmium in the brains of neonates of dams exposed to 5-6.3 mg/kg/day of cadmium acetate in drinking water during gestation and 7-8 mg/kg/day during a 21 day lactation period, was studied by Gupta *et al* (1993). Pup brain and body weights were significantly decreased in the cadmium exposed pups on lactation days 7-21. Cadmium brain accumulation was significantly increased in exposed pups on lactation day 7 and remained at similar levels on days 14 and 21 of lactation.

Saxena *et al*, 1986 reported that exposure to 21 mg Cd/kg/day via drinking water during days 0-20 had no effect on developmental parameters in rats(?). In this study the simultaneous exposure to 20 mg/kg/day lindane via gavage on gestation days 6-14 and cadmium acetate in drinking water at doses that did not individually cause maternal or developmental effects. Maternal toxicity was only observed in the cadmium plus lindane group. Fetal body weight was significantly decreased, as were intrauterine death and the incidence of skeletal anomalies.

Genotoxic Effects

Studies in Humans

The results on the genotoxicity of oral exposure to cadmium are conflicting. Examination of the lymphocytes from women (Itai-Itai patients) exposed to cadmium in the environment has shown an increase in chromosome aberrations (Shiraishi and Yoshida, 1972) and no co-effect (Bui *et al*, 1975). A third more recent study of inhabitants of a cadmium-polluted area in China found an increase in the incidence of chromosomal aberrations that was correlated with the levels of cadmium in urine (Tang *et al*, 1990).

Studies in Animals

Exposure to cadmium at a concentration of 600 ppm in the diet for 1 month had no effect on bone marrow (Deknudt and Gerber 1979). However abnormalities were observed in the bone marrow of mice after daily exposure to cadmium at a dose level of 3.52 mg/kg/day for 1-3 weeks (Mukherjee *et al*, 1988b). However, no evidence of

germ cell mutation was found in male rats orally exposed to cadmium in a dominant lethal test (Sutou *et al*, 1980).

Cancer

Studies in Humans

A few studies of cancer rates among humans orally exposed to cadmium have been performed. No significant increase in cancer rates was found by Inskip and Beral (1982) when they studied the residents of a cadmium polluted village in the UK.

Studies in Animals

In the studies, of the effects chronic oral exposure to cadmium, performed in rats and mice in the 1960-1980s increased incidences of cancer or specific tumour types were not reported. The dose levels used were low, 1mg/kg/day in mice and 3.5mg/kg/day in rats and in general the histopathological examination was limited compared to current standards. In long term studies performed to investigate non-neoplastic effects, the dose levels, though higher, were still low, 4.01 and 8 mg/kg/day in rats and mice respectively (NTIS, 1999).

Studies performed more recently to evaluate e.g. the effects of chronic dietary zinc deficiency on the carcinogenic potential of cadmium in male Wistar rats (Waalkes and Rehm, 1992) employed cadmium concentrations of 25, 50, 100 or 200 ppm and concentrations of 60 (adequate)or 7 (deficient) ppm of zinc in the diet for 77 weeks. In addition, a complete necropsy and ?complete histopathological examination was performed. In this study cadmium did not affect survival rate or food consumption. The incidence of prostatic proliferative lesions both hyperplasias and adenoma was increased when compared with controls (1.8%) in both the zinc adequate (20%) and deficient (14%) groups. In the zinc deficient groups the incidence of prostatic lesions in all the cadmium treated was much lower, though the effect was not dose-related, possibly because the reduced intake of zinc was associated with a marked increase in the incidence of atrophy of the prostate gland. Cadmium treatment resulted in an increase in the incidence of leukaemia in both the zinc groups. In the “adequate” group the increase was significant at 50 and 100 but not 200 ppm. Zinc deficiency reduced the potency of cadmium to induce leukaemia, the increase achieving significance only at the 200 ppm concentration. The incidence of benign interstitial tumours of the testes was increased only in animals given 200 ppm in diets containing adequate levels of Zinc. A significant trend was noted for development of testicular neoplasia as the concentration of cadmium increased. In this study exposure to cadmium by the oral route was associated with tumours of the prostate, testes and haemopoietic system in rats. Diet deficient in zinc has complex apparently inhibitory effects on the carcinogenic potential of cadmium when administered by this route.

In a study in B6C3F1 mice the effects of cadmium exposure on tumour incidence at various times after the initiation of the carcinogenic process were studied (Waalkes 1993). The possible role of metallothionein in the susceptibility of transformed cells

to cadmium induced cytotoxicity was also investigated. The 5 week old mice were given an IP injection of 90 mg/kg NDEA. At 2, 4, 8, 16 or 32 weeks post-NDEA the mice received 1000ppm cadmium ad libitum for up to 48 weeks post-NDEA exposure. There was a marked reduction in the incidence of liver tumours, generally adenomas, when given 32 weeks after the NDEA treatment. Cadmium alone ?eliminated the incidence of liver tumours (0/25) compared to controls (5/25). Exposure to cadmium also resulted in a reduction in the incidence of lung tumours, statistically significant, only for the 16-48 week cadmium treated group pre-treated with NDEA. Cadmium alone ?eliminated the incidence of spontaneously occurring lung tumours compared to the controls. Cadmium also reduced the multiplicity of tumours by 50-80%. The overall size of the tumours in the NDEA plus cadmium treatment groups was smaller than those in the NDEA only groups. Immunochemical and biochemical techniques (liver only) indicated that relatively little metallothionein was present in the liver carcinomas, liver adenomas and lung adenomas. The authors concluded that cadmium can "impair tumour formation in the lungs and liver of male B6C3F1 mice and appears to be able to selectively destroy existing preneoplastic and/or tumour cells (adenomas).

Overall, NTIS (1999) concluded that neither the human nor the animal studies provide sufficient evidence to determine whether or not cadmium is a carcinogen by the oral route.

6.3.5 Toxicity via Oral Exposure

Cadmium chloride and cadmium oxide tend to be the most toxic compounds while the effects of cadmium sulphide and cadmium selenium sulphide (red pigment) are much less severe (by 1 or 2 orders of magnitude). The findings of the review presented above are summarised in Table 6.22.

Table 6.22: Summary of Cadmium Toxicity due to Oral Exposure

Effect		Acute (< 15 days)	Intermediate (15 - 364 days)	Chronic (>1 year)
Death	H	Deaths at 25 mg/kg CdI and 1840 mg/kg $CdCl_2$	No data	No data
	A	Rats: LOAEL < 15 mg/kg Rats/mice: LD_{50} 100-300 mg/kg	Mice: LOAEL < 9.5 mg/kg	No data
Respiratory	H	No data		
	A	No data to suggest effects	S-D rats: LOAEL < 1.2 mg/kg	R. monkeys: NOAEL > 4 mg/kg
Cardiovascular	H	Conflicting evidence suggests no significant effects in humans		
	A	No evidence to suggest effects even at high does	Rats: LOAEL < 2.5 mg/kg *but* small effects and other contradictory evidence	Rats: LOAEL < 0.35 mg/kg *but* small effects and other contradictory evidence
Gastrointestinal	H	LOAEL: 0.07 mg/kg (children)	No data	No data
	A	Rats/mice: LOAEL > 30 mg/kg	Rats: NOAEL > 8 mg/kg	No data

Table 6.22: Summary of Cadmium Toxicity due to Oral Exposure				
Effect		**Acute (< 15 days)**	**Intermediate (15 - 364 days)**	**Chronic (>1 year)**
Haemotological	H	Conflicting evidence suggests no significant effects in humans		
	A	Rats: LOAEL < 66 mg/kg	Mice/rabbits: LOAEL < 0.75 mg/kg	Rats: NOAEL > 0.79 mg/kg; LOAEL < 57 mg/kg
Musculoskeletal	H	No data	No data	Some evidence amongst women in contaminated areas
	A	Acute effects unlikely	Rats: NOAEL 2-8 mg/kg	
Hepatic	H	No significant effects in humans observed		
	A	Rats: NOAEL > 14 mg/kg	Rats: NOAEL > 14 mg/kg *but:* LOAEL < 0.05-15 mg/kg	Rats: LOAEL 0.05-10 mg/kg
Renal	H	No data	No data	NOAEL: 50 years @0.0021 mg/kg
	A	No evidence to suggest effects	Rats: LOAEL < 2mg/kg *but o*ther contradictory evidence	R. monkeys: LOAEL < 1.2 mg/kg *but o*ther contradictory evidence
Endocrine	H	No significant effects in humans observed		
	A	No evidence to suggest effects	Rats: LOAEL < 2.79mg/kg *but S-D* Rats: NOAEL > 8 mg/kg	No data
Dermal		No significant effect in humans/animals		
Ocular		No significant effect in humans/animals		
Body weight	H	No effects in humans observed		
	A	S-D rats: LOAEL < 15.3 mg/kg *but* NOAEL >100 mg/kg (single doses)	Rats: LOAEL < 3 mg/kg	R. monkeys: NOAEL >0.4 mg/kg *and* LOAEL < 12. mg/kg
Metabolic		No significant effect in humans/animals except at high doses (but limited data)		
Immune System	H	No effects in humans observed		
	A	Some evidence to suggest effects in animals - but often contradictory		
Neurological	H	Some evidence to suggest effects in humans - but not yet reliable		
	A	Some neurotoxicity observed		No data
Reproductive	H	No significant effects in humans observed		
	A	Rats: LOAEL 60 mg/kg	Rats: LOAEL <5.8 mg/kg	No data
Development	H	No significant effects in humans observed		
	A	No data	Rats (measured in offspring): LOAEL 0.04 mg/kg	No data
Genotoxic		Conflicting evidence on significant effects in humans/animals		
Cancer	H	No evidence on human carcinogenicity		
	A	No data	Limited and conflicting evidence of effects on numbers of tumours and incidence of leukaemia	
Note: H relates to human effects and A to animal effects				

6.4 Toxicokinetics Relevant to Assessment

6.4.1 Overview

When inhaled or ingested, the toxicokinetics of cadmium depend on the physiological and dietary status of the subject and the form of cadmium/cadmium salt absorbed. Inhaled cadmium is absorbed from the lungs or from the gut after clearance from the airways of the upper respiratory tract. Ingested cadmium is absorbed from the gastrointestinal tract (GIT). When absorption is via the lung it is much higher than that from the GIT. Solubility is an important factor in the bioavailability of the various cadmium compounds.

Cadmium is not absorbed well by the skin (approximately 0.5%) and there is not a significant risk from skin exposure unless the skin is exposed to very high concentrations for long periods of time. As this is unlikely in the general population and steps are taken to prevent this type of occupational exposure by the use of personal protective equipment, dermal exposure is not considered to be a significant source of risk.

6.4.2 Exposure via Inhalation

Overview

As is the case for all inhaled compounds, the deposition of cadmium in the lungs depends on the particle size, the smaller the particles, the greater the deposition. Once deposited in the lung compounds such as cadmium chloride and cadmium oxide (slightly soluble in water) are solubilised and distributed systemically. Clearance of cadmium sulphide occurs mainly via mechanical transport by alveolar macrophages. In addition, larger cadmium particles may also be transported to the GIT by mucocilliary clearance. Estimates of absorption of deposited cadmium oxide have been as high as 90%, while only 10% of deposited cadmium sulphide is absorbed during exposure via the inhalation route (Glaser *et al*, 1986; Oberdorster & Cox, 1989; and Oberdorster, 1992).

Studies in Humans

No data specifically relating to cadmium deposition, retention or absorption in the human lung were found. There are however several studies showing increased levels of cadmium in workers exposed as a result of their occupation which indicate that inhaled cadmium is absorbed (Ellis *et al*, 1985; Elinder *et al*, 1985a,b; Jarup *et al*, 1998; Roels *et al*, 1983; Smith *et al*, 1980; and Thun *et al*, 1989).

Cadmium absorption from cigarettes appears to be greater than absorption of cadmium from aerosols measured in animals due to the very small size of the particles in cigarette smoke and the consequent very high level of deposition in the alveoli of the lung (Nordberg *et al*, 1985).

Studies in Animals

Various studies have shown that the retention of cadmium compounds via inhalation exhibits biphasic behaviour.

The pulmonary retention half-times for cadmium chloride, cadmium oxide and cadmium sulphide administered to rats and monkeys have been determined (Oberdorster & Cox, 1989). The short-term retention half-times were typically measured in days/weeks whereas the long-term retention half-times were measured in months/years. The half-times were shorter for rats than for monkeys.

A similar study by Rhoads and Saunders (1985) in which cadmium oxide was administered by the intratracheal route to rats confirmed that lung clearance was biphasic, though the half-times obtained were shorter, 67% in 4 hours and 13 days for the remaining 33%. The difference in half-times may be due to the particle size in this study being in the fume range rather than the dust range.

Klimisch (1993) also exposed rats to aerosols of cadmium chloride or cadmium sulphide for 6 hours/day for 10 days and found clearance to be biphasic and half times days and months for short-term and long-term half-times respectively.

6.4.3 Exposure via the Oral Route

Studies in Humans

Absorption from the GIT has been found to be relatively low, 3-8% (Järup *et al*, 1998).

Absorption of ingested cadmium compounds is influenced by the chemical form of cadmium and the physiological status of the subject; absorption is increased by low levels of other metals such as calcium, copper, iron and zinc (Nordberg *et al*, 1985).

There are no differences in the bioavailability or the rates of accumulation of cadmium ingested in food and in water available *ad-libitum* when the dose administered was less than 4 mg/kg/day (Ruoff *et al*, 1994).

The bioavailability of cadmium compounds may be influenced more by the contents of the GIT than by the means of exposure.

The placenta may act as a partial barrier to fetal exposure to cadmium. Cadmium concentration in the blood in the foetal cord has been found to be approximately 50% of that in maternal cord blood in several studies in pregnant women who smoke and their non-smoking counterparts (Kuhnert *et al*, 1982; Lauwerys *et al*, 1978; Truska *et al*, 1989). Cadmium levels in the placenta were about 10 times higher than those in maternal blood in studies by Roels *et al* (1978) and Kuhnert (1982), though Truska *et al* (1989) were unable to confirm this (the level of cadmium in the placenta was less than that in either foetal or maternal cord blood in this study).

Metallothionein has been detected in full term placentas (Goyer, 1991) and in foetal cells in humans. Metallothionein was found in cells, which facilitate the transport of substances entering the placenta from maternal blood, in cells, which are phagocytes, in amniotic cells, and decidual cells. The mechanism by which the placenta transports essential metals while limiting the transport of cadmium is unknown but may be associated with the approximately 1000 fold higher concentration of zinc and the higher affinity of cadmium for metallothionein.

Cadmium levels in milk are 5-10% of the levels in blood, possibly as a result of inhibited transfer from blood because of metallothionein binding of cadmium in blood cells (Radisch *et al*., 1987).

Studies in Animals

Gastrointestinal absorption of cadmium compounds in animals is also quite low, 1-2% in rats and mice and 0.5-3% in monkeys (Järup *et al*, 1998). The difference may be due more to the differences between standard laboratory animal diets and typical human diets than physiological factors (Andersen *et al*, 1992).

6.4.4 Distribution and Excretion

Overview

Absorbed cadmium is transported to the liver, where it stimulates the synthesis of metallothionein, a low–molecular-weight protein with a high capacity for binding to cadmium and other metals. Exposure to cadmium, zinc and other metals induces metallothionein in most tissues. The cadmium-metallothionein complex is then released back into the blood and transported to the kidney. Once there it is filtered by the glomerulus and reabsorbed by the cells of the proximal tubules (Foulkes, 1978). Lysosomes proteolyse the metallothionein, releasing free cadmium, which stimulates new metallothionein (NTP, 1994).

Renal damage is believed to be the result of free cadmium, which does not become bound to metallothionein due to localisation or an excessive concentration of cadmium. The binding capacity of kidney metallothionein is lower than that of liver metallothionein, with a result that unbound cadmium is present in the kidney at exposure-levels at which all liver cadmium is bound to metallothionein (Goyer et al., 1989; Kotsonis and Klassen, 1978). According to these authors this tissue-specific difference in binding capacity may account for the sensitivity of the kidney to cadmium.

Studies in Humans

The main route of excretion of inhaled or ingested cadmium is the faeces. Cadmium excretion in the urine of workers exposed as a result of their occupation increases as the body burden of cadmium increases (Roels *et al*, 1981b). As a result of the long retention time of cadmium in the body the amount of cadmium excreted normally is a small fraction of the body burden. When renal damage occurs there is a marked

increase in urinary excretion of cadmium compounds. In subjects with no renal damage, the level of cadmium in the urine appears to be a good reliable marker of cadmium burden, and thus, cumulative exposure to cadmium. In normal subjects, urinary excretion of cadmium is approximately 1 µg/day (Nordberg *et al*, 1985). The blood level of cadmium is considered to reflect the current level of exposure rather than body burden.

Cadmium compounds have a biological half-life of 10-30 years in the kidney and approximately 5-10 years in the liver (Ellis *et al*, 1985). In occupationally exposed workers, the concentration of cadmium compounds in the liver generally increases in proportion to the intensity and duration of exposure (Davison *et al*, 1988; and Ellis *et al*, 1985). After the onset of renal damage, the concentration of cadmium in the kidney begin to decline (Roels *et al*, 1981b). Urinary excretion of cadmium plateaus at human exposure levels above 0.5 mg/m^3 x year, possibly because of renal saturation at this level and the inability of kidney to further increase excretion (Smith *et al*, 1980). Sugita and Tsuchiya, 1995 estimated that the biological half-life of cadmium in the kidney ranged from a few to 100 years. This prolonged half-life when combined with continuous exposure means that a steady state level would be attained near the end of a normal 70 year life span.

Studies in Animals

The placenta may act as a partial barrier to fetal exposure to cadmium. The concentration of cadmium in foetal cord blood is half that of maternal blood (Lauwerys *et al*, 1978). Cadmium may accumulate in the placenta at levels approximately 6-7 times higher than those in maternal or fetal cord blood (Kuhnert *et al*, 1982).

6.4.5 Toxicokinetic Models

Toxicokinetic models have been developed and used to predict cadmium absorption, distribution and half-life using small samples of human data. At the present time none of these models are capable of incorporating factors such as metallothionein induction or the levels of other metals or inter subject variability, which may affect the toxicity developed during long term exposure to cadmium compounds.

6.5 Key Issues for Human Toxicity

6.5.1 Overview

As outlined in Section 6.3, there is a wealth of information on the effects of cadmium (and cadmium compounds) upon humans and other mammals. However, as with many such reviews, it is difficult to build up a comprehensive and consistent picture. One of the more striking features of the summaries presented in Tables 6.16 and 6.22 is that there is often a divergence between the key effects which are observed for

animals in the laboratory and those observed in studies of the effects of human exposure. Nevertheless, animal studies do provide confirmation of the key target organs as well as assisting in the understanding of the toxicokinetics (see Section 6.4).

Within the context of this study, we are interested in two key risk issues:

- the 'safe' levels of cadmium fume/dust in the workplace; and
- the 'safe' level of cadmium intake via oral exposure amongst the EU population.

To these two key issues, we should also add the question of cadmium carcinogenicity which is always of interest to both decision-makers and the general population. These issues are discussed, in turn, below.

6.5.2 Inhalation in the Workplace

Although there are a range of estimates, the 'safe' (NOAEL) level of cadmium dust/fume in the workplace will be of the order of 4 lg/m^3 - based on consideration of the onset of renal dysfunction amongst perhaps 1% of the exposed population.

6.5.3 Tolerable Daily Intake

The current 'tolerable daily intake' (TDI) for cadmium is 1 lg/kg which corresponds to about 70 lg/day for the 'average' EU citizen. This TDI is derived from the Provisional Tolerable Weekly Intake (PTWI) recommended by the Joint Expert Committee on Food Additives of the Food and Agriculture Organisation of the United Nations and the World Health Organisation International Programme on Chemical Safety (JECFA). It is intended that the PTWI is an estimate of the amount of a substance that can be ingested over a lifetime without appreciable risk.

These figures should be compared to a suggested NOAEL of 2 lg/kg/day (as derived from Japanese work) and the 'typical' EU intake of 10 to, say, 30 lg/day[8].

As indicated in the discussions presented earlier, there is ongoing debate as to the precise levels of cadmium which initiate renal dysfunction. In simple terms, it has been suggested that a 'threshold ' intake of 2 lg/kg/day will produce a 'critical' concentration of 200 lg Cd/g in the renal cortex after decades of exposure. However, in certain susceptible individuals, this critical concentration may be only 50 lg Cd/g. This, in turn, would suggest that the 'threshold' intake should be 0.5 lg/kg/day.

Against this background, there are moves to reduce the TDI. Indeed, within the EU, the Scientific Committee for Toxicity, Ecotoxicity and the Environment (CSTEE,

[8] By way of example, in the UK, the mean cadmium dietary intake is 14 lg/day while the 97.5 pecentile is 24 lg/day (*Source: MAFF Data for 1997).*

2000) has suggested that the continuing use of the current PTWI is not acceptable for cadmium risk assessment work.

Within the US, as a result of the major study undertaken by the Agency for Toxic Substances and Disease Registry (NTIS, 1999), the cadmium 'minimum risk level' (MRL) was set to 0.2 lg/kg/day in July 1999. This data relied heavily on the NOAEL of 2 lg/kg/day derived from the Japanese work (Nogawa *et al*, 1989) combined with an 'uncertainty factor' of 10 to allow for variability in the population.

Whilst some would advocate such a limit, its imposition would result in, perhaps, half the EU population being exposed to cadmium intakes above the 'safe' level which, perhaps, could not be justified. A more pragmatic approach would be to adopt a reduced TDI which acknowledges that, in certain susceptible individuals, the 'critical' concentration in the renal cortex may be only 50 lg Cd/g rather than the 200 lg Cd/g associated with the 'general' NOAEL of 2 lg/kg/day. This, in turn, would suggest that the 'threshold' intake should be 0.5 lg/kg/day. On this basis and for the purposes of this analysis, it will be assumed that the TDI should be reduced to half its present value - i.e. 0.5 lg/kg/day.

The adoption of this lower value would indicate that there could be a risk to a small number of susceptible people within those with a cadmium intake at the upper end of the 'normal' range of intakes amongst EU citizens.

6.5.4 Cancer

As can be seen from Tables 6.16 and 6.22 and the associated text, there is room for debate over cadmium carcinogenicity. Since some (but not all) animal studies have shown carcinogenic effects at concentrations of the order of 10 mg/m^3 over a period of 18 months, various authorities have declared cadmium to be a (probable) human carcinogen.

7. RISK CHARACTERISATION

7.1 Overview

The purpose of the risk characterisation is to assess whether the predicted concentrations in the environment exceed those concentrations which produce adverse effects in either the environment or in humans.

7.2 Risks to the Environment

7.2.1 Surface water

The PEC values and PEC/PNEC ratios for surface water are shown in Table 7.1. Where a range of values was calculated for the PEC, the high end of the range has been used here. The outcome of the assessment (i.e. whether the PEC/PNEC ratio is greater or less than 1) would not be affected by choosing the lower end of the range.

Table 7.1: PEC/PNEC Ratios for Surface Water

Scenario		PEC (µg/l)		PEC/PNEC	
		General Environment	**Acidic Environment**	**General Environment**	**Acidic Environment**
Pigment manufacturing sites	A	0.179	0.511	0.41	**1.16**
	B	0.0107	0.0643	0.024	0.15
	C	0.052	0.171	0.12	0.39
	D	0.0205	0.090	0.046	0.20
	E	0.0098	0.062	0.022	0.14
Stabiliser manufacturing sites	F	0.010	0.063	0.022	0.14
	G	0.0275	0.108	0.062	0.25
	H	0.0265	0.106	0.06	0.24
	I	0.0133	0.071	0.03	0.16
	J	-	-	-	-
	K	0.59	1.59	**1.34**	**3.61**
Artists colours – formulation		0.0126	0.0695	0.029	0.16
Cadmium plating sites		0.099	0.301	0.23	0.68
Plated metal use[a]		6.6	17.4	**15**	**39.5**
Waste incineration		-	-	-	-
Sewage sludge incineration		-	-	-	-
Regional sources		9.48×10^{-3}	0.0612	0.22	0.14

Note: a) these estimates are highly uncertain for this application (see earlier).

As can be seen from Table 7.1, the PEC/PNEC ratio exceeds one for one pigment manufacturing site (if in an acidic environment), for one stabiliser manufacturing site and for plated metal use.

7.2.2 Sediment

Sediment concentrations were calculated from the surface water concentrations using the equilibrium partitioning method. The PNEC values were also calculated by the same method. As a result, the PEC/PNEC ratios for sediment are the same as those for surface water, and so are not included separately here. The only exception to this is the regional concentration in sediment which is calculated using the SimpleBox model in EUSES. The highest regional concentrations calculated were 0.474 mg/kg wet weight for the general environment, and 0.186 mg/kg wet weight for the acidic environment. The PEC/PNEC ratios for these concentrations are 0.038 and 0.24 respectively.

7.2.3 Terrestrial compartment

The PEC/PNEC ratios for soil are shown in Table 7.2.

Table 7.2: PEC/PNEC Ratios for Soil

Scenario		PEC (mg/kg wet weight)		PEC/PNEC	
		General Environment	Acidic Environment	General Environment	Acidic Environment
Pigment manufacturing sites	A	0.0363	0.0014	0.03	0.001
	B	0.0364	0.00154	0.03	0.001
	C	0.0378	0.00288	0.03	0.002
	D	0.0375	0.00257	0.03	0.002
	E	0.0360	0.00113	0.03	<0.001
Stabiliser manufacturing sites	F	0.0359	0.00108	0.03	<0.001
	G	0.0360	0.00111	0.03	<0.001
	H	0.0359	0.00109	0.03	<0.001
	I	0.0359	0.00108	0.03	<0.001
	J	0.0359	0.0011	0.03	<0.001
	K	0.0361	0.00123	0.03	<0.001
Artists colours - formulation		0.0359	0.00108	0.03	<0.001
Cadmium plating sites		0.0359	0.0011	0.03	<0.001
Plated metal use		0.0359	0.00108	0.03	<0.001
Waste incineration		0.316	0.268	0.26	0.22
Sewage sludge incineration		0.0362	0.00133	0.03	0.001
Regional sources		0.0359	0.00108	0.03	<0.001

As can be seen from Table 7.2, the PEC/PNEC ratios are well below one in all cases. However, concentrations in industrial/urban soil have also been estimated. For local concentrations from use of plated metal, the concentration predicted was around 35 mg/kg wet wt. At the regional level, the concentration in urban/industrial soil is estimated to be around 5.74 in the general environment and 0.172 in the acidic environment. PEC/PNEC ratios are not calculated for industrial/urban soils in the Technical Guidance Document.

The predicted concentrations in soil, particularly the regional background concentrations which are calculated as steady state values, are very dependent on the removal rates used in the EUSES model (removal is used in the sense of the metal moving to a location or form which is not available to interact with the environment being assessed). The values obtained above were calculated using the EUSES default removal rates for this substance (effectively only leaching loss in this case as no degradation or volatilisation is expected), which are equivalent to a residence time (1/removal rate constant) of 2,245 years for agricultural soil in the general environment and 99 years for agricultural soil in the acidic environment. Since a very long residence time for the substance was used in the calculations, particularly in the general environment, the time to reach the predicted steady state could be of the order of many thousands of years. Over this time frame, it is questionable that a simple steady state model as utilised in EUSES will result in reliable predictions for this type of substance (for example the calculations assume that the same 20 cm depth of soil is exposed over the entire timescale of the calculation, and takes no account of the fact that over a long time period soil can be created and/or removed from the system).

One way around this problem would be to carry out the regional calculations over a shorter time period. WS Atkins (1998a) report a soil residence time of around 100 years for the general environment and around 10 years for the acidic environment. When removal rates appropriate to these lifetimes are used in the EUSES model, the predicted regional concentrations in agricultural soil are around 5×10^{-3} mg/kg wet weight for the general environment and around 4×10^{-4} mg/kg wet weight for the acid environment. Thus the regional concentration in the general environment would be reduced by a factor of around 7 and the regional concentration in the acid environment would be reduced by a factor of around 2.5.

7.2.4 Secondary Poisoning

For secondary poisoning, it is necessary to provide an estimate of dietary cadmium concentrations which would produce a significant effect on the viability of mammalian populations. In broad terms (and with reference to Table 6.22), a daily cadmium intake of the order of 1 mg/kg is required to produce effects over the 'intermediate' period. There are, however, examples of significant effects at lower concentrations including effects amongst rats on the liver at 0.05 mg/kg/day and development effects on the offspring of pregnant rats exposed to 0.04 mg/kg/day.

These intakes can be (approximately) related to dietary concentrations as follows:

rat weight	*500g*
daily diet	*50g food + 50g water*
Cd conc in food	*C mg/kg*
daily Cd intake	*C x 0.05 mg/day = C x 0.1 mg/kg/day*

In other words, a daily intake of 0.04 mg/kg/day would correspond to a cadmium in food concentration (C) of 0.4 mg/kg. Although perhaps conservative, this

corresponds to that of 0.33 mg Cd/kg food derived in the draft risk assessment for cadmium (De Win, 1999). The PEC/PNEC ratios for secondary poisoning (using the Belgian figure) are shown in Tables 7.3 and 7.4.

Table 7.3: PEC/PNEC Ratios for Secondary Poisoning via the Aquatic Food Chain

Scenario		PEC (mg/kg)		PEC/PNEC	
		General Environment	**Acidic Environment**	**General Environment**	**Acidic Environment**
Pigment manufacturing sites	A	0.00239	0.606	0.007	**1.8**
	B	3.7×10^{-4}	0.186	0.001	0.56
	C	9.85×10^{-4}	0.314	0.003	0.95
	D	4.89×10^{-4}	0.21	0.001	0.64
	E	3.63×10^{-4}	0.184	0.001	0.56
Stabiliser manufacturing sites	F	3.6×10^{-4}	0.184	0.001	0.56
	G	3.65×10^{-4}	0.185	0.001	0.56
	H	3.66×10^{-4}	0.185	0.001	0.56
	I	3.61×10^{-4}	0.184	0.001	0.56
	J	3.6×10^{-4}	0.184	0.001	0.56
	K	4.01×10^{-4}	0.192	0.001	0.58
Artists colours - formulation		3.64×10^{-4}	0.184	0.001	0.56
Cadmium plating sites		0.00177	0.477	0.005	**1.44**
Plated metal use[a]		0.126	26.3	0.38	**79.7**
Waste incineration		3.6×10^{-4}	0.184	0.001	0.56
Sewage sludge incineration		3.6×10^{-4}	0.184	0.001	0.56

Note: a) These estimates are highly uncertain for this application

Table 7.4: PEC/PNEC Ratios for Secondary Poisoning via the Terrestrial Food Chain

Scenario		PEC (mg/kg)		PEC/PNEC	
		General Environment	**Acidic Environment**	**General Environment**	**Acidic Environment**
Pigment manufacturing sites	A	0.775	0.0653	**2.3**	0.20
	B	0.776	0.0677	**2.4**	0.21
	C	0.787	0.0897	**2.4**	0.27
	D	0.784	0.0846	**2.4**	0.26
	E	0.773	0.0609	**2.3**	0.18
Stabiliser manufacturing sites	F	0.772	0.060	**2.3**	0.18
	G	0.773	0.0605	**2.3**	0.18
	H	0.772	0.0603	**2.3**	0.18
	I	0.772	0.0601	**2.3**	0.18
	J	0.772	0.0604	**2.3**	0.18
	K	0.773	0.0624	**2.3**	0.18
Artists colours - formulation		0.772	0.060	**2.3**	0.18

Table 7.4: PEC/PNEC Ratios for Secondary Poisoning via the Terrestrial Food Chain				
Scenario	**PEC (mg/kg)**		**PEC/PNEC**	
	General Environment	**Acidic Environment**	**General Environment**	**Acidic Environment**
Cadmium plating sites	0.773	0.0604	**2.3**	0.18
Plated metal use	0.772	0.060	**2.3**	0.18
Waste incineration	2.91	4.46	**8.8**	**13.5**
Sewage sludge incineration	0.774	0.0642	**2.3**	0.19

The comments on the PEC/PNEC ratio for soil also apply here to the PEC/PNEC ratios for secondary poisoning via the terrestrial food chain. For the majority of the calculations the dominant input into the PEC comes from the regional soil concentration, and so would be reduced if a shorter residence time were used - which, in turn, would significantly reduce the PEC/PNEC ratios. This does not apply to waste incineration, where there is a significant input from the local emissions - although there are other uncertainties.

7.3 Risks to Humans

7.3.1 Direct Risks

Historically, workplace concentrations of the order of 10-50 lg/m^3 have been observed in pigment and stabiliser facilities (as outlined in Section 4.2). As these levels are above the suggested NOAEL of 4 lg/m^3, it might be expected that some adverse effects would be observed amongst long term workers. However, given the awareness of the adverse effects of cadmium, measures can (and have) been taken to reduce worker exposures - as indicated by concentrations of the order of 1 lg/m^3 achieved in one of the plating facilities.

7.3.2 Man exposed via the environment

The margins of safety for man exposed via environmental routes are shown in Table 7.5 using a TDI of 0.5 lg/kg/day. As can be seen, the acidic environment results in an increase in cadmium intake and a reduction in the margin of safety. For those near to a very large airport and near to an incinerator with relatively high cadmium emissions, the daily intake has been calculated to exceed the TDI for both the general and acidic environments. In addition, for the acidic environment, the TDI has been exceeded for two of the pigment manufacturing sites.

Table 7.5: Margins of Safety for Man Exposed via the Environment					
Scenario		**Total Daily Human Intake (lg/kg bw/day)**		**MOS (= TDI/Intake)**	
		General Environment	**Acidic Environment**	**General Environment**	**Acidic Environment**
Pigment manufacturing sites	A	0.13	**1.7**	3.8	**0.29**
	B	0.12	0.32	4.2	1.6
	C	0.13	**0.74**	3.8	**0.68**
	D	0.12	0.41	4.2	1.2
	E	0.11	0.31	4.5	1.6
Stabiliser manufacturing sites	F	0.11	0.31	4.5	1.6
	G	0.11	0.31	4.5	1.6
	H	0.11	0.31	4.5	1.6
	I	0.11	0.31	4.5	1.6
	J	0.11	0.31	4.5	1.6
	K	0.12	0.34	4.2	
Artists colours - formulation		0.11	0.31	4.5	1.6
Cadmium plating sites		0.12	0.14	4.2	3.6
Plated metal use		**0.75**	**86**	**0.67**	**0.006**
Waste incineration		**1.37**	**2.66**	**0.36**	**0.19**
Sewage sludge incineration		0.12	0.31	4.2	1.6
Regional sources		0.19	0.32	2.6	1.6

8. COMPARISONS WITH OTHER SOURCES/USES OF CADMIUM

8.1 Cadmium Use in Pigments, Stabilisers and Plating

Cadmium use in pigments, stabilisers and plating is but one source of cadmium emissions to the environment. As discussed in Section 5.5.2, the additional inputs of cadmium to the environment associated with these uses are relatively minor at a regional level when compared to the 'natural' background levels in air, water, sediment and soil.

At the local level (and the results are presented in Section 7), additional inputs of cadmium to the environment from uses in pigments, stabilisers and metal plating are generally minor (less than, say, 10%) when compared to the 'natural' background levels in air, water, sediment and soil. However, for particular facilities, the local Predicted Environmental Concentrations (PECs) may exceed not only background levels but also the Predicted No Effect Concentrations (PNECs).

In relation to human health, the 'typical' overall cadmium intake is of the order of 10-25 lg/day (see Section 4.2.2) and that for occupationally exposed workers (historically at least) may be two or three times greater (see Section 4.2.3). Intakes of cadmium at a local level (see Section 7.3.2) will generally be of a similar order to the typical intake - although, for particular facilities and particular individuals, the intakes could be much higher.

8.2 Major Anthropogenic Sources/Uses

Since cadmium is a naturally occurring metal, there are a range of activities which result in cadmium emissions to the environment. As discussed in Section 4.1.2, these include: refining of non-ferrous metals (such as copper); iron/steel production; fossil fuel combustion; use of phosphate fertilisers; cement production; and waste disposal.

In relation to 'direct' uses of cadmium, the use of cadmium in pigments, stabilisers and metal plating account for about 20% (c1000 tpa) of the EU consumption of cadmium produced from the extraction of zinc (as outlined in Section 3.1). The vast majority of the remaining 80% (c4000 tpa) is used in nickel-cadmium batteries.

Although there are undoubtedly uncertainties associated with particular figures, Table 8.1 provides an indication of the relative importance of various cadmium sources to human exposure. From Table 8.1, it can be seen that cadmium products (essentially in batteries and in pigments, stabilisers and plating) are estimated to account for only 2.5% of human exposure.

Further discussions on the prime source (phosphate fertiliser) and the prime 'direct' cadmium use (batteries) are presented below.

Table 8.1: Significance of Cadmium Sources to Human Exposure	
Source	**Contribution**
Phosphate Fertilisers	41%
Fossil Fuel Combustion	22%
Iron & Steel Production	17%
Natural Sources	8%
Non-Ferrous Metals	6%
Cement Production	2.5%
Cadmium Products	2.5%
Incineration	1%
Source: based on Van Assche (1998)	

8.3 Cadmium in Fertilisers

As discussed, in Section 4.1.2, phosphate fertilisers contain cadmium and it has been estimated that about 270 tpa is added directly to the terrestrial environment through the application of fertilisers.

This figure can be compared with that of about 58 tpa emitted to air, water and soil (i.e. excluding landfills) from uses in pigments, stabilisers and metal plating (derived from the information presented in Figures 4.1 - 4.4).

Given the relative scale of usage, it might be expected that the risks to health and the environment would be greater from the presence of cadmium in some fertilisers - particularly as some fertilisers will be added 'closer' to the food chain - although whether the ratio of risks is as great as suggested by Table 8.1 is a matter for debate.

In order to address concerns over the risks associated with cadmium in fertilisers, the European Commission is currently developing detailed procedures for the assessment of risks to health and the environment from cadmium in fertilisers (as discussed in CSTEE, 2000). Clearly, once these procedures have been developed and applied in EU states, it will become easier to compare risks associated with fertilisers to other sources/uses of cadmium.

8.4 Cadmium in Batteries

As indicated above, the prime 'direct' EU use of cadmium is in batteries. In broad terms, the key issues associated with risks to health and the environment are very similar to those for stabilisers and pigments:

- cadmium fume/dust levels in the workplace are of concern and action is being to minimise worker exposures;
- losses during use are of minimal concern;

- many batteries end up in municipal solid waste leading to potential cadmium emissions from incinerators and landfills; and
- recycling/recovery programmes are being encouraged to limit emissions to the environment.

Given the relative scale of usage (four times greater), it might be expected that the risks to health and the environment would be greater from the use of cadmium in batteries than from the use of cadmium in pigments, stabilisers and plating. This issue is currently being addressed with the preparation of cadmium/cadmium oxide risk assessments reports by the Belgian authorities (De Win *et al*, 1999) under the EU's Existing Substances Regulations. As before, once this work has been completed, it will become easier to compare risks associated with different sources/uses of cadmium.

9. CONCLUSIONS AND RECOMMENDATIONS

9.1 Overview of Study

The earlier work by WS Atkins has been reviewed taking account of the comments made by CSTEE. In broad terms, the pattern of cadmium usage in pigments, stabilisers and metal plating presented by WS Atkins has been validated. Work on PECs (Predicted Environmental Concentrations) and PNECs (Predicted No Effect Concentrations) has been revisited and this has relied heavily on the approach adopted by the Belgian authorities in their ongoing study of the risks associated with cadmium and cadmium oxide. Human toxicity and associated animal studies have been critically reviewed, where this work has relied heavily on a recent US study.

9.2 Overall Findings

Cadmium usage in pigments, stabilisers and metal plating results in a significant emission of cadmium into the environment. While the associated risks to health and environment may be higher for other sources of cadmium (use in batteries, presence in some fertilisers as well as 'natural' background), the risks associated with pigments, stabilisers and metal plating cannot be dismissed.

At a regional level, the level of risk is small both in comparison to the natural background and to the 'safe' levels of cadmium.

At a local (site specific) level, there may be occupational risks (with particular reference to the onset of renal dysfunction) due to continued exposure (over decades) to concentrations of the order of 10 lg/m^3 or more. However, there is a broad drive to reduce occupational levels to 5 lg/m^3 or less (through regulation and best practice) which should practically eliminate such adverse effects (for which a NOAEL has been estimated at 4 lg/m^3).

Local emissions to the environment from the 11 identified pigment and stabiliser manufacturing sites were calculated for both 'general' and 'acidic' environments (to give 22 combinations). For surface water (and sediment), PEC/PNEC ratios of greater than unity were found in three (out of 22) cases. For the terrestrial compartment, PEC/PNEC ratios were less than unity in all cases. For secondary poisoning to mammals via the aquatic food chain, the PEC/PNEC ratio was greater than unity in just one case while that via the terrestrial food chain was greater than unity for all 'general' environment cases. For particularly susceptible individuals with a dietary intake based exclusively on local sources, the Tolerable Daily Intake (TDI - taken as 0.5 lg/kg/day) was exceeded in two cases.

In summary, there would appear to be a slight risk to health and the environment associated with pigment and stabiliser manufacture - subject to further consideration of the secondary poisoning via the terrestrial food chain.

Local emissions to the environment from artists' colours formulators and cadmium plating sites were similarly considered for both the 'general' and 'acidic' environments. For surface water (and sediment) and the terrestrial compartment, PEC/PNEC ratios were less than unity in all cases. For secondary poisoning to mammals via the aquatic food chain, the PEC/PNEC ratio was greater than unity in just one case (plating site/acidic environment) while that via the terrestrial food chain was greater than unity for all 'general' environment cases. For particularly susceptible individuals with a dietary intake based exclusively on local sources, the TDI was not exceeded. In summary, there would appear to be a slight risk to the environment associated with artists' colours formulators and cadmium plating sites - subject to further consideration of the secondary poisoning via the terrestrial food chain.

Emissions to the environment during use of products containing cadmium stabilisers and pigments were determined and were found to be of negligible proportions. By contrast, the emissions from cadmium plated aircraft components accounted for most of the cadmium emitted to the environment associated with the uses under study. For the determination of the associated risks, it was assumed (as a worst case) that 10% of emissions could be assigned to a single major EU airport (and London Heathrow was taken as an example). For surface water (and sediment), PEC/PNEC ratios were greater while those for terrestrial compartment were less than unity. For secondary poisoning to mammals via the aquatic food chain, the PEC/PNEC ratio was greater than unity for the acidic environment while that via the terrestrial food chain was greater than unity for the 'general' environment. For particularly susceptible individuals with a dietary intake based exclusively on local sources, the TDI was exceeded - particularly for the acidic case. In summary, there would appear to be a significant risk to health and the environment associated with use of cadmium plated articles in aircraft. However, the analysis does carry a number of uncertainties and consideration could be given to a 'reality check' based on any cadmium monitoring data in the vicinity of major airports. As a first step, preliminary discussions have been held with the authorities at Heathrow who have indicated that significant levels of cadmium have not been observed - although further confirmatory information has not been forthcoming.

Although emissions from landfill were not determined (as they are outside the Technical Guidance Document), consideration was given to emissions from incinerators (both municipal solid waste - MSW - and sewage sludge). For the determination of the associated risks, it was assumed (as a worst case) that 10% of emissions could be assigned to a single incinerator. For the terrestrial compartment, the PEC/PNEC ratios were less than unity. For secondary poisoning to mammals via the aquatic food chain, the PEC/PNEC ratios were less than unity, while those via the terrestrial food chain were greater than unity - excepting the sewage/acidic case. For particularly susceptible individuals with a dietary intake based exclusively on local sources, the TDI was exceeded for the MSW but not the sewage sludge incinerator. In summary, there would appear to be a significant risk to health and the environment associated with MSW incineration and a lesser risk use associated with sewage sludge incineration. However, the analysis does carry a number of uncertainties and there needs to be some caution in applying the results. For example, although it is highly

unlikely that one incinerator (out of the 500+ MSW incinerators) would handle 10% of the cadmium being incinerated, it is possible that one 'rogue' incinerator could be responsible for a disproportionate amount of cadmium emissions to the atmosphere. Given the current interest of both the public and the regulators in incinerators, it is unlikely that such a situation would be tolerated for long (years).

Overall, using the approach prescribed by the Technical Guidance Note, it would appear that there are certain areas where the risks to health and the environment associated with pigments, stabilisers and cadmium plating merit further consideration.

However, it must be remembered that these 'risks' are based on assumptions which tend to:

- maximise the emissions;
- maximise the exposure; and
- minimise the 'threshold' concentration for effects.

9.3 Recommendations

Given the significance of the secondary poisoning results, further consideration should be given to whether assumptions used in both this and the ongoing study by the Belgian authorities are robust - with particular reference to the 'threshold' value for effects and the likely residence time of cadmium in the soil.

Given that much of the cadmium used in pigments, stabilisers and cadmium plating will ultimately end up in a landfill in one form or another, it would be of assistance if there was an agreed procedure within EUSES to address the associated risks.

Given the potential significance of cadmium losses from plated components on aircraft, it is recommended that further attention be given to cadmium levels around major EU airports.

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ANNEX 1

TERMS OF REFERENCE

Contract No **ETD/99/502487**

ANNEX II

TECHNICAL ANNEX

Description of Tasks

Aim

The aim of the study is to up-date earlier studies on the risks to health and the environment posed by cadmium in certain products. The study will provide a basis on which future discussions on the possible need for further restrictions on the use of cadmium may be based.

Scope of the study

The study will provide an assessment of the risks to health and the environment of cadmium used as a colouring agent, as stabiliser in polymers and for metal plating. All stages of the life cycle will be taken into account including e.g. production, formulation, use, and disposal. The study will also assess the relative risks from cadmium in these applications compared with the other sources of cadmium.

The methodology will be based on the Technical Guidance Document to risk assessments of existing substances under Regulation 93/793 however taking recent developments of methodologies for risk assessment of metals into account.

Tasks

The study will modify earlier assessments of the risks to health and the environment from cadmium in certain products[1,2] made in the context of the review of the cadmium provisions, taking particular account of deficiencies being identified by the Scientific Committee on Toxicity, Ecotoxicity and the Environment in its opinion of 18 January 1999.

Attention will be paid to the following areas where weaknesses have been identified:

$ Human toxicity. The evaluation of toxicity to humans will be expanded taking into consideration the extensive information available

$ Derivation of PEC values. The derivation of PEC values will be revised with regard to methodology and data

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[1] Assessment of the Risks to Health and the Environment of Cadmium in Certain Products, WS Atkins, Final Report September 1998

[2] Additional Assessment of the Risks to Health and the Environment of Cadmium in Certain Products, WS Atkins, Final Report September 1998

- Estimations of daily intakes of cadmium and TDI. The estimation of daily intakes of cadmium and of TDI will be reviewed taking into account especially the variation of measured values.

- Derivation of PNEC values. The derivation of PNEC values will be revised especially with regard to data used, ensuring a proper use of the probabilistic method.

- Risk characterisation. The risk characterisation will be revised and can be further improved by addressing the effects of environmental characteristics on cadmium toxicity and by the inclusion of PNEC values adapted to the water/soil conditions using probabilistic approaches for European water/soil characteristics.

- The study will also assess the relative risks from cadmium used as a colouring agent, as stabiliser in polymers and for metal plating compared with the other sources of cadmium.

Sources of information
The basis of the study are the assessments mentioned under 'Tasks'.
In addition publicly available studies, published reports and information from sources available to the contractor will be used. Previous reports to the Commission on Cadmium and information on on-going risk assessment of cadmium will be provided by the Commission to the contractor.

Other sources of information include contacts with services of the Commission (DGs III, V, XI, XXIV), contacts with Member States and contacts with relevant industries and their representative associations.

Meetings: The contractor may be requested, and should be prepared, to attend a kick-off meeting at the Commission's premises in Brussels and possibly one more meeting in Brussels to discuss the progress of the work. The Commission will reimburse the travel for participation in such meetings in accordance with Article 5 and Annex IV to the Contract which provide, inter alia, for the reimbursement, on the basis of supporting documents, of travel expenses allowed by the contract and a fixed daily subsistence allowance.

The tasks specified above shall be executed on the Contractor's premises, with the exception of the meetings which will be held in Brussels.

ANNEX 2

DIRECTIVE 91/338/EEC

(Specific Restrictions on Cadmium and its Compounds as listed in Annex to the Directive)

24. Cadmium (CAS No 7440-43-9) and its compounds

Conditions of restriction:

1.1 May not be used to give colour to finished products manufactured from the substances and preparations listed below:

- polyvinyl chloride (PVC) (3904 10) (3904 21) (3904 22)[1]
- polyurethane (PUR) (3909 50)[1]
- low-density polyethylene (ld PE), with the exception of low-density polyethylene used for the production of coloured masterbatch (3901 10)[1]
- cellulose acetate (CA) (3912 11) (3912 12)[1]
- cellulose acetate butyrate (CAB) (3912 11) (3912 12)[1]
- epoxy resins (3907 30)[1]

In any case, whatever their use or intended final purpose, finished products or components of products manufactured from the substances and preparations listed above coloured with cadmium may not be placed on the market if their cadmium content (expressed as Cd metal) exceeds 0.01% by mass of the plastic material.

1.2 Section 1.1 also applies from 31 December 1995 for:

(a) finished products manufactured from the following substances and preparations:

- melamine – formaldehyde (MF) (3909 20) [1]
- urea – formaldehyde (UF) (3909 10)[1]
- unsaturated polyesters (UP) (3907 91) [1]
- polyethylene terephthalate (PET) (3907 60)[1]
- polybutylene terephthalate (PBT)
- transparent/general-purpose polystyrene (3903 11) (3903 19)[1]
- acrylonitrile methylmethacrylate (AMMA)
- cross-linked polyethylene (VPE)[1]
- high-impact polystyrene
- polypropylene (PP) (3902 10)[1]

(b) paints (3208) (3209) [1]

However, if the paints have a high zinc content, their residual concentration of cadmium must be as low as possible and at all events not exceed 0.1% of mass.

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[1] Council Regulation (EEC) No 2658/87 of 23 July 1987 on the tariff and statistical nomenclature and on the Common Customs Tariff (OJ No L256, 7.9.1987).

1.3 However, Sections 1.1 and 1.2 do not apply to products to be coloured for safety reasons.

2.1 May not be used to stabilize the finished products listed below manufactured from polymers or copolymers of vinyl chloride:

- packaging materials (bags, containers, bottles, lids) (3923 29 10) (3920 41) (3920 42)[1]
- office or school supplies (3926 10)[1]
- fittings for furniture, coachwork or the like (3926 30)[1]
- articles of apparel and clothing accessories (including gloves) (3926 20)[1]
- floor and wall coverings (3918 10)[1]
- impregnated, coated, covered or laminated textile fabrics (5903 10)[1]
- imitation leather (4202)[1]
- gramophone records (8524 10)[1]
- tubes and pipes and their fittings (3917 23)[1]
- swing doors[1]
- vehicles for road transport (interior, exterior, underbody)[1]
- coating of steel sheet used in construction or in industry[1]
- insulation for electrical wiring[1]

In any case, whatever their use or intended final purpose, the placing on the market of the above finished products or components of products manufactured from polymers or copolymers of vinyl chloride, stabilized by substances containing cadmium is prohibited, if their cadmium is prohibited, if their cadmium content (expressed as Cd metal) exceeds 0.01% by mass of the polymer.

These provisions enter into force on 30 June 1994.

2.2 However, Section 2.1 does not apply to finished products using cadmium-based stabilizers for safety reasons.

3. Within the meaning of this Directive, “cadmium plating” means any deposit or coating of metallic cadmium on a metallic surface.

3.1 May not be used for cadmium plating metallic products or components of the products used in the sectors/applications listed below.

(a) equipment and machinery for:
- food production: (8210) (8417 20) (8419 81) (8421 11) (8421 22) (8422) (8435) (8437) (8438) (8476 11)[1]
- agriculture: (8419 31) (8424 81) (8432) (8433) (8434) (8436)[1]
- cooling and freezing: (8418)[1]
- printing and book-binding (8440) (8442) (8443)[1]

(b) equipment and machinery for the production of:
- household goods (7321) (8421 12) (8450) (8509) (8516)[1]
- furniture (8465) (8466) (9401) (9402) (9403) (9404)[1]
- sanitary ware (7324)[1]
- central heating and air conditioning plant (7322) (8403) (8404) (8415)[1]

In any case, whatever their use or intended final purpose, the placing on the market of cadmium-plated products or components of such products used in the sectors/applications listed in (a) and (b) above and of products manufactured in the sectors listed in (b) above is prohibited.

3.2 The provisions referred to in Section 3.1 are also applicable from 30 June 1995 to cadmium-plated products or components of such products when used in the sectors/applications listed in (a) and (b) below and to products manufactured in the sectors listed in (b) below:

(a) equipment and machinery for the production of:
- paper and board (8419 32) (8439) (8441)[1]
- textiles and clothing (8444)[1] (8445) (8447) (8448) (8449) (8451) (8452)[1]

(b) equipment and machinery for the production of:
- industrial handling equipment and machinery (8425) (8426) (8427) (8428) (8429) (8430) (8431)[1]
- road and agricultural vehicles (chapter 87)[1]
- rolling stock (chapter 86)[1]
- vessels (chapter 89)[1]

3.3 However, Sections 3.1 and 3.2 do not apply to:

- products and components of the products used in the aeronautical, aerospace, mining, offshore and nuclear sectors whose applications require high safety standards and in safety devices in road and agricultural vehicles, rolling stock and vessels.
- electrical contacts in any sector of use, on account of the reliability required of the apparatus on which they are installed.

ANNEX 3

CONSULTEES

CONSULTEES

During the course of this study, many people have been contacted for information, guidance and assistance. We are particularly grateful for the contributions from:

- Akcros Chemicals
- Ashton & Moore
- British Airports Authority
- British Ceramics Association
- British Coatings Federation
- British Colour Makers Association
- British Surface Treatment Suppliers Association
- Chemical Industries Association (UK)
- Daler-Rowney Ltd
- Environment Agency (UK)
- European Council of the Paint, Printing Ink and Artists' Colours Industry (CEPE)
- Institute of Metal Finishing (UK)
- International Cadmium Association
- International Zinc Association
- James M Brown Ltd
- Johnson Matthey Ltd
- Metal Finishing Association (UK)
- Robert Stuart PLC
- Surface Finishing Association (UK)
- Societe Languedocienne de Mircon-Couleurs (SLMC)
- VDMI (Trade Association for Pigments in Germany)

ANNEX 4

EXPOSURE OF MAN VIA THE ENVIRONMENT

(Part 1: General Environment & Part 2: Acidic Environment)

Part 1: General Environment

Endpoint	Use pattern identity in EUSES printout	Concentration in air (mg/m^3)		Concentration in drinking water (mg/l)		Concentration in agricultural soil (average over 180 days) (mg/kg wet wt.)		Concentration in grassland (mg/kg wet wt.)		Annual average concentration in surface water (mg/l)	
		Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper
Pigment manufacturing site A	1 Formulation	9.13E-07	9.19E-07	1.16E-04	1.16E-04	0.0317	0.0363	0.0321	0.0366	1.19E-04	1.19E-04
Pigment manufacturing site B	1 Processing	1.31E-06	1.31E-06	1.19E-05	1.36E-05	0.0319	0.0364	0.0324	0.0369	9.98E-06	1.02E-05
Pigment manufacturing site C	1 Private use	4.92E-06	4.92E-06	4.21E-05	4.24E-05	0.0333	0.0378	0.0352	0.0398	4.22E-05	4.25E-05
Pigment manufacturing site D	1 Recovery	4.07E-06	4.07E-06	1.60E-05	1.63E-05	0.033	0.0375	0.0346	0.0391	1.61E-05	1.64E-05
Pigment manufacturing site E	2 Formulation	1.92E-07	1.98E-07	1.18E-05	1.35E-05	0.0315	0.036	0.0315	0.036	9.36E-06	9.62E-06
Stabiliser manufacturing site F	3 Formulation	4.50E-08	5.00E-08	1.18E-05	1.34E-05	0.0314	0.0359	0.0314	0.0359	9.22E-06	9.48E-06
Stabiliser manufacturing siteG	3 Processing	1.10E-07	1.10E-07	1.18E-05	1.34E-05	0.0314	0.036	0.0315	0.036	9.48E-06	9.74E-06
Stabiliser manufacturing site H	3 Private use	7.90E-08	8.50E-08	1.18E-05	1.34E-05	0.0314	0.0359	0.0314	0.036	9.53E-06	9.79E-06
Stabiliser manufacturing site I	3 Recovery	4.60E-08	5.10E-08	1.18E-05	1.34E-05	0.0314	0.0359	0.0314	0.0359	9.27E-06	9.53E-06
Stabiliser manufacturing site J	4 Formulation	9.60E-08	1.00E-07	1.18E-05	1.34E-05	0.0314	0.0359	0.0314	0.036	9.22E-06	9.48E-06
Stabiliser manufacturing site K	4 Processing	4.10E-08	4.60E-08	1.18E-05	1.35E-05	0.0316	0.0361	0.0315	0.036	1.13E-05	1.16E-05
Artists colours formulation	5 Formulation	3.73E-08	4.26E-08	1.18E-05	1.34E-05	0.0314	0.0359	0.0314	0.0359	9.39E-06	9.65E-06
Cadmium plating	6 Processing	3.73E-08	4.26E-08	8.35E-05	8.37E-05	0.0314	0.0359	0.0314	0.0359	8.32E-05	8.35E-05
Plated metal use	7 Processing	3.73E-08	4.26E-08	6.60E-03	6.60E-03	0.0314	0.0359	0.0314	0.0359	6.60E-03	6.60E-03
Waste incineration	8 Recovery	6.31E-04	7.23E-04	1.05E-04	1.20E-04	0.28	0.321	0.529	0.606	9.22E-06	9.48E-06
Sewage sludge incineration	9 Recovery	7.24E-08	7.29E-08	1.19E-05	1.35E-05	0.0317	0.0362	0.0319	0.0365	9.22E-06	9.48E-06
Regional sources	Regional	3.73E-08	4.26E-08	1.89E-05	2.51E-05	0.0506	0.0671	0.0314	0.0359	9.22E-06	9.48E-06

Part 1: General Environment

Endpoint	Estimated concentration in fish (mg/kg wet weight)		Estimated concentration in grass (mg/kg wet wt)		Estimated concentration in leaf crops (mg/kg wet wt.)		Estimated concentrations in root crops (mg/kg wet wt.)		Estimated concentrations in meat (mg/kg wet wt.)		Estimated concentration in milk (mg/kg wet wt)	
	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper
Pigment manufacturing site A	4.52E-03	4.52E-03	0.00321	0.00366	0.00317	0.00363	0.004755	0.005445	4.77E-03	5.41E-03	7.15E-05	8.12E-05
Pigment manufacturing site B	3.79E-04	3.88E-04	0.00324	0.00369	0.00319	0.00364	0.004785	0.00546	4.69E-03	5.35E-03	7.04E-05	8.02E-05
Pigment manufacturing site C	1.60E-03	1.62E-03	0.00352	0.00398	0.00333	0.00378	0.004995	0.00567	5.14E-03	5.81E-03	7.71E-05	8.71E-05
Pigment manufacturing site D	6.12E-04	6.23E-04	0.00346	0.00391	0.0033	0.00375	0.00495	0.005625	5.02E-03	5.67E-03	7.54E-05	8.51E-05
Pigment manufacturing site E	3.56E-04	3.66E-04	0.00315	0.0036	0.00315	0.0036	0.004725	0.0054	4.56E-03	5.21E-03	6.84E-05	7.82E-05
Stabiliser manufacturing site F	3.50E-04	3.60E-04	0.00314	0.00359	0.00314	0.00359	0.00471	0.005385	4.55E-03	5.20E-03	6.82E-05	7.80E-05
Stabiliser manufacturing siteG	3.60E-04	3.70E-04	0.00315	0.0036	0.00314	0.0036	0.00471	0.0054	4.56E-03	5.21E-03	6.84E-05	7.82E-05
Stabiliser manufacturing site H	3.62E-04	3.72E-04	0.00314	0.0036	0.00314	0.00359	0.00471	0.005385	4.55E-03	5.21E-03	6.82E-05	7.82E-05
Stabiliser manufacturing site I	3.52E-04	3.62E-04	0.00314	0.00359	0.00314	0.00359	0.00471	0.005385	4.55E-03	5.20E-03	6.82E-05	7.80E-05
Stabiliser manufacturing site J	3.50E-04	3.60E-04	0.00314	0.0036	0.00314	0.00359	0.00471	0.005385	4.55E-03	5.21E-03	6.82E-05	7.82E-05
Stabiliser manufacturing site K	4.29E-04	4.41E-04	0.00315	0.0036	0.00316	0.00361	0.00474	0.005415	4.56E-03	5.21E-03	6.84E-05	7.82E-05
Artists colours formulation	3.57E-04	3.67E-04	0.00314	0.00359	0.00314	0.00359	0.00471	0.005385	4.55E-03	5.20E-03	6.82E-05	7.80E-05
Cadmium plating	3.16E-03	3.17E-03	0.00314	0.00359	0.00314	0.00359	0.00471	0.005385	4.63E-03	5.28E-03	6.94E-05	7.91E-05
Plated metal use	2.51E-01	2.51E-01	0.00314	0.00359	0.00314	0.00359	0.00471	0.005385	1.18E-02	1.24E-02	1.77E-04	1.87E-04
Waste incineration	3.50E-04	3.60E-04	0.0529	0.0606	0.028	0.0321	0.042	0.04815	7.80E-02	8.94E-02	1.17E-03	1.34E-03
Sewage sludge incineration	3.50E-04	3.60E-04	0.00319	0.00365	0.00317	0.00362	0.004755	0.00543	4.62E-03	5.29E-03	6.93E-05	7.93E-05
Regional sources	3.50E-04	3.60E-04	0.00314	0.00359	0.00506	0.00671	0.00759	0.010065	4.56E-03	5.21E-03	6.83E-05	7.82E-05

Part 1: General Environment

Endpoint	Estimated daily human intake from drinking water (mg/kg bw/day)		Estimated daily human intake from fish (mg/kg bw/day)		Estimated daily human intake from leaf crops (mg/kg bw/day)		Estimated daily human intake from root crops (mg/kg bw/day)		Estimated daily human intake from meat (mg/kg bw day)		Estimated daily human intake from milk (mg/kg bw/day)	
	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper
Pigment manufacturing site A	3.31E-06	3.31E-06	7.43E-06	7.43E-06	5.43E-05	6.22E-05	2.61E-05	2.99E-05	2.05E-05	2.33E-05	5.73E-07	6.51E-07
Pigment manufacturing site B	3.40E-07	3.89E-07	6.23E-07	6.37E-07	5.47E-05	6.24E-05	2.62E-05	3.00E-05	2.02E-05	2.30E-05	5.64E-07	6.43E-07
Pigment manufacturing site C	1.20E-06	1.21E-06	2.63E-06	2.65E-06	5.71E-05	6.48E-05	2.74E-05	3.11E-05	2.21E-05	2.50E-05	6.18E-07	6.98E-07
Pigment manufacturing site D	4.57E-07	4.66E-07	1.01E-06	1.02E-06	5.66E-05	6.43E-05	2.72E-05	3.09E-05	2.16E-05	2.44E-05	6.04E-07	6.82E-07
Pigment manufacturing site E	3.37E-07	3.86E-07	5.84E-07	6.01E-07	5.40E-05	6.17E-05	2.59E-05	2.96E-05	1.96E-05	2.24E-05	5.48E-07	6.27E-07
Stabiliser manufacturing site F	3.37E-07	3.83E-07	5.76E-07	5.92E-07	5.38E-05	6.15E-05	2.58E-05	2.95E-05	1.96E-05	2.24E-05	5.47E-07	6.25E-07
Stabiliser manufacturing siteG	3.37E-07	3.83E-07	5.92E-07	6.08E-07	5.38E-05	6.17E-05	2.58E-05	2.96E-05	1.96E-05	2.24E-05	5.48E-07	6.27E-07
Stabiliser manufacturing site H	3.37E-07	3.83E-07	5.95E-07	6.11E-07	5.38E-05	6.15E-05	2.58E-05	2.95E-05	1.96E-05	2.24E-05	5.47E-07	6.27E-07
Stabiliser manufacturing site I	3.37E-07	3.83E-07	5.79E-07	5.95E-07	5.38E-05	6.15E-05	2.58E-05	2.95E-05	1.96E-05	2.24E-05	5.47E-07	6.25E-07
Stabiliser manufacturing site J	3.37E-07	3.83E-07	5.76E-07	5.92E-07	5.38E-05	6.15E-05	2.58E-05	2.95E-05	1.96E-05	2.24E-05	5.47E-07	6.27E-07
Stabiliser manufacturing site K	3.37E-07	3.86E-07	7.05E-07	7.24E-07	5.42E-05	6.19E-05	2.60E-05	2.97E-05	1.96E-05	2.24E-05	5.48E-07	6.27E-07
Artists colours formulation	3.37E-07	3.83E-07	5.86E-07	6.02E-07	5.38E-05	6.15E-05	2.58E-05	2.95E-05	1.96E-05	2.24E-05	5.47E-07	6.25E-07
Cadmium plating	2.39E-06	2.39E-06	5.19E-06	5.21E-06	5.38E-05	6.15E-05	2.58E-05	2.95E-05	1.99E-05	2.27E-05	5.56E-07	6.34E-07
Plated metal use	1.89E-04	1.89E-04	4.12E-04	4.12E-04	5.38E-05	6.15E-05	2.58E-05	2.95E-05	5.07E-05	5.35E-05	1.42E-06	1.50E-06
Waste incineration	3.00E-06	3.43E-06	5.76E-07	5.92E-07	4.80E-04	5.50E-04	2.30E-04	2.64E-04	3.36E-04	3.84E-04	9.38E-06	1.07E-05
Sewage sludge incineration	3.40E-07	3.86E-07	5.76E-07	5.92E-07	5.43E-05	6.21E-05	2.61E-05	2.98E-05	1.99E-05	2.27E-05	5.55E-07	6.35E-07
Regional sources	5.40E-07	7.17E-07	5.76E-07	5.92E-07	8.67E-05	1.15E-04	4.16E-05	5.52E-05	1.96E-05	2.24E-05	5.48E-07	6.27E-07

Part 1: General Environment

Endpoint	Estimated daily human intake from air (mg/kg bw day)		Total daily human intake from environmental routes (mg/kg bw day)	
	Lower	Upper	Lower	Upper
Pigment manufacturing site A	1.96E-07	1.97E-07	1.12E-04	1.27E-04
Pigment manufacturing site B	2.81E-07	2.81E-07	1.03E-04	1.17E-04
Pigment manufacturing site C	1.05E-06	1.05E-06	1.12E-04	1.26E-04
Pigment manufacturing site D	8.72E-07	8.72E-07	1.08E-04	1.23E-04
Pigment manufacturing site E	4.11E-08	4.24E-08	1.01E-04	1.15E-04
Stabiliser manufacturing site F	9.64E-09	1.07E-08	1.01E-04	1.15E-04
Stabiliser manufacturing siteG	2.36E-08	2.36E-08	1.01E-04	1.15E-04
Stabiliser manufacturing site H	1.69E-08	1.82E-08	1.01E-04	1.15E-04
Stabiliser manufacturing site I	9.86E-09	1.09E-08	1.01E-04	1.15E-04
Stabiliser manufacturing site J	2.06E-08	2.14E-08	1.01E-04	1.15E-04
Stabiliser manufacturing site K	8.79E-09	9.86E-09	1.01E-04	1.16E-04
Artists colours formulation	7.99E-09	9.13E-09	1.01E-04	1.15E-04
Cadmium plating	7.99E-09	9.13E-09	1.08E-04	1.22E-04
Plated metal use	7.99E-09	9.13E-09	7.32E-04	7.47E-04
Waste incineration	1.35E-04	1.55E-04	1.19E-03	1.37E-03
Sewage sludge incineration	1.55E-08	1.56E-08	1.02E-04	1.16E-04
Regional sources	7.99E-09	9.13E-09	1.50E-04	1.95E-04

Part 1: General Environment

Accumulation/transfer factors			Value	Unit	Comment	
Fish BCF			38	l/kg		
Soil-plant transfer factor for root crops			0.15	kg/kg	on a fresh weight plant basis	
Soil-plant transfer factor for leaf crops and grass			0.1	kg/kg	on a fresh weight plant basis	
Accumulation factor for meat from diet			0.02	d/kg		
Accumulation factor for milk from diet			0.0003	d/kg		

Daily intake figures for humans			
	Human intake		
Drinking water	2	l/day	
Fish	0.115	kg/day	wet weight
Leaf crops	1.2	kg/day	wet weight
Root crops	0.384	kg/day	wet weight
Meat	0.301	kg/day	wet weight
Diary products (milk)	0.561	kg/day	wet weight
Air	20	m^3/day	
Bioavailability inhalation	0.75		TGD default
Bioavailability oral route	1		TGD default
Adult body weight	70	kg	

Daily intake figures for cattle		
	Intake (on a wet weight basis)	
Grass	67.6	kg/day
Soil	0.46	kg/day
Air	122	m^3/day
Drinking water	55	l/day

Part 2: Acidic Environment

Endpoint	Use pattern identity in EUSES printout	Concentration in air (mg/m^3)		Concentration in drinking water (mg/l)		Concentration in agricultural soil (average over 180 days) (mg/kg wet wt.)		Concentration in grassland (mg/kg wet wt.)		Annual average concentration in surface water (mg/l)	
		Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper
Pigment manufacturing site A	1 Formulation	9.13E-07	9.19E-07	3.41E-04	3.43E-04	0.00127	0.00141	0.00157	0.0017	3.39E-04	3.41E-04
Pigment manufacturing site B	1 Processing	1.31E-06	1.31E-06	6.12E-05	6.31E-05	0.00142	0.00155	0.00185	0.00198	6.12E-05	6.32E-05
Pigment manufacturing site C	1 Private use	4.92E-06	4.92E-06	1.46E-04	1.48E-04	0.00278	0.00291	0.00443	0.00457	1.42E-04	1.44E-04
Pigment manufacturing site D	1 Recovery	4.07E-06	4.07E-06	7.71E-05	7.91E-05	0.00246	0.00259	0.00383	0.00397	7.72E-05	7.92E-05
Pigment manufacturing site E	2 Formulation	1.92E-07	1.98E-07	5.96E-05	6.15E-05	0.001	0.00114	0.00105	0.00119	5.96E-05	6.12E-05
Stabiliser manufacturing site F	3 Formulation	4.50E-08	5.00E-08	5.92E-05	6.12E-05	9.45E-04	0.00108	9.48E-04	0.00108	5.92E-05	6.12E-05
Stabiliser manufacturing siteG	3 Processing	1.10E-07	1.10E-07	5.99E-05	6.19E-05	9.72E-04	0.00111	9.92E-04	0.00113	5.99E-05	6.19E-05
Stabiliser manufacturing site H	3 Private use	7.90E-08	8.50E-08	6.00E-05	6.20E-05	9.58E-04	0.00109	9.73E-04	0.00111	6.00E-05	6.20E-05
Stabiliser manufacturing site I	3 Recovery	4.60E-08	5.10E-08	5.93E-05	6.13E-05	9.46E-04	0.00108	9.49E-04	0.00108	5.93E-05	6.13E-05
Stabiliser manufacturing site J	4 Formulation	9.60E-08	1.00E-07	5.92E-05	6.12E-05	9.64E-04	0.0011	9.85E-04	0.00112	5.92E-05	6.12E-05
Stabiliser manufacturing site K	4 Processing	4.10E-08	4.60E-08	6.48E-05	6.68E-05	0.00109	0.00123	0.001	0.00114	6.48E-05	6.68E-05
Artists colours formulation	5 Formulation	3.73E-08	4.26E-08	5.97E-05	6.16E-05	9.43E-04	0.00108	9.43E-04	0.00108	5.97E-05	6.16E-05
Cadmium plating	6 Processing	3.73E-08	4.26E-08	2.55E-04	2.57E-04	9.65E-04	0.0011	9.51E-04	0.00109	2.56E-05	2.57E-05
Plated metal use	7 Processing	3.73E-08	4.26E-08	1.74E-02	1.74E-02	9.42E-04	0.00108	9.42E-04	0.00108	1.74E-02	1.74E-02
Waste incineration	8 Recovery	6.31E-04	7.23E-04	4.56E-03	5.23E-03	0.238	0.273	0.452	0.518	5.92E-05	6.12E-05
Sewage sludge incineration	9 Recovery	7.24E-08	7.29E-08	5.92E-05	6.12E-05	0.0012	0.00134	0.00143	0.00157	5.92E-05	6.12E-05
Regional sources	Regional	3.73E-08	4.26E-08	5.92E-05	6.12E-05	0.00198	0.00263	9.43E-04	0.00108	5.92E-05	6.12E-05

Part 2: Acidic Environment

Endpoint	Estimated concentration in fish (mg/kg wet weight)		Estimated concentration in grass (mg/kg wet wt)		Estimated concentration in leaf crops (mg/kg wet wt.)		Estimated concentrations in root crops (mg/kg wet wt.)		Estimated concentrations in meat (mg/kg wet wt.)		Estimated concentration in milk (mg/kg wet wt)	
	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper
Pigment manufacturing site A	1.02E+00	1.02E+00	0.000314	0.00034	0.000254	0.000282	0.000381	0.000423	8.16E-04	8.55E-04	1.22E-05	1.28E-05
Pigment manufacturing site B	1.84E-01	1.90E-01	0.00037	0.000396	0.000284	0.00031	0.000426	0.000465	5.88E-04	6.26E-04	8.82E-06	9.39E-06
Pigment manufacturing site C	4.26E-01	4.32E-01	0.000886	0.000914	0.000556	0.000582	0.000834	0.000873	1.41E-03	1.45E-03	2.12E-05	2.18E-05
Pigment manufacturing site D	2.32E-01	2.38E-01	0.000766	0.000794	0.000492	0.000518	0.000738	0.000777	1.17E-03	1.21E-03	1.75E-05	1.81E-05
Pigment manufacturing site E	1.79E-01	1.84E-01	0.00021	0.000238	0.0002	0.000228	0.0003	0.000342	3.60E-04	4.01E-04	5.39E-06	6.01E-06
Stabiliser manufacturing site F	1.78E-01	1.84E-01	0.0001896	0.000216	0.000189	0.000216	0.0002835	0.000324	3.30E-04	3.69E-04	4.95E-06	5.54E-06
Stabiliser manufacturing siteG	1.80E-01	1.86E-01	0.0001984	0.000226	0.0001944	0.000222	0.0002916	0.000333	3.44E-04	3.84E-04	5.15E-06	5.76E-06
Stabiliser manufacturing site H	1.80E-01	1.86E-01	0.0001946	0.000222	0.0001916	0.000218	0.0002874	0.000327	3.38E-04	3.79E-04	5.07E-06	5.68E-06
Stabiliser manufacturing site I	1.78E-01	1.84E-01	0.0001898	0.000216	0.0001892	0.000216	0.0002838	0.000324	3.31E-04	3.70E-04	4.96E-06	5.54E-06
Stabiliser manufacturing site J	1.78E-01	1.84E-01	0.000197	0.000224	0.0001928	0.00022	0.0002892	0.00033	3.41E-04	3.81E-04	5.11E-06	5.71E-06
Stabiliser manufacturing site K	1.94E-01	2.00E-01	0.0002	0.000228	0.000218	0.000246	0.000327	0.000369	3.51E-04	3.92E-04	5.26E-06	5.89E-06
Artists colours formulation	1.79E-01	1.85E-01	0.0001886	0.000216	0.0001886	0.000216	0.0002829	0.000324	3.29E-04	3.70E-04	4.94E-06	5.55E-06
Cadmium plating	7.68E-02	7.71E-02	0.0001902	0.000218	0.000193	0.00022	0.0002895	0.00033	5.46E-04	5.88E-04	8.20E-06	8.81E-06
Plated metal use	5.22E+01	5.22E+01	0.0001884	0.000216	0.0001884	0.000216	0.0002826	0.000324	1.94E-02	1.94E-02	2.91E-04	2.92E-04
Waste incineration	1.78E-01	1.84E-01	0.0904	0.1036	0.0476	0.0546	0.0714	0.0819	1.33E-01	1.52E-01	1.99E-03	2.29E-03
Sewage sludge incineration	1.78E-01	1.84E-01	0.000286	0.000314	0.00024	0.000268	0.00036	0.000402	4.65E-04	5.06E-04	6.98E-06	7.60E-06
Regional sources	1.78E-01	1.84E-01	0.0001886	0.000216	0.000396	0.000526	0.000594	0.000789	3.29E-04	3.69E-04	4.93E-06	5.54E-06

Part 2: Acidic Environment

Endpoint	Estimated daily human intake from drinking water (mg/kg bw/day)		Estimated daily human intake from fish (mg/kg bw/day)		Estimated daily human intake from leaf crops (mg/kg bw/day)		Estimated daily human intake from root crops (mg/kg bw/day)		Estimated daily human intake from meat (mg/kg bw day)		Estimated daily human intake from milk (mg/kg bw/day)	
	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper
Pigment manufacturing site A	9.74E-06	9.80E-06	1.67E-03	1.68E-03	4.35E-06	4.83E-06	2.09E-06	2.32E-06	3.51E-06	3.68E-06	9.81E-08	1.03E-07
Pigment manufacturing site B	1.75E-06	1.80E-06	3.02E-04	3.11E-04	4.87E-06	5.31E-06	2.34E-06	2.55E-06	2.53E-06	2.69E-06	7.07E-08	7.53E-08
Pigment manufacturing site C	4.17E-06	4.23E-06	7.00E-04	7.10E-04	9.53E-06	9.98E-06	4.58E-06	4.79E-06	6.07E-06	6.25E-06	1.70E-07	1.75E-07
Pigment manufacturing site D	2.20E-06	2.26E-06	3.80E-04	3.90E-04	8.43E-06	8.88E-06	4.05E-06	4.26E-06	5.01E-06	5.19E-06	1.40E-07	1.45E-07
Pigment manufacturing site E	1.70E-06	1.76E-06	2.94E-04	3.02E-04	3.43E-06	3.91E-06	1.65E-06	1.88E-06	1.55E-06	1.72E-06	4.32E-08	4.82E-08
Stabiliser manufacturing site F	1.69E-06	1.75E-06	2.92E-04	3.02E-04	3.24E-06	3.70E-06	1.56E-06	1.78E-06	1.42E-06	1.59E-06	3.97E-08	4.44E-08
Stabiliser manufacturing siteG	1.71E-06	1.77E-06	2.95E-04	3.05E-04	3.33E-06	3.81E-06	1.60E-06	1.83E-06	1.48E-06	1.65E-06	4.13E-08	4.62E-08
Stabiliser manufacturing site H	1.71E-06	1.77E-06	2.96E-04	3.06E-04	3.28E-06	3.74E-06	1.58E-06	1.79E-06	1.45E-06	1.63E-06	4.07E-08	4.55E-08
Stabiliser manufacturing site I	1.69E-06	1.75E-06	2.92E-04	3.02E-04	3.24E-06	3.70E-06	1.56E-06	1.78E-06	1.42E-06	1.59E-06	3.98E-08	4.44E-08
Stabiliser manufacturing site J	1.69E-06	1.75E-06	2.92E-04	3.02E-04	3.31E-06	3.77E-06	1.59E-06	1.81E-06	1.47E-06	1.64E-06	4.10E-08	4.58E-08
Stabiliser manufacturing site K	1.85E-06	1.91E-06	3.19E-04	3.29E-04	3.74E-06	4.22E-06	1.79E-06	2.02E-06	1.51E-06	1.69E-06	4.22E-08	4.72E-08
Artists colours formulation	1.71E-06	1.76E-06	2.94E-04	3.04E-04	3.23E-06	3.70E-06	1.55E-06	1.78E-06	1.42E-06	1.59E-06	3.96E-08	4.45E-08
Cadmium plating	7.29E-06	7.34E-06	1.26E-04	1.27E-04	3.31E-06	3.77E-06	1.59E-06	1.81E-06	2.35E-06	2.53E-06	6.57E-08	7.06E-08
Plated metal use	4.97E-04	4.97E-04	8.58E-02	8.58E-02	3.23E-06	3.70E-06	1.55E-06	1.78E-06	8.34E-05	8.36E-05	2.33E-06	2.34E-06
Waste incineration	1.30E-04	1.49E-04	2.92E-04	3.02E-04	8.16E-04	9.36E-04	3.92E-04	4.49E-04	5.72E-04	6.55E-04	1.60E-05	1.83E-05
Sewage sludge incineration	1.69E-06	1.75E-06	2.92E-04	3.02E-04	4.11E-06	4.59E-06	1.97E-06	2.21E-06	2.00E-06	2.18E-06	5.59E-08	6.09E-08
Regional sources	1.69E-06	1.75E-06	2.92E-04	3.02E-04	6.79E-06	9.02E-06	3.26E-06	4.33E-06	1.41E-06	1.59E-06	3.95E-08	4.44E-08

Part 2: Acidic Environment

Endpoint	Estimated daily human intake from air (mg/kg bw day)		Total daily human intake from environmental routes (mg/kg bw day)	
	Lower	Upper	Lower	Upper
Pigment manufacturing site A	1.96E-07	1.97E-07	1.69E-03	1.70E-03
Pigment manufacturing site B	2.81E-07	2.81E-07	3.13E-04	3.24E-04
Pigment manufacturing site C	1.05E-06	1.05E-06	7.25E-04	7.36E-04
Pigment manufacturing site D	8.72E-07	8.72E-07	4.01E-04	4.12E-04
Pigment manufacturing site E	4.11E-08	4.24E-08	3.02E-04	3.11E-04
Stabiliser manufacturing site F	9.64E-09	1.07E-08	3.00E-04	3.11E-04
Stabiliser manufacturing siteG	2.36E-08	2.36E-08	3.03E-04	3.14E-04
Stabiliser manufacturing site H	1.69E-08	1.82E-08	3.04E-04	3.15E-04
Stabiliser manufacturing site I	9.86E-09	1.09E-08	3.00E-04	3.11E-04
Stabiliser manufacturing site J	2.06E-08	2.14E-08	3.00E-04	3.11E-04
Stabiliser manufacturing site K	8.79E-09	9.86E-09	3.28E-04	3.39E-04
Artists colours formulation	7.99E-09	9.13E-09	3.02E-04	3.12E-04
Cadmium plating	7.99E-09	9.13E-09	1.41E-04	1.42E-04
Plated metal use	7.99E-09	9.13E-09	8.63E-02	8.63E-02
Waste incineration	1.35E-04	1.55E-04	2.35E-03	2.66E-03
Sewage sludge incineration	1.55E-08	1.56E-08	3.02E-04	3.12E-04
Regional sources	7.99E-09	9.13E-09	3.05E-04	3.18E-04

Part 2: Acidic Environment

Accumulation/transfer factors			Value	Unit	Comment	
Fish BCF			3000	l/kg		
Soil-plant transfer factor for root crops			0.3	kg/kg	on a fresh weight plant basis	
Soil-plant transfer factor for leaf crops and grass			0.2	kg/kg	on a fresh weight plant basis	
Accumulation factor for meat from diet			0.02	d/kg		
Accumulation factor for milk from diet			0.0003	d/kg		

Daily intake figures for humans			
	Human intake		
Drinking water	2	l/day	
Fish	0.115	kg/day	wet weight
Leaf crops	1.2	kg/day	wet weight
Root crops	0.384	kg/day	wet weight
Meat	0.301	kg/day	wet weight
Diary products (milk)	0.561	kg/day	wet weight
Air	20	m^3/day	
Bioavailability inhalation	0.75		TGD default
Bioavailability oral route	1		TGD default
Adult body weight	70	kg	

Daily intake figures for cattle		
	Intake (on a wet weight basis)	
Grass	67.6	kg/day
Soil	0.46	kg/day
Air	122	m^3/day
Drinking water	55	l/day

ANNEX 5

EUSES RESULTS

This Annex contains an example summary EUSES printout for one of the scenarios considered in the main report.

As explained in the main text, the available plant uptake data for cadmium is in a form that cannot be readily included in the EUSES model and so the calculations for the concentrations in the food chain for human consumption are contained in Annex 4. These have been carried out using the same assumptions as included in the TGD and should replace the values presented in the EUSES printout for Humans Exposed Via the Environment (due to the input data available for cadmium, EUSES calculates the correct concentrations for intake through drinking water, fish and air only).

EUSES Summary report | Single substance

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Explanation status column: 'O' = Output; 'D' = Default; 'S' = Set; 'I' = Imported

Name	Reference	Value	Units	Status
IDENTIFICATION OF THE SUBSTANCE				
General name	Cadmium	Cadmium		S
CAS-No				D
EC-notification no.				D
EINECS no.				D
Molecular weight	112.41	112.41	[g.mol-1]	S

EUSES Summary report | Single substance

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
PHYSICO-CHEMICAL PROPERTIES				
Melting point	320.9	320.9	[oC]	S
Boiling point	765	765	[oC]	S
Vapour pressure at 25 [oC]	1E-06	1E-06	[Pa]	S
Water solubility	9.6	9.6	[mg.l-1]	S
Octanol-water partition coefficient.	-1	-1	[log10]	S
Henry's law constant	1.17094E-05	1E-09	[Pa.m3.mol-1]	S

EUSES Summary report | Single substance

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION				
Tonnage of substance in Europe	0	5.5E+03	[tonnes.yr-1]	S
Regional production volume of substance	0	550	[tonnes.yr-1]	S
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [USE PATTERN 1]				
Industry category	11 Polymers industry	11 Polymers industry		S
Use category	10 Colouring agents	10 Colouring agents		S
Extra details on use category	Polymer processing	Polymer processing		S
Extra details on use category	Thermoplastics: additives, pigments, fillers	Thermoplastics: additives, pigments, fillers		S
Fraction of tonnage for application	0.111	0.111	[-]	O
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [FORMULATION]				
Main category formulation	III Multi-purpose equipment	III Multi-purpose equipment		D
Fraction of tonnage released to air	2.5E-03	0	[-]	S
Fraction of tonnage released to waste water	0.02	0	[-]	S
Fraction of tonnage released to industrial soil	1E-04	0	[-]	S
Fraction of the main local source	1	1	[-]	O
Number of emission days per year	300	230	[-]	S
Local emission to air during episode	0	5E-03	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [PROCESSING]				
Main category processing	III Non-dispersive use	III Non-dispersive use		D
Fraction of tonnage released to air	5E-04	0	[-]	S
Fraction of tonnage released to waste water	5E-04	0	[-]	S
Fraction of tonnage released to industrial soil	1E-04	0	[-]	S
Fraction of the main local source	0.5	0.25	[-]	O
Number of emission days per year	1	231	[-]	S
Local emission to air during episode	0	7.186E-03	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [PRIVATE USE]				
Fraction of tonnage released to air	0	0	[-]	O
Fraction of tonnage released to waste water	0	0	[-]	O
Fraction of tonnage released to industrial soil	0	0	[-]	O
Fraction of the main local source	0	0	[-]	O
Number of emission days per year	1	276	[-]	S

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE (Continued) RELEASE ESTIMATION [PRIVATE USE]				
Local emission to air during episode	0	0.0232	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [RECOVERY]				
Fraction of tonnage released to air	0	0	[-]	O
Fraction of tonnage released to waste water	0	0	[-]	O
Fraction of tonnage released to industrial soil	0	0	[-]	O
Fraction of the main local source	0	0	[-]	O
Number of emission days per year	1	230	[-]	S
Local emission to air during episode	0	0.02304	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [USE PATTERN 2]				
Industry category	11 Polymers industry	11 Polymers industry		S
Use category	10 Colouring agents	10 Colouring agents		S
Extra details on use category	Polymer processing	Polymer processing		S
Extra details on use category	Thermoplastics: additives, pigments, fillers	Thermoplastics: additives, pigments, fillers		D
Fraction of tonnage for application	0.111	0.111	[-]	O
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [FORMULATION]				
Main category formulation	III Multi-purpose equipment	III Multi-purpose equipment		D
Fraction of tonnage released to air	2.5E-03	0	[-]	S
Fraction of tonnage released to waste water	0.02	0	[-]	S
Fraction of tonnage released to industrial soil	1E-04	0	[-]	S
Fraction of the main local source	1	1	[-]	O
Number of emission days per year	300	145	[-]	S
Local emission to air during episode	0	1.4E-03	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [USE PATTERN 3]				
Industry category	11 Polymers industry	11 Polymers industry		S
Use category	49 Stabilizers	49 Stabilizers		S
Extra details on use category	Polymer processing	Polymer processing		S

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE (Continued) RELEASE ESTIMATION [USE PATTERN 3]				
Extra details on use category	Thermoplastics: additives, pigments, fillers	Thermoplastics: additives, pigments, fillers		D
Fraction of tonnage for application	0.111	0.111	[-]	O
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [FORMULATION]				
Main category formulation	III Multi-purpose equipment	III Multi-purpose equipment		D
Fraction of tonnage released to air	2.5E-03	0	[-]	S
Fraction of tonnage released to waste water	0.02	0	[-]	S
Fraction of tonnage released to industrial soil	1E-04	0	[-]	S
Fraction of the main local source	1	1	[-]	O
Number of emission days per year	300	2.22	[-]	S
Local emission to air during episode	0	4.5E-03	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [PROCESSING]				
Main category processing	III Non-dispersive use	III Non-dispersive use		D
Fraction of tonnage released to air	5E-04	0	[-]	S
Fraction of tonnage released to waste water	5E-04	0	[-]	S
Fraction of tonnage released to industrial soil	1E-04	0	[-]	S
Fraction of the main local source	0.5	0.25	[-]	O
Number of emission days per year	1	5.33	[-]	S
Local emission to air during episode	0	0.01667	[kg.d-1]	S
Local emission to wastewater during episode	0	0.0104	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [PRIVATE USE]				
Fraction of tonnage released to air	0	0	[-]	O
Fraction of tonnage released to waste water	0	0	[-]	O
Fraction of tonnage released to industrial soil	0	0	[-]	O
Fraction of the main local source	0	0	[-]	O
Number of emission days per year	1	6.67	[-]	S
Local emission to air during episode	0	8.333E-03	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [RECOVERY]				
Fraction of tonnage released to air	0	0	[-]	O

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE (Continued)				
RELEASE ESTIMATION				
[RECOVERY]				
Fraction of tonnage released to waste water	0	0	[-]	O
Fraction of tonnage released to industrial soil	0	0	[-]	O
Fraction of the main local source	0	0	[-]	O
Number of emission days per year	1	5	[-]	S
Local emission to air during episode	0	2.22E-03	[kg.d-1]	S
Local emission to wastewater during episode	0	2.22E-03	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE				
RELEASE ESTIMATION				
[USE PATTERN 4]				
Industry category	11 Polymers industry	11 Polymers industry		S
Use category	49 Stabilizers	49 Stabilizers		S
Extra details on use category	Polymer processing	Polymer processing		S
Extra details on use category	Thermoplastics: additives, pigments, fillers	Thermoplastics: additives, pigments, fillers		S
Fraction of tonnage for application	0.111	0.111	[-]	O
ENVIRONMENT-EXPOSURE				
RELEASE ESTIMATION				
[FORMULATION]				
Main category formulation	III Multi-purpose equipment	III Multi-purpose equipment		D
Fraction of tonnage released to air	2.5E-03	0	[-]	S
Fraction of tonnage released to waste water	0.02	0	[-]	S
Fraction of tonnage released to industrial soil	1E-04	0	[-]	S
Fraction of the main local source	1	1	[-]	O
Number of emission days per year	300	1.44	[-]	S
Local emission to air during episode	0	0.05385	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE				
RELEASE ESTIMATION				
[PROCESSING]				
Main category processing	III Non-dispersive use	III Non-dispersive use		D
Fraction of tonnage released to air	5E-04	0	[-]	S
Fraction of tonnage released to waste water	5E-04	0	[-]	S
Fraction of tonnage released to industrial soil	1E-04	0	[-]	S
Fraction of the main local source	0.5	0.25	[-]	O
Number of emission days per year	1	1.33	[-]	S
Local emission to air during episode	0	3.333E-03	[kg.d-1]	S
Local emission to wastewater during episode	0	0.342	[kg.d-1]	S
Intermittent release	No	No		D

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [USE PATTERN 5]				
Industry category	6 Public domain	6 Public domain		S
Use category	10 Colouring agents	10 Colouring agents		S
Extra details on use category	No extra details necessary	No extra details necessary		S
Extra details on use category	No extra details necessary	No extra details necessary		D
Fraction of tonnage for application	0.111	0.111	[-]	O
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [FORMULATION]				
Main category formulation	III Multi-purpose equipment	III Multi-purpose equipment		D
Fraction of tonnage released to air	2.5E-03	0	[-]	S
Fraction of tonnage released to waste water	0.02	0	[-]	S
Fraction of tonnage released to industrial soil	1E-04	0	[-]	S
Fraction of the main local source	1	1	[-]	O
Number of emission days per year	300	20	[-]	S
Local emission to air during episode	0	0	[kg.d-1]	S
Local emission to wastewater during episode	0	1.85E-03	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [USE PATTERN 6]				
Industry category	16 Engineering industry, civil and mechanical	16 Engineering industry, civil and mechanical		S
Use category	55/0 Others	55/0 Others		D
Extra details on use category	No extra details necessary	No extra details necessary		D
Extra details on use category	No extra details necessary	No extra details necessary		D
Fraction of tonnage for application	0.111	0.111	[-]	O
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [PROCESSING]				
Main category processing	III Non-dispersive use	III Non-dispersive use		D
Fraction of tonnage released to air	1E-03	0	[-]	S
Fraction of tonnage released to waste water	0.1	0	[-]	S
Fraction of tonnage released to industrial soil	1E-02	0	[-]	S
Fraction of the main local source	1	0.8	[-]	O
Number of emission days per year	1	300	[-]	S
Local emission to air during episode	0	0	[kg.d-1]	S
Local emission to wastewater during episode	0	0.0533	[kg.d-1]	S
Intermittent release	No	No		D

EUSES Summary report | Single substance

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [USE PATTERN 7]				
Industry category	16 Engineering industry, civil and mechanical	16 Engineering industry, civil and mechanical		S
Use category	55/0 Others	55/0 Others		S
Extra details on use category	No extra details necessary	No extra details necessary		D
Extra details on use category	No extra details necessary	No extra details necessary		D
Fraction of tonnage for application	0.111	0.111	[-]	O
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [PROCESSING]				
Main category processing	III Non-dispersive use	III Non-dispersive use		D
Fraction of tonnage released to air	1E-03	0	[-]	S
Fraction of tonnage released to waste water	0.1	0	[-]	S
Fraction of tonnage released to industrial soil	1E-02	0	[-]	S
Fraction of the main local source	1	0.8	[-]	O
Number of emission days per year	1	365	[-]	S
Local emission to air during episode	0	0	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [USE PATTERN 8]				
Industry category	15/0 Others	15/0 Others		D
Use category	55/0 Others	55/0 Others		D
Extra details on use category	No extra details necessary	No extra details necessary		D
Extra details on use category	No extra details necessary	No extra details necessary		D
Fraction of tonnage for application	0.111	0.111	[-]	O
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [RECOVERY]				
Fraction of tonnage released to air	0	0	[-]	O
Fraction of tonnage released to waste water	0	0	[-]	O
Fraction of tonnage released to industrial soil	0	0	[-]	O
Fraction of the main local source	0.5	0.5	[-]	O
Number of emission days per year	150	365	[-]	S
Local emission to air during episode	0	2.6	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D

EUSES Summary report | Single substance

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [USE PATTERN 9]				
Industry category	15/0 Others	15/0 Others		D
Use category	55/0 Others	55/0 Others		D
Extra details on use category	No extra details necessary	No extra details necessary		D
Extra details on use category	No extra details necessary	No extra details necessary		D
Fraction of tonnage for application	0.111	0.111	[-]	O
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [FORMULATION]				
Main category formulation	III Multi-purpose equipment	III Multi-purpose equipment		D
Fraction of tonnage released to air	2.5E-03	0	[-]	S
Fraction of tonnage released to waste water	0.02	0	[-]	S
Fraction of tonnage released to industrial soil	1E-04	0	[-]	S
Fraction of the main local source	1	1	[-]	O
Number of emission days per year	300	300	[-]	O
Local emission to air during episode	0	0	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [PROCESSING]				
Main category processing	III Non-dispersive use	III Non-dispersive use		D
Fraction of tonnage released to air	1E-03	0	[-]	S
Fraction of tonnage released to waste water	0.1	0	[-]	S
Fraction of tonnage released to industrial soil	1E-02	0	[-]	S
Fraction of the main local source	1	0.8	[-]	O
Number of emission days per year	1	20	[-]	O
Local emission to air during episode	0	0	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	S
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE RELEASE ESTIMATION [PRIVATE USE]				
Fraction of tonnage released to air	0	0	[-]	O
Fraction of tonnage released to waste water	0	0	[-]	O
Fraction of tonnage released to industrial soil	0	0	[-]	O
Fraction of the main local source	0	4E-08	[-]	O
Number of emission days per year	1	200	[-]	O
Local emission to air during episode	0	0	[kg.d-1]	O
Local emission to wastewater during episode	0	0	[kg.d-1]	O
Intermittent release	No	No		D

EUSES Summary report | Single substance

Printed on: 20/09/00 14:10:13
Study: Gen. Env. - Scen. A - No sludge In.
Substance: Cadmium
Defaults: Standard
Assessment types: 1A, 1B, 2, 3A, 3B
Base set complete: No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE				
RELEASE ESTIMATION				
[RECOVERY]				
Fraction of tonnage released to air	0	0	[-]	O
Fraction of tonnage released to waste water	0	0	[-]	O
Fraction of tonnage released to industrial soil	0	0	[-]	O
Fraction of the main local source	0.5	0.5	[-]	O
Number of emission days per year	150	365	[-]	S
Local emission to air during episode	0	2.47E-03	[kg.d-1]	S
Local emission to wastewater during episode	0	0	[kg.d-1]	O
Intermittent release	No	No		D
ENVIRONMENT-EXPOSURE				
RELEASE ESTIMATION				
TOTAL REGIONAL EMISSIONS TO COMPARTMENTS				
Total regional emission to air	0	956.18	[kg.yr-1]	S
Total regional emission to wastewater	0	128.14	[kg.yr-1]	S
Total regional emission to industrial soil	0	4.64535E+03	[kg.yr-1]	S
Total regional emission to surface water	0	203.22	[kg.yr-1]	S
ENVIRONMENT-EXPOSURE				
RELEASE ESTIMATION				
TOTAL CONTINENTAL EMISSIONS TO COMPARTMENTS				
Total continental emission to air	0	8.55655E+03	[kg.yr-1]	S
Total continental emission to wastewater	0	1.15043E+03	[kg.yr-1]	S
Total continental emission to industrial soil	0	4.18081E+04	[kg.yr-1]	S
Total continental emission to surface water	0	1.80523E+03	[kg.yr-1]	S
ENVIRONMENT-EXPOSURE				
PARTITION COEFFICIENTS				
SOLIDS-WATER PARTITION COEFFICIENTS				
Solids-water partition coefficient in soil	3.90302E-03	3.029E+03	[l.kg-1]	S
Solids-water partition coefficient in sediment	9.75754E-03	8.51E+04	[l.kg-1]	S
Solids-water partition coefficient suspended matter	0.0195151	1.3E+05	[l.kg-1]	S
ENVIRONMENT-EXPOSURE				
PARTITION COEFFICIENTS				
BIOTA-WATER PARTITION COEFFICIENTS				
Bioconcentration factor for aquatic biota	1.41254	38	[l.kg-1]	S
ENVIRONMENT-EXPOSURE				
PARTITION COEFFICIENTS				
Fraction of chemical associated with aerosol particles	0.0598478	1	[-]	S
ENVIRONMENT-EXPOSURE				
DEGRADATION AND TRANSFORMATION				
Characterization of biodegradability	Not biodegradable	Not biodegradable		D
Degradation calculation method in STP	First order, standard OECD/EU tests	First order, standard OECD/EU tests		D
Rate constant for biodegradation in STP	0	0	[d-1]	O

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE (Continued)				
DEGRADATION AND TRANSFORMATION				
Rate constant for biodegradation in surface water	0	0	[d-1]	O
Rate constant for biodegradation in bulk soil	6.93E-07	6.93E-07	[d-1]	O
Rate constant for biodegradation in aerated sediment	6.93E-07	6.93E-07	[d-1]	O
Rate constant for hydrolysis in surface water	6.93E-07	6.93E-07	[d-1]	O
Rate constant for photolysis in surface water	6.93E-07	6.93E-07	[d-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 1][FORMULATION]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	0	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	0.0229	[mg.l-1]	S
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	S
PEC for micro-organisms in the STP	0	0.0229	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 1][PROCESSING]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	0	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	3.4E-03	[mg.l-1]	S
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	S
PEC for micro-organisms in the STP	0	3.4E-03	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 1][PRIVATE USE]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	0	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	0.087	[mg.l-1]	S
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	0.087	[mg.l-1]	O

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Study | Gen. Env. - Scen. A - No sludge In.
Substance | Cadmium
Defaults | Standard
Assessment types | 1A, 1B, 2, 3A, 3B
Base set complete | No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 1][RECOVERY]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	0	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	8E-03	[mg.l-1]	S
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	8E-03	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 2][FORMULATION]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	0	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	0.0435	[mg.l-1]	S
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	S
PEC for micro-organisms in the STP	0	0.0435	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 3][FORMULATION]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	0	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	1.01E-03	[mg.l-1]	S
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	1.01E-03	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 3][PROCESSING]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	5.2E-03	[mg.l-1]	O

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE (Continued)				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 3][PROCESSING]				
OUTPUT				
Concentration of chemical (total) in the STP-effluent	0	5.2E-04	[mg.l-1]	O
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	3.22E-04	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	5.2E-04	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 3][PRIVATE USE]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	0	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	5.06E-03	[mg.l-1]	S
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	5.06E-03	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 3][RECOVERY]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	1.11E-03	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	1.11E-04	[mg.l-1]	O
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	6.86E-05	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	1.11E-04	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 4][FORMULATION]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	0	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	0	[mg.l-1]	O
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	0	[mg.l-1]	O

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Study | Gen. Env. - Scen. A - No sludge In.
Substance | Cadmium
Defaults | Standard
Assessment types | 1A, 1B, 2, 3A, 3B
Base set complete | No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 4][PROCESSING]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	0.171	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	0.0171	[mg.l-1]	O
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0.0106	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	0.0171	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 5][FORMULATION]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	9.25E-04	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	9.25E-05	[mg.l-1]	O
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	5.72E-05	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	9.25E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 6][PROCESSING]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	O
Concentration in untreated wastewater	0	0.0267	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	2.67E-03	[mg.l-1]	O
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	1.65E-03	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	2.67E-03	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 7][PROCESSING]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	S
Concentration in untreated wastewater	0	0	[mg.l-1]	O

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Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE (Continued)				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 7][PROCESSING]				
OUTPUT				
Concentration of chemical (total) in the STP-effluent	0	0.195	[mg.l-1]	S
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	0.195	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 8][RECOVERY]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	O
Concentration in untreated wastewater	0	0	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	0	[mg.l-1]	O
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	0	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
LOCAL STP[USE PATTERN 9][RECOVERY]				
OUTPUT				
Fraction of emission directed to air by STP	0	0	[-]	S
Fraction of emission directed to water by STP	0	0.1	[-]	S
Fraction of emission directed to sludge by STP	0	0.9	[-]	S
Fraction of the emission degraded in STP	0	0	[-]	O
Concentration in untreated wastewater	0	0	[mg.l-1]	O
Concentration of chemical (total) in the STP-effluent	0	0	[mg.l-1]	O
Concentration in effluent exceeds solubility	No	No		O
Concentration in dry sewage sludge	0	0	[mg.kg-1]	O
PEC for micro-organisms in the STP	0	0	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
REGIONAL STP				
Fraction of emission directed to air	0	0	[-]	S
Fraction of emission directed to water	0	0.1	[-]	S
Fraction of emission directed to sludge	0	0.9	[-]	S
Fraction of the emission degraded	0	0	[-]	S
ENVIRONMENT-EXPOSURE				
SEWAGE TREATMENT				
CONTINENTAL STP				
Fraction of emission directed to air	0	0	[-]	S
Fraction of emission directed to water	0	0.1	[-]	S
Fraction of emission directed to sludge	0	0.9	[-]	S

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Study | Gen. Env. - Scen. A - No sludge In.
Substance | Cadmium
Defaults | Standard
Assessment types | 1A, 1B, 2, 3A, 3B
Base set complete | No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE (Continued)				
SEWAGE TREATMENT				
CONTINENTAL STP				
Fraction of the emission degraded	0	0	[-]	S
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 1][FORMULATION]				
Concentration in air during emission episode	0	1.39E-06	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	8.76E-07	[mg.m-3]	O
Concentration in surface water during emission episode	0	1.69E-04	[mg.l-1]	O
Annual average concentration in surface water	0	1.07E-04	[mg.l-1]	O
Local PEC in surface water during emission episode	0	1.79E-04	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	1.16E-04	[mg.l-1]	O
Local PEC in sediment during emission episode	0	5.06	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0362	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0362	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0366	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.36E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 1][PROCESSING]				
Concentration in air during emission episode	0	2E-06	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	1.26E-06	[mg.m-3]	O
Concentration in surface water during emission episode	0	1.17E-06	[mg.l-1]	O
Annual average concentration in surface water	0	7.39E-07	[mg.l-1]	O
Local PEC in surface water during emission episode	0	1.07E-05	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	1.02E-05	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.301	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0364	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0364	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0369	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.36E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 1][PRIVATE USE]				
Concentration in air during emission episode	0	6.45E-06	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	4.88E-06	[mg.m-3]	O
Concentration in surface water during emission episode	0	4.35E-05	[mg.l-1]	O
Annual average concentration in surface water	0	3.29E-05	[mg.l-1]	O
Local PEC in surface water during emission episode	0	5.3E-05	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	4.24E-05	[mg.l-1]	O
Local PEC in sediment during emission episode	0	1.5	[mg.kgwwt-1]	O

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE (Continued)				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 1][PRIVATE USE]				
Local PEC in agric. soil (total) averaged over 30 days	0	0.0378	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0378	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0397	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.41E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 1][RECOVERY]				
Concentration in air during emission episode	0	6.41E-06	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	4.04E-06	[mg.m-3]	O
Concentration in surface water during emission episode	0	1.08E-05	[mg.l-1]	O
Annual average concentration in surface water	0	6.78E-06	[mg.l-1]	O
Local PEC in surface water during emission episode	0	2.02E-05	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	1.63E-05	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.572	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0374	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0375	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0391	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.4E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 2][FORMULATION]				
Concentration in air during emission episode	0	3.89E-07	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	1.55E-07	[mg.m-3]	O
Concentration in surface water during emission episode	0	3.48E-07	[mg.l-1]	O
Annual average concentration in surface water	0	1.38E-07	[mg.l-1]	O
Local PEC in surface water during emission episode	0	9.83E-06	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	9.62E-06	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.278	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.036	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.35E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 3][FORMULATION]				
Concentration in air during emission episode	0	1.25E-06	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	7.61E-09	[mg.m-3]	O
Concentration in surface water during emission episode	0	7.03E-07	[mg.l-1]	O

Printed on 20/09/00 14:10:13
Study Gen. Env. - Scen. A - No sludge In.
Substance Cadmium
Defaults Standard
Assessment types 1A, 1B, 2, 3A, 3B
Base set complete No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE (Continued)				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 3][FORMULATION]				
Annual average concentration in surface water	0	4.28E-09	[mg.l-1]	O
Local PEC in surface water during emission episode	0	1.02E-05	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	9.49E-06	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.288	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.34E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 3][PROCESSING]				
Concentration in air during emission episode	0	4.63E-06	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	6.77E-08	[mg.m-3]	O
Concentration in surface water during emission episode	0	1.76E-05	[mg.l-1]	O
Annual average concentration in surface water	0	2.57E-07	[mg.l-1]	O
Local PEC in surface water during emission episode	0	2.71E-05	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	9.74E-06	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.766	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.34E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 3][PRIVATE USE]				
Concentration in air during emission episode	0	2.32E-06	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	4.23E-08	[mg.m-3]	O
Concentration in surface water during emission episode	0	1.7E-05	[mg.l-1]	O
Annual average concentration in surface water	0	3.1E-07	[mg.l-1]	O
Local PEC in surface water during emission episode	0	2.65E-05	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	9.79E-06	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.748	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.34E-05	[mg.l-1]	O

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 3][RECOVERY]				
Concentration in air during emission episode	0	6.17E-07	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	8.45E-09	[mg.m-3]	O
Concentration in surface water during emission episode	0	3.76E-06	[mg.l-1]	O
Annual average concentration in surface water	0	5.15E-08	[mg.l-1]	O
Local PEC in surface water during emission episode	0	1.32E-05	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	9.54E-06	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.374	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.34E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 4][FORMULATION]				
Concentration in air during emission episode	0	1.5E-05	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	5.91E-08	[mg.m-3]	O
Concentration in surface water during emission episode	0	0	[mg.l-1]	O
Annual average concentration in surface water	0	0	[mg.l-1]	O
Local PEC in surface water during emission episode	0	9.48E-06	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	9.48E-06	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.268	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.34E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 4][PROCESSING]				
Concentration in air during emission episode	0	9.27E-07	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	3.38E-09	[mg.m-3]	O
Concentration in surface water during emission episode	0	5.8E-04	[mg.l-1]	O
Annual average concentration in surface water	0	2.11E-06	[mg.l-1]	O
Local PEC in surface water during emission episode	0	5.89E-04	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	1.16E-05	[mg.l-1]	O
Local PEC in sediment during emission episode	0	16.7	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.036	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.036	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.036	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.35E-05	[mg.l-1]	O

Printed on 20/09/00 14:10:13
Study Gen. Env. - Scen. A - No sludge In.
Substance Cadmium
Defaults Standard
Assessment types 1A, 1B, 2, 3A, 3B
Base set complete No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 5][FORMULATION]				
Concentration in air during emission episode	0	0	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	0	[mg.m-3]	O
Concentration in surface water during emission episode	0	3.14E-06	[mg.l-1]	O
Annual average concentration in surface water	0	1.72E-07	[mg.l-1]	O
Local PEC in surface water during emission episode	0	1.26E-05	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	9.66E-06	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.357	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.34E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 6][PROCESSING]				
Concentration in air during emission episode	0	0	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	0	[mg.m-3]	O
Concentration in surface water during emission episode	0	9.03E-05	[mg.l-1]	O
Annual average concentration in surface water	0	7.43E-05	[mg.l-1]	O
Local PEC in surface water during emission episode	0	9.98E-05	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	8.37E-05	[mg.l-1]	O
Local PEC in sediment during emission episode	0	2.82	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.34E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 7][PROCESSING]				
Concentration in air during emission episode	0	0	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	0	[mg.m-3]	O
Concentration in surface water during emission episode	0	6.61E-03	[mg.l-1]	O
Annual average concentration in surface water	0	6.61E-03	[mg.l-1]	O
Local PEC in surface water during emission episode	0	6.62E-03	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	6.62E-03	[mg.l-1]	O
Local PEC in sediment during emission episode	0	187	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0359	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.34E-05	[mg.l-1]	O

EUSES Summary report | Single substance

Printed on: 20/09/00 14:10:13
Study: Gen. Env. - Scen. A - No sludge In.
Substance: Cadmium
Defaults: Standard
Assessment types: 1A, 1B, 2, 3A, 3B
Base set complete: No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 8][RECOVERY]				
Concentration in air during emission episode	0	7.23E-04	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	7.23E-04	[mg.m-3]	O
Concentration in surface water during emission episode	0	0	[mg.l-1]	O
Annual average concentration in surface water	0	0	[mg.l-1]	O
Local PEC in surface water during emission episode	0	9.48E-06	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	9.48E-06	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.268	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.315	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.321	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.606	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.2E-04	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
LOCAL SCALE				
[USE PATTERN 9][RECOVERY]				
Concentration in air during emission episode	0	6.87E-07	[mg.m-3]	O
Annual average concentration in air, 100 m from point source	0	6.87E-07	[mg.m-3]	O
Concentration in surface water during emission episode	0	0	[mg.l-1]	O
Annual average concentration in surface water	0	0	[mg.l-1]	O
Local PEC in surface water during emission episode	0	9.48E-06	[mg.l-1]	O
Annual average local PEC in surface water (dissolved)	0	9.48E-06	[mg.l-1]	O
Local PEC in sediment during emission episode	0	0.268	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 30 days	0	0.0362	[mg.kgwwt-1]	O
Local PEC in agric. soil (total) averaged over 180 days	0	0.0362	[mg.kgwwt-1]	O
Local PEC in grassland (total) averaged over 180 days	0	0.0364	[mg.kgwwt-1]	O
Local PEC in groundwater under agricultural soil	0	1.35E-05	[mg.l-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
REGIONAL AND CONTINENTAL SCALE				
CONTINENTAL				
Continental PEC in air (total)	0	1.15E-08	[mg.m-3]	O
Continental PEC in surface water (dissolved)	0	1.45E-06	[mg.l-1]	O
Continental PEC in agricultural soil (total)	0	0.0114	[mg.kgwwt-1]	O
Continental PEC in pore water of agricultural soils	0	4.27E-06	[mg.l-1]	O
Continental PEC in natural soil (total)	0	9.67E-03	[mg.kgwwt-1]	O
Continental PEC in industrial soil (total)	0	0.593	[mg.kgwwt-1]	O
Continental PEC in sediment (total)	0	0.0725	[mg.kgwwt-1]	O
ENVIRONMENT-EXPOSURE				
DISTRIBUTION				
REGIONAL AND CONTINENTAL SCALE				
REGIONAL				
Regional PEC in air (total)	0	4.26E-08	[mg.m-3]	O

Printed on 20/09/00 14:10:13
Study Gen. Env. - Scen. A - No sludge In.
Substance Cadmium
Defaults Standard
Assessment types 1A, 1B, 2, 3A, 3B
Base set complete No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE (Continued)				
DISTRIBUTION				
REGIONAL AND CONTINENTAL SCALE				
REGIONAL				
Regional PEC in surface water (dissolved)	0	9.48E-06	[mg.l-1]	O
Regional PEC in agricultural soil (total)	0	0.0671	[mg.kgwwt-1]	O
Regional PEC in pore water of agricultural soils	0	2.51E-05	[mg.l-1]	O
Regional PEC in natural soil (total)	0	0.0359	[mg.kgwwt-1]	O
Regional PEC in industrial soil (total)	0	5.74	[mg.kgwwt-1]	O
Regional PEC in sediment (total)	0	0.474	[mg.kgwwt-1]	O
ENVIRONMENT-EXPOSURE				
BIOCONCENTRATION				
Bioconcentration factor for fish	1.41254	38	[l.kg-1]	S
Bioconcentration factor for earthworms	3.30381	15	[kg.kg-1]	S
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 1][FORMULATION]				
Concentration in fish from surface water for predators	0	2.39E-03	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.775	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 1][PROCESSING]				
Concentration in fish from surface water for predators	0	3.74E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.776	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 1][PRIVATE USE]				
Concentration in fish from surface water for predators	0	9.85E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.787	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 1][RECOVERY]				
Concentration in fish from surface water for predators	0	4.89E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.784	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 2][FORMULATION]				
Concentration in fish from surface water for predators	0	3.63E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.773	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 3][FORMULATION]				
Concentration in fish from surface water for predators	0	3.6E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.772	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 3][PROCESSING]				
Concentration in fish from surface water for predators	0	3.65E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.773	[mg.kg-1]	O

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 3][PRIVATE USE]				
Concentration in fish from surface water for predators	0	3.66E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.772	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 3][RECOVERY]				
Concentration in fish from surface water for predators	0	3.61E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.772	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 4][FORMULATION]				
Concentration in fish from surface water for predators	0	3.6E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.772	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 4][PROCESSING]				
Concentration in fish from surface water for predators	0	4.01E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.773	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 5][FORMULATION]				
Concentration in fish from surface water for predators	0	3.64E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.772	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 6][PROCESSING]				
Concentration in fish from surface water for predators	0	1.77E-03	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.773	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 7][PROCESSING]				
Concentration in fish from surface water for predators	0	0.126	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.772	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 8][RECOVERY]				
Concentration in fish from surface water for predators	0	3.6E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	2.91	[mg.kg-1]	O
ENVIRONMENT-EXPOSURE				
SECONDARY POISONING[USE PATTERN 9][RECOVERY]				
Concentration in fish from surface water for predators	0	3.6E-04	[mg.kg-1]	O
Local concentration in earthworms from agricultural soil	0	0.774	[mg.kg-1]	O

EUSES Summary report | Single substance

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Study | Gen. Env. - Scen. A - No sludge In.
Substance | Cadmium
Defaults | Standard
Assessment types | 1A, 1B, 2, 3A, 3B
Base set complete | No

Name	Reference	Value	Units	Status
ENVIRONMENT - EFFECTS				
MICRO-ORGANISMS				
EC50 for micro-organisms in a STP	??	??	[mg.l-1]	D
EC10 for micro-organisms in a STP	??	??	[mg.l-1]	D
NOEC for micro-organisms in a STP	??	??	[mg.l-1]	D
PNEC for micro-organisms in a STP	??	??	[mg.l-1]	O
Assessment factor applied in extrapolation to PNEC micro	??	??	[-]	O
ENVIRONMENT - EFFECTS				
AQUATIC ORGANISMS				
EC50 for algae	??	??	[mg.l-1]	D
L(E)C50 for Daphnia	??	??	[mg.l-1]	D
LC50 for fish	??	??	[mg.l-1]	D
LC50 for other aquatic species	??	??	[mg.l-1]	D
NOEC for algae	??	??	[mg.l-1]	D
NOEC for Daphnia	??	??	[mg.l-1]	D
NOEC for fish	??	??	[mg.l-1]	D
NOEC for other aquatic species	??	??	[mg.l-1]	D
PNEC for aquatic organisms	??	??	[mg.l-1]	O
PNEC for aquatic organisms, intermittent releases	??	??	[mg.l-1]	O
PNEC for aquatic organisms with statistical method	??	??	[mg.l-1]	O
ENVIRONMENT - EFFECTS				
TERRESTRIAL ORGANISMS				
LC50 for plants	??	??	[mg.kgwwt-1]	D
LC50 for earthworms	??	??	[mg.kgwwt-1]	D
EC50 for microorganisms	??	??	[mg.kgwwt-1]	D
LC50 for other terrestrial species	??	??	[mg.kgwwt-1]	D
NOEC for plants	??	??	[mg.kgwwt-1]	D
NOEC for earthworms	??	??	[mg.kgwwt-1]	D
NOEC for microorganisms	??	??	[mg.kgwwt-1]	D
NOEC for other terrestrial species	??	??	[mg.kgwwt-1]	D
PNEC for terrestrial organisms	??	??	[mg.kgwwt-1]	O
Equilibrium partitioning used for PNEC in soil?	Yes	Yes		O
Assessment factor applied in extrapolation to PNEC Terr	??	??	[-]	O
PNEC for terrestrial organisms with statistical method	??	??	[mg.kgwwt-1]	O
ENVIRONMENT - EFFECTS				
SEDIMENT DWELLING ORGANISMS				
PNEC for sediment-dwelling organisms	??	??	[mg.kgwwt-1]	O
ENVIRONMENT - EFFECTS				
BIRDS AND MAMMALS				
LC50 in avian dietary study (5 days)	??	??	[mg.kg-1]	D
NOAEL	??	??	[mg.kg-1.d-1]	D
NOEC via food	??	??	[mg.kg-1]	O
Duration of (sub-)chronic oral test	Chronic	Chronic		D
Oral NOAEL	??	??	[mg.kg-1.d-1]	O
NOEC via food	??	??	[mg.kg-1]	O
Duration of (sub-)chronic oral test	28 days	28 days		D
PNEC for secondary poisoning of birds and mammals	??	??	[mg.kg-1]	O

EUSES Summary report | Single substance

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Study: Gen. Env. - Scen. A - No sludge In.
Substance: Cadmium
Defaults: Standard
Assessment types: 1A, 1B, 2, 3A, 3B
Base set complete: No

Name	Reference	Value	Units	Status
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 1][FORMULATION]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 1][PROCESSING]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 1][PRIVATE USE]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 1][RECOVERY]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 2][FORMULATION]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 3][FORMULATION]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O

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Study Gen. Env. - Scen. A - No sludge In.
Substance Cadmium
Defaults Standard
Assessment types 1A, 1B, 2, 3A, 3B
Base set complete No

Name	Reference	Value	Units	Status
ENVIRONMENT - RISK CHARACTERIZATION (Continued)				
LOCAL[USE PATTERN 3][FORMULATION]				
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION				
LOCAL[USE PATTERN 3][PROCESSING]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION				
LOCAL[USE PATTERN 3][PRIVATE USE]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION				
LOCAL[USE PATTERN 3][RECOVERY]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION				
LOCAL[USE PATTERN 4][FORMULATION]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION				
LOCAL[USE PATTERN 4][PROCESSING]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O

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Study Gen. Env. - Scen. A - No sludge In.
Substance Cadmium
Defaults Standard
Assessment types 1A, 1B, 2, 3A, 3B
Base set complete No

Name	Reference	Value	Units	Status
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 5][FORMULATION]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 6][PROCESSING]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 7][PROCESSING]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 8][RECOVERY]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION LOCAL[USE PATTERN 9][RECOVERY]				
RCR for the local water compartment	??	??	[-]	O
RCR for the local soil compartment	??	??	[-]	O
RCR for the local sediment compartment	??	??	[-]	O
RCR for the sewage treatment plant	??	??	[-]	O
RCR for fish-eating birds and mammals	??	??	[-]	O
RCR for worm-eating birds and mammals	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION REGIONAL[USE PATTERN 1]				
RCR for the regional water compartment	??	??	[-]	O
RCR for the regional soil compartment	??	??	[-]	O
RCR for the regional sediment compartment	??	??	[-]	O

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Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
ENVIRONMENT - RISK CHARACTERIZATION REGIONAL[USE PATTERN 2]				
RCR for the regional water compartment	??	??	[-]	O
RCR for the regional soil compartment	??	??	[-]	O
RCR for the regional sediment compartment	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION REGIONAL[USE PATTERN 3]				
RCR for the regional water compartment	??	??	[-]	O
RCR for the regional soil compartment	??	??	[-]	O
RCR for the regional sediment compartment	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION REGIONAL[USE PATTERN 4]				
RCR for the regional water compartment	??	??	[-]	O
RCR for the regional soil compartment	??	??	[-]	O
RCR for the regional sediment compartment	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION REGIONAL[USE PATTERN 5]				
RCR for the regional water compartment	??	??	[-]	O
RCR for the regional soil compartment	??	??	[-]	O
RCR for the regional sediment compartment	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION REGIONAL[USE PATTERN 6]				
RCR for the regional water compartment	??	??	[-]	O
RCR for the regional soil compartment	??	??	[-]	O
RCR for the regional sediment compartment	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION REGIONAL[USE PATTERN 7]				
RCR for the regional water compartment	??	??	[-]	O
RCR for the regional soil compartment	??	??	[-]	O
RCR for the regional sediment compartment	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION REGIONAL[USE PATTERN 8]				
RCR for the regional water compartment	??	??	[-]	O
RCR for the regional soil compartment	??	??	[-]	O
RCR for the regional sediment compartment	??	??	[-]	O
ENVIRONMENT - RISK CHARACTERIZATION REGIONAL[USE PATTERN 9]				
RCR for the regional water compartment	??	??	[-]	O
RCR for the regional soil compartment	??	??	[-]	O
RCR for the regional sediment compartment	??	??	[-]	O

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Study | Gen. Env. - Scen. A - No sludge In.
Substance | Cadmium
Defaults | Standard
Assessment types | 1A, 1B, 2, 3A, 3B
Base set complete | No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
Purification factor for surface water	1	1	[-]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
BIOCONCENTRATION AND BIOACCUMULATION FACTORS				
Bioconcentration factor for fish	1.41254	38	[l.kg-1]	S
Bioaccumulation factor for meat	7.94328E-07	0.02	[d.kg-1]	S
Bioaccumulation factor for milk	7.94328E-06	3E-04	[d.kg-1]	S
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 1][FORMULATION]				
Local concentration in wet fish	0	4.42E-03	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.26E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.14E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.22E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.16E-04	[mg.l-1]	O
Local concentration in meat (wet weight)	0	4.83E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	7.24E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 1][FORMULATION]				
Daily dose through intake of drinking water	0	3.32E-06	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	7.26E-06	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.57E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.92E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	2.08E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	5.8E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	1.97E-07	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 1][FORMULATION]				
Fraction of total dose through intake of drinking water	??	0.253	[-]	O
Fraction of total dose through intake of fish	??	0.553	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0119	[-]	O
Fraction of total dose through intake of root crops	??	5.27E-03	[-]	O
Fraction of total dose through intake of meat	??	0.158	[-]	O
Fraction of total dose through intake of milk	??	4.42E-03	[-]	O
Fraction of total dose through intake of air	??	0.015	[-]	O
Local total daily intake for humans	0	1.31E-05	[mg.kg-1.d-1]	O

EUSES Summary report | Single substance

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Study Gen. Env. - Scen. A - No sludge In.
Substance Cadmium
Defaults Standard
Assessment types 1A, 1B, 2, 3A, 3B
Base set complete No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 1][PROCESSING]				
Local concentration in wet fish	0	3.88E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.27E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.17E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.3E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.36E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	3.74E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	5.6E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 1][PROCESSING]				
Daily dose through intake of drinking water	0	3.89E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	6.38E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.57E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.95E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.61E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.49E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	2.8E-07	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 1][PROCESSING]				
Fraction of total dose through intake of drinking water	??	0.122	[-]	O
Fraction of total dose through intake of fish	??	0.2	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0494	[-]	O
Fraction of total dose through intake of root crops	??	0.0218	[-]	O
Fraction of total dose through intake of meat	??	0.504	[-]	O
Fraction of total dose through intake of milk	??	0.0141	[-]	O
Fraction of total dose through intake of air	??	0.0879	[-]	O
Local total daily intake for humans	0	3.19E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 1][PRIVATE USE]				
Local concentration in wet fish	0	1.61E-03	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.32E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.53E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	1E-05	[mg.kg-1]	O
Local concentration in drinking water	0	4.24E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	4.41E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	6.62E-06	[mg.kg-1]	O

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Study: Gen. Env. - Scen. A - No sludge In.
Substance: Cadmium
Defaults: Standard
Assessment types: 1A, 1B, 2, 3A, 3B
Base set complete: No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 1][PRIVATE USE]				
Daily dose through intake of drinking water	0	1.21E-06	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	2.65E-06	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.63E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	7.22E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.9E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	5.31E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	1.05E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 1][PRIVATE USE]				
Fraction of total dose through intake of drinking water	??	0.171	[-]	O
Fraction of total dose through intake of fish	??	0.373	[-]	O
Fraction of total dose through intake of leaf crops	??	0.023	[-]	O
Fraction of total dose through intake of root crops	??	0.0102	[-]	O
Fraction of total dose through intake of meat	??	0.267	[-]	O
Fraction of total dose through intake of milk	??	7.48E-03	[-]	O
Fraction of total dose through intake of air	??	0.149	[-]	O
Local total daily intake for humans	0	7.1E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 1][RECOVERY]				
Local concentration in wet fish	0	6.18E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.3E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.45E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.85E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.63E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	4.04E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	6.06E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 1][RECOVERY]				
Daily dose through intake of drinking water	0	4.65E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	1.02E-06	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.62E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	7.16E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.74E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.86E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	8.74E-07	[mg.kg-1.d-1]	O

EUSES Summary report | Single substance

Printed on | 20/09/00 14:10:13
Study | Gen. Env. - Scen. A - No sludge In.
Substance | Cadmium
Defaults | Standard
Assessment types | 1A, 1B, 2, 3A, 3B
Base set complete | No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 1][RECOVERY]				
Fraction of total dose through intake of drinking water	??	0.106	[-]	O
Fraction of total dose through intake of fish	??	0.232	[-]	O
Fraction of total dose through intake of leaf crops	??	0.037	[-]	O
Fraction of total dose through intake of root crops	??	0.0164	[-]	O
Fraction of total dose through intake of meat	??	0.397	[-]	O
Fraction of total dose through intake of milk	??	0.0111	[-]	O
Fraction of total dose through intake of air	??	0.2	[-]	O
Local total daily intake for humans	0	4.37E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 2][FORMULATION]				
Local concentration in wet fish	0	3.66E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.25E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.06E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.08E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.35E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	3.62E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	5.43E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 2][FORMULATION]				
Daily dose through intake of drinking water	0	3.84E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	6.01E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.55E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.86E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.56E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.35E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	4.23E-08	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 2][FORMULATION]				
Fraction of total dose through intake of drinking water	??	0.135	[-]	O
Fraction of total dose through intake of fish	??	0.211	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0545	[-]	O
Fraction of total dose through intake of root crops	??	0.0241	[-]	O
Fraction of total dose through intake of meat	??	0.546	[-]	O
Fraction of total dose through intake of milk	??	0.0153	[-]	O
Fraction of total dose through intake of air	??	0.0148	[-]	O
Local total daily intake for humans	0	2.85E-06	[mg.kg-1.d-1]	O

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 3][FORMULATION]				
Local concentration in wet fish	0	3.61E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.25E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.05E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.05E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.34E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	3.61E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	5.41E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 3][FORMULATION]				
Daily dose through intake of drinking water	0	3.84E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	5.92E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.55E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.85E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.55E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.34E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	1.08E-08	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 3][FORMULATION]				
Fraction of total dose through intake of drinking water	??	0.137	[-]	O
Fraction of total dose through intake of fish	??	0.211	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0553	[-]	O
Fraction of total dose through intake of root crops	??	0.0244	[-]	O
Fraction of total dose through intake of meat	??	0.553	[-]	O
Fraction of total dose through intake of milk	??	0.0155	[-]	O
Fraction of total dose through intake of air	??	3.83E-03	[-]	O
Local total daily intake for humans	0	2.81E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 3][PROCESSING]				
Local concentration in wet fish	0	3.7E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.25E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.06E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.06E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.34E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	3.61E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	5.42E-06	[mg.kg-1]	O

EUSES Summary report | Single substance

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 3][PROCESSING]				
Daily dose through intake of drinking water	0	3.84E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	6.08E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.55E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.86E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.55E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.34E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	2.36E-08	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 3][PROCESSING]				
Fraction of total dose through intake of drinking water	??	0.135	[-]	O
Fraction of total dose through intake of fish	??	0.214	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0547	[-]	O
Fraction of total dose through intake of root crops	??	0.0242	[-]	O
Fraction of total dose through intake of meat	??	0.548	[-]	O
Fraction of total dose through intake of milk	??	0.0153	[-]	O
Fraction of total dose through intake of air	??	8.33E-03	[-]	O
Local total daily intake for humans	0	2.84E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 3][PRIVATE USE]				
Local concentration in wet fish	0	3.72E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.25E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.05E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.06E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.34E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	3.61E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	5.42E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 3][PRIVATE USE]				
Daily dose through intake of drinking water	0	3.84E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	6.11E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.55E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.86E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.55E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.34E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	1.82E-08	[mg.kg-1.d-1]	O

EUSES Summary report | Single substance

Printed on 20/09/00 14:10:13
Study Gen. Env. - Scen. A - No sludge In.
Substance Cadmium
Defaults Standard
Assessment types 1A, 1B, 2, 3A, 3B
Base set complete No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 3][PRIVATE USE]				
Fraction of total dose through intake of drinking water	??	0.135	[-]	O
Fraction of total dose through intake of fish	??	0.216	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0548	[-]	O
Fraction of total dose through intake of root crops	??	0.0242	[-]	O
Fraction of total dose through intake of meat	??	0.548	[-]	O
Fraction of total dose through intake of milk	??	0.0153	[-]	O
Fraction of total dose through intake of air	??	6.42E-03	[-]	O
Local total daily intake for humans	0	2.83E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 3][RECOVERY]				
Local concentration in wet fish	0	3.62E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.25E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.05E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.05E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.34E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	3.61E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	5.41E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 3][RECOVERY]				
Daily dose through intake of drinking water	0	3.84E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	5.95E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.55E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.85E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.55E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.34E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	1.09E-08	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 3][RECOVERY]				
Fraction of total dose through intake of drinking water	??	0.137	[-]	O
Fraction of total dose through intake of fish	??	0.212	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0552	[-]	O
Fraction of total dose through intake of root crops	??	0.0244	[-]	O
Fraction of total dose through intake of meat	??	0.552	[-]	O
Fraction of total dose through intake of milk	??	0.0154	[-]	O
Fraction of total dose through intake of air	??	3.89E-03	[-]	O
Local total daily intake for humans	0	2.81E-06	[mg.kg-1.d-1]	O

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 4][FORMULATION]				
Local concentration in wet fish	0	3.6E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.25E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.05E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.06E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.34E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	3.61E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	5.42E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 4][FORMULATION]				
Daily dose through intake of drinking water	0	3.84E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	5.92E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.55E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.86E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.55E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.34E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	2.18E-08	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 4][FORMULATION]				
Fraction of total dose through intake of drinking water	??	0.136	[-]	O
Fraction of total dose through intake of fish	??	0.21	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0551	[-]	O
Fraction of total dose through intake of root crops	??	0.0243	[-]	O
Fraction of total dose through intake of meat	??	0.551	[-]	O
Fraction of total dose through intake of milk	??	0.0154	[-]	O
Fraction of total dose through intake of air	??	7.73E-03	[-]	O
Local total daily intake for humans	0	2.82E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 4][PROCESSING]				
Local concentration in wet fish	0	4.41E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.25E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.09E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.06E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.35E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	3.61E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	5.42E-06	[mg.kg-1]	O

Printed on	20/09/00 14:10:13
Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 4][PROCESSING]				
Daily dose through intake of drinking water	0	3.85E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	7.24E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.56E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.88E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.55E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.34E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	9.85E-09	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 4][PROCESSING]				
Fraction of total dose through intake of drinking water	??	0.131	[-]	O
Fraction of total dose through intake of fish	??	0.246	[-]	O
Fraction of total dose through intake of leaf crops	??	0.053	[-]	O
Fraction of total dose through intake of root crops	??	0.0234	[-]	O
Fraction of total dose through intake of meat	??	0.528	[-]	O
Fraction of total dose through intake of milk	??	0.0148	[-]	O
Fraction of total dose through intake of air	??	3.35E-03	[-]	O
Local total daily intake for humans	0	2.94E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 5][FORMULATION]				
Local concentration in wet fish	0	3.67E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.25E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.05E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.05E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.34E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	3.61E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	5.41E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 5][FORMULATION]				
Daily dose through intake of drinking water	0	3.84E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	6.03E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.55E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.85E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.55E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.34E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	9.12E-09	[mg.kg-1.d-1]	O

EUSES Summary report | Single substance

Printed on | 20/09/00 14:10:13
Study | Gen. Env. - Scen. A - No sludge In.
Substance | Cadmium
Defaults | Standard
Assessment types | 1A, 1B, 2, 3A, 3B
Base set complete | No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 5][FORMULATION]				
Fraction of total dose through intake of drinking water	??	0.136	[-]	O
Fraction of total dose through intake of fish	??	0.214	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0551	[-]	O
Fraction of total dose through intake of root crops	??	0.0244	[-]	O
Fraction of total dose through intake of meat	??	0.551	[-]	O
Fraction of total dose through intake of milk	??	0.0154	[-]	O
Fraction of total dose through intake of air	??	3.24E-03	[-]	O
Local total daily intake for humans	0	2.81E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 6][PROCESSING]				
Local concentration in wet fish	0	3.18E-03	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.25E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.05E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.05E-06	[mg.kg-1]	O
Local concentration in drinking water	0	8.37E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	4.38E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	6.57E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 6][PROCESSING]				
Daily dose through intake of drinking water	0	2.39E-06	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	5.23E-06	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.55E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.86E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.88E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	5.27E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	9.12E-09	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 6][PROCESSING]				
Fraction of total dose through intake of drinking water	??	0.244	[-]	O
Fraction of total dose through intake of fish	??	0.534	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0159	[-]	O
Fraction of total dose through intake of root crops	??	7E-03	[-]	O
Fraction of total dose through intake of meat	??	0.192	[-]	O
Fraction of total dose through intake of milk	??	5.38E-03	[-]	O
Fraction of total dose through intake of air	??	9.32E-04	[-]	O
Local total daily intake for humans	0	9.79E-06	[mg.kg-1.d-1]	O

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Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 7][PROCESSING]				
Local concentration in wet fish	0	0.252	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.25E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.05E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.05E-06	[mg.kg-1]	O
Local concentration in drinking water	0	6.62E-03	[mg.l-1]	O
Local concentration in meat (wet weight)	0	7.63E-03	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	1.14E-04	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 7][PROCESSING]				
Daily dose through intake of drinking water	0	1.89E-04	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	4.13E-04	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.55E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.85E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	3.28E-05	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	9.17E-07	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	9.12E-09	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 7][PROCESSING]				
Fraction of total dose through intake of drinking water	??	0.297	[-]	O
Fraction of total dose through intake of fish	??	0.649	[-]	O
Fraction of total dose through intake of leaf crops	??	2.44E-04	[-]	O
Fraction of total dose through intake of root crops	??	1.08E-04	[-]	O
Fraction of total dose through intake of meat	??	0.0515	[-]	O
Fraction of total dose through intake of milk	??	1.44E-03	[-]	O
Fraction of total dose through intake of air	??	1.43E-05	[-]	O
Local total daily intake for humans	0	6.36E-04	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 8][RECOVERY]				
Local concentration in wet fish	0	3.6E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.12E-04	[mg.kg-1]	O
Local concentration in leaves of plant	0	8.1E-05	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	1.53E-04	[mg.kg-1]	O
Local concentration in drinking water	0	1.2E-04	[mg.l-1]	O
Local concentration in meat (wet weight)	0	7.73E-03	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	1.16E-04	[mg.kg-1]	O

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Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 8][RECOVERY]				
Daily dose through intake of drinking water	0	3.43E-06	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	5.92E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.39E-06	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.13E-07	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	3.33E-05	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	9.3E-07	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	1.55E-04	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 8][RECOVERY]				
Fraction of total dose through intake of drinking water	??	0.0176	[-]	O
Fraction of total dose through intake of fish	??	3.03E-03	[-]	O
Fraction of total dose through intake of leaf crops	??	7.12E-03	[-]	O
Fraction of total dose through intake of root crops	??	3.14E-03	[-]	O
Fraction of total dose through intake of meat	??	0.17	[-]	O
Fraction of total dose through intake of milk	??	4.77E-03	[-]	O
Fraction of total dose through intake of air	??	0.794	[-]	O
Local total daily intake for humans	0	1.95E-04	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA[USE PATTERN 9][RECOVERY]				
Local concentration in wet fish	0	3.6E-04	[mg.kg-1]	O
Local concentration in root tissue of plant	0	1.26E-05	[mg.kg-1]	O
Local concentration in leaves of plant	0	9.12E-06	[mg.kg-1]	O
Local concentration in grass (wet weight)	0	9.19E-06	[mg.kg-1]	O
Local concentration in drinking water	0	1.35E-05	[mg.l-1]	O
Local concentration in meat (wet weight)	0	3.68E-04	[mg.kg-1]	O
Local concentration in milk (wet weight)	0	5.52E-06	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
DOSES IN INTAKE MEDIA[USE PATTERN 9][RECOVERY]				
Daily dose through intake of drinking water	0	3.87E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	5.92E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	1.56E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	6.9E-08	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	1.58E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	4.42E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	1.56E-07	[mg.kg-1.d-1]	O

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Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL SCALE				
FRACTIONS OF TOTAL DOSE[USE PATTERN 9][RECOVERY]				
Fraction of total dose through intake of drinking water	??	0.129	[-]	O
Fraction of total dose through intake of fish	??	0.198	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0524	[-]	O
Fraction of total dose through intake of root crops	??	0.0231	[-]	O
Fraction of total dose through intake of meat	??	0.53	[-]	O
Fraction of total dose through intake of milk	??	0.0148	[-]	O
Fraction of total dose through intake of air	??	0.0523	[-]	O
Local total daily intake for humans	0	2.99E-06	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
REGIONAL SCALE				
CONCENTRATIONS IN INTAKE MEDIA				
Regional concentration in wet fish	0	3.6E-04	[mg.kg-1]	O
Regional concentration in root tissue of plant	0	2.34E-05	[mg.kg-1]	O
Regional concentration in leaves of plant	0	1.69E-05	[mg.kg-1]	O
Regional concentration in grass (wet weight)	0	1.69E-05	[mg.kg-1]	O
Regional concentration in drinking water	0	2.51E-05	[mg.l-1]	O
Regional concentration in meat (wet weight)	0	6.74E-04	[mg.kg-1]	O
Regional concentration in milk (wet weight)	0	1.01E-05	[mg.kg-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
REGIONAL SCALE				
DOSES IN INTAKE MEDIA				
Daily dose through intake of drinking water	0	7.17E-07	[mg.kg-1.d-1]	O
Daily dose through intake of fish	0	5.92E-07	[mg.kg-1.d-1]	O
Daily dose through intake of leaf crops	0	2.9E-07	[mg.kg-1.d-1]	O
Daily dose through intake of root crops	0	1.28E-07	[mg.kg-1.d-1]	O
Daily dose through intake of meat	0	2.9E-06	[mg.kg-1.d-1]	O
Daily dose through intake of milk	0	8.1E-08	[mg.kg-1.d-1]	O
Daily dose through intake of air	0	9.12E-09	[mg.kg-1.d-1]	O
HUMAN HEALTH - EXPOSURE ASSESSMENT				
HUMANS EXPOSED VIA THE ENVIRONMENT				
REGIONAL SCALE				
FRACTIONS OF TOTAL DOSE				
Fraction of total dose through intake of drinking water	??	0.152	[-]	O
Fraction of total dose through intake of fish	??	0.126	[-]	O
Fraction of total dose through intake of leaf crops	??	0.0615	[-]	O
Fraction of total dose through intake of root crops	??	0.0272	[-]	O
Fraction of total dose through intake of meat	??	0.615	[-]	O
Fraction of total dose through intake of milk	??	0.0172	[-]	O
Fraction of total dose through intake of air	??	1.93E-03	[-]	O
Regional total daily intake for humans	0	4.72E-06	[mg.kg-1.d-1]	O

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Study Gen. Env. - Scen. A - No sludge In.
Substance Cadmium
Defaults Standard
Assessment types 1A, 1B, 2, 3A, 3B
Base set complete No

Name	Reference	Value	Units	Status
HUMAN HEALTH - EFFECTS ASSESSMENT				
(SUB)CHRONIC DATA				
Oral NOAEL	??	??	[mg.kg-1.d-1]	D
Oral LOAEL	??	??	[mg.kg-1.d-1]	D
Dermal NOEC in a medium	??	??	[mg.cm-3]	D
Dermal LOEC in a medium	??	??	[mg.cm-3]	D
Inhalatory (fibre) NOAEL	??	??	[fibres.m-3]	D
Inhalatory (fibre) LOAEL	??	??	[fibres.m-3]	D

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Study	Gen. Env. - Scen. A - No sludge In.
Substance	Cadmium
Defaults	Standard
Assessment types	1A, 1B, 2, 3A, 3B
Base set complete	No

Name	Reference	Value	Units	Status
HUMAN HEALTH - RISK CHARACTERIZATION				
CURRENT CLASSIFICATION				
Corrosive (C, R34 or R35)	No	No		D
Irritating to skin (Xi, R38)	No	No		D
Irritating to eyes (Xi, R36)	No	No		D
Risk of serious damage to eyes (Xi, R41)	No	No		D
Irritating to respiratory system (Xi, R37)	No	No		D
May cause sensitisation by inhalation (Xn, R42)	No	No		D
May cause sensitisation by skin contact (Xi, R43)	No	No		D
May cause cancer (T, R45)	No	No		D
May cause cancer by inhalation (T, R49)	No	No		D
Possible risk of irreversible effects (Xn, R40)	No	No		D
HUMAN HEALTH - RISK CHARACTERIZATION				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL[USE PATTERN 1][FORMULATION]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL[USE PATTERN 1][PROCESSING]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL[USE PATTERN 1][PRIVATE USE]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL[USE PATTERN 1][RECOVERY]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL[USE PATTERN 2][FORMULATION]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION				
HUMANS EXPOSED VIA THE ENVIRONMENT				
LOCAL[USE PATTERN 3][FORMULATION]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O

Printed on 20/09/00 14:10:13
Study Gen. Env. - Scen. A - No sludge In.
Substance Cadmium
Defaults Standard
Assessment types 1A, 1B, 2, 3A, 3B
Base set complete No

Name	Reference	Value	Units	Status
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT LOCAL[USE PATTERN 3][PROCESSING]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT LOCAL[USE PATTERN 3][PRIVATE USE]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT LOCAL[USE PATTERN 3][RECOVERY]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT LOCAL[USE PATTERN 4][FORMULATION]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT LOCAL[USE PATTERN 4][PROCESSING]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT LOCAL[USE PATTERN 5][FORMULATION]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT LOCAL[USE PATTERN 6][PROCESSING]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT LOCAL[USE PATTERN 7][PROCESSING]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O

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Study Gen. Env. - Scen. A - No sludge In.
Substance Cadmium
Defaults Standard
Assessment types 1A, 1B, 2, 3A, 3B
Base set complete No

Name	Reference	Value	Units	Status
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT LOCAL[USE PATTERN 8][RECOVERY]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT LOCAL[USE PATTERN 9][RECOVERY]				
MOS local, exposure via air	1E+60	??	[-]	O
MOS local, total exposure via all media	1E+60	??	[-]	O
HUMAN HEALTH - RISK CHARACTERIZATION HUMANS EXPOSED VIA THE ENVIRONMENT REGIONAL				
MOS regional, exposure via air	1E+60	??	[-]	O
MOS regional, total exposure via all media	1E+60	??	[-]	O

ANNEX 6

ENVIRONMENTAL TOXICITY DATA

The following tables reproduce those in the draft risk assessment report on cadmium oxide produced by Belgium for the EU Existing Substances Regulation (De Win *et al*, 1999). Data underlined in these tables are those selected by the rapporteur to be used in the effects assessment in that report. In this work a subset of these data has been used as indicated in the text.

Table A6.1: Toxicity to Fish/Amphibians (Table 3.2.3 from De Win, 1999)

test substance	organism	medium	test conditions	Nominal/ Measured	Duration (d)	Acute/chronic	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1})	EC_{50} (µg L^{-1})	LC_{50} (µg L^{-1})	references	R.I.
$CdCl_2$	Salmo salar	municipal water charcoal filtered and UV sterilised; BC 0.13 µg Cd/L; pH 6.5-7.3; T 5-10; DO 11.1-12.5; Al 14-17; H 19-28	semi-static	M	24	A	mortality				34	Rombough and Garside, 1982	2
					46	C	total biomass	0.47	0.78(28)				2
Cd-solution	Oncorhynchus tsahwytscha	continuous flow; aerated UV sterilised well water; T 11.6-12.8; Al 22; H 23; DO 10.2; pH 7.1; Cd < 0.2 µg L^{-1}	newly hatched alevins	M	4	A	mortality				> 27	Chapman, 1978	3
			swim-up alevins			A					1.3		3
			5-8m old parr			A					1.0		3
			smolts			A					> 2.9		3
			newly hatched alevins		8.3	A					> 27		3
			swim-up alevins			A					1.3		3
			5-8m old parr			A					0.9		3
			smolts			A					1.6		3
	Salmo gairdneri		newly hatched alevins		4	A					> 26		3
			swim-up alevins			A					1.8		3
			5-8m old parr			A					3.5		3
			smolts			A					> 2.9		3
			newly hatched alevins		8.3	A					> 26		3
			swim-up alevins			A					1.6		3
			5-8m old parr			A					2.0		3
			smolts			A					2.3		3
$CdCl_2$	Salmo gairdneri	water; T 15; pH 7.4; DO 90%; H 320	continuous flow	N	4 months	C	activity of liver enzymes	1	10(10)			Arillo et al., 1984	4
						C	blood enzymes	1	10(36)				4
						C	mitochondrial enz	1		10(75)			4
						C	gill sialic acid content	1	10(20)				4
						C	mucus lysozyme	10					4
$CdCl_2$	Brachydanio rerio	synthetic water (changed ISO) ; T 24; DO >80%; H 100; pH 7.2	semi-static; adults	N	4	A	mortality				3500	Bresch ., 1982	3
			larvae		24	C	reproduction		10(35)				
					36	C		1					

Table A6.1: Toxicity to Fish/Amphibians (Table 3.2.3 from De Win, 1999)

test substance	organism	medium	test conditions	Nominal/ Measured	Duration (d)	Acute/chronic	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1})	EC_{50} (µg L^{-1})	LC_{50} (µg L^{-1})	references	R.I.
$CdCl_2$	Catostomus commersoni	sand filtered Lake Superior Water; continuous flow; DO 10.3; H 45; Al 41; Ac 3; pH 7.6	T 18.1	M	10 30	C	standing crop (biomass)	4.2	12			Eaton et al., 1978	2
	Esox lucius		T 15.9		7 28	C		4.2	12.9				2
	Oncorhynchus kisutch (sac fry)		T 10.1	M	0 27	C	biomass	1.3	3.4				2
	Oncorhynchus kisuch		T 9.7		20 27	C		4.1	12.5				2
	Salvelinus namaycush		T 9.6		10 31	C		4.4	12.3				2
	Salvelinus fontinalis		T 9.7		24 126	C		1.1	3.8				2
	Salmo trutta		T 9.7		50 60	C		3.8	11.7				2
	Salmo trutta (late eyed eggs)		T 10		2 61	C		1.1	3.7				2
$CdCl_2$	Salvelinus fontinalis	sterilised Lake Superior water; H 42-47; pH 7-8; Al 38-46; Ac 1-10; DO 4-12; T 9-15	continuous flow	M	3 years	C	mortality	1.7			3.4	Benoit et al, 1976	2
						C	growth (weight) of 16 week old juveniles	1.7		3.4(56)			2
						C	total weight of young /female of the 2th generation	0.9	1.7(31)				2
						C	reproduction	6.4					2

Table A6.1: Toxicity to Fish/Amphibians (Table 3.2.3 from De Win, 1999)

test substance	organism	medium	test conditions	Nominal/ Measured	Duration (d)	Acute/chronic	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1})	EC_{50} (µg L^{-1})	LC_{50} (µg L^{-1})	references	R.I.
$CdCl_2$	Brachydanio rerio	tap water; continuous flow;	H 170	M	1	A	mortality				7000	Canton and Slooff,	3
	Oryzias latipes	T 20			2	A					4200	1982	
		synthetic water (Dutch	H 200		1	A					>2600		
		standard water); semi-static;			2	A					1800		
		T 24			3	A					170		
					4	A					130		
					1	A	mortality and				>2600		
					2	A	abn. behaviour				470		
					3	A					160		
					4	A					70		
			H 100		1	A	mortality						
					2	A					>2800		
					3	A					350		
					4	A					350		
					1	A	mortality and				>2800		
					2	A	abn. behaviour				320		
					3	A					120		
	Poecilia reticulata				4	A					70		
		synthetic water (Dutch	H 200		1	A	mortality				33000		
		standard water); semi-static;			2	A					20500		
		T 24			3	A					14400		
					4	A					11100		
					1	A	mortality and				31000		
					2	A	abn. behaviour				19500		
					3	A					12100		
					4	A					11100		
			H 100		1	A	mortality				10400		
					2	A					5700		
					3	A					4300		
					4	A					3800		
					1	A	mortality and				7100		
					2	A	abn. behaviour				5900		
	Salmo gairdneri				3	A					3700		
					4	A					3400		
		tap water; continuous flow;	H 170		1	A	inhibition of						
	Oryzias latipes	T 20					respiration						
							frequency						
			H 200		18	C	mortality	30	35(25)		50		
			H 100			C		6	23(25)		40		
			H 200		18	C	mortality and	6	70(35)		20		
			H 100			C	abn. behaviour	3	13(25)		30		

Table A6.1: Toxicity to Fish/Amphibians (Table 3.2.3 from De Win, 1999)

test substance	organism	medium	test conditions	Nominal/ Measured	Duration (d)	Acute/chronic	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1})	EC_{50} (µg L^{-1})	LC_{50} (µg L^{-1})	references	R.I.
	Xenopus laevis	tap water; continuous flow; T 20	H 170		1	A	mortality				4000		
					2	A					3200		
					100	C	mortality	30			1500		
							inhibition of larvae development	9		650			
							body weight	30					
$CdCl_2$	Jordanella floridae	untreated Lake Superior water; T 25; DO 8.3; H 44; Al 42; Ac 2.4; pH 7.1-7.8	continuous flow	M	4	A	mortality				2500	Spehar, 1976	2
					100	C	mortality	8.1			16(72)		2
						C	growth	8.1	16(27)				2
						C	reproduction	4.1		8.1(52)			2
$CdSO_4$	Brachydanio rerio	synthetic water (ISO 1977) ; T 25; pH 8.3; H 100	static; juvenile fish (0.25g each)	N	1	A	mortality				2400	Dave et al., 1981	3
					4	A					1700		3
					6	A					1700		3
	Salmo gairdneri		semi-static; embryo-larva		10	C	median survival	50(HT)					4
			semi-static; T 8; embryo-larva		50	C	time	4	5(15)				3
$CdCl_2$	Salmo gairdneri	aerated well water; T 10; O_2 7.5; H 375-390; pH 8-8.6	continuous flow	M	84	C	mortality	12	36 (10) HT			Lowe-Jinde and Niimi, 1984	2
$CdSO_4$	Pimephallus promelas	pond water diluted with carbon filtered demineralised tap water; H 201-204; DO 6.5-6.6; pH 7.6-7.7; Al 145-161; Ac 8-12; T 16-27	continuous flow; pond fish	M	60	C	survival of developing embryos	37	57(26)			Pickering and Gast, 1972	3
						C	hatchability of eggs	37	57				3
						C	growth	350					3
						C	reproduction	13	37(26)				3
					104	C	mortality				68		3
			continuous flow; 3 week old fry from laboratory		60	C	survival of developing embryos	27	57(22)				3
						C	reproduction	14	27(24)				3
					30	C	growth	110					3
			static		4	A	mortality				30000		3
			continuous flow		4	A					2000		3
Cd	Brachydanio rerio	OECD-203-test water; T 22; pH 7.42; DO 9.3; H 233.4	static	M	4	A	mortality		320(20)			Janssen Pharmaceutica, 1993a	4
CdO	Brachydanio rerio	OECD-203-test water; T 22; pH 7.82; DO 9.5; H 243.6	static	M	4	A	mortality	1600				Janssen Pharmaceutica, 1993b	4
$CdCl_2$	Brachydanio rerio	synthetic water; T 25; pH 6.9-7.2; H 12.4	continuous flow	N	42	C	mortality		3 (40)LT		10 (60)	Karlsson-Norrgren et al., 1985	3
$CdCl_2$	Cyprinus carpio	tap water; T 18-19; pH 6.8; Al 14.8; H 18, BC 0.001 mg L^{-1}; food < 0.05 µg L^{-1}	semi-static	N	47	C	vertebral column damage		10LT			Muramoto, 1981	3

Table A6.1: Toxicity to Fish/Amphibians (Table 3.2.3 from De Win, 1999)

test substance	organism	medium	test conditions	Nominal/ Measured	Duration (d)	Acute/chronic	endpoint	NOEC ($\mu g\ L^{-1}$)	LOEC ($\mu g\ L^{-1}$)	EC_{50} ($\mu g\ L^{-1}$)	LC_{50} ($\mu g\ L^{-1}$)	references	R.I.
$CdCl_2$	Lepomis macrochirus	dechlorinated, carbon-filtered tap water	static; T 22; DO 8.5; H 18; Al 16; pH 7.4-7.7	M	4	A	mortality				2300	Bishop and McIntosh, 1981	2
			continuous flow; T 14.5-16; pH 7.8-8.2; DO 6.2-8.1; H 340-360; Al 248-264; Cd < 1 $\mu g\ L^{-1}$	N	3	A	cough rate		50(35)LT				4
$CdSO_4$	Salvelinus fontinalis	synthetic soft water (EPA); pH 7.3 - 7.7; T 12	+ 3.10^{-3} M $CaCO_3$ H 340 - 344; Al 327-332	N	4	A	mortality				26	Carroll et al., 1979	3
			+ 3.10^{-3} M $CaSO_4$ H 332 - 348; Al 28-30			A					29		3
			+ 3.10^{-3} M $MgCO_3$ H 348 - 360; Al 314-324			A					3.8		3
			+ 3.10^{-3} M $MgSO_4$ H 324 - 336; Al 27-32			A					4.4		3
			+ 3.10^{-3} M Na_2SO_4 H 44 -46; Al 27-34			A					2.4		3
$CdCl_2$	Pimephales promelas	lake water; T 22.5; DO 7.5; H 44.4; Al 45.4; pH 7.1-7.8	continuous flow	M	4	A	mortality				1500	Phipps and Holcombe, 1985	2
	Carassius auratus				4	A					748		2
	Ictalurus punctatus				4	A					4480		2
	Lepomis macrochirus(juv)				4	A	mortality				6470		2
$CdCl_2$	Pimephales promelas	well water; pH 7.7; H 200; Al 140; T 22; BC negligible	static	N	2	A	mortality				100	Hall et al., 1986	3
					4	A					90		
$Cd(NO_3)_2$	Gambusia affinis	0.15µm filtered pond water rich in TOC; continuous flow; DO > 90%; Al 4; H 10; pH 5.6; Cd 0.02 $\mu g\ L^{-1}$	T 30.2	N	4	A	mortality				1300	Giesy et al., 1977	3
			T 30.7			A					1500		3
			T 28			A					2600		3
		0.15 µm filtered well water pour in TOC; continuous flow; ; DO > 90%; Al 9.7; H 11.1; pH 6.5; Cd 0.023 $\mu g\ L^{-1}$	T 30.2			A					900		3
			T 28			A					2200		3
$CdCl_2$	Carassius auratus	aerated dechlorinated and aged city water; T 22-25; Cd < 10 $\mu g\ L^{-1}$; Al 14-18; DO 90%	static; H 20	N	2	A	mortality				2760	McCarty et al., 1978	3
					4	A					2130		3
					10	A					1780		3
			H 140		2	A					46900		3
					4	A					46800		3
					10	A					40200		3

Table A6.1: Toxicity to Fish/Amphibians (Table 3.2.3 from De Win, 1999)

test substance	organism	medium	test conditions	Nominal/ Measured	Duration (d)	Acute/chronic	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1})	EC_{50} (µg L^{-1})	LC_{50} (µg L^{-1})	references	R.I.
$CdCl_2$	Barytelphusa guerini	tap water; pH 7.2-7.4; DO 7.8-8 mg L^{-1}, Al 102; H 112; male fish	semi-static	N	4 30	A C C	mortality activity of antioxidase lipid peroxidase			 620(80) 620(52)	1820	Venugopal et al., 1997	2 4 4
$CdCl_2$	Pimephales promelas Lepomis macrochirus Carassius auratus Lebistes reticulatus Lepomis cyanellus Pimephales promelas Lepomis cyanellus	synthetic water (5% natural limestone spring water + 95% deionised water); static hard limestone spring water	T 25; DO 7.8; pH 7.5; Al 18; H 20 T 25; DO 7.8; pH 8.2; Al 300; H 360	N	4	A A A A A A A	mortality				630-1050 1940 2340 1270 2840 72600-73500 66000	Pickering and Henderson, 1966	4 4 4 4 4 4 4
Soluble Cd	Perca fluviatilis	River Emån water; T 20-22; H 40-50; pH 6.7; female fish	field study	M	whole life	C	immune defense			0.1-0.2(45-100)LT		Sjöbeck et al., 1984	4

*T = temperature (°C); H = hardness (as mg CaCO3/L); DO = dissolved oxygen (mg O2/L); Al = alkalinity (mg CaCO3/L); Ac = acidity (mg CaCO3/L); ** days of exposure of embryos and larvae-juveniles*

Table A6.2: Toxicity to Aquatic Invertebrates (Table 3.2.5 in De Win, 1999)

test substance	Organism	medium	test conditions	Nominal/Measured	duration (d)	acute/ chronic	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1}) (%effect)	EC_{50} (µg L^{-1}) (%effect)	LC_{50} (µg L^{-1}) (%effect)	references	RI
$CdCl_2$	Daphnia magna	unchlorinated, carbon	static	N	2	A	mortality				178	Elnabarawy et al.,	3
	Daphnia pulex	filtered well water, aerated				A					319	1986	3
	Ceriodaphnia reticulata	to saturation; H 240; Al 230; pH 8; DO >5; T 23; Cd <				A					184		3
	Daphnia magna	0.01 µg Cd/L	semi-static		14	C	mortality	25(HT)					4
	Daphnia pulex					C		25(HT)					4
	Ceriodaphnia reticulata					C		25(HT)					4
	Daphnia magna				14	C	reproductive impairment	2.5		7.5 (75)			3
	Daphnia pulex					C		7.5	25 (32)				3
	Ceriodaphnia reticulata					C		0.25	0.75 (20)				4
$CdCl_2$	Daphnia magna	20 µm cloth filtered Lake Superior water; pH 7.7; H 45.3; Al 42.3; DO 9; T 18; Cd < 0.1µg L^{-1}	semi-continuous flow; without food	N	2	A	mortality				65	Biesinger and Christensen, 1972	3
			with food		21	C	mortality				5		3
						C	reproductive impairment		0.17 (16)	0.7			4
						C	weight/animal	1		0.7			3/4
						C	protein conc./animal	1					3
						C	GOT activity/animal		1(15)				3
$CdCl_2$	Daphnia pulex	Whatman N° 1 filtered Lake Champlain water; pH 7.7; Al 42.4; H 65; Cd < 1µg L^{-1}	static	N	3	A	mortality				62	Bertram and Hart,	3
					4	A	mortality				47	1979	3
			semi-continuous flow		104	C	longevity	1		5(57)			3
						C	brood size		1(37)LT				3
						C	generation time in days		1(19)LT				3
$CdCl_2$	Crustacean plankton communities	Lake Michigan; BC 0.1 µg Cd /L;H 120	batch in situ	N	21	C	biodiversity index		1.2(14)	5.2		Marshall and	3
				N	21	C	crustacean density	2.5		5(72)		Mellinger ,1980	
$CdCl_2$	Daphnia magna	Dutch standard water; T 19	semi-static H 200	M(>20µg L^{-1})	2	A	mortality				30	Canton and Slooff,	3
			H 100		2	A	mortality				30	1982	3
			H200		20	C	mortality	0.37			0.67		4
			H100		20	C	reproduction	0.5		4.2			4/3
$CdSO_4$	Daphnia magna	synthetic water; H 90; Al 65; T 25	static	N	7	C	mortality	3(HT)				Winner, 1988	4
	Ceriodaphnia dubia					C		1.5	2(40)		3(70)		3
	Daphnia magna					C	reproduction	2	3(30)				3
	Ceriodaphnia dubia					C		0.5	1(45)				4/3
$CdCl_2$	Daphnia magna	synthetic water; T 25; pH 8; H 11; DO 69%	semi-static	M	1	A	mortality			1900		Kühn et al., 1989	2
					21	C	reproduction	0.6	1.9				2

Table A6.2: Toxicity to Aquatic Invertebrates (Table 3.2.5 in De Win, 1999)

test substance	Organism	medium	test conditions	Nominal/Measured	duration (d)	acute/ chronic	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1}) (%effect)	EC_{50} (µg L^{-1}) (%effect)	LC_{50} (µg L^{-1}) (%effect)	references	RI
$CdCl_2$	Daphnia magna	aerated well water; DO >70%; pH 8; T 22; H 300; Al 250	continuous	M	21	C	mortality	4.3			7.2 (100)	Knowles and McKee, 1987	2
						C	reproduction	0.8		2.1(54)			2
$CdCl_2$	Daphnia magna	NPR synthetic water; pH 8.4; T 20; H 200	semi-static	N	21	C	intrinsic rate of natural increase	1	1.8(32.5)	32(100)		Van Leeuwen et al., 1985	3
						C	mortality	1	1.8 (17)		3.2 (88)		3
		50 µm filtered and sterilised Lake IJssel water; pH 8.1; T 20; H 224	semi-continuous flow	M	21	C	yield		0.3(36)LT				3
			semi-static	N	21	C	intrinsic rate of natural increase	3.2	10 (14.5)	32 (100)			3
$CdCl_2$	Daphnia galeata mendotae	10 µm filtered Lake Michigan water; T 18.5 H 120	semi-continuous flow	N	154	C	carrying capacity			7.7(50)		Marshall, 1978	3
						C	number of individuals	2	4(23)	8(58)			3
						C	average biomass	2	4(9)				3
						C	average birth rate	2		4(71)			3
						C	average death rate	2		4(70)			3
						C	brood size	2	4(36)	5(54)			3
						C	dry weight	4		8(71)			3
						C	life expectancy			5(50)			3
$CdCl_2$	Daphnia magna	hard synthetic water; H 250; T 20; DO 66-100%; pH 7.2-8.2	semi-static	N	16	C	ALA-D activity			0.1(80)		Berglind, 1985	4
						C	haemoglobin content		0.1(20)				4
						C	growth	1.6(HT)					4
$CdCl_2$	Daphnia magna	culture medium; pH8.4; H 150; T 20	semi-continuous flow	N	25	C	mortality	2.5			10(60)	Bodar et al., 1988a	3
						C	biomass production/female	2.5		10(71)			3
						C	intrinsic rate of natural increase	5	10(34)	20(72)			3
$CdCl_2$	Simocephalus serrulatus	synthetic water; H 39-48; Al 26-42; Ac 1.9-5.7; pH 7-7.9	static T 20; <1d old	M	2	A	mortality				24.5	Spehar and Carlson, 1984	3
	Gammarus pseudolimnaeus	unfiltered river water; static;	T 17; 0.1g		4	A					68.3		3
	Daphnia magna	H 55-79 Ac 2-4.2; Al 41-65;	T 20; <1d old		2	A					166		3
	Ceriodaphnia reticulata	pH 7.2-7.8	T 20; <1d old		2	A					129		3
					9	C	reproduction	3.4	15.2				
	Simocephalus vetulus		T 20; <1d old		2	A	mortality				89.3		3
	Simocephalus serrulatus		T 20; <1d old		2	A					123		3
	Gammarus pseudolimnaeus		T 17; 0.1g		4	A					54.4		3
	Hyalella azteca		T 7; 1µg		4	A					285		3
	Paraleptophlebia praepedita		T 12; 2µg		4	A					449		3

Table A6.2: Toxicity to Aquatic Invertebrates (Table 3.2.5 in De Win, 1999)

test substance	Organism	medium	test conditions	Nominal/Measured	duration (d)	acute/ chronic	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1}) (%effect)	EC_{50} (µg L^{-1}) (%effect)	LC_{50} (µg L^{-1}) (%effect)	references	RI
$CdCl_2$	Aplexa hypnorum mature immature	Lake Superior water; DO 7.5; T 24	continuous flow H 44.8; Al 40.7; pH 7.4-7.5; H 45.3; Al 40.3; pH 7.3-7.6	M	 4 26 C	 A C C	 mortality growth mortality+hatchability	 <u>4.41</u> 4.41		 <u>4.79</u>(47) 4.79(62)	 93 	Holcombe et al., 1984	 2 2 2
$CdCl_2$	Dugesia sp. Cyclops sp. Cypridopsis sp. Hyalella sp. Procambarus sp.	non aerated spring water; T 23; H 20	static	N	4	A A A A A	mortality				<u>4900</u> <u>340</u> <u>190</u> <u>85</u> <u>5000</u>	Fennikoh et al., 1978	3 3 3 3 3
Cd (filtrate of dispersion)	Daphnia magna	OECD 202-test medium; pH 7.76; DO 9.6; H 274; T 20.2	static	M	2	A	mortality		<u>40</u>(30)		<u>110</u>(70)	Janssen Pharmaceutica, 1993c	1 2
$CdCl_2$	Physa integra Ephemerella sp. Pteronarcys dorsata Hydropsyche betteni	untreated Lake Superior water; pH 7.1-7.7; T 15; DO 10-11; H 44-48; Al 40-44; Ac 1.9-3	semi-static	M	28 28 28 28	C C C C	mortality	<u>8.3</u> 238 (HT) 238 (HT)			<u>10.4</u> <u>3</u>(70)	Spehar et al., 1978	2 2 4 4
CdO (filtrate of dispersion)	Daphnia magna	OECD 202-test medium; pH 8.05; DO 9.3; H 226; T 19.1	static	M	2	A	mortality				<u>750</u>(70)	Janssen Pharmaceutica, 1993d	2
$CdCl_2$	Daphnia magna	Dutch Standard water NPR 6503 (1980); pH 8.4; H 150; T 20	semi-continuous flow	N	2	A	mean survival time of embryos		<u>1000</u>(37)	<u>10000</u>(87)		Bodar et al., 1989	3
$CdCl_2$	Tetrahymena sp.	Osterhouts medium		N	15 min.	A	ciliate chemotactic response inhibition		250	475		Berk et al., 1985	4
$CdCl_2$	Daphnia magna	Dutch Standard water NPR 6503 (1980); pH 8.4; H 150; T 20	semi-continuous flow	N	14	C C	body weight consumption rate	 1		<u>1</u>(56) 5(60)		Bodar et al., 1988b	3 3
$CdSO_4$	Tubifex tubifex	dilution water for BOD without phosphate buffer dilution water for BOD with phosphate buffer: drinking water	pH 6.85; H 34.2; Al 1.5 pH 6.85; H 34.2; Al 22.5 pH 7.32; H 261; Al 234; T 20	N	2	A A A	mortality				<u>31</u> <u>45</u> <u>720</u>	Brkovic-Popovic and Popovic, 1977	3 3 3

Table A6.2: Toxicity to Aquatic Invertebrates (Table 3.2.5 in De Win, 1999)

test substance	Organism	medium	test conditions	Nominal/Measured	duration (d)	acute/ chronic	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1}) (%effect)	EC_{50} (µg L^{-1}) (%effect)	LC_{50} (µg L^{-1}) (%effect)	references	RI
$Cd(NO_3)_2$	Simocephalus serrulatus	filtered (0.15µm) well water; Cd 0.023 µg L^{-1}; pH 6.5; H 11.1; Al 9.7; T 22; DO >80%	filtered well water	N	2	A	mortality				7	Giesy et al., 1977	3
			filtered well water + F1*			A					8.6		3
			filtered well water + F2*										
			filtered well water + F3*			A					12		3
			filtered well water + F4*										
						A					16.5		3
						A					3.6		3
		Skinface pond water; filtered through 0.15µm; Cd 0.02 µg L^{-1}; pH 5.6; H 10; Al 4; T 22; DO >80%				A					35		3
$CdCl_2$	Daphnia magna	dechlorinated Montreal city water; pH 6.95; T 20; Al 80; H 130; Cd 1µg L^{-1}	continuous flow	N	1.5	A	mortality				203.8	Attar and Maly, 1982	2
					2	A					58.16		2
					2.5	A					15.8		2
					3	A					8.88		2
					4	A					5		2
$CdCl_2$	Daphnia magna	synthetic water; static; pH 8-8.5	H 160-180; T 20	N	2	A	mortality				38	Lewis and Horning, 1991	2
	Daphnia pulex		H 80-100; T 20								42		2
Cd-solution	Gammarus pulex	dechlorinated tap water; pH 7.7; T 12; H 152; DO >96%	continuous flow	M	4	A	mortality				20	Williams et al., 1985	3
	Asellus aquaticus					A					600		3
	Baetis rhodani					A					500		3
	Physa fontinalis					A					800		3
	Limnodrilus hoffmeisteri					A					2400		3
	Ephemerella ignita					A					13000		3
	Leuctra inermis												
	Polycelis tenius					A					32000		3
	Chironomus riparius					A					74000		3
						A					300000		3
	Hydropsyche angustipennis					A					520000		3
$CdCl_2$	Daphnia magna	well water; T 19.5; H 32-76; Al 31-69; pH 6.8-7.8	semi-static <4h old	M	2	A	mortality				109	Nebeker et al., 1986a	3
			<24h old			A					46		3
			1d old			A					48		3
			2d old			A					164		3
			3d old			A					63		3
			4d old			A					82		3
			5d old			A					49		3
			6d old			A					23		3

Table A6.2: Toxicity to Aquatic Invertebrates (Table 3.2.5 in De Win, 1999)

test substance	Organism	medium	test conditions	Nominal/Measured	duration (d)	acute/ chronic	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1}) (%effect)	EC_{50} (µg L^{-1}) (%effect)	LC_{50} (µg L^{-1}) (%effect)	references	RI
$CdCl_2$	Daphnia pulex	synthetic water; pH 7.8; T 22	static; H 120; Al 110	N	2	A	mortality				90	Hall et al., 1986	3
	Daphnia magna					A					35		3
	Ceriodaphnia reticulata					A					110		3
	Daphnia pulex	well water; pH 7.7; T 22	static; H 200; Al 140;		2	A					90		3
	Daphnia magna					A					65		3
	Ceriodaphnia reticulata					A					80		3
$CdCl_2$	Daphnia magna	soft well water; H 26-32; Al 30; T 20; DO 7.5-9; pH 6.6-7.8	static	M	2	A	mortality				36	Schuytema et al., 1984	2
			continuous			A					49		2
		slurry; T 20; H 15-23; Al 10-15; DO 7.5-9; pH 6.1-7.1 (LC50 expressed on dissolved fraction)	100 mg L^{-1} total solids, static			A					39		2
			continuous 1000 mg L^{-1} total solids, static			A					144		2
			continuous			A					44		2
						A					97		2
$CdSO_4$	Daphnia magna	synthetic water (ISO 1977); pH 7.8; H 200; T 20-23	static	N	1	A	mortality				309	Dave et al., 1981	3
					2	A					69		3
					3	A					40		3
$CdSO_4$	Cyclops abyssorum prealpinus	5µm filtered Lake Monate water; pH 7.2; H 40.7; T 10		N	2	A	mortality				3800	Baudouin and Scoppa, 1974	3
	Eudiaptomus padanus padanus					A					550		3
	Daphnia hyalina					A					55		3
$CdCl_2$	Daphnia magna	synthetic water; T 20; pH 8.3; H 250	semi-static; small neonates	N	2	A	mortality				98	Enserink et al., 1990	3
			large neonates			A					294		3
$CdCl_2$	Daphnia magna	synthetic water; T 20; pH 8.4; H 150	static; pre-exposed to control	N	2	A	mortality				320	Bodar et al., 1990	3
			pre-exposed to 1µg Cd/L		2	A					391		3
			pre-exposed to 5 µg Cd/L		2	A					424		3
Cd-solution	Physa gyrina mature	synthetic water; T 20-22; DO 10-14; H 200; Al 130; pH 6.73; Cd < 0.5µg L^{-1}	static	N	1	A	mortality				7600	Wier and Walter, 1976	3
					2	A					4250		3
					4	A					1370		3
					9.5	A					830		3
	immature				2	A					690		3
					4	A					410		3

Table A6.2: Toxicity to Aquatic Invertebrates (Table 3.2.5 in De Win, 1999)

test substance	Organism	medium	test conditions	Nominal/Measured	duration (d)	acute/ chronic	endpoint	NOEC ($\mu g\ L^{-1}$)	LOEC ($\mu g\ L^{-1}$) (%effect)	EC_{50} ($\mu g\ L^{-1}$) (%effect)	LC_{50} ($\mu g\ L^{-1}$) (%effect)	references	RI
$CdCl_2$	Brotia hainanensis	aerated artificial pond water; T 20: pH 7.4;H 200	upstream adolescents downstream adolescents upstr. juv. <2d downstr. juv. <2d upstr. juv. >7d downstr. juv. >7d	N	4	A A A A A A	mortality				15210 35940 770 1090 1180 1220	Lam, 1996	3 3 3 3 3 3
$CdSO_4$	Daphnia magna	filtered aerated tubewell hard water; H 240, T 13; pH 7.6; DO 5.6; Al 400	static	M	2	A	mortality				1880	Khangarot and Ray, 1989	3
$CdSO_4$	Acroneuria lycorias Ephemerella subvaria Hydropsyche betteni	carbon filtered Lake Superior tap water; pH 7-7.3; T 18.5; DO 8; Al 54-60; Ac 6-12; H 52-56		N	14 4 10	A A A	mortality				32000 2000 32000	Warnick and Bell, 1969	3 3 3

T = temperature (°C); H = hardness (as mg CaCO3/L); DO = dissolved oxygen (mg O2/L); Al = alkalinity (mg CaCO3/L); Ac = acidity (mg CaCO3/L);
**organic fractions F1: > 0.0183 µm; F2: 0.0183 - 0.0032 µm; F3: 0.0032 - 0.0009 µm; F4: < 0.0009 µm; GOT: glutamic oxalacetic transaminase.*

Table A6.3: Toxicity to Aquatic Primary Producers (Table 3.2.7 in De Win, 1999)

test substance	organism	medium	test conditions	nominal/ measured	duration (d)	endpoint	NOEC ($\mu g\ L^{-1}$)	LOEC ($\mu g\ L^{-1}$) (%effect)	EC_{50} ($\mu g\ L^{-1}$) (%effect)	references	RI
$CdCl_2$	Scenedesmus quadricauda	AM; H 28; T 21-30	static	N	2-16	average cell number	0.6		6.1(52%)	Klass et al., 1974	4/3
Cd filtrate of dispersion	Selenastrum capricornutum	modified ISO 6341 medium; 0.2 µm filtered; T 20.3-25.6; pH 7.7-10.4 H 49	static	M	3	cell number growth rate	2.5 9.5	10 (24)	23 89	LISEC, 1998a	1 1
$CdSO_4$	Coelastrum proboscideum	AM;H 32;T 31;pH 5.3;	static	M	1	biomass	6.3	27(36)		Müller and Payer 1979	2
$CdCl_2$	Chlamydomonas reinhardii	AM;H 42;pH 6.7; T 20	continuous	N	7	steady state cell number	7.5	10(22)		Lawrence et al. 1989	3
$CdCl_2$	Asterionella formosa Fragilaria crotonensis	AM; pH 8; H 121	static	M	1	growth rate	0.85 8.5HT	1.9(18)LT		Conway and Williams 1979	2 4
CdO filtrate of dispersion	Selenastrum capricornutum	modified ISO 6341 medium; 0.2 µm filtered; H 49;pH 7-10		M	3	cell number growth rate	9.5(LT)	9.5(37)LT 48(39	18 79	LISEC, 1998b	1 1
Cd filtrate of dispersion	Selenastrum capricornutum	AM; H 23; pH 7-9		M	3	growth rate	15	60(45)	70	Janssen Pharmaceutica, 1993e	1
$CdCl_2$	Scenedesmus subspicatus	AM;H 60;T 24; pH 8	static	N	3	biomass growth rate (0-3d)			62 136	Kühn and Pattard 1990	3 3
$Cd(NO_3)_2$	Scenedesmus quadricauda	AM; pH 7	static; T 27; H 55	N	7	biomass (OD)	31			Bringmann and Kühn, 1980	3
CdO filtrate of dispersion	Selenastrum capricornutum	AM H 23; pH 7-8		M	3	growth rate	50		120	Janssen Pharmaceutica, 1993f	1
$CdCl_2$	Lemna paucicostata	AM; T 25	static; pH > 6; H 120 pH 5.1; H 120 pH 5.1; H	N	7	number of fronds	 5 10 10	 10(19) 100(35) 50(20)		Nasu and Kugimoto, 1981	 3 3 3
$Cd(NO_3)_2$	Ankistrodesmus falcatus Chlorella vulgaris Scenedesmus quadricauda Chlorella pyrenoidosa	AM; H=34;T=20 AM; H=34;T=20 AM; H=34;T=20 AM; H=34;T=20 AM; H=34;T=20	static static static static static	N	6 1 1 1 1	cell number $^{14}CO_2$ uptake	500		1000(60%) 600(50) 700(50) 20(80)LT 100(50)	Wong et al., 1979	3 3 3 3 3
$CdCl_2$	Selenastrum capricornutum	AM;H=15;pH=7.1;T=24	static	N	4	biomass		50(32)LT		Bartlett et al. 1974	3

Table A6.3: Toxicity to Aquatic Primary Producers (Table 3.2.7 in De Win, 1999)

test substance	organism	medium	test conditions	nominal/ measured	duration (d)	endpoint	NOEC (µg L^{-1})	LOEC (µg L^{-1}) (%effect)	EC_{50} (µg L^{-1}) (%effect)	references	RI
$CdCl_2$	Selenastrum capricornutum	AM; pH 7.6; H 15; T 24	continuous	N	1	growth rate			13	Chen and Lin, 1997	3
			static		1	growth rate			341		3
$CdCl_2$	Selenastrum capricornutum	AM; pH 7.5; H 15;T24;	static	N	1	growth rate			32	Lin et al., 1996	3
$CdCl_2$	Selenastrum capricornutum	AM;H=15;pH=7.1	static	N	14-21	biomass			57	Turbak et al., 1986	3
$CdCl_2$	Chlorella vulgaris	AM;H=82;T=21	static	N	33	cell number	0.75	18(28)	60(50)	Rosko and Rachlin, 1977	4/3/3
$Cd(NO_3)_2$	Chlorella vulgaris	AM Lefevre Czarda; T 20	static	N	4	biomass (OD)			1220	Jouany et al., 1983	4
			pseudo-dynamic		4				550		4
Cd-salt	Dinobryon bavaricum	lake water	in situ continuous; epilimnion	M	12	cell density			3.5(>90)	DeNoyelles F. et al., 1980	4
	Dinobryon sertularia								3.5(>90)		4
	Elakatothrix sp					cell density		3.5(+80%)			4
	Rhabdoderma gorskii							3.5(+40%)			4
$CdCl_2$	Chlorella vulgaris	AM;H33;T 28	static	N	7	chlorophyll a content			393(77)LT	Kosakowska et al. 1988	4
	Anabaena variabilis								393(79)LT		

T = temperature (°C); H= water hardness (mg CaCO3/L); AM, artificial medium; OD, optical density

Table A6.4: Toxicity to Soil Microflora (Table 3.2.15 in De Win, 1999)

test substance	organism	medium	pH[a]	%OC[b]	% clay	Equil. Period (d)	duration (d)	endpoint	NOEC ($\mu g\ g^{-1}$)	LOEC ($\mu g\ g^{-1}$) (%effect)	$EC_{x\geq 50}$ ($\mu g\ g^{-1}$) (%effect)	references	R.I.
CdOAc.	native soil microflora	phaosem	6.9	1.3	21	>1 week	84(min.)	substrate induced respiration rate	3.6	7.1(15)	60	Reber, 1989	2
		neutral sandy hortisol	7	1.5	3		84(min.)	substrate induced respiration rate	3.6	7.1(15)	70		2
		acidic cambisol	5.6	1	7		84(min.)	substrate induced respiration rate	14.3	28.6(18))	>228(HT)		2
$CdSO_4$	native soil microflora	sandy soil	4.9	2.2	5.2	0	14 56	respiration	100HT 5	 10(17)		Cornfield 1977	4 3
$CdSO_4$	Rhizobium leguminoasorum bv. trifolii	sandy loam	6.5	/	9		540	cell number (survival)	4	7.1		Chaudri et al., 1992	3
$CdCl_2$	native soil microflora	forest soil (0-4.5cm) loamy sand	4.8	1.1	0-15	0	1.5 23	respiration rate: 47ppm Zn added no Zn added with or without Zn added	 1.3 6.7HT 6.7HT	 6.7(38)		Chaney et al., 1978	 4 4 4
$CdCl_2$	native soil microflora	grassland soil	7.4	5.7	24	0	33	nitrification: NO_3^- production rate $-NH_4^+$substrate	 50	 100(12*		Dušek, 1995	2
								$+NH_4^+$substrate	100	500(45)			
			7.6	2.9	19		33	nitrification: NO_3^- production rate $-NH_4^+$substrate	 50	 100(19)			
								$+NH_4^+$substrate	10	100 (13)	500(60)		
$Cd(NO_3)_2$	native soil microflora	loamy sand	5.8	2.7	16	>98	1 50	24h respiration ammonification: NH_4^+ found after 50 days incubation	14.3 58HT	29.1(36)		Walter and Stadelman, 1979	2 4
$CdCl_2$	native soil microflora	brown earth loamy sand	4.6	1-20	0-15	15	45	cellulolytic activity: unplanted soil oat grown soil	10	50(17) 10(34)LT		Khan and Frankland, 1984	3 3
								wheat grown soil		50(43)LT			3
$CdCl_2$	native soil microflora	inceptisol	5.2	1.4	8	0	28	substrate induced respiration	50	250(27)		Saviozzi et al. 1997	3
$CdCl_2$	native soil microflora	sand silty loam	7 7.7	1.1 2.6	2 19	560 560	1-2 1-2	glutamic acid decomposition time glutamic acid decomposition time	 55	 150(15)	150(56)	Haanstra and Doelman, 1984	2 2
		clay	7.5	3.4	60	560	1-2	glutamic acid decomposition time	150	400(10)			2
		peat	4.5	13.6	5	560	1-2	glutamic acid decomposition time	1000HT				4
$CdCl_2$	native soil	sand	7	1.1	2	490	42-70	respiration	150	400(23)		Doelman and Haanstra,	2
	microflora	sandy loam	6	6.1	9	301	42-70	respiration	150	400(20)		1984	
		silty loam	7.7	2.6	19	630	42-70	respiration	200	400(17)			
		clay	7.5	3.4	60	560	42-70	respiration	200	400(17)			
		peat	4.5	13.6	5	574	42-70	respiration	500	1000(19)			

Table A6.4: Toxicity to Soil Microflora (Table 3.2.15 in De Win, 1999)													
test substance	**organism**	**medium**	**pH[a]**	**%OC[b]**	**% clay**	**Equil. Period (d)**	**duration (d)**	**endpoint**	**NOEC (µg g^{-1})**	**LOEC (µg g^{-1}) (%effect)**	**$EC_{x\geq50}$ (µg g^{-1}) (%effect)**	**references**	**R.I.**
$Cd(NO_3)_2$	native soil microflora	silt loam	6.7	1.9	28	0	14	denitrification		10LT	100	Bollag and Barabasz, 1979	3
$CdSO_4$	native soil microflora	surface soil	4.8	3.8	9	0	4	nitrification: % NO_3^- of total N		50(30)LT	1000(54)	Bewley and Stotzky, 1983	4
$CdSO_4$	native soil microflora	surface soil	5.8	2.8	23	0	10	nitrification: NO_3^- + NO_2^- accumulation after 10 days with addition of NH_4^+			560(94)LT	Liang and Tabatabai, 1978	4
			7.8	3.9	30		10	nitrification: NO_3^- + NO_2^- accumulation after 10 days with addition of NH_4^+			560 (70)LT		
			7.4	5.7	34		10	nitrification: NO_3^- + NO_2^- accumulation after 10 days with addition of NH_4^+			560(67)LT		
$CdCl_2$	native soil microflora	sandy soil	7	1.1	2	42 560	5h 5h	urease activity urease activity			340 120	Haanstra and Doelman, 1991	2
						42	1h	phosphatase activity			840		
						560	1h	phosphatase activity			330		
						42	2h	arylsulphatase activity			2206		
						560	2h	arylsulphatase activity			121		
		sandy loam	6	6.1	9	42	5h	urease activity			>8000		
						42	1h	phosphatase activity					
						42	2h	arylsulphatase activity			>8000		
						560	2h	arylsulphatase activity			1792		
		silty loam	7.7	2.6	19	42	5h	urease activity			970		
						560	5h	urease activity			520		
						42	1h	phosphatase activity			5488		
						560	1h	phosphatase activity			235		
						42	2h	arylsulphatase activity			1882		
						560	2h	arylsulphatase activity			137		
		clay	7.5	3.4	60	42	5h	urease activity			4460		
						560	5h	urease activity			520		
						42	1h	phosphatase activity			9744		
						560	1h	phosphatase activity			5264		
						42	2h	arylsulphatase activity			9486		
						560	2h	arylsulphatase activity			1016		2

Table A6.4: Toxicity to Soil Microflora (Table 3.2.15 in De Win, 1999)													
test substance	**organism**	**medium**	**pH[a]**	**%OC[b]**	**% clay**	**Equil. Period (d)**	**duration (d)**	**endpoint**	**NOEC (µg g^{-1})**	**LOEC (µg g^{-1}) (%effect)**	**$EC_{x\geq 50}$ (µg g^{-1}) (%effect)**	**references**	**R.I.**
		peat	4.5	13.6	5	42	5h	urease activity			3260		
						560	5h	urease activity			490		
						42	2h	arylsulphatase activity			3181		
$CdCl_2$	native soil microflora	forest soil				0	24	respiration	0.01	10(36)		Bond et al., 1976	4
[a] pH-water ;[b]% OC= %OM/1.7;LT: Lowest Tested concentration;HT: Highest Tested concentration; estimated OC - clay content													

Table A6.5: Toxicity to Soil Fauna (Table 3.2.17 in De Win, 1999)

test substance	organism	medium	pH[a]	%OC[b]	clay	Equil. Period (d)	duration (d)	endpoint	NOEC (µg g^{-1})	LOEC (µg g^{-1}) (%effect)	$EC_{x\geq50}$ (µg g^{-1}) (%effect)	$LC_{x\geq50}$ (µg g^{-1}) (%effect)	references	R.I.
$Cd(NO_3)_2$	Eisenia fetida	OECD soil	6.3	5.9	20		56	mortality cocoon production	300HT 5		 46		Spurgeon et al, 1994	4 3
$CdCl_2$	Eisenia andrei	OECD soil	6.3	5.9	20	7	21	growth cocoon production juvenile/adult ratio	100HT 10	 10 18(38)			Van Gestel et al., 1993	4 3 3
$CdSO_4$	Aporrectodea caliginosa	natural forest soil	7.05	12.5		0	42	growth		5(40)LT)	68		Khalil et al., 1996a	3
$CdSO_4$	Aporrectodea caliginosa	natural forest soil	7.05	12.7		0	56	mortality cocoon production		 10(28)LT	 35	540	Khalil et al., 1996b	3 3
$Cd(NO_3)_2$	Dendrobaena rubida	C-horizon of sandy coniferous forest soil+well decomposed cattle dung	4.5 5.5 6.5 5.5	4.5-6.9	0	4 weeks	110 270	cocoon production cocoon production hatching success embryonic development	10 10	 100(47) 10LT	100(72%) 100(78%)		Bengtsson et al., 1986	2 2 2 2
$CdCl_2$	Eisenia andrei	OECD soil	6.7	5.9	20		84	growth mortality sexual development	18	 10(37)LT	33 27	 253	van Gestel et al., 1991	3 3 3
$CdCl_2$	Folsomia candida	OECD-soil	6.1*	5.9	20		42	fresh weight reproduction	50		322 51		van Gestel and Hensbergen, 1997	1
$CdCl_2$	Plectus acuminatus	OECD soil	5.5	5.9	20	5h	21	juvenile/adult ratio	32	100(24)	321		Kammenga et al., 1996	3
$CdCl_2$	Lumbricus rubellus	sandy loam soil	7.3	4.7	17	0	42	mortality weight	150 150			1000(100)	Ma, 1982	2
$CdSO_4$	nematode community	top 10cm of an arable field on a sandy soil	4.1	1.9	4		14	mortality	160HT				Korthals et al. , 1996	4
$CdSO_4$	trophic groups of nematode and microanthropod communities	top 10cm of A-horizon of mature oak-beech forest soil	3.8[a]	3.4	11	0	7	mortality	200HT				Parmelee et al., 1997	4
$CdCl_2$	Folsomia candida	OECD-soil	6.3*	5.9	20	0	42	mortality at 25% MC 45%MC 55%MC fresh weight at 25%MC 35%MC 45%MC 55%MC reproduction at 25%MC 35%MC 45%MC 55%MC	 160 320 80 160	 320 160 320	 523 640(73) 253 481 80(56)LT 80(57)LT 80(46)LT 80(43)LT	1275 868 617	van Gestel and van Diepen, 1997	2

Table A6.5: Toxicity to Soil Fauna (Table 3.2.17 in De Win, 1999)														
test substance	**organism**	**medium**	**pH[a]**	**%OC[b]**	**clay**	**Equil. Period (d)**	**duration (d)**	**endpoint**	**NOEC ($\mu g\ g^{-1}$)**	**LOEC ($\mu g\ g^{-1}$) (%effect)**	**$EC_{x\geq50}$ ($\mu g\ g^{-1}$) (%effect)**	**$LC_{x\geq50}$ ($\mu g\ g^{-1}$) (%effect)**	**references**	**R.I.**
$CdCl_2$	Eisenia fetida	sandy soil	4.1*	1	4.3	0	14	mortality				320-560	Van Gestel & van Dis.	2
													1988	
		OECD soil	7*	5.9	10.4	0		mortality				>1000HT		1
$CdCl_2$	Folsomia candida	OECD soil; T 20; 50% WHC	5.4-5.9*	1.2	20	0	35	growth			246		Crommentuijn et al., 1997b	2
								reproduction			125			
								mortality				323		
			5.4*	2.1				growth			356			
								reproduction			44			
								mortality				684		
			5.7*	3.1				growth			389			
								reproduction			82			
								mortality				758		
			5.9*	4				growth			651			
								reproduction			193			
								mortality				940		
			5.9*	4.9				growth			615			
								reproduction			130			
								mortality				890		
			5.8*	5.9				growth			824			
								reproduction			193			
								mortality				1261		
			3.1*	5.9				growth			302			
								reproduction			102			
								mortality				743		
			3.7*	5.9				growth			316			
								reproduction			102			
								mortality				1276		
			4.3*	5.9				growth			542			
								reproduction			164			
								mortality						
			5.7*	5.9				growth			697			
								reproduction			177			
			7*	5.9				growth			583			
								reproduction			113			
			7.3	5.9				growth			601			
								reproduction			306			

Table A6.5: Toxicity to Soil Fauna (Table 3.2.17 in De Win, 1999)

test substance	organism	medium	pH[a]	%OC[b]	clay	Equil. Period (d)	duration (d)	endpoint	NOEC ($\mu g\ g^{-1}$)	LOEC ($\mu g\ g^{-1}$) (%effect)	$EC_{x\geq50}$ ($\mu g\ g^{-1}$) (%effect)	$LC_{x\geq50}$ ($\mu g\ g^{-1}$) (%effect)	references	R.I.
$CdCl_2$	Folsomia candida	OECD soil	6	5.9	20	0	19	mortality	326	707		917	Crommentuijn et al., 1994	2
								number of offspring			/			
								growth	148	326	448			
							23	mortality	326	707		778		
								number of offspring	71	148	159			
								growth	148	326	376			
							26	mortality	326	707		822		
								number of offspring	71	148	204			
								growth	326		707			
							30	mortality	326	707		893		
								number of offspring	71	148	227			
								growth	148	326	541			
							35	mortality	326			854		
								number of offspring	148	326				
								growth	326	707	807			
$CdCl_2$	Folsomia candida	OECD soil	6.6	5.9	20	0	35	mortality				972	Crommentuijn et al., 1995	1
								growth			633			
								number of juveniles			153			
								population increase			152			
$Cd(NO_3)_2$	Eisenia fetida	OECD-soil	6.5	5.8	20	0	14	mortality				374	Fitzpatrick et al., 1996	1
	Lumbricus terrestris						14	mortality				256		3
$CdCl_2$	Caenorhabditis elegans	sandy loam	6.2	1	16	1	1	mortality	225	700(30)		742	Donkin and Dusenbery, 1994	2
		sandy loam	5.1	1.7	16			mortality	225	450(42)		506		
		loam	6.1	2	20			mortality	225	700(18)		922		
		clay loam	6.2	1.3	39			mortality	112	225(28)		337		
$Cd(NO_3)_2$	Eisenia fetida	OECD soil	6	5.9	20	0	14	mortality				1843	Neuhauser et al., 1985	2
$CdCl_2$	Enchytraeus albidus	OECD-soil	6.5	5.9	20	0	28	mortality				3680	Römbke, 1989	3
$Cd(NO_3)_2$	Lumbricus terrestris	artificial-soil				0	16	sperm-count			100		Cikutovic et al., 1993	4

*[a] pH-water; *pH-KCl; [b] OM = OC*1.7; LT: Lowest Tested concentration; HT: Highest Tested concentration*
MC: moisture content.

Table A6.6: Toxicity to Higher Plants (Table 3.2.19 in De Win, 1999)

test substance	plant	medium	pH[a]	%OC[b]	% clay	Equil. time prior to plant growth (d)	growing period (d)	pot (P) or field (F) trial	endpoint	NOEC ($\mu g\ g^{-1}$)	LOEC ($\mu g\ g^{-1}$) (%effect)	$EC_{x \geq 50}$ ($\mu g\ g^{-1}$) (%effect)	references	R.I.
$CdCl_2$	Picea sitchensis	peaty clay	3.3	45	40-100	2	100	P	root length	1.8		2.8(59)	Burton et al, 1984	2
$CdCl_2$	Glycine max	silt loam	4.5	1-20	0-28	4 drying and	28	P	shoot dry weight	1		10(52)	Miller et al., 1976	4/3
			6.1		0-28	rewetting				1	10(33)	100(66)		4/3
			7.0		0-28	cycles				1	10(33)	100(50)		4/3
			7.9		0-28					10	100(22)			3
			6.0		0-28					10		100(69)		3
			5.5		0-28						10(26)			3
			6.5		0-28					5	10(12)	100(47)		3
		clay loam	6.1		28-40					10		100(66)		3
		loamy sand	5.7		0-15					1		10(77)		4/3
$CdCl_2$	Phaseolus vulgaris	loamy sand	5.1	1	0-15		up to maturity	F	shoot dry weight	6.7HT			Sajwan et al., 1996	4
$CdCl_2$	Raphanus sativus	loamy sand	5.4	1-20	0-15	15	42	P	shoot dry weight	10	50(30)	70	Khan and Frankland,	3
CdO											100(29)LT	190	1983	3
$CdCl_2$	Glycine max	silty clay loam	6.7	2.5	28-40		35	P	shoot dry weight			10(50)	Haghiri, 1973	3
	Triticum aestivum						35	P	shoot dry weight		2.5(21)LT			3
	Raphanus sativus						26	P	root dry weight		2.5(36)LT			3
	Lactuca sativa						37	P	shoot dry weight		2.5(40)LT			3
	Capsicum frutescens						112	P	pepper dry weight	10HT				4
	Apium graveolem						117	P	leaf dry weight	10HT				4
$CdCl_2$	Poa pratensis	sandy	4.8	1.1	0-15	7-10	42	P	shoot dry weight	10		30(90)	Miles and Parker, 1979	4
	Liatris spicata								shoot dry weight		10(30)			
	Rhus radicans								shoot dry weight	10		30(63)		
	Andropgong scoparius								shoot dry weight		10(21)			
	Rudbeckia hirta								shoot dry weight			10(79)		
	Monarda fistulosa								shoot dry weight	10		30(68)		
$CdCl_2$	Pinus strobus	forest soil	4.8	1.1	0-15	several	120	P	shoot dry weight	15		100(57)	Kelly et al., 1979	4
	Pinus taeda	0-14cm				weeks			shoot dry weight	15		100(55)		
	Liriodendron tulipifera	sandy							shoot dry weight	15		100(78)		
	Betula alleghaniensis								shoot dry weight	15		100(82)		
	Prunus virginiana								shoot dry weight	15		100(62)		
CdOAc	Triticum aestivum	phaeosem	6.9	1.3	21	>84	28	P	shoot dry weight	7.1	14.3(15)		Reber, 1989	2
		neutral sandy hortisol	7.0	1.4	3					29	57(15)			2
		acidic cambisol	5.6	0.9	7						3.6(11)			2
$Cd(NO_3)_2$	Lactuca sativa	soil	3.9	1.2	8		42	P	shoot dry weight	3	32 (30)		Jasiewicz,1994	3
$CdCl_2$	Avena sativa	loamy sand	5.4	1-20	0-15	15	42	P	root dry weight		10(24)LT		Khan and Frankland,	3
	Triticum aestivum					15	42	P	root dry weight			50(61)LT	1984	
CdO	Triticum aestivum					15	42	P	root dry weight		100(47)LT			

Table A6.6: Toxicity to Higher Plants (Table 3.2.19 in De Win, 1999)

test substance	plant	medium	pH[a]	%OC[b]	% clay	Equil. time prior to plant growth (d)	growing period (d)	pot (P) or field (F) trial	endpoint	NOEC (µg g^{-1})	LOEC (µg g^{-1}) (%effect)	$EC_{x\geq50}$ (µg g^{-1}) (%effect)	references	R.I.
$CdSO_4$	Phaseolus vulgaris	silt-loam soil	7.5		0-28		up to	P	bean dry weight	20	40 (25)		Bingham et al., 1975	3
	Glycine max	amended with					maturity		bean dry weight	2.5	5 (25)			3
	Triticum aestivum	1% clean sludge							grain weight	20	50 (25)			3
	Zea mays								kernel weight	10	18 (25)			3
	Lycosperisicon esculentum								ripe fruit weight	80	160 (25)			3
	Cucurbita pepo								fruit weight	80	160 (25)			3
	Brassica oleracea								head weight	120	170 (25)			3
	Lactuca sativa								head weight	5	13 (25)			3
	Lepidium sativum								shoot weight	5	8 (25)			3
	Spinacia oleracea								shoot weight	1.25	4 (25)			4/3
	Brassica rapa								tuber weight	10	28 (25)			3
	Raphanus sativus								tuber weight	40	96 (25)			3
	Daucus carota								tuber weight	10	20 (25)			3
	Oryza sativa								grain weight	160				3
$CdSO_4$	Lactuca sativa	surface soils	4.8	2.6	8.3	14	63	P	shoot dry weight	40	80(20)	260	Mahler et al., 1978	3
	Beta vulgaris	amended with				>77			shoot dry weight	20	80(35)	110		
	Lactuca sativa	1% clean sludge	5.0	3.3	14.6	14				40	160(22)	270		
	Beta vulgaris					>77				20	80(30)	135		
	Lactuca sativa		5.3	0.9	8.9	14				10	40(35)	100		
	Beta vulgaris					>77				40	80(25)	110		
	Lactuca sativa		5.7	3.0	37.5	14				20	80(25)	160		
	Beta vulgaris					>77				40	160(40)	185		
	Lactuca sativa		7.4	1.4	18.7	14				20	80(35)	195		
	Beta vulgaris					>77				40	160(25)	320		
	Lactuca sativa		7.5	0.6	4.4	14				2.5	10(25)	80		
	Beta vulgaris					>77				20	80(25)	105		
	Lactuca sativa		7.7	0.9	40.6	14				5	10(30)	18		
	Beta vulgaris					>77				40	160(35)	195		
	Lactuca sativa		7.8	0.7	15.2	14				10	40(38)	58		
	Beta vulgaris					>77				80		320		
$CdSO_4$	Zea mays	calcareous chernozem		1.8			season	F	grain yield	456HT			Kádár, 1995	4
$Cd(NO_3)_2$	Nicotinia tabacum	sandy clay	5.3	0.9	9.4	21	60	P	total plant dry weight	5.4			Mench et al., 1989	4
	Nicotinia rustica								total plant dry weight	5.4				
	Zea mays								total plant dry weight	0.4	5.4(21)			

[a] pH-water; *pH-KCl; [b] OM = OC*1.7; LT: Lowest Tested concentration; HT: Highest Tested concentration; estimated OC - clay content

Table A6.7: Toxicity to Benthic Organisms (Table 3.2.25 in De Win, 1999)

test substance	organism	medium	test conditions	Nominal/ Measured	Equilibration period (d)	Duration (d)	endpoint	NOEC µg g^{-1}_{dw}	NOEC µg L^{-1}	LOEC µg g^{-1}_{dw}	LOEC µg L^{-1}	$EC_{x\geq50}$ µg g^{-1}_{dw}	$EC_{x\geq50}$ µg L^{-1}	references	R.I.
$CdCl_2$		uncontaminated freshwater sediment from:	semi-static; sed./water:1:3 (vol)	M-total	/	10	mortality							Di Toro et al., 1992	
	Helisoma sp.	Pequaywan Lake	AVS: 42 µmol/g												
		East River	AVS: 8.8 µmol/g					3390				4520			3
		West Bearskin Lake	AVS: 3.6 µmol/g					2260				3340			3
	Lumbricus variegatus	Pequaywan Lake	AVS: 42 µmol/g					340				790			3
		East River	AVS: 8.8 µmol/g					3390				4520			3
		West Bearskin Lake	AVS: 3.6 µmol/g					680				1130			3
	Hyalella azteca	contaminated freshwater sediment from Foundry cove	AVS: 0.1-47 µmol/g; SEM (Ni+Cd) 0.3-1000 µmol/g					340				680			3
												17 (100)			4
$CdCl_2$	Hyalella azteca	natural sediment (Soap Creek Pond - Oregon State University); 200 ml spiked natural sediment + 800 ml well water	static; T 19°C; sediment characteristics: 3% organic carbon, 15% sand, 29% silt, 56% clay; water characteristics: pH 7.1, H 54 mg L^{-1} $CaCO_3$, BC < 0.5 µg L^{-1}. AVS unknown	M (diss.)	0.5	4	mortality	128	1.1	256(26)	3.2		6.6	Nebeker et al., 1986b	3
$CdCl_2$	Rana pipiens	natural stream sediment; 250 g_{dw} sediment + 25 ml Cd-solution or distilled deionized water (control) +350 ml reconstituted water	DO 6.6-8.1 mg L^{-1}, T 22.1-22.5 °C, pH 7.9-8.4; sed: OM 2.3%, Cd_T 1.02 mg kg^{-1}, Zn_T 108.2 mg kg^{-1}, Fe_T 5.52%; 5.52% sand, 35.4% silt, 12% clay	M	0.42	7	mortality	1074	77					Francis et al., 1984	4
	Micropterus salmoides							540	22	1079 (14)	44 (14)				3
$CdCl_2$			sediment AVS content:	M	4	10	mortality							Carlson et al., 1991	2
	Lumbricus variegatus	Pequaywan Lake	38-32 µmol/g					3000				6000			
		East River sediment	6.8-7.3 µmol/g					800				1400			
		West Bearskin Lake	2.8-3.2 µmol/g					380				700			
	Helisoma sp.	Pequaywan Lake	38-32 µmol/g												
		East River sediment	6.8-7.3 µmol/g					3000				6200			
		West Bearskin Lake	2.8-3.2 µmol/g					2300				4100			
			test water: sand filtered Lake Superior water; T 21-22 °C, alkalinity 45-46 mg L^{-1}, hardness 44-45 mg L^{-1}, pH 7.9-8, dissolved oxygen concentration >6 mg L^{-1}, continuous flow; T 23°C; 1.5L Cd sol. + 1L sed.					380				810			

Table A6.7: Toxicity to Benthic Organisms (Table 3.2.25 in De Win, 1999)

test substance	organism	medium	test conditions	Nominal/ Measured	Equilibration period (d)	Duration (d)	endpoint	NOEC µg g^{-1}_{dw}	NOEC µg L^{-1}	LOEC µg g^{-1}_{dw}	LOEC µg L^{-1}	$EC_{x\geq50}$ µg g^{-1}_{dw}	$EC_{x\geq50}$ µg L^{-1}	references	R.I.
Cd^{2+}	Hyalella azteca	natural sediment: Foundry cove	% total organic carbon: 0.55-16.4 µg/g, total Cd: 0.4-38900 µg/g, total Cu: 18-143 µg/g, total Ni: 18-31500 µg/g, total Pb: 6.1-357 µg/g, total Zn: 65-403 µg/g, sum metals: 2.9-893, SEM: 0.2-779 µmol/g, AVS: 0.4-64.6 µmol/g, SEM/AVS: 0.02-139	M-total	/	10	mortality	72		363(20)				Hansen et al., 1996	4

TOC: total organic carbon; AVS: acid volatile sulphides; H: water hardness (mg CaCO3 L-1)