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LIST OF SYNONYMS, CONCEPTS AND ABBREVIATIONS

Collective analysis  Chemical analysis whereby only the total amount of mono-, di- and tri-chlorine etc. is determined
Congeners  Compounds within the same series/class of compounds (e.g. 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD are PCDD congeners)
Dioxins  The seventeen 2,3,7,8-chlorosubstituted polychlorodibenzo-p-dioxins (PCDDs) and polychlorodibenzofurans (PCDFs)
GI '89  Guideline Incineration 1989
Isomers  Compounds with the same molecular formula (e.g. the isomers 1,2,3,7,8-PeCDD and 2,3,4,7,8-HeSDF both have the molecular formula C_{12}H_5Cl_5O)
PCDDs  polychlorodibenzo-p-dioxins; group of 75 chemical compounds with increasing chlorination level (molecular formula: C_{12}H_{x}Cl_{8-x}O_2, where x=1-8); where in this report PCDDs are mentioned, the seven 2,3,7,8-chlorosubstituted dibenzo-p-dioxins are meant, unless stated otherwise
PCDFs  polychlorodibenzofurans; a group of 135 chemical compounds with increasing chlorination level (molecular formula: C_{12}H_{x}Cl_{8-x}O, where x=1-8); where in this report PCDFs are mentioned the ten 2,3,7,8-chlorosubstituted dibenzofurans are meant, unless stated otherwise
PCBs  polychlorobiphenyls; group consisting of 209 chemical compounds with increasing chlorination level (molecular formula: C_{12}H_{x}Cl_{10-x}, where x=1-10); to distinguish between the 209 PCBs the so-called IUPAC numeration is frequently used
planar PCBs  PCB-congeners which spatially can adopt a planar structure. It is assumed that these compounds act as toxins similar to 2,3,7,8-TCDD. The PCBs with IUPAC numbers 77 (3,3',4,4'-TeCB), 126 (3,3',4,4',5-PeCB) and 169 (3,3',4,4',5,5'-HxCB) belong to these planar PCBs
TDI  Tolerable Daily Intake
TEF  toxicity equivalency factor in relation to 2,3,7,8-TCDD as proposed by the Working Group TEF (Van Zorge et al., 1989)
TEQ  2,3,7,8-TCDD toxicity equivalents
I-TEQ  TEQ-value calculated by adding up the values of the seventeen 2,3,7,8-chlorosubstituted PCDDs and PCDFs, each multiplied by the TEF-value determined for the compound concerned
und  under normal conditions dry
WI  Waste Incinerator
WTP  Wastewater Treatment Plant
SUMMARY

This document contains data on dioxins, concerning sources and distributions, risks based on a comparison of exposure levels and hazardous concentrations, as well as the possibilities for (further) reducing these risks.

The name dioxins stands for the collective of polychlorodibenzo-p-dioxins and -dibenzofurans (PCDDs and PCDFs). This is a group of 210 tricyclic compounds, the toxicity of which is ascribed to seventeen congeners with the 2,3,7,8-chloro-substitution pattern. In the Netherlands the collective toxicity of these mixtures, using so-called International Toxicity Equivalence Factors (I-TEFs), is expressed in toxic equivalence (I-TEQs) of the 2,3,7,8-TCDD, which is the most extensively studied dioxin.

Dioxins have received considerable attention since the end of the 1970s. This attention has arisen from the identification of PCDDs and PCDFs in emissions from waste incinerators (WIs). Studies on emissions from WIs completed in 1989 showed that, based on calculations, the tolerable daily intake (TDI) of PCDDs and PCDFs were likely to have been exceeded locally. This was confirmed by elevated dioxin levels found in bovine milk and dairy products from locations in the vicinity of WIs. On the basis of the above-mentioned emission and emission control studies and within the framework of the Guideline Incineration 1989, an emission limit value of 0.1 ng I-TEQ. m\(^{-3}\) und (under normal conditions dry) was determined for incinerators, which will come into effect as from 30 November 1993. At the same time, a maximally acceptable value (6 pg I-TEQ. g\(^{-1}\) milkfat) was established for milk and milk(fat)products, based on possible exceedings of the TDI and departing from average consumption patterns. No (official) standards were set for the environment.

Only limited data are required for the quantification of dioxin emissions. Dioxins are not produced commercially and have no application; they are produced in incineration processes and as (undesired) byproducts in industrial processes. Although many potential sources exist, the study was mostly focused on incinerators for domestic and comparable industrial waste, the greatest dioxin source in the early 1990s (appr. 2/3 of the total emission). Other important sources are incineration of chemical waste, evaporation from wood preserved with pentachlorophenol and the basic metal industry. The emissions to air are the most significant (appr. 600 g I-TEQ/year). The direct emissions to surface water (appr. 4 g I-TEQ/year) and soil (appr. 3 g I-TEQ/year) are slight as compared to the contribution from other sources (situation end 1990). Partly due to this, deposition (appr. 325 g I-TEQ/year) and the application of sludge (appr. 7 g I-TEQ/year) contribute significantly to the total load of the
soil in the Netherlands, whereas for the load of surface water (especially via the Rhine and the Meuse), apart from the contribution via deposition (appr. 45 g I-TEQ/year), the contribution of trans-boundary import (appr. 55-80 g I-TEQ/year) is relatively large. Departing from the estimated and calculated data on 1990 the amount of dioxins in the Dutch soil appears to have increased by appr. 330 g I-TEQ and in sediments by more than 65-90 g TEQ that year.

An overview of the flows and the accumulation of dioxins in the environment of the Netherlands is given in figure A.

![Diagram](image)

**Figure A**  Schematic overview of dioxin emissions in the Netherlands, contributions from abroad, and the flows and reservoirs in the environment of the Netherlands (in grammes I-TEQ per year, situation end 1990)

Measuring low levels of dioxins is complex and labour-intensive and only since the end of the eighties it is possible to carry out these (relatively costly) measurements in a reliable isomer-specific way. As a result information on the occurrence of dioxins is extremely limited and usually estimated on incidental measurements and data from abroad, in combination with calculations.

With regard to ambient concentration levels, with due caution, the following may be stated. In soil (topmost 10 cm) the level is estimated at 2 to 5 ng I-TEQ kg⁻¹ d.m., whereas in the
vicinity of WIs levels of up to appr. 250 ng I-TEQ kg$^{-1}$ d.m. are to be expected. Based on physico-chemical properties of dioxins it is assumed that the dioxin levels (dissolved) in groundwater and surface water are in general extremely low (< 0.005 ng.l$^{-1}$). Concentrations in sediments suggest a level of appr. 10 ng I-TEQ kg$^{-1}$ d.m., however, locally (near sources or locally elevated sedimentation) levels are found of over 100 ng I-TEQ kg$^{-1}$ d.m. (Laurenhaven, Chemiehaven). The annual average concentration in air amounts to 0.01-0.04 pg I-TEQ m$^{-3}$ in rural areas, 0.05-0.1 pg I-TEQ m$^{-3}$ in urban/industrial areas up to appr. 1 pg I-TEQ m$^{-3}$ near point sources.

For humans sufficient data for the determination of a recommended value are only available for the most toxic congener, 2,3,7,8-TCDD. The limited data for the other congeners are incorporated in the toxicity equivalency factors (TEF). Although 2,3,7,8-TCDD has been found to be carcinogenic in experimental animals there is insufficient evidence for carcinogenicity in humans. Epidemiologic studies have shown chloracne to be the only effect consistently related to 2,3,7,8-TCDD-exposure. Based on data from genotoxicity studies, mechanistic studies and the mode of action of 2,3,7,8-TCDD, it was departed from a threshold value in the determination of the recommended value. During the WHO/EURO meeting held in December 1990, a tolerable daily intake (TDI) of 10 pg.kg$^{-1}$ body weight was derived for 2,3,7,8-TCDD via a pharmaco-kinetic approach and using a safety factor of 10. This WHO recommendation is endorsed. With respect to the assessment of mixtures it is suggested to interpret the recommended value as 10 pg I-TEQ per kg body weight per day. For the general population food is the main source of exposure; for adults the contribution via food is estimated to be at least 90 - 95%, milk and milk products as well as products on a basis of fish oil contributing considerably. The median intake via food amounts to appr. 70 pg I-TEQ per day for adults, corresponding to appr. 1 pg I-TEQ per kg body weight per day. For children the daily intake per kg body weight is higher (appr. 3 pg I-TEQ). At the current exposure levels of PCDDs and PCDFs, from food as well as via other sources, adverse effects are not to be expected either in children or in adults. Partly due to measures taken in the framework of the Commodities Act Dioxins in milk, exceeding of the TDI is considered unlikely, even in the vicinity of sources such as WIs.

In breast-fed infants the recommended value is exceeded. The average daily intake is estimated to be appr. 150 pg I-TEQ per kg body weight. At present, however, insufficient data are available to assess the effects of such exceedings.

Based on limited ecotoxicity data indicative maximally tolerable risk levels (MTRs) have
been derived for soil, sediments and surface water. For soil and water two MTRs have been derived, one for the protection of organisms living in the compartment concerned (MTR_{ecosystem}) and one for the protection of predators (mammals, birds) which prey on organisms from that compartment (MTR_{predator}). In addition, using the equilibrium partition method, this has resulted in two MTRs for sediments. Since for groundwater neither ecotoxicological data nor measurements are available, a risk assessment is not possible. However, because of the immobile nature of PCDDs and PCDFs in the soil, groundwater organisms are unlikely to form a group at risk.

For the soil a MTR_{ecosystem} has been derived of 500,000 ng I-TEQ.kg^{-1} d.m. and a MTR_{predator} of 3 ng I-TEQ.kg^{-1} d.m. The MTR_{ecosystem} is not known to be exceeded anywhere in the Netherlands. The MTR_{predator} corresponds with background levels measured in the Netherlands as well as abroad. In the Netherlands, in the vicinity of WIs, levels have been found which are 8-80 times higher than the MTR_{predator}, whereas locally, near illegal incineration sites of cables and cars and in the Volgermeer waste tip, levels have been found of up to 32,000 times higher than the MTR_{predator}. At such levels effects are likely to occur in predators preying on worms.

Based on available data it is concluded that the risk for soil ecosystems on a national and regional scale is limited. On a regional scale (in the vicinity of WIs) and a local scale (particularly at illegal incineration sites) there is possibly, respectively very likely, an unacceptable risk for predators preying on worms. The same may possibly apply to other carnivorous mammals and birds. However, attention should be drawn to the uncertainties in the MTR_{predator} and to the fact that heavily polluted sites are usually relatively small as compared to the entire feeding grounds of predators.

For surface water a MTR_{ecosystem} has been determined of 0.0012 ng I-TEQ.l^{-1} (dissolved fraction) and a MTR_{predator} of 0.00005 ng I-TEQ.l^{-1} (dissolved fraction). Owing to the lack of measuring data in water a direct risk assessment is not possible. Although for sediments measuring data are available, there are no toxicity data for benthic organisms so that also in this case a direct risk assessment is not possible. For sediments, by means of the equilibrium partition method, MTRs have been determined of 378 ng I-TEQ.kg^{-1} d.m. (based on the MTR_{ecosystem}) and of 15 ng I-TEQ.kg^{-1} d.m. (based on the MTR_{predator}). With the exception of several heavily loaded harbours, the MTR_{ecosystem} is generally not exceeded in the Netherlands. From 1983 - 1985 in almost all big rivers, canals and lakes the MTR_{predator} was exceeded sometimes up to several tens of times (in some heavily loaded harbours to more than 150 times). In estuaries and the marine environment the level (1987-1990) approximates the MTR_{predator}. Based on the above-mentioned data it is concluded that the risk for aquatic
ecosystems is in general limited, with the exception of several heavily polluted locations (harbours). On a national and a local scale there is a possible, probable, respectively, unacceptable risk for fish-eating predators. However, here again, attention should be drawn to the uncertainties in the MTR\textsubscript{predator} and to the fact that heavily polluted locations are usually relatively small as compared to the entire feeding grounds of predators.

Current standard-setting is particularly aimed at emission reduction of specific incinerators and at the protection of humans by means of a product standard. In the Guideline Incineration 1989 (GI'89), for new as well as for modernized WIs, based on the best available techniques, an emission limit value of 0.1 ng I-TEQ.m\textsuperscript{-3} und has been determined. Assuming the emissions from other sources and those from abroad will remain unchanged, it may be calculated that in this situation the contribution of WI emitted dioxins to cow's milk from dairy cattle farms in the near vicinity will be negligible. Based on this outcome it is concluded that the measures taken in the framework of GI'89 will lead to a situation where humans, even at a local scale, are sufficiently protected. Under similar conditions the levels in the soil will increase by appr. 0.1 ng I-TEQ.kg\textsuperscript{-1} d.m. annually. Soil ecosystems will hardly be affected by this increase. However, since the MTR\textsubscript{predator} is already at the background level, any increase will lead to a higher risk for predators preying on worms. Emissions to air will decrease through autonomous developments (implemented policies), it is therefore expected that in the year 2000 dioxin emissions will have decreased by appr. 75\% from appr. 600 to appr. 125 g I-TEQ per year. Significant sources will then be the application of pesticides, metal industry and wood combustion. Direct emissions to water and soil remain at the same level. At a deposition rate of 12 ng I-TEQ.m\textsuperscript{-2}.year\textsuperscript{-1} (situation as from the date the Guideline Incineration 1989 came into effect) it is estimated that the concentrations in the soil will increase annually by appr. 0.1 ng I-TEQ.kg\textsuperscript{-1} d.m. Also in Germany, Austria and Sweden an emission limit value of 0.1 ng I-TEQ.m\textsuperscript{-3} is in effect. At a European level measures have not yet been taken. Therefore it is expected that the influence from sources abroad will only slowly decrease. The effect of emission control measures on dioxin levels in solid residues, still needs to be studied. It is not known whether the residuals can be used without causing everlasting concerns or that they should be discharged under IBC-criteria control.

Additional measures may result in a further decrease of the emissions to air and the soil of appr. 100 and 0.2 g I-TEQ per year, respectively, further measures concerning PCP-polluted wood not being taken into account.
INTRODUCTION

Government effect-oriented environmental policy is aimed at achieving and maintaining an environmental quality which guarantees the health and well-being of people and the preservation of animals, plants, goods and patterns of utilization in a general sense. In that framework integrated criteria documents have by now been drawn up for most priority substances. These documents aim at providing information which serves as a scientific basis for the formulation of the effect-oriented environmental policy.

Integrated criteria documents are drawn up under the auspices of the National Institute of Public Health and Environmental Protection (RIVM). This document was prepared by the RIVM in cooperation with the Consultancy Bongaerts, Kuyper and Huiswaard (BKH). Government, industry and representatives of scientific institutes were involved in the drawing up. The content of this document has been integrally checked by a review committee of the RIVM. Support in the preparation of this document was rendered by an interdepartmental Counselling group consisting of staff members of the Ministries of Public Housing, Physical Planning and the Environment (VROM), of Transport and Public Works (V&W), of Welfare, Health and Cultural Affairs (WVC) and of Agriculture, Nature Conservation and Fisheries (LNV). Industry, by means of the Liaison Group Criteria Documents of the Department of Environment and Physical Planning of the Dutch Labour Unions VNO and NCW, provided important, often confidential information. In cases of differences of opinion an addendum may be added to the document, drawn up under the auspices of the Liaison Group. This is also possible for environmental groups through the Foundation Nature and the Environment.

This document concerns dioxins, which is a collective name for polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDDs and PCDFs), a series of chlorinated aromatic hydrocarbons, which has received considerable attention since the Seveso (Italy) disaster. In 1989 dioxins were prominent in the news media following pollution of milk from the Lickebaart area in the vicinity of the Rijnmond waste incinerator. It is known that dioxins are formed as undesired by-products (as a mixture) during some chemical processes, waste incineration being the most significant. In all there are more than 200 different chlorinated dibenzodioxins and dibenzofurans. The difference between the congeners is the number and the position of the chlorine atoms in the molecule. This difference may result in considerable
variance in the extent of toxicity. Some dioxins are hardly toxic, and others are extremely so, 2,3,7,8-TCDD belonging to the latter category. Seventeen dioxins and dibenzofurans appear to be responsible for the extent of toxicity of mixtures of these compounds. The toxicity of such mixtures is usually expressed in toxic equivalents (TEQ) of the 2,3,7,8-TCDD, the most extensively studied dioxin.

In this criteria document attention will primarily be given to the 17 toxic PCDD- and PCDF-congeners. Other compounds which show an action similar to that of 2,3,7,8-TCDD, such as planar and mono-ortho-planar PCBs and bromo/chloro dioxins, will only be touched upon in the toxicological assessment. In accordance with the assignment the following items will be emphasized: emissions, concentrations in the environment, exposure levels, the biological availability and behaviour in biota, as well as human toxicity. Important items such as ecotoxicity, emission control measures and the effects of emissions from abroad will also be discussed, however, less extensively. With respect to various sections, as much information as possible will be drawn from existing reports and review articles; the original literature will only be consulted in case data or conclusions are inconsistent in the review articles or when specific data need to be used for the determination of the toxicological recommended values.

Concerning the occurrence of dioxins, a CCRX document has recently been drawn up. With respect to studies on emissions, distribution and risks of dioxins as a result of domestic waste incineration, a review article has been prepared in 1992 (HIMH, TNO and RIVM) summarizing and evaluating the various data from studies carried out in the previous years. These data contributed considerably to the information necessary for the preparation of this document. In order to get an understanding of the problems posed by dioxins as quickly as possible the various research data have been combined with those on other (potential) dioxin sources (as far as available by the end of 1991) and the current views on both the human toxicological and ecotoxicological fields have been summarized. Subsequently, based on the available data, a risk assessment for humans and the ecosystem is given, and the developments to be expected in the years to come are discussed.
1. PROPERTIES AND EXISTING STANDARDS

1.1 PROPERTIES

The polychlorodibenzo-\textit{para}-dioxins and dibenzofurans (abbreviated to PCDDs and PCDFs, respectively) together form a series consisting of 210 tricyclic, planar, aromatic compounds (75 PCDDs and 135 PCDFs, collectively also referred to as 'dioxins'). Figure 1.1 shows the tricyclic, planar structure of these compounds. The basic structure is formed by two benzene rings which, in the dibenzodioxins, are connected by two oxygen atoms and, in the dibenzofurans, by one oxygen atom. On each benzene ring one or more (maximally 4) hydrogen atoms are substituted by chlorine atoms. Table 1.1 shows the number of isomers possible as a function of the number of chlorine atoms.

![Figure 1.1](image)

\textit{Figure 1.1} Structural formulas of PCDDs (A) and PCDFs (B). $x+y=1-8$

<p>| Number of PCDD- and PCDT-isomers as a function of the number of chlorine atoms |
|-----------------------------------|---------------------------------|-----------------|</p>
<table>
<thead>
<tr>
<th>number Cl-atoms</th>
<th>number CDDs</th>
<th>number CDFs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>10</td>
<td>16</td>
</tr>
<tr>
<td>3</td>
<td>14</td>
<td>28</td>
</tr>
<tr>
<td>4</td>
<td>22</td>
<td>38</td>
</tr>
<tr>
<td>5</td>
<td>14</td>
<td>28</td>
</tr>
<tr>
<td>6</td>
<td>10</td>
<td>16</td>
</tr>
<tr>
<td>7</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>135</td>
</tr>
</tbody>
</table>
In table 1.2 the names and molecular formulas for several PCDD- and PCDF-isomers from each homologous series are summarized. The Chemical Abstracts Service registration numbers are also given for the seventeen toxic 2,3,7,8-chlorine substituted PCDDs and PCDFs. The term 'isomers' refers to compounds with the same molecular formulas, and the term 'congeners' to those within the same series of compounds (PCDDs, PCDFs), however, with a different number of chlorine atoms. Table 1.2 gives an overview of several basic data relevant to the environment. The data have been derived from recent review articles by Van der Bolt (1990), WHO (1989) and Shiu et al. (1988).

PCDDs and PCDFs show similar physical and chemical properties. These properties may be summarized as follows: (Shiu et al. 1988; WHO, 1989; Van der Bolt, 1990)

- **Structure and Kow.** PCDDs and PCDFs are nonpolar and lipophilic compounds. The structure (figure 1.1) shows that the compounds contain no unstable functional groups. The frequently used lipophility parameter Log (K\text{ow}) increases with the number of chlorine atoms and ranges from appr. 4.75 for MCDD and 6.80 for 2,3,7,8-TCDD to appr. 8.20 for OCDD (Shiu et al., 1988, table 1.3).

- **Reactivity.** PCDDs and PCDFs are generally very stable in the presence of strong acids and bases as well as in the presence of highly reductive and oxidative agents. Substitution reactions and photochemical dechlorination have been observed for 2,3,7,8-TCDD. OCDD is decomposed in alkaline environment at elevated temperatures (Albro and Corbett, 1977).

- **Thermostability.** PCDDs and PCDFs remain stable at extremely high temperatures. Decomposition of 2,3,7,8-PCDD only takes place at temperatures above 750°C.

- **Solubility.** The compounds are hardly or not at all soluble in (pure) water. Solubility decreases with an increasing number of chlorine atoms (table 1.3) and varies from less than 1 pg.l\text{−1} (OCDD) to appr. 400 μg.l\text{−1} for MCDD (appr. 200 ng.l\text{−1} for 2,3,7,8-PCDD).

- **Volatility.** Volatility decreases with an increasing number of chlorine atoms. The relative volatility can be described with the Henry constant (H, table 1.3) which can be derived for compounds with extremely low solubility from the ratio of the (measured) vapour pressure of the pure compound and the (measured) water solubility at the same temperature. At an increasing number of chlorine atoms a decrease in the Henry constant can be derived from the reported data.

The properties of the PCDFs are less extensively studied than those of the PCDDs (table
The scanty data on the water solubility and the $K_{ow}$-values are, however, in the same range and are more or less similar to those for the corresponding dioxins.

<table>
<thead>
<tr>
<th>Compound</th>
<th>Abbreviation</th>
<th>Mol. weight formula</th>
<th>CAS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dibenzo-p-dioxin</td>
<td>DD</td>
<td>184.2 $\text{C}_{12}\text{H}_8\text{O}_2$</td>
<td></td>
</tr>
<tr>
<td>1-chloro-</td>
<td>1-MCDD</td>
<td>218.6 $\text{C}_{12}\text{H}_7\text{Cl}_2\text{O}_2$</td>
<td></td>
</tr>
<tr>
<td>2,3-dichloro-</td>
<td>2,3-DCDD</td>
<td>253.1 $\text{C}_{12}\text{H}_6\text{Cl}_3\text{O}_2$</td>
<td></td>
</tr>
<tr>
<td>1,2,4-trichloro-</td>
<td>1,2,4-TrCDD</td>
<td>287.5 $\text{C}_{12}\text{H}_5\text{Cl}_4\text{O}_2$</td>
<td></td>
</tr>
<tr>
<td>2,3,7,8-tetrachloro-</td>
<td>2,3,7,8-TCDD</td>
<td>322.0 $\text{C}_{12}\text{H}_4\text{Cl}_4\text{O}_2$</td>
<td>1746-01-6</td>
</tr>
<tr>
<td>1,2,3,7,8-pentachloro-</td>
<td>1,2,3,7,8-PeCDD</td>
<td>356.4 $\text{C}_{12}\text{H}_3\text{Cl}_5\text{O}_2$</td>
<td>40321-76-4</td>
</tr>
<tr>
<td>1,2,3,4,7,8-hexachloro-</td>
<td>1,2,3,4,7,8-HxCDD</td>
<td>390.9 $\text{C}_{12}\text{H}_2\text{Cl}_6\text{O}_2$</td>
<td>39227-28-6</td>
</tr>
<tr>
<td>1,2,3,6,7,8-hexachloro-</td>
<td>1,2,3,6,7,8-HxCDD</td>
<td>390.9 $\text{C}_{12}\text{H}_2\text{Cl}_6\text{O}_2$</td>
<td>57653-85-7</td>
</tr>
<tr>
<td>1,2,3,7,8,9-hexachloro-</td>
<td>1,2,3,7,8,9-HxCDD</td>
<td>390.9 $\text{C}_{12}\text{H}_2\text{Cl}_6\text{O}_2$</td>
<td>19408-74-3</td>
</tr>
<tr>
<td>1,2,3,4,6,7,8-heptachloro-octachloro-</td>
<td>1,2,3,4,6,7,8-HpCDD</td>
<td>425.3 $\text{C}_{12}\text{HCl}_2\text{O}_2$</td>
<td>35822-46-9</td>
</tr>
<tr>
<td>Dibenzo[a]furan</td>
<td>DF</td>
<td>168.2 $\text{C}_{12}\text{H}_8\text{O}$</td>
<td></td>
</tr>
<tr>
<td>1-chloro-</td>
<td>1-MCDF</td>
<td>202.6 $\text{C}_{12}\text{H}_7\text{Cl}_2\text{O}$</td>
<td></td>
</tr>
<tr>
<td>2,8-dichloro-</td>
<td>2,8-DCDF</td>
<td>237.1 $\text{C}_{12}\text{H}_6\text{Cl}_3\text{O}_2$</td>
<td></td>
</tr>
<tr>
<td>1,2,3-trichloro-</td>
<td>1,2,3-TrCDF</td>
<td>271.5 $\text{C}_{12}\text{H}_5\text{Cl}_4\text{O}_2$</td>
<td></td>
</tr>
<tr>
<td>2,3,7,8-tetrachloro-</td>
<td>2,3,7,8-TCDF</td>
<td>306.0 $\text{C}_{12}\text{H}_4\text{Cl}_4\text{O}_2$</td>
<td>51207-31-9</td>
</tr>
<tr>
<td>1,2,3,7,8-pentachloro-</td>
<td>1,2,3,7,8-PeCDF</td>
<td>340.4 $\text{C}_{12}\text{H}_3\text{Cl}_5\text{O}_2$</td>
<td>57117-41-6</td>
</tr>
<tr>
<td>2,3,4,7,8-pentachloro-</td>
<td>2,3,4,7,8-PeCDF</td>
<td>340.4 $\text{C}_{12}\text{H}_3\text{Cl}_5\text{O}_2$</td>
<td>57117-31-4</td>
</tr>
<tr>
<td>1,2,3,4,7,8-hexachloro-</td>
<td>1,2,3,4,7,8-HxCDD</td>
<td>374.9 $\text{C}_{12}\text{H}_2\text{Cl}_6\text{O}_2$</td>
<td>70648-26-9</td>
</tr>
<tr>
<td>1,2,3,6,7,8-hexachloro-</td>
<td>1,2,3,6,7,8-HxCDD</td>
<td>374.9 $\text{C}_{12}\text{H}_2\text{Cl}_6\text{O}_2$</td>
<td>57117-44-9</td>
</tr>
<tr>
<td>1,2,3,7,8,9-hexachloro-</td>
<td>1,2,3,7,8,9-HxCDD</td>
<td>374.9 $\text{C}_{12}\text{H}_2\text{Cl}_6\text{O}_2$</td>
<td>72918-38-8</td>
</tr>
<tr>
<td>2,3,4,6,7,8-hexachloro-</td>
<td>2,3,4,6,7,8-HxCDD</td>
<td>374.9 $\text{C}_{12}\text{H}_2\text{Cl}_6\text{O}_2$</td>
<td>60851-34-5</td>
</tr>
<tr>
<td>1,2,3,4,6,7,8-heptachloro-octachloro-</td>
<td>1,2,3,4,6,7,8-HpCDD</td>
<td>409.3 $\text{C}_{12}\text{HCl}_2\text{O}_2$</td>
<td>67562-39-4</td>
</tr>
<tr>
<td>1,2,3,4,7,8,9-heptachloro-octachloro-</td>
<td>1,2,3,4,7,8,9-HpCDD</td>
<td>409.3 $\text{C}_{12}\text{HCl}_2\text{O}_2$</td>
<td>55673-89-7</td>
</tr>
<tr>
<td>OCDF</td>
<td></td>
<td>443.8 $\text{C}_{12}\text{Cl}_6\text{O}_2$</td>
<td>39001-02-0</td>
</tr>
</tbody>
</table>
### Table 1.3 Physico-chemical properties of several PCDDs and PCDFs

<table>
<thead>
<tr>
<th>Compound</th>
<th>Melting point (°C) (25°C, ng/l)</th>
<th>Solubility</th>
<th>Log(Kow)</th>
<th>Vapour pressure solid</th>
<th>H(25°C)</th>
<th>liquid</th>
<th>Pa.m^3.mol^1</th>
</tr>
</thead>
<tbody>
<tr>
<td>DD</td>
<td>868000</td>
<td>4.3</td>
<td></td>
<td></td>
<td>5.5×10^2</td>
<td>5.1×10^1</td>
<td>12.29</td>
</tr>
<tr>
<td>1,3,4,7-TCDD</td>
<td>164</td>
<td>14900</td>
<td>5.6</td>
<td></td>
<td>3.9×10^4</td>
<td>9.3×10^3</td>
<td>6.61</td>
</tr>
<tr>
<td>1,2,4,9-TCDD</td>
<td>128</td>
<td>8410</td>
<td>6.4</td>
<td></td>
<td>1.0×10^4</td>
<td>4.8×10^2</td>
<td>3.84</td>
</tr>
<tr>
<td>1,2,3,4,7,8-TCDD</td>
<td>219</td>
<td>6700</td>
<td>6.9</td>
<td></td>
<td>1.0×10^4</td>
<td>5.8×10^3</td>
<td>0.77</td>
</tr>
<tr>
<td>1,2,3,4,7,8-TCDD</td>
<td>219</td>
<td>660</td>
<td>8.3</td>
<td></td>
<td>8.3×10^3</td>
<td>2.8×10^2</td>
<td>1.29</td>
</tr>
<tr>
<td>1,2,3,4,7,8,9-TCDD</td>
<td>275</td>
<td>2681</td>
<td>7.4</td>
<td></td>
<td>8.3×10^3</td>
<td>4.3×10^2</td>
<td>0.26</td>
</tr>
<tr>
<td>OCDD</td>
<td>305</td>
<td>200</td>
<td>6.8</td>
<td></td>
<td>4.5×10^5</td>
<td>2.6×10^3</td>
<td>1.32</td>
</tr>
<tr>
<td>OCDD</td>
<td>196</td>
<td>120</td>
<td>7.4</td>
<td></td>
<td>8.3×10^3</td>
<td>4.3×10^2</td>
<td>0.26</td>
</tr>
<tr>
<td>1,2,3,4,7,8-HxCDD</td>
<td>275</td>
<td>24 (20°C)</td>
<td>8.0</td>
<td></td>
<td>1.8×10^7</td>
<td>1.8×10^7</td>
<td>0.13</td>
</tr>
<tr>
<td>1,2,3,4,7,8,9-HcCDD</td>
<td>239</td>
<td>2.4 (20°C)</td>
<td>8.1</td>
<td></td>
<td>8.7×10^6</td>
<td>8.1×10^6</td>
<td>0.26</td>
</tr>
<tr>
<td>OCDF</td>
<td>330</td>
<td>0.4 (20°C)</td>
<td>8.1</td>
<td></td>
<td>1×10^-10</td>
<td>1.2×10^7</td>
<td>0.68</td>
</tr>
</tbody>
</table>

**References:**
1.2 CALCULATIONS OF 2,3,7,8-TCDD-EQUIVALENTS (TEQ)

In table 1.4 several examples are given of approaches which have been developed over time to relate the toxicity of mixtures of PCDDs and PCDFs to those of 2,3,7,8-TCDD. In these approaches, concentrations of the individual compounds are multiplied by the corresponding 2,3,7,8-TCDD toxicity equivalency factor (TEF) and subsequently the sum of these products is calculated. The total amount expressed in toxic equivalents 2,3,7,8-TCDD is also abbreviated to TEQ. For the way in which the various TEF approaches have been developed, reference is made to section 5.1.5.

International TEFs (I-TEFs, table 1.4) are currently more and more frequently used (NATO/CCMS, 1988a and 1988b; Van Zorge et al. 1989). In this document, therefore, the I-TEFs are used for the transformation of values of separate PCDD- and PCDF-congeners to values expressed in I-TEQs wherever possible. When the information needed for the transformation to I-TEQs is lacking, it will be explained in the text how the TEQ-values in these cases have been derived.

In order to be able to distinguish the different TEF-approaches, TEQ values calculated from I-TEFs will be shown as I-TEQ values.
<table>
<thead>
<tr>
<th>PCDD/PCDF</th>
<th>Toxicity Equivalency Factors according to:</th>
<th>BGA</th>
<th>EPA**</th>
<th>Nordic</th>
<th>I-TEF</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCDDs thru TrCDDs</td>
<td></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2,3,7,8-TCDD</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>other TCDDs</td>
<td></td>
<td>0</td>
<td>0.01</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1,2,3,7,8-TeCDD</td>
<td></td>
<td>0.1</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>other PeCDDs</td>
<td></td>
<td>0</td>
<td>0.005</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1,2,3,4,7,8-HxCDD</td>
<td></td>
<td>0.1</td>
<td>0.04</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>1,2,3,6,7,8-HxCDD</td>
<td></td>
<td>0.1</td>
<td>0.04</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>1,2,3,7,8,9-HxCDD</td>
<td></td>
<td>0.1</td>
<td>0.04</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>other HxCDDs</td>
<td></td>
<td>0</td>
<td>0.0004</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1,2,3,4,6,7,8-HpCDD</td>
<td></td>
<td>0.001</td>
<td>0.001</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>other HpCDDs</td>
<td></td>
<td>0</td>
<td>0.00001</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>OCDD</td>
<td></td>
<td>0.001</td>
<td>0</td>
<td>0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>MCDFs thru TrCDFs</td>
<td></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2,3,7,8-TCDF</td>
<td></td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>other TCDFs</td>
<td></td>
<td>0</td>
<td>0.001</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1,2,3,7,8-HeCDF</td>
<td></td>
<td>0.1</td>
<td>0.1</td>
<td>0.01</td>
<td>0.05</td>
</tr>
<tr>
<td>2,3,4,7,8-HeCDF</td>
<td></td>
<td>0.1</td>
<td>0.1</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>other PeCDFs</td>
<td></td>
<td>0</td>
<td>0.001</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1,2,3,4,7,8-HxCDF</td>
<td></td>
<td>0.1</td>
<td>0.01</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>1,2,3,6,7,8-HxCDF</td>
<td></td>
<td>0.1</td>
<td>0.01</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>1,2,3,7,8,9-HxCDF</td>
<td></td>
<td>0.1</td>
<td>0.01</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>2,3,4,6,7,8-HxCDF</td>
<td></td>
<td>0.1</td>
<td>0.01</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>other HxCDFs</td>
<td></td>
<td>0</td>
<td>0.0001</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1,2,3,4,6,7,8-HpCDF</td>
<td></td>
<td>0.01</td>
<td>0.001</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>1,2,3,4,7,8,9-HpCDF</td>
<td></td>
<td>0.01</td>
<td>0.001</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>other HpCDFs</td>
<td></td>
<td>0</td>
<td>0.00001</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>OCDF</td>
<td></td>
<td>0.001</td>
<td>0</td>
<td>0.001</td>
<td>0.001</td>
</tr>
</tbody>
</table>

References*:

1: UBA/BGA (1985); 2: Bellin and Barnes (1986); 3: Ahlborg (1989); 4: NATO/CCMS (1988a); 5: NATO/CCMS (1988b); 6: Van Zorg et al. (1989)

** Concerns dated EPA TEFs. Since 1989, EPA is using I-TEFs (Federal Register, 1989).
1.3 EXISTING STANDARDS AND GUIDELINES

Soil and groundwater

The preliminary value for soil in residential areas derived for the protection of humans is put at 1000 ng I-TEQ.kg$^{-1}$ d.m. For soil in grazing lands exceedings of 10 ng I-TEQ.kg$^{-1}$ d.m. is considered undesirable (Van Zorge, 1987).

Surface water and sediments

For sediments a remediation value has been proposed of 100 ng I-TEQ.kg$^{-1}$ d.m. (Van Zorge, 1987). For waste water, dioxins are considered black-list compounds, for the reduction of which the best available techniques need to be applied and zero emission should be aimed at.

Air

For air no limit value has been determined. In the framework of the Guideline Incineration 1989 an emission limit value of 0.1 ng I-TEQ.m$^{-3}$ d.m. (Nijpels, 1989) has been determined for existing waste incinerators and those to be constructed, incinerators for RDF and other waste categories. The guideline will become effective as from 30 November 1993.

Food and drinking water

The Regulation Dioxin in milk (Commodities Act), regulating that the dioxin content in milk or milk products may not exceed 6 pg I-TEQ.g$^{-1}$ milkfat, is the only statutory Dutch standard for dioxins (Staatscourant, 1991). The standard is established based on the WHO recommended Tolerable Daily Intake (TDI) of 10 pg 2,3,7,8-TCDD per kg body weight per day (WHO/EURO, 1991). This recommendation has been adopted in the Netherlands in July 1991 and is interpreted as 10 pg I-TEQ per kg body weight per day.
2. **FORMATION, SOURCES AND EMISSIONS**

2.1 **FORMATION**

Dioxins are not produced commercially and have no technical use. They are mainly formed by human activities, only slight amounts are of a natural origin. Forest fires are the only natural sources that may possibly contribute to the occurrence of dioxins.

Studies on sediments from lakes in North-America and Switzerland indicate that before 1940 dioxins occurred only in small quantities and that a relation exists between the dioxin level increases over time and the production of chloro-aromatics (Czuczwa and Hites, 1986; Buser and Müller, 1986). Hardly any dioxins have been found in eskimos frozen for centuries (Tong et al., 1990).

2.1.1 **Formation of dioxins**

Dioxin formation near waste incinerators has been extensively studied. In other incineration processes the same mechanisms as in WIs will occur. There may be various causes for the presence of dioxins in smoke gases from waste incinerators, namely:

- dioxins present in waste are not destroyed during incineration;
- dioxins are formed from precursors, such as chlorinated phenols and poly-chlorobiphenols;
- dioxins are formed from greatly differing precursors by chemical reactions;
- the de-novo synthesis, where, under the influence of a catalyst (especially copper), dioxins are formed from non-chlorinated organic compounds and chlorine sources.

During proper incineration dioxins present in waste will for the greater part be decomposed, which also holds for the precursors. The de-novo synthesis is generally assumed to be the main cause for the presence of dioxins. In this process the hydrochloric acid formed during incineration from various sources is catalytically transformed into chlorine via the Deacon reaction:

\[ 4\text{HCl} + \text{O}_2 \rightarrow \rightarrow 2\text{Cl}_2 + 2\text{H}_2\text{O} \]
The chlorine formed reacts with organic compounds, via precursors such as chlorophenols and polychlorobiphenyls, to PCDD and PCDF. The temperature range within which dioxin formation can occur via this route is between 200 and 450 °C.

The above-mentioned shows that dioxin formation depends on the completeness of the incineration as well as on the residence time of flue gases at 200-450 °C. The completeness of the incineration determines the flue gas concentration of organic components. At a long residence time in the temperature range mentioned (in WIs in the steam boilers and the E-filter) the above-mentioned reaction will take place. It is generally assumed that catalyst surface (fly ash) and hydrochloric acid are present in excess. Therefore, the dioxin formation depends little on these compounds.

2.2 SOURCES AND EMISSIONS

In this section the dioxin sources in the Netherlands are mentioned and estimates are given of the dioxin emission. Estimates are made based on literature data, dioxin determinations in the Netherlands and on a survey carried out into processes in the Netherlands where dioxins may be formed. The majority of the data mentioned in this chapter and chapter 6 are extracted from Bronnen van dioxines in Nederland (Dioxin sources in the Netherlands) (Bremmer, 1991) and Inventarisatie van processen waarbij dioxinen kunnen ontstaan (A Survey of processes during which dioxins may be formed) (Bremmer and Hesseling, 1991).

It has been attempted to estimate the dioxin emissions based on existing conditions. The dioxin emissions to air near waste incinerators may be estimated based on measurements recently carried out. In addition a number of measurements has been carried out in the metal industry. With regard to all other sources estimates have been made based on literature data, sometimes on several dioxin determinations and on process data from installations and industries in the Netherlands.

It may be clear that the accuracy of the estimates of dioxin emissions during processes or in branches of industry in the Netherlands based on literature data, on one or more dioxin analyses and process data is only slight. More dioxin determinations may make estimates more accurate. However, even for WIs, where three determinations were carried out near all installations, the accuracy of the estimate can only be approximated. This is caused by varying process conditions and varying composition of the waste, the small number of
determinations per installation and the staggering of the determinations over time. Where possible, the estimates are provided with an indication of the accuracy, for instance by giving the range of the determinations carried out in the Netherlands. All available determinations near dioxin sources are indicated in the following discussion of these sources. A measuring programme is presently being carried out on sources of dioxin other than waste incinerators ("Metten overige bronnen", MOB). The aim of these studies is to obtain better estimates if the dioxin emission in the Netherlands. The results of these studies will become available in 1993. Data from these studies have not been incorporated in this report; in this chapter only data available before 1 January 1992 have been used.

According to Bremmer (1991) data show that no or negligible amounts of dioxins are formed in the pulp and paper industry and during smoking. It is assumed that hardly any or no dioxins are formed in asphalt mixing installations during the combustion of coal and in crematoriums (Bremmer, 1991). The proposed measuring program ‘other sources’, including measurements to these sources, should reveal whether these assumptions are justified.

Amounts of dioxin are, where possible, expressed in I-TEQ (table 1.4, section 1.2). Many dated measuring data refer to the total levels of tetra-, penta-, hexa-, hepta- and octachlorodibenzo-p-dioxins and -dibenzofurans (the ‘collective determination’). The amount TEQ can, in that case, not be calculated in the usual way. In estimating the TEQ-value it is assumed that all isomers within the same homologous series are present in equal quantities (for example: within the series of tetrachlorodibenzo-p-dioxins all 22 TCDDs occur in equal quantities). Departing from the I-TEFs (table 1.4), transformation factors for the total levels of tetra-, penta-, hexa-, hepta- and octachlorodibenzo-p-dioxins and -dibenzofurans can subsequently be calculated (Bremmer, 1991). This calculation method results in a value approximating the I-TEQ and is only used when no isomer-specific data are available.

2.2.1 Waste incinerators

On 1 January 1990 twelve waste incinerators were in operation in the Netherlands. Emissions to air near all WIs have been extensively studied by TNO in cooperation with the RIVM (Slob et al., 1992).

Because of too high emissions of dioxin, particles and hydrochloric acid, or too high dioxin levels in cow’s milk in the near vicinity of WIs, four installations were closed down in 1990 (Alkmaar, Zaandam, Leeuwarden and Leyden). In 1990 additional flue gas cleaning systems
were installed in the WIs of Arnhem and Dordrecht, possibly decreasing the dioxin emissions near these installations tenfold. In table 2.1 data concerning the conditions in 1989 and the end of 1990 are indicated. In table 2.2 data from the studies are given based on the situation at the end of 1990.

**Table 2.1**  
*Dioxin emission to air from WIs (Bremmer, 1991)*

<table>
<thead>
<tr>
<th></th>
<th>1989</th>
<th>end 1990</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amount incinerated waste (ktonnes/year)</strong></td>
<td>3079</td>
<td>2663</td>
</tr>
<tr>
<td><strong>Weighted average emission (ng I-TEQ·m⁻³, und, 11% O₂)</strong></td>
<td>52</td>
<td>35</td>
</tr>
<tr>
<td><strong>Weighted average emission per tonne waste (ng I-TEQ/tonne waste)</strong></td>
<td>0.26</td>
<td>0.15</td>
</tr>
<tr>
<td><strong>Emission (g I-TEQ/year)</strong></td>
<td>789</td>
<td>410</td>
</tr>
</tbody>
</table>

Since the emission was measured during three consecutive days, it may be questioned whether the emissions measured are to be considered representative for the average dioxin emissions over a longer period. The replication measurements discussed in Slob et al. (1992) appeared to be surprisingly stable over the three days (with the exception of only one determination in Zaanstad). No clear measuring results are available on possible fluctuations in emission over longer periods, for instance between seasons. Since it appeared to be possible, via model calculations, to relate the dioxin emission obtained to the levels found in soil and milk, there are indications that the dioxin emissions from waste incinerators in the Netherlands are reasonably representative. In fact in the Rijnmond area the levels in 12 milk samples taken in different months were predicted with reasonable accuracy, while in all these cases the predictions were based on the same emission level measured. However, some caution is called for, especially when deviations in the emission are found as in the case of Zaanstad.

During the survey of possible dioxin sources (Bremmer and Hesseling, 1991) two companies were surveyed where waste was incinerated very similar to municipal waste and where the incineration conditions are comparable to those in WIs. The total emission to air from these two installations is estimated at 1.6 g I-TEQ·year⁻¹.
Table 2.2  Dioxin emission to air from WIs (situation end of 1990, Slob et al., 1992)

<table>
<thead>
<tr>
<th>WI</th>
<th>tonnes waste per year</th>
<th>emission to air (ng I-TEQ.m⁻³)*</th>
<th>(g I-TEQ.year⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amsterdam</td>
<td>508</td>
<td>4.8</td>
<td>14</td>
</tr>
<tr>
<td>The Hague</td>
<td>336</td>
<td>8.8</td>
<td>15</td>
</tr>
<tr>
<td>Arnhem</td>
<td>225</td>
<td>6</td>
<td>6.1</td>
</tr>
<tr>
<td>Roosendaal</td>
<td>33</td>
<td>14</td>
<td>1.4</td>
</tr>
<tr>
<td>Dordrecht</td>
<td>133</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>Nijmegen</td>
<td>69</td>
<td>5.8</td>
<td>2.8</td>
</tr>
<tr>
<td>Rotterdam</td>
<td>386</td>
<td>92</td>
<td>107</td>
</tr>
<tr>
<td>Rijamond</td>
<td>967</td>
<td>53</td>
<td>253</td>
</tr>
</tbody>
</table>

* und, 11% O₂

In recent studies on emissions from WIs (Slob et al., 1992) only the dioxin levels in flue gas were determined and not those in E-filter ash and slag. Based on German data (Hagenmaier et al., 1987; Hagenmaier et al., 1990) and data on the Dutch situation, obtained from collective determination (Sein et al., 1989), the best estimate for the dioxin level in E-filter ash is 15 ng I-TEQ.g⁻¹ filter ash. Departing from the situation at the end of 1990 a total of appr. 1200 g I-TEQ.year⁻¹ is removed with E-filter ash in the Netherlands. Approximately 35% of the amount of E-filter ash produced is applied as a base for asphalt. The remaining E-filter ash in the Netherlands is dumped under IBC-conditions (Anthonissen, 1991).

For slag in German installations, Schmidt-Tegge (1990) reports 80 pg I-TEQ.g⁻¹ slag. Based on these data 64 g I-TEQ would be removed with slag from WIs in the Netherlands. WI slag is mainly applied as levelling material in the construction of roads and canals (Anthonissen, 1991).

2.2.2 Incineration of chemical waste

In this chapter, chlorine containing waste is understood to include:

- Chlorine-containing compounds which, according to the Chemical Waste Act are considered as such. As opposed to what is stated in the act, no exception is made here for compounds that are reprocessed within installations.

- Chlorine-containing gaseous waste flows, such as process- and ventilation gases.

A total of 25 installations were surveyed where dioxins were possibly formed (Bremmer and Hesseling, 1991). With respect to the process three combustion processes can be
distinguished: incinerators, recovery-ovens and afterburners. In table 2.3 data on incineration of chemical waste are given.

**Table 2.3 Incineration of chlorine-containing waste**

<table>
<thead>
<tr>
<th>Oven</th>
<th>Installations</th>
<th>Waste (tonnes/year)</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incinerators</td>
<td>12</td>
<td>appr. 105,000</td>
<td>of which 90,000 tonnes/year by AVR-Chemie and 13,500 tonnes/year by Shell (two installations)</td>
</tr>
<tr>
<td>Recovery ovens</td>
<td>6</td>
<td>appr. 48,000</td>
<td>of which 41,000 by means of HCl-recovery (Akzo 30,000 tonnes/year, Shell 11,000 tonnes/year)</td>
</tr>
<tr>
<td>Afterburners</td>
<td>7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In incinerators, chemical waste is combusted with the aim to destroy it, mainly waste from chemical concerns is combusted in those facilities. All these installations are provided with some form of flue gas cleaning device (E-filter, fabric filter, scrubber and afterburner). In the AVR, apart from municipal waste, chemical waste is also incinerated in separate installations, this part of the plant is called *AVR-Chemie*. In AVR-chemie emissions have recently been measured (Poot, 1990; Poot and Troost, 1991). The old type of oven (DTO-7) emitted 27 ng I-TEQ.m⁻³ flue gas und, the new type (DTO-8) 77 ng I-TEQ.m⁻³ flue gas und.

In the guarantee measurements of DTO-8 (TAUW, 1987), under favourable conditions, by adding chlorophenols and PCBs to the waste, an emission of 1 to 2.3 ng I-TEQ.m⁻³ flue gas und was found. This very great difference cannot be explained. Based on recent data (Poot , 1990; Poot and Troost, 1991) the emission from AVR-chemie can be estimated at 41 g I-TEWQ.year⁻¹.

In recovery ovens chemical waste is combusted, during this process part of the compounds is recovered. In the companies surveyed metal recovery took place, particularly silver and tin, and hydrochloric acid was formed from chemical waste with a high chlorine content (30-85%). Emissions to water from Shell and Akzo incinerators and recovery ovens are known (calculated with collective determination, Turkstra and Pols, 1986). The emissions per installation ranged from 1 to 750 mg I-TEQ.year⁻¹. Considering the high combustion temperature and the rapid cooling of the flue gases the emissions from these installations to air are expected to be slight.
In afterburners mainly organic-chemical ventilation gases or liquids from production processes are combusted. Seven afterburners with possible dioxin emission were surveyed. (Bremmer and Hesseling, 1991). Given the literature data, the temperatures in the afterburners, the possibly low levels of chlorine and the absence of considerable amounts of (fly) ash it is assumed that generally small amounts of dioxin are formed in these processes. For the combustion of chemical waste the following dioxin emissions are estimated:

- For incinerators and recovery ovens: for the AVR an emission to air of 41 g I-TEQ.year\(^{-1}\) and bound to E-filter ash 41 g I-TEQ.year\(^{-1}\). For the other large installations (Akzo and Shell) an emission of 2 ng I-TEQ.m\(^{-3}\) und; for the small installations 20 ng I-TEQ.m\(^{-3}\) und.

- For afterburners: an emission to air of less than 1 ng I-TEQ.m\(^{-3}\) und.

This means that during the combustion of chemical waste in the Netherlands approximately 43 g I-TEQ.year\(^{-1}\) is emitted to air. Approximately 41 g I-TEQ is removed with E-filter ash (only AVR has an E-filter). Filter ash is transported to the C2 depot. Mainly based on measurements carried out by the RIZA (collective determinations) (Turkstra and Pols, 1986), the emission to water is estimated to be about 1 g I-TEQ.year\(^{-1}\).

2.2.3 Rubbish tip gas, sludge

Rubbish tip gas

Rubbish tip gas is formed in rubbish tips of municipal waste. This gas usually contains a chlorine level of appr. 50 mg chlorine.m\(^{-3}\). It is combusted or fed into the gas mains in purified form (<5 mg chlorine.m\(^{-3}\)). During the survey (Bremmer and Hesseling, 1991) nine rubbish tip sites were found where rubbish tip gas is extracted and combusted without purification. In most cases the gas was combusted in a gas engine to generate electricity. It is estimated that, in the manner mentioned above, a total of appr. 10,000 m\(^3\) is combusted per hour.

Gas formed during anaerobic fermentation of sludge from sewage treatment plants and industrial sludge is also combusted (gas engines). It is assumed that this sludge gas is more or less similar to rubbish tip gas.

In the combustion of impurified rubbish tip gas dioxin emissions have been found ranging from 0.1 to 1 ng I-TEQ.m\(^{-3}\) flue gas und (Hasemann and Rieskamp, 1989; Müller and Hör,
1989; Müller et al., 1990).
At an average emission of 0.5 ng I-TEQ.m⁻³ flue gas und, at a combustion of 50,000 m³ chlorine containing gas per hour the emission to air amounts to 0.2 g I-TEQ per year.

**Dioxins in sludge**

Based on data from the Netherlands (Pols, 1988) and abroad (Hagenmaier and Brunner, 1990; Klöpffer et al., 1990; Näf et al., 1990) a dioxin level of 70 pg I-TEQ.g⁻¹ dry matter is estimated in Dutch purification sludge.

In the effluent of water treatment plants dioxins can hardly be detected. The effluent contributes to the dioxin load of surface water (Pols, 1988).

Of the total amount of purification sludge 280,000 tonnes is municipal sludge. This means appr. 20 g I-TEQ in municipal purification sludge. Calculations with the STRAVERA-model show that at a deposition of an average of 20 ng I-TEQ.m⁻² on drained paved surface within built-up areas this amount of 20 g I-TEQ in purification sludge can be fully ascribed to deposition. Starting from the following percentages: 95% connection to water treatment plants, 12% separate sewerage, 22% excess flux in mixed sewerage, and a 90% efficiency of the water treatment plant, approximately 13 g I-TEQ will end up in surface water via run-off from paved surfaces and municipal discharges.

The total amount of purification sludge in the Netherlands is appr. 460,000 tonnes of dry sludge per year (Duvoort-van Engers, 1991), containing appr. 32 g I-TEQ. The fate of purification sludge is: agriculture, 22 %; compost/black soil, 14%; dumped, 55%; combusted, 2.6%; and miscellaneous, 6.6% (Duvoort-van Engers, 1991).

**Incineration of sludge**

In the incineration of sludge mostly small amounts of dioxins are found, usually in the order of 0.1 ng I-TEQ.m⁻³ und (NATO-CCMS, 1988; Southerland et al., 1987; Orthofer and Vesely, 1990; Hagenmaier and Brunner, 1990). In the Netherlands the dioxin emission in the sludge incinerator in Amsterdam was determined. The emission amounted to 0.15 ng I-TEQ.m⁻³ und (11% oxygen; TAUW, 1990a). Concerning incineration of sludge the following kinds of sludge can be distinguished:

- purification sludge, originating from water treatment plants;
- industrial sludge, originating from production processes and wet washing installations.

Incineration of sewage sludge in the Netherlands takes place in three installations.
Incineration of industrial sludge also takes place in three installations. A total of appr. 16,000 tonnes of (dry) sludge is incinerated per year (appr. 8000 tonnes of purification sludge and appr. 8,000 tonnes of industrial sludge per year). Assuming the dioxin levels in flue gas to be an average of 0.5 ng I-TEQ.m\(^{-3}\) und, 11% O\(_2\), the total emission to air is appr. 0.05 g I-TEQ per year.

2.2.4 Contaminated pesticides

Pesticides containing chlorphenols, chlorobenzenes or their derivatives may be contaminated with dioxins (NATO-CCMS, 1988).

Agricultural pesticides

Recent analyses are only available for a few pesticides registered in the Netherlands (Hagenmaier, 1987; Hagenmaier and Brunner, 1987; Wegman et al., 1987; NATO-CCMS, 1988). The amount of pesticides containing chlorphenols, chlorobenzenes or their derivatives applied in 1984 in agriculture, horticulture and veterinary practice was appr. 1,500 tonnes (Bremmer et al., 1988). Based on the limited data it is assumed that 20% of these pesticides is contaminated with an average of 2 ng I-TEQ.g\(^{-1}\). The total amount of dioxins in pesticides applied in agriculture, horticulture and veterinary practice is thus 0.6 g I-TEQ per year.

Wood preservatives

Compounds expected to be contaminated with dioxins are no longer allowed for the preservation of wood in the Netherlands. Pentachlorophenol (PCP) as a wood preservative is banned in the Netherlands as from 1 January 1989. Literature data (Ree, 1984, 1984; DHV, 1986; Hagenmaier, 1987; Hagenmaier and Brunner, 1987; Wegman et al., 1987) show that pentachlorophenol is contaminated with an average of appr. 3000 ng I-TEQ.g\(^{-1}\) PCP (i.e. 3 mg I-TEQ.kg\(^{-1}\) PCP). Considerable amounts of PCP and dioxins are still present in preserved wood. Through the import of wood and wood products, particularly vegetable and fruit crates, pallets and parquet, an estimated 130 tonnes PCP enters the Netherlands annually. Departing from the above-mentioned dioxin level, the total amount involved is appr. 400 g I-TEQ.
The total PCP emission to air from wood is estimated at 55 tonnes.year\(^{-1}\) (Haskoning, 1989; Slooff et al., 1990). Dioxins present in pentachlorophenol treated wood evaporate from the wood, as does PCP (Eckrich, 1987; Sagunski et al., 1989; Püpke et al., 1989). The total amount of dioxins now present in pentachlorophenol treated wood is appr. 4500 I-TEQ (Bremmer, 1991).

Two methods can be followed to estimate, based on these data, the dioxin emission as a result of evaporation from PCP contaminated wood:

- Based on physico-chemical properties it is assumed that the evaporation of dioxin from preserved wood is ten times slower than that of PCP. Departing from this assumption it can be calculated that the emission of dioxins from PCP preserved wood is in the order of 25 g I-TEQ per year.

- Püpke et al. (1989) have studied the relationship between dioxins and PCP in air. There appeared to be no direct relationship between these two parameters. At elevated dioxin concentrations in indoor air the mean ratio PCP:dioxin (in I-TEQ) was about 1 : 5*10\(^{-6}\). At an annual evaporation of 55 tonnes PCP from wood in the Netherlands this would involve an emission of 275 g I-TEQ per year.

It is emphasized that both calculation methods indicate an order of magnitude. Since Püpke et al. did not find a direct relationship between dioxin and PCP, the first method is preferred. It is assumed that the dioxin emission from PCP contaminated wood is about 50 g I-TEQ per year. PCP impregnated wood (for instance pallets, vegetable crates and demolition wood) constitutes a considerable source of dioxins present in municipal waste.

**Preservation**

In the formulation of pesticides a small amount of pentachlorophenol is in some cases added as a fungicide in pesticides containing organic material, such as snail granules (Slooff et al., 1990). This application leads to a diffuse emission to the soil of 500-1000 kg PCP per year, causing a diffuse emission to the soil of appr. 2 g I-TEQ per year.

Pentachloroilaureate is applied as an anti-putrefaction and fungicide in products such as tents, sun-blinds, sponges and chamois leathers. The use of PCP-laureate in products in the Netherlands is estimated at appr. 29 tonnes that is 18 tonnes.year\(^{-1}\) PCP (Slooff et al., 1990). The emissions of dioxin to air, water and soil amount to an estimated 0.06; 0.09 and 0.15 g I-TEQ.year\(^{-1}\), respectively. Approximately 7 g I-TEQ.year\(^{-1}\) is removed with domestic and industrial waste (Bremmer, 1991).
2.2.5 **Metal industry**

**Basic metal industry**

In the basic metal industry dioxins are formed by the steel production. The emission of dioxin to air in the cinder plant of *Hoogovens* amounts to appr. 3 ng I-TEQ.m\(^{-3}\) flue gas, corresponding to 24 g I-TEQ.year\(^{-1}\) (Kuipers, 1990). Dioxin emission may possibly also occur in the coke plant, the pallet plant and in the blast furnaces itself. These emissions are expected to be lower than those in the cinder plant. The dioxin emission to air from the basic metal industry is estimated at appr. 35 g I-TEQ.year\(^{-1}\).

**Secondary non-ferro industry**

Dioxins may be formed during the smelting of aluminum-, lead- and copper scrap when chlorine-containing metal-processing liquids and/or PVC are present in the materials used. In the survey (Bremmer and Hesseling, 1991), 4 aluminum smelters (throughput of 3 of the 4 smelters: 47,000 tonnes.year\(^{-1}\)), 12 lead smelters and a copper-bronze smelter were found where, given the base materials, dioxin emission may occur.

The filter ash from secondary aluminum smelters contained 3-4 ng I-TEQ.g\(^{-1}\). In the flue gases 2.9 ng I-TEQ.m\(^{-3}\) was found (Rodenburg et al., 1991; Bremmer, 1988). In a lead smelter 1.3 ng I-TEQ.m\(^{-3}\) was found in the flue gases and 7.7 and 17.8 ng I-TEQ.g\(^{-1}\) in the filter ash (Rodenburg et al., 1991). Sludge from a secondary bronze smelter contained 62 ng I-TEQ.g\(^{-1}\) (Rodenburg et al., 1991).

Departing from the survey (which probably did not fully cover this branch of industry) and the dioxin emissions measured, it is assumed that in appr. 10 non-ferro companies dioxins are emitted. The emission to air from secondary non-ferro companies is estimated to be 7 g I-TEQ per year and the emission to water appr. 0.5 g I-TEQ per year. The amount of dioxins removed with filter ash and sludge is estimated at 15 g I-TEQ per year.

**Secondary ferro industry**

In iron/steel foundries/smelters dioxins can be formed when chlorine containing metal-processing liquids and/or PVC are present in the raw materials used.

In the Netherlands 60,000-200,000 tonnes of machining waste with adhering cutting compounds is generated per year. Almost all machining waste is exported, only a few percent of the total amount is processed in the Netherlands (Scheepens, 1988). In the Netherlands
only a few companies were found which process iron- and steel machining waste. In the flue gases from an iron foundry, using machining waste with adhering metal-processing liquids as raw material, dioxins were found. (Rodenburg et al., 1991). This corresponds with the expectations based on process conditions (Rodenburg et al., 1991).

In the survey (Bremmer and Hesseling, 1991) inquiries were made about raw materials in ferro foundries/smelters. Companies using raw materials containing chlorine containing metal-processing liquids and/or PVC were not surveyed.

In iron/steel foundries/smelters processing engine blocks small amounts of chlorine containing compounds will occur in the adhering engine oil. In a number of companies PVC will be present in scrap. To what extent dioxins will be formed in these companies depends on the process conditions.

Based on the number and the extent of the Dutch ferro foundries/smelters, emission data from abroad and the conditions in the Netherlands it is assumed, for the time being, that in ferro foundries/smelters appr. 3 g I-TEQ/year is emitted to air.

2.2.6 Cable burning

During the burning off of cables dioxins may be formed (Eberhardt, 1986; Bergvall, 1987; Hagenmaier, 1987; Souderland et al., 1987; EPA, 1987, Christmann et al., 1989; Rappe et al., 1987). At incineration sites, where cables are burnt off in the open air, highly elevated dioxin levels were found (Liem et al., 1991). The dioxin emission depends on, among other things, the kind of oven, the gas purification, the temperature, the amount of oxygen and the presence of chlorine sources such as PVC.

Underground cables are burnt off batch-wise in simple ovens. PVC-coated cables may not be burnt off in the Netherlands. In the Netherlands there are 7-10 wire reclamation incinerators, 6 of which are situated in Gelderland. From studies on flue gases from 3 wire reclamation incinerators (TAUW, 1988) it appeared that in the underground cables that were burnt off an amount of PVC was present.

The total amount of cables that are burnt off in the Netherlands is estimated at 20,000 tonnes (TAUW, 1988; Rotteveel, 1989). An estimated amount of 13,000 tonnes is burnt off in wire reclamation incinerators and appr. 7,000 tonnes per year illegally in the open air.

Illegally burning off of cables especially takes place near the border with Germany.
(Overijssel, Gelderland and Limburg). A considerable amount of these cables originates from Germany and Belgium. It is obvious that the amount of cable illegally burnt off is indicative (Bremmer and Hesseling, 1991).

In a wire reclamation incinerator, where bitumen underground cables and a mixture of underground cables and PVC containing cables were burnt off, dioxin emissions were measured. In the underground cables 0.8 ng I-TEQ.m\(^{-3}\) flue gas was found and in the mixture 2.5 ng I-TEQ.m\(^{-3}\) flue gas. Given the normal process conditions in wire reclamation incinerators, the values measured at Culemborg may not reflect the average emission of wire reclamation incinerators. The dioxin levels in milkfat in the near vicinity of the wire reclamation incinerators at Culemborg (Kootstra et al., 1991) indicate that the current emission is considerable and (or) that the amounts of dioxin in the top layer of the soil, as a result of emissions in the past, are great.

Departing from the above-mentioned data the dioxin emission in wire reclamation incinerators in the Netherlands is estimated at half the average emission per installation in WIs, that is 0.13 mg I-TEQ per tonne waste. Based on these data legal wire reclamation incinerators emit appr. 2 g I-TEQ per year.

It is difficult to estimate the PCDD/PCDF emissions from illegal cable burning in the open air. During illegal burning off, PVC coated cables will probably also be burnt off. The emission per tonne waste will, also given the manner in which the burning off takes place, probably be considerably higher than that in legal wire reclamation incinerators. The emission from cable burning in the open air is provisionally estimated at twice the average emission to air from WIs, which amounts to appr. 4 g I-TEQ per year.

2.2.7 Traffic

Exhaust gas from cars running on leaded petrol contains many halogen compounds (Müller and Buser, 1986), including dioxins (Marklund et al., 1987; Marklund et al., 1990; Bingham et al., 1989; Larssen et al., 1990; Hagenmaier and Brunner, 1990). Apart from dioxins the corresponding bromine- and bromine/chlorine compounds are formed (Haglund et al., 1988). Leaded petrol contains dichloroethane and dibromoethane as a scavenger to transform inorganic lead compounds into volatile compounds, such as lead chloride and lead bromide, which are removed with the exhaust gas. With respect to the composition of Swedish automobile fuels Haglund et al. (1988) reported a level of 61 ppm Br and 63 ppm Cl for
leaded petrol, less than 0.4 ppm Br and 14 ppm Cl for unleaded petrol and in diesel fuel. Br and Cl could not be detected. Larssen et al. (1990) determined the dioxin levels in ("clean") air drawn into two tunnels and in the air expelled. From these data, emission factors for the dioxin emission were calculated. On workdays they found an emission factor much higher than that on week-ends. Larssen et al. explain the much lower value in the week-end by attributing an emission factor to lorries, which run on diesel fuel, appr. 20 times higher than for passenger cars.

Hagenmaier et al. (1990) report that the emission of tetra- to octachloro-compounds from cars running on leaded petrol is relatively small, and that mainly brominated and mixed chloro-bromo, mono- to tetra-halogenated dibenzofurans are emitted. For Germany they estimate the emission from exhaust gases from cars running on unleaded petrol at 10%, from cars with a catalytic converter at 1% and from diesel engines at 1-10% of the emission from cars running on leaded petrol.

In the Netherlands 4470*10^6 l petrol and 4050*10^6 l diesel fuel was used in 1990 by road traffic (CBS, 1991). Of the petrol 51% was leaded (CBS, 1991a). The emission estimate was based on emissions from leaded petrol of 100 pg I-TEQ.km^-1 and from unleaded petrol of 1 pg I-TEQ.km^-1. For cars running on diesel fuel the situation is still unclear.

For diesel fuel therefore it is departed from a conservative estimate of 100 pg I-TEQ.km^-1. Based on the above-mentioned assumptions the total emission from road traffic in 1990 amounts to appr. 6 g I-TEQ per year. About 10% of the contamination in exhaust gases settles on the road surface, appr. 20% of which ends up in surface water, resulting in an emission of appr. 0.1 g I-TEQ to water.

2.2.8 Hospital waste incinerators

Dioxins have been demonstrated in flue gases and in filter ash from hospital waste incinerators (Hagenmaier, 1987; Hagenmaier and Brunner, 1990; National Agency of Environmental Protection in Denmark, 1989; Lindner et al., 1990; Ozvacic et al., 1990). Hospital waste can be distinguished in waste removed as industrial waste and in specific hospital waste. Specific hospital waste contains glass, synthetic material, pathological waste, carcasses of experimental animals and packing material. The chemical composition is not known (Vos, 1989). The amount of specific hospital waste is estimated to be 5000 tonnes.year^-1 (Vos, 1989). This specific hospital waste is partly combusted in incinerators
of the hospitals. Part is collected by specialized companies and incinerated in the AVR as chemical waste. The survey (Bremmer and Hesseling, 1991) shows that the number of hospitals with internal incinerators has rapidly decreased the last few years. The current number of incinerators is estimated to be appr. 25. Hospital waste incinerators are in general small installations combusting several tonnes of waste per year. There are only a few installations with a capacity greater than 1000 tonnes.year⁻¹.

The incinerators in hospitals generally operate in an unsatisfactory manner. The environmental protection requirements with respect to incomplete combustion and emissions are not met (Tebodin, undated). Based on literature data on process management, the incineration conditions and the composition of the waste, the dioxin emission in hospital waste incinerators is estimated to be on average 3 times as high as the average emission in WIs (situation 1989), that is 0.78 mg I-TEQ.tonne⁻¹ waste. Based on these assumptions and an annual amount of 5000 tonnes of waste combusted in hospital incinerators, 4 g I-TEQ is emitted per year.

2.2.9 Chemical production processes

In the production of chlorine containing chemicals dioxins may be formed. Processes during which dioxins may be formed, in order of declining importance as potential sources, are the following (NATO-CCMS, 1988; Hutzinger and Fiedler, 1989):

- formation of chlorophenols, chlorobenzenes and chlorophenol and chlorobenzene derivatives;
- synthesis of aliphatic chlorine compounds;
- various chemical processes
  * syntheses with chlorinated intermediates;
  * inorganic chlorine chemistry, metal chlorides;
  * processes in which chlorinated solvents or catalysts are applied;
  * reactivation of active carbon;
  * cinder processes.

In processes with temperatures lower than 150°C the possibility of dioxin being formed is only slight.
Chlorophenols, chlorobenzenes and derivatives

In the Netherlands pesticides containing chlorophenols, chlorobenzenes or their derivatives are produced in only a few companies (Bremmer et al., 1988). In the pharmaceutical industry and in the production of chemicals, particularly specialties for the pharmaceutical industry, chlorination processes are carried out and chlorobenzenes and derivatives of chlorophenols and chlorobenzenes are processed.

Data from the Emission Registration show that in less than 20 companies in the Netherlands emissions occurred of chlorinated compounds to water or air. This may be a good indication of the number of companies where chlorobenzenes are applied as solvent or as basic material and/or chlorination processes are carried out. It is expected that the dioxin emissions to water and air in companies using chlorobenzenes and/or chlorophenol- and chlorobenzene derivatives will be slight. Waste produced by these companies may contain dioxins. This waste is removed as chemical waste and incinerated as such.

Aliphatic chlorine compounds

Dioxins have been determined in chloroaliphates in only a limited number of cases. Since chloroaliphates are usually rather volatile it is expected that in the case of dioxins being formed these will occur in other reaction products, such as distillate residues (NATO-CCMS, 1988; Fuchs et al., 1990). Heindl and Hutzinger (1987) have demonstrated dioxins in 5 of the 23 analyzed aliphatic chlorine compounds, levels of up to 0.7 pg I-TEQ.g\(^{-1}\). Possible dioxin contamination in aliphatic chlorine compounds may be slight and negligible as a source of dioxin.

The production of chloroaliphates in the Netherlands amounts to appr. 1.3 million tonnes per year. A considerable proportion of this quantity is an intermediate product in the production of other halogen hydrocarbons (Bremmer et al., 1988). Most of the amount of these chloroaliphates is used for the production of PVC. In the oxychlorination of ethane, a process preceding PVC production, dioxins are formed (Evers, 1989; Turkstra and Pols, 1986). Residues released in the production of chloroaliphates are chemical waste. The major part of the distillate residues is incinerated. In the large-scale application of chloroaliphates small dioxin emissions may occur to water and to air. Dioxin emission from distillate residues is probably nil.
Various chemical processes

Reactivation of active carbon for reuse takes place by means of heating. When carbon is treated with considerable amounts of total organic chlorine, dioxins may occur in the flue gases and in the flue gas cleaning residues (Lykins et al., 1988; EPA, 1987). It has recently been reported in the literature that dioxins may be formed in the regeneration of the catalyst used in reforming processes (Thompson et al., 1990a). During reforming processes normal petrol is converted into petrol with a higher octane number with hydrogen and a catalyst at high temperatures and high pressure.

From Bremmer and Hesseling (1991) it appeared that in addition to the above-mentioned processes two large-scale cinder processes occur where dioxin emission may be expected. These two processes are the production of synthetic gravel from E-central fly ash and powder carbon, where consumed oil is reused as fuel, and the production of pellets from phosphor ore, clay and return material from the phosphate production.

It is concluded that in the waste from a number of processes dioxins may occur. This waste is for the greater part incinerated as chemical waste. The number of companies where, given the kind of chemicals in use and the production process, formation of dioxins may be possible is appr. 25. The emission of dioxins to air originating from chemical production processes is difficult to determine. In view of the uncertainty the emission is provisionally estimated to be 3 g I-TEQ per year, the emission to water is estimated at 2 g I-TEQ per year. The emission in chemical processes outside the chemical industry is estimated at 2 g I-TEQ per year.

2.2.10 Incineration of waste oil

In the incineration of PCB contaminated oil and used oil, dioxins may be formed (Erickson, 1989; desRosiers and Lee, 1986; Bröker and Gliwa, 1986; Nielsen and Blenkinsjerg, 1989). TNO has carried out studies on the emissions from used oil-fired garage heaters (Kiers and Spierings, 1987). In the flue gases from eight samples unprocessed and processed (i.e. purified) used oil maximally 0.4 ng TEQ.m\(^{-3}\) flue gas was found (collective determination). During the incineration of PCB-containing transformer oil in a heater intended for waste oil 24,000 ng I-TEQ were found in the soot (Brinkmann et al., 1988), while in the soot from oil-fired heaters in Germany 0.147 and 0.907 ng I-TEQ.g\(^{-1}\), respectively, was found (Thoma,
The total amount of used oil that was released in 1989 in the Netherlands amounted to appr. 100,000 tonnes (Nieuwenhuis et al., 1990). Of this amount 53,000 tonnes was recycled to fuel. Waste oil is collected by specialized companies, which redistribute it as fuel following simple recycling. The recycling procedure is such that the chlorine content does not decrease. This processed used oil is applied as bunker oil in shipping, particularly in the ocean-going trade and in big installations such as gras-dryers, asphalt mixing installations and for the heating of hothouses (Bremmer and Hesseling, 1991). Approximately 20,000 tonnes of used oil is incinerated annually without being processed, of which 10,000 tonnes in garage heaters and appr. 10,000 tonnes illegally. TAUW (1990) reported an organic chlorine content in used oil of 218 mg chlorine per kg.

Based on the available data it is difficult to estimate the dioxin emission caused by the incineration of waste oil. It is assumed that the dioxin emission in the incineration of used oil will be an average of a few ng.m\(^{-3}\) at most. The total emission is estimated at 2 g per year. Based on the literature data it may be assumed that during the incineration of oil, not belonging to the waste oil category, hardly any or no dioxins will be emitted.

With the data presently known it may be assumed that dioxins will be found in the soot from used oil-fired heaters, possibly in the order of ng I-TEQ.g\(^{-1}\). The amount of dioxins bound to soot originating from the incineration of oil is roughly estimated to be 10 g I-TEQ.year\(^{-1}\).

2.2.11 Incineration of wood

In the incineration of untreated wood dioxins were found in the soot and the bottom ash (Clement et al., 1985; Thoma, 1988; Esmil, 1982; Aittola and Viinikainen, 1989). In the flue gases from the incineration of untreated wood no dioxins were found by Aittola and Viinikainen (1989), whereas the National Agency of Environmental Protection in Denmark (1990) did demonstrate dioxins in the incineration of clean wood (appr. 10 ng I-TEQ.m\(^{-3}\) und). In the incineration of contaminated wood the dioxin emission will depend on the kind of contamination and the incineration conditions.

In the incineration of PCP-contaminated wood (e.g. in pallets and vegetable crates) an emission of 1000 ng I-TEQ.m\(^{-3}\) und has been measured (National Agency of Environmental Protection, Denmark, 1990). To what extent forest fires constitute a (natural) source of
dioxins is not yet clear, - further studies are needed (Tashiro et al., 1990). In view of the small number of forest fires in the Netherlands this source will probably be of very little importance. Dioxin emissions resulting from incineration of wood are difficult to estimate with data currently available, since the literature concerning this matter is not unequivocal. It is assumed that the emission during the incineration of clean wood is negligible, the emission during the incineration of treated and untreated wood together with chlorine containing compounds amounts to an average of 0.3 mg I-TEQ per tonne waste, and that dioxins are present in soot, possibly in the order of μg·g⁻¹.

In the Netherlands appr. 100,000 tonnes of scrap wood are applied annually in industrial installations. In fireplaces and woodstoves appr. 980,000 tonnes of wood is incinerated annually (Okken et al., 1992). In fireplaces and woodstoves wood from pallets, painted/impregnated wood and demolition wood is often used as fuel by 7%, 2% and 8% of the users, respectively, and sometimes used by 18%, 8% and 25% of the users, respectively. Irregular fuels such as paper briquets, plastic, empty milk cartons, leather and rubber are applied by less than 1% of the users (Okken et al., 1992).

From the above-mentioned data it is estimated that 5% of the incinerated wood is contaminated with chlorine containing compounds. The dioxin emission by incineration of wood in stoves and fireplaces is calculated at appr. 16 g I-TEQ per year. The amount of dioxins bound to soot resulting from the incineration of wood is roughly estimated at 80 g I-TEQ per year.

2.2.12 Fires

The number of PCB-containing transformers and large condensers still in use is fairly small (Bremmer, 1991). As it is assumed that in the Netherlands very few fires occur involving considerable amounts of PCBs, these may be ignored as a source of dioxins. However, if such fires do occur, dioxin emission will be very likely. In fires involving PCBs, PCDFs will be the main constituent of the emission.

Literature data concerning fires where PVC containing material is involved (Deutsch and Goldfort, 1988; Christmann et al., 1989; Theisen et al., 1989; Funcke et al., 1988; Marklund et al., 1989) show that:
- dioxins are formed during incineration;
- the amount of dioxin depends on the conditions during the fire (e.g. temperature, humidity, oxygen concentration);
- incinerated PVC containing materials and soot from the near vicinity of the seat of the fire may contain considerable amounts of dioxins;
- in the cases described the dioxin emission to air is slight as compared to the amount of dioxin bound to soot found in the near vicinity of the seat of the fire;
- in laboratory experiments, intended to simulate PVC-fires, up to 500 ng I-TEQ g\(^{-1}\) was found.

In 1987 the number of buildings in the Netherlands where fires took place amounted to appr. 11,000. The number of fires at sites other than buildings (containers, cars, ships) and possibly involving PVC amounted to appr. 10,000 (CBS, 1988). In addition to these registered fires, PVC is also incinerated in open fires, particularly in rural areas by private individuals and enterprises (for instance farmers).

Dioxins may also be formed in other fires, for instance in the incineration of automobile tyres (Thompson et al., 1990; Steer et al., 1990) and of straw (Nielsen and Blinksbjerg, 1989). In the Netherlands the burning of straw in the open field is small-scale. Incineration of straw in stoves is very little practiced in the Netherlands.

In the survey of Bremmer and Hesseling (1991) twelve flares were found, none of which were used to incinerate chlorinated compounds.

Based on current data the dioxin emissions as a result of fires are difficult to estimate. For the time being it is assumed that the emission to air is in the order of 90 g I-TEQ per year (50 fires with an emission of 30 mg, 500 fires with an emission of 3 mg, 10,000 fires with an emission of 0.3 mg, 100,000 fires with an emission of 0.03 mg I-TEQ). The amount of dioxins formed that, bound to soot, remains in the near vicinity of the seat of the fire is estimated at 50 g I-TEQ per year.

2.2.13 Other processes

Apart from the above processes there is a number that do not fit into any of the categories mentioned. Given the incineration conditions, presence of chlorine and flue gas cleaning, a number of processes was found in the survey where dioxins may be formed: rubber production, soil purification, production of poroso-stone, incineration of industrial waste, drying of fly ash, cement ovens.
For the time being it is assumed that in these other processes a total of 15 ng I-TEQ is emitted.

2.2.14 Sources abroad

In "De verspreiding en depositie van dioxinen in Nederland" (Schutter and van Jaarsveld, 1992) dioxin emissions abroad have been estimated. These data have been used to calculate the contributions from abroad for the deposition distribution of dioxins in the Netherlands (section 4.4.3). The estimates are indicated in table 2.4. The calculated values are given in the table, although this suggests too high a level of accuracy. However, in the rounding of the figures relevant information is lost. From the countries mentioned in the table extensive data on the emission of dioxin are available. From other European countries only few data are available, or none at all.

The amount of dioxins entering the Netherlands from sources abroad amounts to an estimated 500 g I-TEQ per year via air (see 4.6) and 55-80 g I-TEQ per year via surface water (see 4.3).
2.3 SUMMARY AND CONCLUSIONS

Dioxins are not produced commercially and are formed during incineration processes (200-450°C) in the presence of a chlorine source and in industrial production processes. At this moment, the dioxin emission to air from waste incinerators can be estimated on the basis of determinations recently carried out. Additionally, a number of measurements has been carried out in the metal industry. With respect to all other sources estimates have been made based on literature data, sometimes on one or several dioxin determinations and process data from installations and industries in the Netherlands.

In table 2.5 the various sources and the estimated amounts of dioxins annually emitted from these sources are summarized for the Dutch situation (end 1990). The calculated values are indicated in the table, however they suggest too high a level of accuracy. For an indication of the accuracy the sources are provided with a classification based on the amount of data available.

The total amount of dioxins emitted annually (situation end 1990) is estimated at appr. 600 g I-TEQ, 400 g I-TEQ of which originating from WIs. The total amount of dioxins which, bound to filter ash, soot, sludge wood and textile is formed or imported into the Netherlands in the form of products is estimated at appr. 1800 g I-TEQ per year (situation end 1990).

<table>
<thead>
<tr>
<th>Country</th>
<th>Waste WIs (tonnes/y)</th>
<th>Emission factor WIs (µg/tonne)</th>
<th>Emission WIs WIs (g/y)</th>
<th>Emission other sources (g/y)</th>
<th>Total emission (g/y)</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Netherlands</td>
<td>2663 (1990)</td>
<td>155</td>
<td>412</td>
<td>204</td>
<td>616</td>
</tr>
<tr>
<td>West Germany</td>
<td>9000 (1989)</td>
<td>44</td>
<td>400</td>
<td>491</td>
<td>891</td>
</tr>
<tr>
<td>Denmark</td>
<td>1755 (1987)</td>
<td>20</td>
<td>34</td>
<td>83 1)</td>
<td>117</td>
</tr>
<tr>
<td>Sweden</td>
<td>1400 (1985)</td>
<td>35</td>
<td>49</td>
<td>159</td>
<td>208</td>
</tr>
<tr>
<td>Austria</td>
<td>460 (1989)</td>
<td>9</td>
<td>3</td>
<td>104</td>
<td>107</td>
</tr>
</tbody>
</table>

1) A Danish estimate for the emission from hospital waste incinerators is 13.5 g TEQ/y
<table>
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<tr>
<th>Indication for accuracy</th>
<th>Air</th>
<th>Emission Water</th>
<th>Soil</th>
<th>Bound 2)</th>
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<tr>
<td>- Incineration of municipal waste</td>
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<td>* WIs</td>
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<td>- Incineration of chemical waste</td>
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<td>43</td>
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<td>- Incineration of rubbish tip gas, biogas, sludge</td>
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<td>* purification sludge</td>
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<td>* rubbish tip gas, biogas</td>
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<td>- Application pesticides</td>
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<td>* agricultural pesticides</td>
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<td>0.6</td>
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<tr>
<td>* wood preservatives (PCP) 4)</td>
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<td>50</td>
<td></td>
<td>400 5)</td>
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<tr>
<td>* PCP as fungicide</td>
<td>c</td>
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<td>2.2</td>
<td>7 6)</td>
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<tr>
<td>- Metal industry</td>
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<tr>
<td>* basic metal industry</td>
<td>b</td>
<td>35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>* secondary non-ferroindustry</td>
<td>b</td>
<td>7</td>
<td>0.5</td>
<td>15</td>
</tr>
<tr>
<td>* iron/steel-smelters/foundries</td>
<td>b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Cable burning</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>* &quot;legal&quot;</td>
<td>b</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>* in the open air</td>
<td>d</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Traffic</td>
<td>c</td>
<td></td>
<td>6 7)</td>
<td></td>
</tr>
<tr>
<td>- Hospital waste incinerators</td>
<td>c</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Chemical processes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>* production of chlorphenols-,benzenes aliphatic chlorine compounds, and the like</td>
<td>c</td>
<td>3</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>* outside the chemical industry</td>
<td>c</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Incineration of oil (garage heaters, large furnaces, bunker oil</td>
<td>b</td>
<td>2</td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>- Incineration of wood in stoves, fireplaces, industrial installations)</td>
<td>d</td>
<td>16</td>
<td></td>
<td>80</td>
</tr>
<tr>
<td>- Fires, torches</td>
<td>d</td>
<td>9</td>
<td></td>
<td>50</td>
</tr>
<tr>
<td>- Various processes (e.g. in industrial production, incineration industrial waste, soil purification installations)</td>
<td>d</td>
<td>15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1) Accuracy indication: based on several measurements; b: based on one or several measurements in NL; c: estimate based on emission data from abroad and process conditions in NL; d: order of magnitude.
2) Bound to: fly ash, filter ash, soot, sludge, wood, textile
3) Situation beginning 1990: 789 g I-TEQ/year
4) The total amount of dioxins bound to wood is in the order of 4500 g I-TEQ
5) Caused by importation of wood
6) Bound to textile (particularly tents and sun-blinds) and sponges
7) Only polychlorodibenzo-p-dioxins and -dibenzofurans
3. DISTRIBUTION AND TRANSFORMATION

3.1 BEHAVIOUR IN SOIL AND GROUNDWATER

As is the case with other organic compounds, the behaviour of dioxins in soils is the resultant of a number of sub-processes: transport processes (volatilization, advection and diffusion, influenced by adsorption) and transformation processes (chemical, photochemical and microbiological).

3.1.1 Transport in the soil

Adsorption

Adsorption leads, in particular, to a slowing down of the rate of migration of a substance in the soil. The stronger the sorption, the slower will be the migration. For organic substances, the mechanism determining organic soil content levels is commonly used to establish relationships. The more organic substance and the higher the log Kow, the higher will be the sorption.

In view of the high log Kow values of dioxins (5-8, see table 1.3), a very strong adsorption is to be expected. In experiments with soil materials, extremely high Koc values have indeed been found (Des Rosiers, 1986; Jackson et al., 1986; Walters and Guiseppe-Elle, 1988; Walters et al., 1989; Lodge and Cook, 1989).

Walters and Guiseppe-Elle (1988) observed that sorption can reach an equilibrium very quickly (1 day), but may require more time (30 days) in the case of higher organic substance levels in the soil.

In addition, Esposito et al. (1980) suggested the occurrence of an irreversible binding process when contact between soil and dioxins is protracted. This was confirmed by Hüttner and Philippi (1982), on the basis of poor recovery determinations. Muir et al. (1985) found a non-extractable residue of 5-15%.

Transport with groundwater/mobility

The water solubility of dioxins is relatively low (Table 1.3). Since sorption is also high, the transport with groundwater will be small and strongly reduced. High water transport velocities may lead to a relatively enhanced transport of substances where there is no
equilibrium situation with respect to sorption. Dioxins should be able to penetrate more deeply in the soil as a result of high water transport velocities. However, high mobility rates could not be obtained in experiments with high water transport velocities (Matsumura and Benezet, 1973; Arsenault, 1976). With the aid of a transport model, Murphy (1989) calculated a negligible water transport rate for 2,3,7,8-TCDD. Laboratory studies with TLC-and column experiments have confirmed this. Helling (1971) did not find a detectable migration rate for 2,7-DCDD and 2,3,7,8-TCDD in various kinds of soil, with different clay and organic compound levels (TLC experiments). In column experiments, Matsumura and Benezet (1973) recovered almost 98% of 2,3,7,8-TCDD in the top most 0.5 cm, while Arsenault (1976) found that less than 0.0005% of OCDD was washed out from a soil column of 20 cm.

In general, dioxins may be recovered from a few centimeters below the soil surface, if the loading has taken place at the top. In Seveso, the bulk of the contamination was found in the top 10 cm (Di Domenico et al., 1980a). Young et al. (1975, 1987) found no TCDD below 15 cm in a field where 2,3,7,8-TCDD had been deposited through spraying with 2,4,5-T and Agent Orange. Neither Nash and Beall (1980), nor Muir et al. (1985) found any indication for mobility. Kitunen and Salkinoja-Salonen (1990) observed that dioxins (present as a result of PCP contamination near a wood preservation plant) did not occur below a depth of 5 cm. Much of the literature on the distribution of dioxins is in agreement with this picture.

It has become clear from various studies that an increased mobility may be found when solvents (Palausky et al., 1986; Young et al., 1987), surface-active substances (Yanders et al., 1989) or other contaminants (Puri et al., 1990: chlorophenols, etc.; Puri et al., 1989: waste oil) are present. Kapila et al. (1989) suggest that, in the presence of waste oil, the downward flow is in the form of a colloidal suspension. Other mechanisms which may enhance mobility include adsorption of dioxins to organic carbon, dissolved in the water (humic acids, etc.) (Webster et al., 1986) and particles (Matsumura and Benezet, 1973).

Sometimes, soil profiles reveal a high mobility which cannot be readily explained (Esposito et al., 1980; Di Domenico et al., 1980b). For example, six months after the accident in Seveso dioxin was found at a depth of 1 metre (Di Domenico et al., 1980b). Apart from the mechanisms already mentioned, preferential flow channels (e.g. through crevices) or biological activity may be responsible for this phenomenon (Di Domenico et al., 1980b; Monteriolo et al., 1982). Esposito et al. (1980) attributed it to soil erosion.
Volatilization

Although volatilization (mass flux through soil-air boundary) is negligible over short periods of time, it appears that this process can contribute significantly to dioxin losses from soil in the long run (comparable to DDT; Kearney et al., 1972). The interpretation of data is complicated by the confusion with photochemical transformation. Isensee and Jones (1971) found high volatilization rates for 2,7-DCDD and 2,3,7,8-TCDD, but in the dark no such volatilization appeared to take place (Crosby and Wong, 1977). Work undertaken by Crosby et al. (1971) and Nash and Beall (1980) confirms that short-term volatilization is small, but Nash and Beall point out that its contribution is significant in the longer term.

Studies with volatilization models (Eduljee, 1987), as well as experiments on profiles (Freeman and Schroy, 1985; Palausky et al., 1986) indicate that volatilization may indeed take place. Using a model, Freeman and Schroy (1989) predict a flux of 6 ng.cm\(^{-2}.d^{-1}\) as a result of volatilization. Eduljee (1987) calculated a half-life of 104 days for 2,3,7,8-TCDD, while Thibodeaux and Lipsky (1985) reported a half-life of 190 days. Fachetti et al. (1986) observed contamination of emergent parts of plants cultivated on non-contaminated soils, which was explained as resulting from deposition from contaminated soil nearby (blown up dust).

In general, it may also be stated that the volatilization rate depends on a large number of factors, sometimes in inconsistent ways. Higher levels of organic substances in the soil lead to reduced volatilization as a result of increased sorption. Higher temperatures lead to increased volatilization.

Soil erosion

Young et al. (1987), Muir et al. (1985), as well as Monteriolo et al. (1982) determined that soil particles contaminated with dioxins are transported by wind and water erosion.

Young et al. (1975) suggested soil erosion as a transport process, because 2,3,7,8-TCDD was found in silt from water flows near soils that had been treated with TCDD-contaminated 2,4,5-T, but Young et al. (1983) indicate that this phenomenon does not contribute significantly (\(<\ < \, 1\%\)) to the overall removal process.

Esposito et al. (1980) make a similar suggestion, based on higher levels of dioxins in low-lying terrains.
3.1.2 Transformation in the soil

Dioxins are chemically stable and apart from photochemical processes, chemical transformation does not affect them. In addition, biological transformation might be important. The purification of soils by chemical processes could have some potential (Botré et al., 1979; Des Rosiers, 1986), although soils are affected by the substances added.

Photochemical transformation

Dioxins and dibenzofurans may be rapidly transformed photochemically. Since the wavelength needed forms part of the solar spectrum, transformation under natural conditions is a possibility.

The most important conclusions from numerous photochemical studies are the following. Photochemical transformation is a very fast process in the presence of a hydrogen donor. In the soil hardly any, or no transformation has been observed (Crosby et al., 1971; Crosby et al., 1973; Crosby and Wong, 1977; Plimmer, 1978). Half-lives may run into weeks or years, while hours or days suffice in solutions.

The reduced photochemical transformation found in soils is caused by the shading effect of soil particles and the strong sorption (Crosby and Wong, 1977). In addition, sunlight penetration is limited, so that the process is restricted to the top layers. Miller et al. (1989) only found a significant transformation down to 0.13 mm.

In the presence of hydrogen donors, half-lives in the top layers of soils are found to be in the order of days. The addition of a xylene-ethylene mixture to dioxin-contaminated soil from Seveso (Liberti et al., 1978; Bertoni et al., 1978) resulted in rapid photochemical transformation. Wipf et al. (1978) obtained a similar result with olive oil. Miller et al. (1989) found a 30-50% transformation of OCDD after 5 days, with or without ethylene being present.

In the field, the highest soil levels have not been found in the top layer (several millimetres), probably as a result of photochemical transformation in that layer (Di Domenico et al., 1980a).

The rate of photochemical transformation seems to decrease with increasing chlorine substitution (Crosby et al., 1971; Nestrick et al., 1980). However, these data are not consistent, as the position of the chlorine substitution appears to have an equally important effect, if not greater. Nestrick et al. (1980) have derived a relationship between the rate of
photochemical transformation and the occupation of symmetrical positions by chlorine substituents.

The photochemical transformation in or on solid phases leads to a preferential loss of chlorine on the 1-, 4-, 6- and 9-positions (Nestrick et al., 1980; Lamparski and Stehl, 1980), leading to the formation of more toxic components. The most stable isomer is then 2,3,7,8-TCDD. Miller et al. (1989) confirm this for photolysis of OCDD in soil, which results in the formation of relatively many 2,3,7,8-isomers. In solution, this is exactly the other way round (Nestrick et al., 1980; Dobbs and Grant, 1979; Rappe et al., 1979).

The most important mechanism of photochemical transformation is a reductive dechlorination (Crosby et al., 1971; Desideri et al., 1979; Miller et al., 1989), resulting in dioxins with lower chlorination rates.

Photochemical transformation on plant leaves is also rapid, probably as a result of the presence of hydrogen donors in or near the cuticula (Crosby and Wong, 1977).

(Micro)biological transformation

The only consistent evidence for microbiological transformation stems from laboratory studies. In general, it concerns a co-oxidization process, requiring external sources of carbon and energy (Klecka and Gibson, 1980 - succinate, biphenyl). The rate of oxidation of the dioxins decreased with larger numbers of chlorine substituents (Klecka and Gibson, 1980). Klecka and Gibson (1980) found hydroxylated compounds (probably cis-dihydrodioles) as transformation products of 1- and 2-monochlorodioxin, but not after the oxidation of dichlorodioxins (2,7; 2,3 and 2,8).

Microbiological transformation is hardly, if ever, observed in soils, where other processes (e.g. irreversible binding) complicate the interpretation of data (Arsenault, 1976; Camoni et al., 1982). Kearney et al. (1972) observed some transformation of 2,7-DCDD, but not of 2,3,7,8-TCDD. With the transformation of 2,7-DCDD they detected 5% CO₂, as well as one unidentified polar metabolite. After one year, more than 50% of TCDD could still be determined. After 8 months incubation, Quensen and Matsumura (1983) found up to 1% transformation of 2,3,7,8-TCDD, but demonstrated higher rates of transformation in pure cultures. Muir et al. (1985) detected a polar metabolite with the transformation of 1,3,6,8-TCDD in the soil, at percentages of 0.7, 2.5 and 0% after respectively 131, 321 and 495 days. Hüttner and Philippi (1982) could not detect a significant transformation, nor any CO₂ production, from 2,3,7,8-TCDD, although 1-2% of a hydroxylated metabolite was found (1-
OH-2,3,7,8-TCDD; Philippi et al., 1982).

Current understanding suggests that the transformation of dioxins in soil is extremely slow or negligible, as a consequence of combined characteristics of the compounds.

Anaerobic transformation, which on the basis of what is known about other chlorinated compounds such as chlorophenols (Slooff et al., 1990) and chlorobenzenes (Slooff et al., 1991) might be assumed to contribute significantly, has recently been reported for the first time. Adriaens and Grob'-Galic' (1991) found some indication for anaerobic reductive dechlorination of a number of dioxin congeners in sediments, but not in aquifer material.

Half-lives in soil
Kearney et al. (1972) determined an "overall" half-life of 435-650 days for 2,3,7,8-TCDD in sand and a clay-loam soil. Six years after the application of TCDD-contaminated 2,4-D and 2,4,5-T on a sandy soil for three years, Woolson et al. (1973) could find no residues of 2,3,7,8-TCDD. Di Domenico et al. (1980c) calculated half-lives for 2,3,7,8-TCDD in Seveso and found that these increased over time: 1 year one month after contamination and 10 years 17 months later. Cerlesi et al. (1989) confirmed this, establishing a half-life of 9.1 years under stable conditions.

Muir et al. (1985) determined "overall" half-lives of 130-400 days for 1,3,6,8-TCDD in soil. A half-life in the order of 2-4 years can be calculated for OCDD from data produced by Arsenault (1976).

On the basis of field observations, Freeman and Schroy (1985) found a negligible transformation of 2,3,7,8-TCDD after 12 years.

McKinney (1978) calculated half-lives from 190 to 330 days for 2,3,7,8-TCDD in three experimental plots (sandy loam, silt loam, clay loam). Westing (1978) reports half-lives from 1 to 3 years for 2,3,7,8-TCDD. In a contaminated location (treated with 2,4,5-T and Herbicide Orange), Young et al. (1975) found that 95% of 2,3,7,8-TCDD has disappeared after 8 years. Eight years later, this percentage had increased to 99% (Young et al., 1987). Based on other studies, these authors (Young et al., 1978) reported half-lives of 230 and 320 days for 2,3,7,8-TCDD. At two highly contaminated locations (Agent Orange storage: Johnston Island and NCBC, Gulfport), Young et al. (1983) observed no significant reduction of 2,3,7,8-TCDD within 3 years.

Following soil contamination, Camoni et al. (1982) noted a 25% reduction of 2,3,7,8-TCDD
after 480 days, irrespective of the addition of organic compost. After 15 months, Orazio et al. (1992) reported a measurable decomposition for di- and trichloro-congeners only. Short half-lives may be explained by processes immediately following contamination (photochemical transformation), while long half-lives reflect phenomena in the second phase, after a strong sorption has been established and the only significant contribution remaining is made by volatilization.

3.2 BEHAVIOUR IN SURFACE WATER

3.2.1 Distribution

The dispersal of dioxins in and from surface water occurs through volatilization, adsorption in soil, adsorption from the air, diffusion, transport with suspended soil particles, and uptake by organisms.

Adsorption in sediment and suspended materials
The adsorption in soil has already been discussed in 3.1.1. The adsorption of dioxins in sediment may be described with the same data.

Diffusion in the water phase
Dioxins dissolve particularly poorly in pure water, but bind strongly to soil and sediment (3.1.1). In nature, diffusion of the free, dissolved, substance will therefore usually be less important than transport by means of carriers such as humic acids and suspended sediment. Dissolved humic acids and suspended particles may increase the solubility of these substances in water considerably, and thus enhance their mobility in the water phase.

Volatilization from the water phase
Dioxins may get into air from the water phase, for instance by volatilization (evaporation). Under normal temperatures, the vapour pressure is very low (table 1.3). However, since the solubility of dioxins in water is also low, evaporation may still contribute substantially to the disappearance of dissolved dioxins from the water phase. In this connection, the value of the
Henry constant (H) is of importance (table 1.3).

The role of evaporation in the removal of these substances from the water phase has been studied by adding dioxins to experimental ponds (either by broadcasting salt crystals, coated with a thin layer of the substance, or by evenly injecting a dioxin solution in acetone/benzene under the water surface). Studies of this kind, undertaken with OCDD (Marcheterre et al., 1985) and 1,3,6,8-TCDD (Corbet et al., 1983, 1988), showed a significant evaporation effect, especially at the start of the experiment. After the first day, dioxin levels in the air above the water surface dropped steeply. This is probably due to adsorption of the dissolved substance to sediment, suspended particles or dissolved organic materials, which results in much decreased levels of the substance in the water phase. An estimate of this effect for 1,3,6,8-TCDD shows that it could be responsible for a 10-fold reduction of the evaporation in experimental pond studies (Corbet et al., 1988).

Podoll et al. (1986) give half-lives from 16 to 32 days for the volatilization of dioxins from water (lakes and rivers). Neidhard and Herrmann (1987) calculated half-lives for 2,3,7,8-TCDD and OCDD of 75-250 and 90-300 days, respectively.

No further estimates are given in the literature concerning the contribution of volatilization to the removal of dioxins from surface water. After adsorption and absorption processes in dead and living materials, volatilization is widely regarded as the most important factor in the removal of these substances from surface water.

3.2.2 Transformation

Abiotic transformation

Dioxins have a very stable structure and are little reactive. Hydrolysis and chemical oxidization in surface water may be practically ruled out.

Photochemical transformation

In solution, half-lives have been found in the order of hours. In most of these cases hydrogen donors were present: methanol (Crosby et al., 1971), 1,4-dioxane (Koshioka et al., 1989), iso-octane (Steil et al., 1973), n-hexadecane (Nestrick et al., 1980), and a cationic surfactant (Botré et al., 1978).

Photolysis of dioxins consists of a step-wise reductive dechlorination. The decomposition rate
in solution, under the influence of light, is a pseudo first-order reaction. A relationship exists between the rate and the molecular structure: the number of chlorine atoms and their position inside the dioxin molecule. The decomposition rate diminishes with increasing chlorine substitution of a compound. In aqueous solutions, PCDDs with chlorine atoms on the 2,3,7 and/or 8 positions are more sensitive to light than those with chlorine atoms on different positions: 2,3,7,8-TCDD is the most sensitive to light of all TCDDs.

The above needs to be regarded with some reservation, however. Many of the experiments have not been carried out on solutions in water, but in organic solvents (acetonitrile, hexadecane, etc.), or in mixtures of these with water. The values found by various authors often vary widely. As an example, the half-lives of two TCDDs may be quoted: an analysis of results may lead to the conclusion that 1,3,6,8-TCDD is broken down faster when exposed to sunlight than 2,3,7,8-TCDD, as suggested by half-lives found of 0.3 days and 6 days respectively (Choudry and Webster, 1986: based on an experiment in water/acetonitrile; Dulin et al., 1986), but the opposite may be found too: half-lives of 507 minutes and 57 minutes respectively (Nestrick et al., 1980: experiment in hexadecane). Half-lives have been calculated for eight dioxins in different seasons at 40° N.L. (important in connection with the position of the sun) (table 3.1). Averaged over one year, these half-lives varied from 1.5 days (1,3,6,8-TCDD) to 2,400 days (1,2,3,4,6,7,8-HpCDD) (Choudry and Webster, 1986).

As factors like water transparency, depth, mixing rate, etc., play a role in practice, these values should be regarded as indications only. It is assumed that the behaviour of dibenzofurans does not differ from that of dioxins. Hutzinger et al. (1973) demonstrated that photolytic behaviour of DCBFs and OCBFs does not differ from that of dioxins.

**Table 3.1**  Calculated half-lives (in days) for photochemical transformation of dioxins in surface water at 40° N.L.

<table>
<thead>
<tr>
<th>Dioxin congener</th>
<th>Spring</th>
<th>Summer</th>
<th>Autumn</th>
<th>Winter</th>
<th>Overall</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>2,3,7-TrCDD</td>
<td>2.1</td>
<td>1.8</td>
<td>3.2</td>
<td>5.4</td>
<td>8.7</td>
<td>2</td>
</tr>
<tr>
<td>1,2,3,7-TCDD</td>
<td>0.3</td>
<td>0.3</td>
<td>0.5</td>
<td>0.8</td>
<td>1.5</td>
<td>2</td>
</tr>
<tr>
<td>2,3,7,8-TCDD</td>
<td>1.1</td>
<td>0.9</td>
<td>2.1</td>
<td>4.9</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>2,3,7,8-TCDD</td>
<td>6.0</td>
<td>6.0</td>
<td></td>
<td></td>
<td>6.0</td>
<td>1</td>
</tr>
<tr>
<td>1,2,3,4,7-PeCDD</td>
<td>18</td>
<td>15</td>
<td>29</td>
<td>52</td>
<td>77</td>
<td>2</td>
</tr>
<tr>
<td>1,2,3,4,7,8-HxCDD</td>
<td>7.6</td>
<td>6.3</td>
<td>12</td>
<td>22</td>
<td>32</td>
<td>2</td>
</tr>
<tr>
<td>1,2,3,4,6,7,8-HpCDD</td>
<td>56</td>
<td>47</td>
<td>88</td>
<td>156</td>
<td>239</td>
<td>2</td>
</tr>
<tr>
<td>OCDD</td>
<td>21</td>
<td>18</td>
<td>31</td>
<td>50</td>
<td>863</td>
<td>2</td>
</tr>
</tbody>
</table>

References: 1: Dulin et al. (1986); 2: Choudry and Webster (1986); 3: Podoll et al. (1986)
Biodegradation

Mainly because of their poor solubility, the decomposition of dioxins in surface water or sediment has been studied less than their decomposition in soil.

Organisms, in particular bacteria and fungi, are capable of transforming dioxins into other compounds under laboratory conditions. In natural situations, the transformation of these substances hardly ever takes place, as they resist being broken down and the conditions are seldom favourable. The persistence of dioxins is mostly due to the fact that few organisms are capable of breaking the (unnatural) bond between carbon and chlorine. An example of unfavourable conditions is the low level of availability of dioxins in the environment, because of their high degree of adsorption to solid materials.

In 1973, Matsumura and Benezet demonstrated that, out of approximately 100 strains of bacteria that were capable of breaking down persistent pesticides, five were able to transform a small portion of the 2,3,7,8-TCDD offered. The adaptation of these bacteria to the digestion of other chlorinated compounds has probably contributed to the fact that a relatively large number of them was able to transform dioxins.

The degree of transformation of 2,3,7,8-TCDD by pure cultures of the bacterial species Nocardia sp. and Bacterium megaterium appears to be closely linked to the solvent used. The highest rate of transformation is found when ethylacetate is used as a solvent (Quensen and Matsumura, 1983), probably as a result of greater biological availability.

The transformation of dioxins by micro-organisms appears to take place through co-oxidization. This means that another source of carbon and energy is present in addition to these substances (Klecka and Gibson, 1980; Parsons et al., 1990; Parsons and Storms, 1989). The fungus Phanerochaete chrysosporium is capable of mineralizing 2,3,7,8-TCDD to CO₂ (4% after 60 days), by means of co-oxidization (Bumpus et al., 1985).

No micro-organisms have as yet been described which destroy dioxins completely by using it as their only growth substrate (Keuning and Janssen, 1987).

The more chlorine atoms a dioxin contains, the slower is its rate of transformation. For instance, after adaptation to the decomposition of monochlorobiphenyls, the bacterium Alcaligenes denitrificans is capable of breaking down 2-MCDD and 2,8-DCDF, but 1,3,7,8-TCDD will be hardly affected (Parsons et al., 1990).

A bacterium species of the genus Beijerinckia co-oxidized 1-MCDD faster than non-chlorinated dibenzodioxin. When the number of chlorine atoms of the dioxin was increased in this
experiment, the rate of transformation decreased sharply. It was furthermore observed that bacterial growth was reduced by the presence of the waste products formed (Klecka and Gibson, 1980).

As the metabolites are difficult to measure, little is known about the breakdown pathways of dioxins. Klecka and Gibson (1980) found that various MCDD-, DCDD- and TCDD-congeners are oxidized to the corresponding cis-dihydrodioles. 1-hydroxy-2,3,7,8-TCDD has been identified as a metabolite of microbial transformation of 2,3,7,8-TCDD (Philippi et al., 1982). When 2-MCDF is broken down by the bacterium *Alcaligenes denitrificans*, 5-chlorosalicylic acid is one of the metabolites. From the presence of this substance it is evident that bacteria are capable of opening the dibenzofuran ring (Parsons et al., 1990).

In laboratory experiments, the half-lives of dioxins subjected to biological transformation in aqueous solutions varies from a few days for 2-MCDF and 1,3-DCDD (Parsons et al., 1990; Parsons, 1991) to at least 600 days for 2,3,7,8-TCDD (Ward and Matsumura, 1978) in a sediment-water system. Yockim et al. (1978) found a reduction of 18% in 2,3,7,8-TCDD levels in sediments in a microcosm study, from which it was possible to calculate a half-life of 333 days.

Recently, the anaerobic transformation of dioxins in sediment was described for the first time (Adriaens and Grob’ Galic’, 1991). Following the addition of 1,2,4,6,8-PeCDF, 1,2,3,4,6,9-HxCDD and 1,2,3,4,6,7,9-HpCDD, a reductive dechlorination was observed, leading to the formation of HxCDD from the HpCDD congener. However, studies on sediment nuclei demonstrate that anaerobic transformation of various dioxin congeners has hardly, if ever, taken place in Dutch sediments over a period of 20-40 years (Beurskens and Van de Guchtte, 1992).

### 3.3 Behaviour in Air

#### 3.3.1 General

In connection with the behaviour of dioxins in air, the most important processes are those determining persistence and residence time. These processes are: transformation to other substances (less hazardous ones) and deposition (wet and dry). When dioxins are present in the atmosphere in the gas phase, these processes will be determined by the specific chemical
properties of the compounds concerned. When, however, dioxins are mostly bound to dust particles, a description of the physical characteristics of these particles will be sufficient. It is therefore of crucial importance to distinguish between the gas phase and the particulate phase.

The binding to dust particles appears to be mostly a function of the vapour pressure of the congener concerned and the available particle surface (Bidleman, 1988). As the vapour pressure of a certain compound depends heavily on temperature, the gas phase/particulate phase ratio is also strongly determined by temperature. It follows from a bibliography made by Shiu et al. (1988) that vapour pressures, as determined by different authors, may vary several orders of magnitude. Predictions of gas phase/particulate phase ratios, based on vapour pressure values, are therefore highly uncertain. On the basis of vapour pressures of various congeners it may, however, be stated that the less chlorinated dioxins (TCDD, TCDF) will be mostly found in the gas phase and the highly chlorinated ones mostly in the particulate phase.

Various authors have studied the PCDD/PCDF ratio in the gas phase and in the particulate phase. It has been concluded (Nakano et al., 1990) that these compounds are predominantly found in the particulate phase (appr. 85%), although the tetra-, penta- and hexa-CDD/CDF occur mostly in the gas phase, on the basis of individual congener distributions. This has been confirmed by measurements undertaken in North America (Eitzer and Hites, 1989). Table 3.2 gives the fractions of dioxins in air, bound to particles, as measured by these authors in Bloomington, Indiana, over a period of more than one year. For comparative purposes, the ratios predicted in accordance with the Junge method, described in Noordijk and De Leeuw (1991), have also been included. The saturation vapour pressures for the various congeners have been taken from Eitzer and Hites (1989). When comparing measured and predicted fractions, it should be realised that persistence of particles in the atmosphere is less than that of dioxins in the gas phase. At greater distances from sources, this will lead to a relative increase of dioxins in the gas phase.
Table 3.2  Particulate dioxin fractions in air

<table>
<thead>
<tr>
<th>Congener group</th>
<th>Fraction determined (Eitzer and Hites, 1989)</th>
<th>Fraction calculated (Junge’s model)¹⁾</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt; 3⁰ C</td>
<td>16-20⁰ C</td>
</tr>
<tr>
<td>Tetra-CDD/F</td>
<td>0.5</td>
<td>0.1</td>
</tr>
<tr>
<td>Penta-CDD/F</td>
<td>0.8</td>
<td>0.3</td>
</tr>
<tr>
<td>Hexa-CDD/F</td>
<td>0.95</td>
<td>0.6</td>
</tr>
<tr>
<td>Hepta-CDD/F</td>
<td>1.0</td>
<td>0.9</td>
</tr>
<tr>
<td>Octa-CDD/F</td>
<td>1.0</td>
<td>0.95</td>
</tr>
</tbody>
</table>

¹⁾ Junge (1977) has described a model for the adsorption of (semi)volatile organic substances to aerosols. The fraction which is reversibly adsorbed (φ), the saturation vapour pressure (P₀) and the aerosol surface totally available (θ) are mutually related as: \( \phi = c \theta /(P₀ + c \theta) \), where c is a constant. Bidleman (1988) advises to use the saturation vapour pressure of the liquid phase for \( P₀ \).

3.3.2 Deposition

Important removal mechanisms are the following:
- Dry deposition, the uptake of substances at ground level by soil, water or vegetation;
- Wet deposition, the uptake in cloud- or rainwater; the substances are transported to ground level by precipitation.

Dry deposition

In general, it may be stated that the rate of deposition is determined by meteorological factors and the (chemical) characteristics of a compound, together with the characteristics of the kind of surface (vegetation, soil, water) on which the deposition takes place. The dry deposition flux of a compound may be estimated from the product of the level in air and the deposition rate. Deposition rates of (gaseous) dioxins have not been measured.

On the basis of characteristics such as water solubility and chemical reactivity, it may be assumed that the dry deposition rate of gaseous dioxins will be very low. Dry deposition of gaseous dioxins may therefore be ignored as a removal mechanism. Dioxins on fir needles, as found by Reischl et al. (1987), might hence be attributed to dry deposition of dioxins bound to particles.

For particulate contamination (aerosol), the diameter and the specific mass of the particle determine the rate of deposition (Seinfeld, 1986). A minimal deposition rate is found for
particles with a diameter between 0.1 and 1 \( \mu m \). Smaller particles behave more or less like gases, and show higher rates of deposition. Gravity becomes steadily more important for particles larger than 1 \( \mu m \). Dry deposition is a particularly efficient removal mechanism for coarse particles.

**Wet deposition**

In the case of wet deposition, the following distinction can be made:

- **rain-out/cloud scavenging**: a process which takes place in the clouds, resulting in the compounds being taken up by cloud water and subsequently removed with precipitation;
- **wash-out/below-cloud scavenging**: a process with takes place underneath the clouds, by which gaseous or particulate contaminants are being washed down by raindrops falling down.

As far as gases of poor water solubility are concerned, as is the case with many organic compounds, cloud water will already be too close to saturation levels of these compounds for additional uptake by falling raindrops. For such compounds, rain-out is therefore more important than wash-out. The transition from aerosols to the water phase takes place in a very efficient manner, inside as well as underneath the clouds, especially with aerosols larger than 1 \( \mu m \). Wet deposition close to a source is almost entirely determined by the wash-out mechanism, partly because the plume has not yet interacted with the clouds, and partly because the concentration in the plume is much higher than in the clouds.

The effective removal rate during rain may be expressed as follows:

\[
K_n = W \times \frac{R_i}{H_m} \quad [\text{h}^{-1}]
\]

where \( R_i \) is the rain intensity (m/h) and \( H_m \) the mixing layer height (in m) and \( W \) the ration between the average concentrations in rain water \( (C_w) \) and air \( (C_a) \):

\[
W = \frac{C_w}{C_a}
\]

The (equilibrium) ratio between the level of a compound in water and in air may be related to the Henry coefficient for the compound in question, as follows:

\[
H = R \times \frac{T}{W}
\]

where \( R \) is the gas constant \( (8.3 \text{ Pa.m}^3\text{.mol}^{-1}\text{.K}^{-1}) \), \( T \) the temperature \( (^\circ \text{K}) \) and \( H \) the Henry coefficient \( (\text{Pa.m}^3\text{.mol}^{-1}) \).
Shiu et al. (1988) have published Henry coefficients of 7-10 for tetra-CDD (as representatives of dioxins predominantly occurring in gaseous form). These produce \( W \) values between 240 and 3,500. At these levels, the yearly average removal term under average conditions in the Netherlands will amount to 0.003, respectively 0.055 \( \% \cdot \text{h}^{-1} \). For aerosols, \( W \) is in the order of \( 10^5 \) to \( 10^6 \), depending on particle size distribution. The corresponding averages for the wet removal terms are 1, respectively 4 \( \% \cdot \text{h}^{-1} \). The differences between wet deposition rates for the gas and particulate phase are confirmed by measurements carried out in the USA by Eitzer and Hites (1989). The congener patterns found in rainwater by these authors correlate strongly with those in the gas phase. In addition, they found relatively more highly chlorinated dioxins in rainwater and sediment than in air.

3.3.3 Transformation

Apart from deposition processes, (photo)chemical transformations also determine the persistence of dioxins in air. In this case it is equally important whether the congeners are considered in the gas phase or in the particulate phase. In the latter case, photochemical transformation will probably not play an important role, least of all when the dioxin is mostly "locked up" inside the particles. Decomposition of more highly chlorinated dioxins will lead to a loss of chlorine atoms and hence possibly to the formation of more toxic congeners. Buser (1979) and Dobbs and Grant (1979) convincingly argue that this is not the case.

Two decomposition mechanisms may be distinguished:

[1] **Photochemical oxidisation under the influence of OH-radicals**

Neidhard and Herrmann (1987) and Podoll et al. (1986) start from reaction rates of 2,3,7,8-TCDD with OH-radicals of respectively \( 3.3 \times 10^{-13} \) and \( 5.0 \times 10^{-13} \) molec\(^{-1}\)cm\(^3\)s. In doing so, they estimate the average OH-radicals levels at \( 5.0 \times 10^5 \), respectively \( 1.8 \times 10^6 \) molec.cm\(^{-3}\). The resulting lifespan estimates in the troposphere are 50 and 10 days, respectively.

[2] **Decomposition by photolysis**

A more important mechanism is decomposition under the influence of solar UV light.
UV light absorbed by 2,3,7,8-TCDD shows an absorption maximum at 305 nm (Mill, 1983). Experiments in the laboratory demonstrate that photolysis may be effective, especially for TCDDs and TCDFs. Nestrick et al. (1980) have measured photolytic rates which result in half-lives in the order of 1 hour for 2,3,7,8-TCDD in hexane solutions to 140 hours for other TCDDs. However, the same substances tested on a glass plate show half-lives of 140 hours for 2,3,7,8-TCDD, and down to 1 hour for other TCDDs. Dobbs and Grant (1979) conclude from laboratory experiments that the most toxic dioxins are at the same time the most sensitive to photodecomposition.

No measurements have been undertaken of photolytic rates of dioxins in the atmosphere. Neidhard and Herrmann (1987) estimated the persistence of 2,3,7,8-TCDD in air under Central European conditions from minimally 1 day (clear summer weather) to twenty or thirty days (dark winter weather).

**Table 3.3**

<table>
<thead>
<tr>
<th>Overview of rates of loss and resulting residence times in the atmosphere</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry</td>
</tr>
<tr>
<td>Deposition</td>
</tr>
<tr>
<td>% h(^{-1})</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>2,3,7,8-TCDD (gaseous)</td>
</tr>
<tr>
<td>Particles</td>
</tr>
<tr>
<td>Particles</td>
</tr>
<tr>
<td>Particles</td>
</tr>
</tbody>
</table>

\(^1\) assuming complete mixing in a mixing layer of 800 m

\(^2\) assuming a dry deposition rate of 0.0005 m s\(^{-1}\)

### 3.3.4 Atmospheric residence time

Table 3.3 provides a comparison of half-lives for the various removal processes. These lead to the conclusion that gaseous dioxins are removed mostly through photochemical decomposition, dioxins on particles mostly through wet deposition and dioxins on coarse particles mostly through dry deposition. During summer, removal through transformation will play a more important role than during winter, on the one hand because of the relatively higher proportion of (lesser chlorinated) dioxins in the gas phase and, on the other hand, because of the higher rate of photolytic processes.
3.4 BEHAVIOUR IN BIOTA

3.4.1 Aquatic organisms

The data in this paragraph have been based on corresponding data in Chapter 5. Most of the data on accumulation of dioxins in aquatic organisms are based on studies with freshwater fish. The uptake of dioxins can take place from various media, such as water, sediment and food. Equilibrium bio-concentration factors (BCF-values, calculated from uptake- and elimination rate constants), determined for a number of dioxins on the basis of accumulation studies in freshwater fish vary between approximately 100 to 86,000. By far the highest equilibrium BCF-values were determined for 2,3,7,8-TCDD; for this compound a value of around 40,000 is considered the most reliable. From two experiments with fish in which "2,3,7,8-TCDD" levels were determined with different analytical methods (scintillation counts of $^{14}$C or $^{3}$H following oxidative destruction of the fish, or GC-MS analysis following 2,3,7,8-TCDD extraction), it appears that by far the largest proportion of "2,3,7,8-TCDD" found in fish consisted of un-metabolized 2,3,7,8-TCDD. For the remaining compounds (especially PCDDs) the equilibrium BCF-values usually vary between 500 and 2,500-5,000. In accumulation studies, some BCF-values have also been calculated for 2,3,7,8-TCDD from the $C_{\text{organism}}/C_{\text{water}}$ quotient. These resulted in the following values: 740-3,700 for snails, 1,800- 7,100 for Daphnias and 5,800-28,700 for fish. In the same manner, BCF-values for 2,3,7,8-TCDF in fish were found to be between 2,450 and 6,050. From the BCF-values mentioned (all of them based on fresh body weights of the organisms), it appears that dioxins can be accumulated (concentrated) to a considerable extent from the water phase. However, the equilibrium BCF-values based on experiments are usually much lower than those calculated from (log)Kow values. The BCF$_{b}$-values ($C_{\text{organism}}/C_{\text{water}}$) of various dioxins, determined in sediment-water systems with contaminated sediment, are usually < 1 (invertebrates; fish). Irrespective of the exposure route, the highest accumulation levels in aquatic organisms were found for certain congeners with 4 to 6 chlorine atoms, including 2,3,7,8-substituted congeners.

3.4.2 Terrestrial organisms

Of dioxins present in the soil, probably little is taken up by plants. Even if uptake does occur
through the roots, little of it will be transported to the emergent parts. Although levels "in" roots may still be higher than those in soil, it is unclear to what extent this reflects uptake in the roots (absorption), or absorption to the outer surface of the roots (external contamination through absorption). In crops with demonstrable levels "in" emergent parts, this is probably related to external contamination, involving deposition from the air as well as volatilization from the soil.

Concentration factors (CF-values) for the accumulation of 2,3,7,8-TCDD in earthworms vary from 0.07 to 9.4; except one, all of these values are around 1 or < 1. These values (C_{organic}/C_{soil}) are based on the level of un-metabolized 2,3,7,8-TCDD in the worms (based on fresh body weight). When these experimentally determined CF-values are standardized for a soil with 10% organic matter, this results in CF_{st.}-values from 0.2 to 4.7, with a geometric mean of 0.7. Most standardized values are < 1. From these data it follows that 2,3,7,8-TCDD is hardly, if ever, concentrated in rainworms from soil. At high levels of 2,3,7,8-TCDD in soil, however, correspondingly high levels may be found in earthworms, which is partly attributable to the low susceptibility for this compound in earthworms. For instance, 2,3,7,8-TCDD levels of 0.75 - 2.2 mg.kg\(^{-1}\) fresh body weight were found in earthworms, exposed to soil with a level of 5 mg.kg\(^{-1}\), - the highest non-lethal dose known. In dead earthworms, exposed to a lethal level of 10 mg.kg\(^{-1}\), levels were found of 1.8 - 3 mg.kg\(^{-1}\) fresh body weight. CF-values for other dioxins are hardly available, if not at all. In one study only, CF-values have been determined for a number of PCDFs in earthworms, which resulted in values of 0.004 - 2.4 (fresh weight).

From various field studies it appears that selective accumulation of certain 2,3,7,8-substituted PCDDs and PCDFs with 4 to 8 chlorine atoms takes place in higher animal species, in fish-eating birds of prey as well as mammals (preying on worms). Bio-magnification, or the accumulation in the food chain, has been demonstrated for a number of these dioxins. Among other things, this applies to 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 2,3,7,8-TCDF and 2,3,4,7,8-PeCDF, which belong to the most toxic dioxins. However, the data with regard to bio-magnification are inconsistent. The differences found may partly be explained by species-specific differences, associated with habits (nutrition) and kinetics.
3.5 SUMMARY AND CONCLUSIONS

As a consequence of their physico-chemical properties, dioxins absorb quite strongly to organic materials in soils and sediments, sometimes leading to irreversible binding. As a result, very little transport takes place in soil and groundwater. Only when transport is facilitated (by the presence of other compounds, such as solvents, organic compounds in solution, or by preferential flow channels), transport may take place to deeper layers. In surface water solubility may be significantly increased by the presence of (halogenated) compounds in solution or particles.

Under natural conditions, adsorption to sediments is the most common mechanism for the removal of dioxins from surface water. In addition, volatilization plays a certain role (half-lives 10 to hundreds of days). In soil, volatilization is a relatively slow process, but in the absence of other (transport) processes it might contribute significantly in the long-term (half-lives a hundred days or more).

The only abiotic transformations of importance in surface water and soil are the decomposition reactions under the influence of light. In this process, chlorine atoms are removed from the molecule one by one, in particular through reductive dechlorination, resulting in lower chlorinated congeners. In general, a higher chlorination seems to be associated with a lower rate of decomposition in water as well as in soil, but the position of the chlorine substitutes also has a role to play. In water, it seems as if chlorine atoms on the 2,3,7, and 8 positions are the most easily removed, while the 2,3,7,8-substituted congeners are the most stable ones in soil.

In practice, the photochemical behaviour of dioxins will strongly depend on the characteristics of the surface water concerned. For eight dioxins, half-lives of 1.5 - 2,400 days have been determined.

In soil, photochemical transformation can only contribute in the top layer, where various interactions (presence of hydrogen donor, sorption) determine the rate. Half-lives in the top layer of soil vary from several days to weeks.

Although transformation of dioxins by bacteria and fungi has been demonstrated under laboratory conditions, it cannot be demonstrated in the field because of conditions unfavourable for decomposition. Dioxins cannot be utilized as the sole source of carbon and energy. The transformation takes the form of co-oxidisation. Higher levels of chlorination generally lead to lower rates of transformation. Half-lives in water vary from several days
for lower chlorinated dioxins (1 or 2 chlorine atoms) to hundreds of days for higher chlorinated dioxins. Overall half-lives in soil vary from one to more than 10 years. The transformation produces hydroxylated metabolites. Although little is known about anaerobic transformation, reductive dechlorination seems to be a possibility, even the limited field data available do not suggest this.

In air, dioxins occur in the gas phase as well as bound to particles. The binding to particles is a function of characteristics of the dioxins (vapour pressure) and the carrier particles, and the environmental conditions (temperature, for example). In particular lower chlorinated dioxins (up to and including tetra) occur in the gas phase, while higher chlorinated dioxins are predominantly bound to particles.

The behaviour of dioxins is strongly associated with their distribution. Removal mechanisms include deposition and transformation. In particular dioxins bound to particles in air are removed by dry and wet deposition. Gaseous dioxins are removed mostly through chemical transformation, by one of two mechanisms: photochemical oxidation under the influence of OH-radicals (half-life: 10 - 50 days), or photolysis (half-life: one day to several weeks). Chlorine atoms are lost during these processes, which leads to less toxic congeners being produced. Dioxins bound to particles are hardly, if ever, subjected to transformation. Atmospheric persistence of dioxins varies from 2 to 60 hours for particulate dioxins and from 20 to 200 hours for gaseous dioxins.

As a result of high Kow-values, a strong accumulation from the water phase takes place in aquatic organisms. BCF-values measured for various congeners vary from 100 to 86,000 (generally lower than those based on Kow calculations). For 2,3,7,8-TCDD, a measured BCF-value of approximately 40,000 is considered the most reliable. BCF-values in respect of contaminated sediment are usually below 1, with the highest values found for congeners with 4 - 6 chlorine atoms. Earthworms are hardly capable, or not at all, to accumulate 2,3,7,8-TCDD from soil. From standardisation of experimentally determined CF-values for 2,3,7,8-TCDD in worms to a soil with 10% organic matter, a CF_{st.}-value is derived of 0.2 - 4.7, with a geometric mean of 0.7. In fish-eating birds and mammals preying on worms, findings suggest the biomagnification of 2,3,7,8-substituted dioxins with 4 - 8 chlorine atoms. Uptake of dioxins by plants or crops from soil is not likely. Contamination of crops is probably due to external contamination (below ground level through adsorption to roots, above ground as a result of deposition from air and volatilization from soil).
4. CONCENTRATIONS, FLUXES AND EXPOSURE LEVELS

4.1 MEASURING METHODS

4.1.1 History

In the early seventies methods were developed for the analysis of dioxins for studies on their formation in various industrial and chemical processes, and their occurrence in products such as PCBs (Vos et al., 1970), chlorophenols and their derivatives (Buser and Bosshardt, 1976), in pesticides (Courtney and Moore, 1971), as well as in ashes and flue gases from waste incineration (Olie et al., 1977). In the methods packed column gas chromatography (GC) was used combined with mass-spectrometric detection (GCMS). Due to the poor separating ability of these GC-columns, it was only possible to carry out collective determinations or total dioxin determinations of the various congener groups (with 4 to 8 Cl-atoms) and compound classes (CDD/F) without any specification into levels of individual components. By the end of the 70s techniques became available rendering possible the first specific determinations, particularly the determination of 2,3,7,8-TCDD, considered to be the most toxic dioxin occurring as a byproduct in products such as 2,4,5-T and chlorophenols that were used at a large scale (Rappe, 1978).

In the subsequent years the technique became increasingly sophisticated which led to the current possibilities, facilitating the determination of practically every individual toxic isomer. The introduction of the TEF principle (Bradlow and Casterline, 1979; Van Zorge et al., 1989) contributed to this development, leading to the shift in interest in not further specified total dioxin levels to the concentrations of the toxic isomers. Initially this involved the twelve 2,3,7,8-substituted tetra- to hexa-substitued PCDDs and PCDFs (the so-called 'dirty dozen'; McConnel et al., 1976), however, at present it includes all seventeen substances including the hepta- and octachlorinated ones.

The latest developments in the field of dioxin analysis include what is called the bio-assay (Knutson et al., 1982). Bio-assays are based on a sensitivity (morphological or toxicological responses) of certain biological systems towards dioxins and/or toxicologically related compounds. The technique is still struggling with imperfections, hampering a broad application. Lack of selectivity and too low a sensitivity are disadvantages. In the near future the technique could be used as a screening method for the GCMS analysis which is
considerably more costly (Schiller et al., 1987).

4.1.2 Overview of measuring methods

For the quantitative determination of PCDDs and PCDFs highly reliable methods and strategies are required in both the sampling and the chemical analysis. In table 1.4 an overview is given of the methods that have been applied in recent studies in the Netherlands. Since techniques used for clean-up and analysis are for the greater part identical, these have not been incorporated (see discussion on clean-up and analysis later in this section). For each of the methods indicated only a limited number of references is given.

Sampling
As compared to other determinations reliable analyses of PCDDs and PCDFs are very costly (appr. Dfl. 4000.- per analysis). This is a result of the high degree of complexity of the determination, the use of expensive reagents, solvents, reference compounds and analysis equipment. In addition they are very time-consuming. Therefore, considerable attention is given to a well-founded sampling strategy and the use of adequate sampling equipment and techniques, in order to bring down the cost of analysis involved in such studies.

The sampling strategy should be aimed at obtaining samples which are representative for the system, process or species to be studied and relevant for the studies concerned. In the sampling strategy attention should be given to the proper timing of sampling, the location (geographical, depth, altitude etc.), the sampling density and/or sampling frequency (dynamic processes) and other internal or external factors. The technique used may also influence the final results, which for instance is quite evident in emission determinations at stationary sources. Studies have shown (Marklund, 1990) that dioxin emissions from waste incinerators may highly fluctuate. In addition, a lack of certainty exists about the validity and the comparability between the various types of sampling equipment at lower emission levels. At the request of the Ministry of Housing, Physical Planning and Environment a national interlaboratory comparison study has been initiated on dioxin determination in flue gases. The results will be reported early in 1993. In addition, a programme was started in 1991 by the Committee for European Normalization (CEN) aimed at the standardization of dioxin emission determinations (CEN TC/264) concerning sampling and analysis, which should lead to a validated method in 1993.
Extraction

In the literature a large number of methods has been described for the analysis of dioxins in a wide range of matrices. Several important methods are those for drinking water (Hileman et al., 1984), industrial waste flows (Swanson et al., 1988), soil/sediment (Exner et al., 1987), flue gases and fly ash (Marklund et al., 1987), ambient air (Smith et al., 1987), plant material (Hummel, 1989), chemical products (Lilienfeld, 1989) and human and animal tissue, milk and blood (Lindström, 1988). The choice of the extraction method mainly depends on the nature of the sampling material as well as on the sampling technique. Thus, as a result of their hydrophobic nature, dioxins in a particle-free water sample are easily obtained by extraction with a non-water miscible solvent (benzene, toluene), or by adsorption to XAD-2 or other appropriate absorbents (LeBel et al., 1986; O’Keefe et al., 1986). However, in the case of the water sample containing particles such as suspended sludge, an additional method will need to be used for the isolation of the particle bound dioxins. To this end various methods have been developed, of which the most frequently used is the so-called soxhlet extraction (Lamparski and Nestrick, 1980). Biota, such as fish, human and animal tissue and plant material, belong to the most difficult sampling matrices and have recently been reviewed by Firestone (1991). In a number of cases sampling these matrices requires additional pretreatment in the form of a digestion of the sample (Firestone et al., 1979) to obtain a higher extraction efficiency of intracellular PCDD and PCDF. In some methods, for the same reasons, matrices such as fly ash and soil, were treated with acid (Korfmacher et al., 1985). Extraction methods for dioxins in biological materials are based on the fact that dioxins, because of their lipophilic nature, mainly occur in/on the fat fraction. Recoveries from such methods are in general quantitative (among others Liem et al., 1990a). It is far more difficult to determine the extraction efficiencies for particle bound dioxins. They are usually determined by means of recovery of known additions, whereby it is still uncertain whether the behaviour of the added dioxins is fully representative for the native dioxins.

Clean-up

The aim of cleaning up extracts is to remove as much as possible the bulk of co-extracted matrix components and compounds possibly interfering in the GCMS analysis. The required degree of clean-up partly depends on the relative concentration compared to other chemical contaminations in the samples as well as on the selectivity and robustness of the analytical technique. Intensive clean-up procedures are usually labour-intensive and may be coupled
with increased risks of losing the component(s) to be determined. These disadvantages should be in balance with the advantages such as a reduced risk of false positive or false negative results, a better signal-noise ratio for quantification and an improvement of the capillary GC performance by removal of the excess of matrix components. Clean-up methods are usually based on the Smith et al. method (1984), and consist of a removal of organic co-extractants by means of fractioning over acid/base impregnated silica gel and activated carbon. Variants are: size exclusion chromatography (Niemann et al., 1983), gel permeation chromatography (Van Rhijn et al., 1992) and high pressure liquid chromatography (HPLC) with activated carbon on silica gel (Korfmacher, 1985) followed by a separation of chlorinated organic compounds (such as PCBs) by means of acid or base activated aluminum oxide-column chromatography.

Analysis

In the past 20 years analytical determination of PCDDs and PCDFs have developed dramatically. Methods have been improved from those in which the presence of non-specified PCDDs and PCDFs at the parts per million (ppm) level can be detected to the quantitative, isomer-specific analysis of practically all 2,3,7,8-substituted congeners at the parts per trillion (ppt) level. In this combination the gas chromatography (GC) offers the separation and information required for unequivocal identification of individual isomers while the mass spectrometry (MS) distinguishes between the various congeners (number of Cl-atoms) and classes (PCDD or PCDF). The identification and quantification is carried out using $^{13}$C$_{12}$-labelled reference compounds which at present, for each of the seventeen congeners to be determined, can be obtained commercially. These compounds are added to the analytical sample in known quantities prior to sample processing. During the processing procedure these additives are supposed to behave in a way similar to that of the compounds to be determined. The compounds show an almost identical retention behaviour in the gas chromatography, thus they behave as markers and are, because of the difference in mass, easily to be distinguished from the compounds to be analyzed by MS-detection.

The mass spectrometric separation and detection can be carried out at low (quadrupole MS) and high resolution (HR), and recently also by means of tandem MS (MS/MS; Tondeur et al., 1987). A considerable improvement can be reached with modern high resolution sector instruments with which measurements can be carried out at a sufficient rate. This -as compared to the previous low resolution measurements- leads to a considerable improvement
of the selectivity. Since it links this high selectivity with extremely high sensitivity the HRGC-HRMS is considered the standard method for analyses of trace levels in relatively highly contaminated samples. MS/MS methods offer an even higher selectivity than HRMS and are therefore extremely suitable for samples containing many closely related compounds. However, MS/MS methods are, as far as equipment is concerned, complex, costly and less sensitive (De Jong et al., 1989).

**Gaschromatography**

In the gaschromatography two types of GC columns are generally used, that is columns with a polar stationary phase (90% polycyanopropylsiloxane; CPSil-88, SP-2331 and equivalents) or columns with an non-polar stationary phase (95% methylpolysiloxane; CPSil-5, DB-55 and equivalents). With polar columns practically any 2,3,7,8-substituted PCDD or PCDF can be separated from the other isomers, whereas on non-polar columns the toxic congeners can only be mutually separated. The choice of the column therefore depends on the objectives - isomer-specific, collective determination or profile - and by the dioxin composition of the sample. Environmental and emission samples almost always contain the whole spectrum of PCDDs and PCDFs (210 congeners), whereas biological samples, in particular in mammals, as a result of specific bio-retention of 2,3,7,8-substituted compounds, almost exclusively contain the seventeen toxic congeners. As a result a non-polar column can be used for the latter samples, which in practice offers some advantages such as a shorter duration of analysis and a longer life span.

A complete isomer specificity is not possible with columns at present commercially available. Interferences with non-toxic isomers occur for instance with 2,3,7,8-TCDF (2,3,4,8-TCDF); 1,2,3,7,8-PeCDF (1,2,3,4,8-PeCDF) and 1,2,3,4,7,8-HxCDF (1,2,3,4,7,9-HxCDF) (Buser et al., 1985). As a result reported TCDD equivalence levels (TEQ) are in many cases of a conservative nature (overestimation). Another problem attached to the use of the polar column is that decomposition (dechlorination) may take place for highly chlorinated PCDDs and PCDFs (Olie et al., 1989). In a number of protocols this is taken into account, by carrying out the analysis of hepta- and octa-CDD and -CDF separately on a non-polar column (BCR).

**Mass spectrometry**

Electron impact (EI) is the most frequently applied ionization method. Furthermore negative
ion-chemical ionization (NCI) is used mainly with low resolution (quadrupole) instruments. This technique offers a slightly higher sensitivity and selectivity (Buser, 1980), however, a disadvantage being that the toxicologically most significant isomer, 2,3,7,8-TCDD, is less sensitive to NCI-detection, rendering frequent re-analysis under EI-conditions necessary (Rappe et al., 1983). Identification of individual congeners takes place based on the retention time and the measured $^{35}\text{Cl}^{37}\text{Cl}$ intensity ratio of at least two ions from the molecular ion-cluster. Determination limits depend on the available and/or workable sample size. In table 4.1 the required amounts of starting material per analytical sample and the detection (lower) limits to be achieved are given for a number of relevant matrices.

**Quality Assurance-Quality Control Procedures**

The determination limits indicated in table 4.1 highly depend on the pattern and, if expressed in I-TEQ, on the distribution of the toxicity equivalents over the congeners. If samples mainly contain hepta- and octa-CDD/F (pentachlorophenol pattern) this will result in a lower determination limit on a TEQ basis because of the low relative toxicity (TEF) of these congeners (0.01 and 0.001, respectively). Quality Assurance-Quality Control procedures in most cases indicate determination limits which should be more appropriate than three times the lowest detectable concentration to be expected, with recoveries of the $^{13}\text{C}_{12}$ labelled standards between 50 and 120%. Other general quality requirements are: reporting on the reproducibility of the method (short and long term), calibration of quantification standards, measurements for the control of contamination of the materials and instruments used, and the use of unequivocal criteria for identification and quantification. Results from interlaboratory comparison studies show that the comparability of data for analyses in routinely used matrices lies within 25%, providing the laboratories have the required experience. Data from recent studies are: WHO, human milk, 25% (Stephens et al., 1991), RIVM, cow’s milk, 12% (De Jong et al., 1993), BGA, paper, 36-46% (Beck et al., 1989), RIVM, soil, appr. 10% (Liem et al., 1990). At present standardization and interlaboratory comparison studies are carried out for items such as flue gas measurements near WIs for emissions smaller than 0.1 ng I-TEQ.m$^{-3}$ (CEN, 1991-93; VROM, 1991-92), cow’s milk (BCR, 1992) and biota (WHO, 1991-92).

These efforts will in the near future lead to a high degree of standardization in dioxin measurements for various matrices, which will improve the reliability of the methods and the comparability of international data.
### Table 4.1
**Sampling strategy, techniques and measuring methods applied in measurements of PCDDs and PCDFs in the Netherlands**

<table>
<thead>
<tr>
<th>Compartent</th>
<th>Sampling method</th>
<th>Principle</th>
<th>Amount of sample</th>
<th>Analytical method (Ref)</th>
<th>Detection limit (Ref)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emission</td>
<td>Isokinetic, NPR 2788</td>
<td>Separation of particulate matter</td>
<td>10 mg</td>
<td>1. solvent extr. after HCI</td>
<td>0.1-1 ng 1-TEQ/m³</td>
</tr>
<tr>
<td>(flue gas)</td>
<td>using Stoebel dilution method</td>
<td>Air bound tetra- and penta CDD/F on glass fiber</td>
<td></td>
<td>2. clean-up</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chlorinated compounds</td>
<td></td>
<td>3. GC/MS</td>
<td></td>
</tr>
<tr>
<td>Ambient air</td>
<td>High Volume Sampler with glass filter/PUFs</td>
<td>Filtration of particulate matter</td>
<td>1000-5000 mg</td>
<td>1. solvent extr., toluene</td>
<td>5 ng 1-TEQ/m³</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Filtered and gaseous components</td>
<td></td>
<td>2. clean-up</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3. GC/MS</td>
<td></td>
</tr>
<tr>
<td>Water</td>
<td>Continuous flow</td>
<td>Separation of suspended matter</td>
<td>1000 l water (100 g dm³)</td>
<td>1. solvent extr., toluene</td>
<td>1-10 ng 1-TEQ/kg dm³</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2. clean-up</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3. GC/MS</td>
<td></td>
</tr>
<tr>
<td>Soil</td>
<td>Each location several spots at different depths (0-5 or 0-10 cm)</td>
<td>Mixed samples</td>
<td>25-50 g dm³</td>
<td>1. solvent extr., toluene</td>
<td>1 ng 1-TEQ/kg dm³</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2. clean-up</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3. GC/MS</td>
<td></td>
</tr>
<tr>
<td>Biological material:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>* (cow/milk)</td>
<td>Mixture of 6 samples per cow per month</td>
<td>Analyses of monthly milk samples</td>
<td>150 g milk (5-6 g milk fat)</td>
<td>1. addition sodilumurate</td>
<td>0.1-1 ng 1-TEQ/kg fat</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2. addition methanol</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3. diethyl-ether, ether extr.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4. clean-up</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5. GC/MS</td>
<td></td>
</tr>
<tr>
<td>* (meat/fat)</td>
<td>Sampling of fat or liver</td>
<td>Amount correspond to fat melting down at 40 C</td>
<td>5-10 g fat</td>
<td></td>
<td>0.1-1 ng 1-TEQ/kg fat</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7</td>
</tr>
<tr>
<td>* fish</td>
<td>Several samples each location</td>
<td>Analyses of mixed sample</td>
<td>5-10 g fat</td>
<td>1. drying with sodium sulphate</td>
<td>0.1-1 ng 1-TEQ/kg fat</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2. dichloromethane extr.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3. see milk, steps 4 and 5</td>
<td></td>
</tr>
<tr>
<td>* Food prod.</td>
<td>Sampling in 4 regions</td>
<td>National mixed sample</td>
<td>Amount correspond to 5-10 g fat</td>
<td>1. extr. with org. solvent</td>
<td>0.3 ng 1-TEQ/kg fat</td>
</tr>
<tr>
<td>(others) at any given supplier</td>
<td>Sample composed of 4 regional mixed samples</td>
<td></td>
<td>2. clean-up</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3. GC/MS</td>
<td></td>
</tr>
</tbody>
</table>

References: 1: Sibol et al. (1992); 2: Bolt and De Jong (1992); 3: Wegman et al. (1989); 4: Lien et al. (1990b); 5: Van Rijn et al. (1992); 6: Lien et al. (1990a); 7: De Jong et al. (1990); 8: Lien et al. (1991)

## 4.2 OCCURRENCE IN SOIL AND GROUNDWATER

Current understanding of the occurrence of dioxins in soil and groundwater is as yet based on poorly comparable data. In some cases only one or a few isomers (usually 2,3,7,8-TCDD) have been determined, in other cases only the totals per group of homologues and sometimes only the totals. Data for the calculation of I-TEQs are very limited indeed.

Gradually systematic studies on the occurrence in soil are being carried out. However, studies on the occurrence in groundwater have hardly been performed.
4.2.1 Occurrence in soil

Data on the occurrence of dioxins in soil have been collected over the past fifteen years. Initially it involved the occurrence as a result of accidents. Only in the past five years have background levels been determined.

Since only relatively few data are involved, the relevant data from abroad will also be discussed. Data concerning the occurrence in soil can be distinguished into two categories:

1. occurrence at a regional scale;
   a. atmospheric deposition (particularly WIs);
   b. background levels;

2. occurrence at a local scale;
   a. accidents, such as Times Beach and Seveso;
   b. pesticide industry: 2,4,5-T, Agent Orange and pentachlorophenol contaminations, as a result of both application and waste flows (Diemerzeedijk, Volgermeer polder).

Regional data from the Netherlands

Because of the possible contribution of dioxins in soil to the load in cows in particular, studies were started in 1990 on the occurrence of dioxins in the soil near several waste incinerators (De Jong et al., 1990 and 1991; Matthijsen et al., 1991, Bremmer et al., 1991). The selected locations generally concerned grazing lands.

In table 4.2 the dioxin levels in the soil at various depths from several locations in the Netherlands are indicated in ng I-TEQ.kg⁻¹ dry matter.

The first soil analysis data from the Dutch source studies show that:

- the lowest levels occur at the reference location (Bergambacht);
- near the WI Zaanstad, the highest levels so far have been found, up to a factor 5 times higher than the highest values at other locations;
- levels decrease below 2 cm ground level and that below 10 cm ground level they are practically similar to those found at reference locations, confirming the immobility of dioxins.

The congener patterns of the samples from the various locations correspond reasonably well and all suggest dioxins to originate from fly ash deposition. No significant differences in pattern have been found either between the samples taken at various distances from the WI Leeuwarden. This may indicate an emission to particles, since otherwise a distinction in
distance of the low and high chlorinated dioxins would have been expected.

As a consequence of the combination of properties of dioxins (low mobility, persistence) the levels in the soil reflect the deposition history on that soil, provided the soil concerned has not been cultivated during the deposition period.

In figure 4.1 the deposition on the various locations (situation end of 1989) has been indicated on the map of the Netherlands (see 4.4). On this map the available soil concentrations measured have been indicated. Qualitatively as well as quantitatively the comparisons seem to be reliable, which means that outside of the very local deposition areas and the Randstad, (background) levels in soil may be expected in the range of 2 to 5 ng I-TEQ.kg\(^{-1}\).

**Local data from the Netherlands**

In table 4.2 data from other studies in the Netherlands are also indicated.

The soil samples from the Bommelerwaard originate from grazing lands bordering on a ditch with discharge points from mushroom farms (Liem et al., 1990). After PCP-contaminated sludge was dredged from the ditch the neighbouring grazing lands were suspected to be contaminated with PCP and the concomitant dioxin residues. The congener pattern indicates a typical pentachlorophenol (PCP-) contamination.

Turkstra and Pols (1986) report that through the deposition of sludge during the inundation of river forelands elevated levels may be found. In the two samples studied levels were found of 4.2 and 176 ng I-TEQ.kg\(^{-1}\) dry matter, respectively.

In a study on dioxin levels in the soil at illegal burning sites of cables and cars, levels of 60 to 98,000 ng I-TEQ.kg\(^{-1}\) dry matter were determined (Liem et al., 1991). Levels at the car scrap-yard (60-160 ng I-TEQ.kg\(^{-1}\) dry matter) were lower than those at the illegal burning sites. The highest level of 2,3,7,8,-TCDD found was 3.4 ng.kg\(^{-1}\). It is striking that at these levels in the soil a deviating congener pattern is observed, mainly with furans, the hexa- and heptachlorofurans in particular.

At the rubbish tip Volgermeerpolder a maximum of 1929 ng 2,3,7,8-TCDD.kg\(^{-1}\) was found in the soil (Heida and Olie, 1989) and in the soil of the Diemerzeedijk 41 ng I-TEQ.kg\(^{-1}\) was found (Heida et al., 1989) at open air burning sites for chemical waste, however, no 2,3,7,8-TCDD was detected. In the residue in the centre of the burning site a 2,3,7,8-TCDD level of 50,000 ng.kg\(^{-1}\) was determined.
Figure 4.1  Calculated deposition distribution over the Netherlands, including contributions from abroad for the situation end 1989 (Van Jaarsveld, personal communication, 1990), compared with the levels in the soil at various depths for a number of locations in the Netherlands. The depositions are expressed in ng I-TEQ.m\(^2\).year\(^{-1}\) and the soil levels in ng I-TEQ.kg\(^{-1}\) dry matter.
### Table 4.2
Data on the regional and local occurrence of dioxins in soil (based on ng 1-TEQ/kg d.m.) in the Netherlands

<table>
<thead>
<tr>
<th>References</th>
<th>Number of samples</th>
<th>Levels in ng 1-TEQ/kg dm</th>
<th>Remarks concerning location</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>minimally</td>
<td>maximally</td>
</tr>
<tr>
<td><strong>Regional scale:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>De Jong et al., 1991</td>
<td>5</td>
<td>13</td>
<td>252</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>12</td>
<td>46</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>De Jong et al., 1990</td>
<td>5</td>
<td>18</td>
<td>51</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>13</td>
<td>55</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>10</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>nd</td>
<td>nd</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>2</td>
<td>9</td>
</tr>
<tr>
<td>Matthijsen et al., 1991</td>
<td>10</td>
<td>2.5</td>
<td>22.6</td>
</tr>
<tr>
<td>Bremmer et al., 1991</td>
<td>1</td>
<td>2.6</td>
<td>22.6</td>
</tr>
<tr>
<td><strong>Local scale:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Turkstra and Pols, 1986</td>
<td>2</td>
<td>4</td>
<td>180</td>
</tr>
<tr>
<td>Liem et al., 1991</td>
<td>4</td>
<td>60</td>
<td>160</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>380</td>
<td>98,000</td>
</tr>
<tr>
<td>Liem et al., 1990</td>
<td>2</td>
<td>60</td>
<td>1,110</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heida and Olie, 1985</td>
<td>9</td>
<td>&lt;20 TCDD</td>
<td>1929 TCDD</td>
</tr>
<tr>
<td>Heida et al., 1989</td>
<td>4</td>
<td>0.7</td>
<td>41.1</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>3.2</td>
<td>3.2</td>
</tr>
</tbody>
</table>

### Data from abroad
In tables 4.3 and 4.4 data from abroad have been listed. Since, in general, no data were available to enable a direct calculation of the I-TEQ values, the formula given by Birmingham (1990) has been used to make an estimate based on a given congener distribution.
Comparison the Netherlands with foreign countries

Data on the regional occurrence of dioxins in soil in the Netherlands and in foreign countries are in general comparable. Background values may highly vary, however, on average they approximate 10 ng I-TEQ.kg\(^{-1}\). Elevated levels are observed near WIs, as well as in industrial and urban areas.

The highest levels are found near local pollution. It is striking that the levels in soils as a result of application of sludge are higher than those as a result of atmospheric deposition. When sludge is concerned the dioxins often originate from pentachlorophenol.

Extremely high levels have been found in metallurgic industries, in the Netherlands (illegal cable burning sites) as well as in Germany. The highest reported levels are found in the case of accidents. The highest reported level amounts to 33 mg 2,3,7,8-TCDD.kg\(^{-1}\) (Kimbrough et al., 1977). The highest reported level in the Netherlands is 98 µg I-TEQ.kg\(^{-1}\) (Liem et al., 1991).

4.2.2 Occurrence in groundwater

Data on the occurrence of dioxins in groundwater are scanty, and concerning the Netherlands not available at all. This is probably due to the low water solubility of the dioxins and the resulting analytical possibilities.

During a survey in the USA of 1555 water samples "no dioxin presence" was reported in any of the cases. During similar studies in Canada Jobb et al. (1990) report that in 399 water samples OCDD was found 37 times with a concentration of 9-175 pg.l\(^{-1}\) and TCDD only once (no 2,3,7,8-TCDD, however) with a concentration of 40 pg.l\(^{-1}\). In Germany a groundwater load was found only in two instances: in the near vicinity of a municipal waste dump in Baden-Württemberg and on a former factory site at Hamburg (Umwelt, 1989). Götz et al. (1986) report the occurrence of dioxins in the perculate of an industrial waste dump (Georgswerder, Hamburg), however, without being able to properly carry out a quantification. Based on sediment levels concentrations of up to 30 pg.l\(^{-1}\) were calculated.
Table 4.3  Data on the regional occurrence of dioxins in soil (based on ng I-TEQ/kg d.m.) outside of the Netherlands

<table>
<thead>
<tr>
<th>References</th>
<th>Number of samples</th>
<th>Level in ng I-TEQ/kg dm</th>
<th>Remarks</th>
<th>Concerning</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>minimum</td>
<td>maximum</td>
<td>location</td>
</tr>
<tr>
<td>Creaser et al. (1989)</td>
<td>66</td>
<td>0.63 #</td>
<td>39 #</td>
<td>rural area</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>2.3 #</td>
<td>12 #</td>
<td>urban area</td>
</tr>
<tr>
<td>Rotard et al. (1991)</td>
<td>7</td>
<td>0.3</td>
<td>2.7</td>
<td>farmland</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>0.7</td>
<td>30</td>
<td>grazing land</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>5.4</td>
<td>112</td>
<td>forest</td>
</tr>
<tr>
<td>Birmingham (1990)</td>
<td>30</td>
<td>nd</td>
<td>2.2 #</td>
<td>rural area</td>
</tr>
<tr>
<td></td>
<td>47</td>
<td>nd</td>
<td>78 #</td>
<td>urban area</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>1.7 #</td>
<td>102 #</td>
<td>industrial area</td>
</tr>
<tr>
<td>NRW (1991)</td>
<td>69</td>
<td>0.6</td>
<td>11</td>
<td>rural area</td>
</tr>
<tr>
<td></td>
<td>28</td>
<td>0.8</td>
<td>27</td>
<td>urban area</td>
</tr>
<tr>
<td>Rappe &amp; Kjeller (1987)</td>
<td>3</td>
<td>1.4</td>
<td>5.6</td>
<td>rural area</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>55</td>
<td>251</td>
<td>industrial area</td>
</tr>
<tr>
<td>Pearson et al. (1990)</td>
<td>23</td>
<td>nd</td>
<td>55 #@</td>
<td>urban area</td>
</tr>
<tr>
<td></td>
<td>43</td>
<td>nd</td>
<td>2.8 #@</td>
<td>rural area</td>
</tr>
<tr>
<td></td>
<td>29</td>
<td>nd</td>
<td>110 #@</td>
<td>urban area, background value</td>
</tr>
<tr>
<td>Creaser et al. (1990)</td>
<td>19</td>
<td>12 #</td>
<td>140 #</td>
<td>urban area</td>
</tr>
<tr>
<td>Rotard et al. (1991)</td>
<td>4</td>
<td>11</td>
<td>1976</td>
<td>Halle</td>
</tr>
<tr>
<td>Morselli et al. (1989)</td>
<td>5</td>
<td>11 #</td>
<td>65 #</td>
<td>WI vicinity</td>
</tr>
<tr>
<td>Berlincioni &amp; DiDomenico (1987)</td>
<td>7</td>
<td>nd</td>
<td>9.1 #</td>
<td>not cultivated, WI</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>2.1 #</td>
<td>4.0 #</td>
<td>cultivated land</td>
</tr>
<tr>
<td>McLaughlin et al. (1989)</td>
<td>11</td>
<td>0.05 #</td>
<td>41 #</td>
<td>WI Hamilton</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.8 #</td>
<td>36 #</td>
<td>background levels</td>
</tr>
<tr>
<td>Stenhouse &amp; Badsha (1990)</td>
<td>12</td>
<td>3</td>
<td>20</td>
<td>chem.waste incineration</td>
</tr>
<tr>
<td>Eduljee et al. (1986)</td>
<td>8</td>
<td>4.1 #</td>
<td>39 #</td>
<td>chem.waste incineration</td>
</tr>
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<td></td>
<td>1</td>
<td>1.1 #</td>
<td></td>
<td>record sample 1877</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>3.7 #</td>
<td>173 #</td>
<td>background levels</td>
</tr>
<tr>
<td>Reed et al. (1990)</td>
<td>0.8</td>
<td>9.2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* only 2,3,7,8-TCDD  
# I-TEQ value estimated by means of the Birmingham method, 1990  
@ I-TEQ value estimated based on minimum/maximum levels given
Table 4.4  Data on local occurrence of dioxins in soil (based on ng I-TEQ/kg dry matter) outside of the Netherlands

<table>
<thead>
<tr>
<th>References</th>
<th>Number</th>
<th>Level in ng I-TEQ/kg dm</th>
<th>Remarks concerning</th>
<th>location</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>samples</td>
<td>minimum</td>
<td>maximum</td>
<td></td>
</tr>
<tr>
<td>Hagenmaier (1990)</td>
<td>9</td>
<td>0.1 #</td>
<td>2.2 #</td>
<td>without sludge application with sludge application</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>4.4 #</td>
<td>261 #</td>
<td>Germany</td>
</tr>
<tr>
<td>Rotard et al. (1990)</td>
<td>16</td>
<td>0.3</td>
<td>149</td>
<td>sludge residue paper mill on land</td>
</tr>
<tr>
<td>Prinz et al. (1990)</td>
<td>4</td>
<td>27 #</td>
<td>79 #</td>
<td>wire reclamation incinerators background</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>2.5 #</td>
<td>20 #</td>
<td></td>
</tr>
<tr>
<td>Spindelbalk et al. (1990)</td>
<td>20</td>
<td>3</td>
<td>332</td>
<td>metal smelter</td>
</tr>
<tr>
<td>Riss et al. (1990)</td>
<td>1</td>
<td>5</td>
<td>19</td>
<td>highway forest</td>
</tr>
<tr>
<td>Wendling et al. (1989)</td>
<td>1</td>
<td>500000 *</td>
<td>1400000 *</td>
<td>waste oil, Times Beach Newark, 24ST-production</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td>USA</td>
</tr>
<tr>
<td>Yanders et al. (1989)</td>
<td>68 *</td>
<td>176 *</td>
<td></td>
<td>Times Beach</td>
</tr>
<tr>
<td>Young et al. (1987)</td>
<td>56</td>
<td>2375 *</td>
<td></td>
<td>Florida, Eglin AFB</td>
</tr>
<tr>
<td>Young et al. (1983)</td>
<td>234</td>
<td>3400000 *</td>
<td></td>
<td>Gulfport and Johnston Island, USA storage Agent Orange</td>
</tr>
<tr>
<td>DiDomenico et al. (1980)</td>
<td></td>
<td>86000 *</td>
<td></td>
<td>Seveso, 1976</td>
</tr>
<tr>
<td>Sivers &amp; Friesel (1989)</td>
<td>900 *</td>
<td>874000 *</td>
<td></td>
<td>chemical waste dump</td>
</tr>
<tr>
<td>Jürgens &amp; Roth (1991)</td>
<td></td>
<td>9000000 *</td>
<td></td>
<td>pesticide plant</td>
</tr>
<tr>
<td>Mineiro et al. (1991)</td>
<td></td>
<td>3400</td>
<td></td>
<td>pesticide plant</td>
</tr>
<tr>
<td>Kimbrough et al. (1977)</td>
<td>9</td>
<td>33000000 *</td>
<td></td>
<td>soil in horse arena</td>
</tr>
</tbody>
</table>

* only 2,3,7,8-TCDD
# TEQ-value, calculated with BGA-TEFs
4.3 OCCURRENCE IN WATER AND SEDIMENTS

Studies on sediments from lakes in North America and Switzerland show that since the 40s an ever increasing amount of dioxins may be observed and that a relationship exists between the increase of the amounts of dioxins over time and the production of chloroaromates (Czuczwa and Hites, 1986; Buser and Müller, 1986). The homologue profiles (group totals) in Switzerland agreed well with those in the USA as well as those of dioxins present on dust particles originating from urban areas (Czuczwa et al., 1985). These data may indicate (a comparison on the basis of homologue profiles instead of congeners profiles is less reliable) that the chemical industry and combustion processes can be considered as significant sources for dioxins in surface water (CCRX, 1991).

The presence of dioxins in the Dutch surface water is mainly caused by deposition from the air and the transfrontier load in the form of suspended sludge via the big rivers. In this section data have been indicated which may enable the quantification of these dioxin flows (see also 4.6).

Measurements in the water phase have scarcely been carried out or not at all. Because of the low solubility of dioxins, their high lipophilicity and extremely high adsorption to suspended particles, transport and storage of dioxins mainly take place via particles, the lightest material being the most significant (CCRX, 1991). The following data have been extracted from studies on sediment samples collected at various locations from 1984 to 1990, and from a recent study on sediment profiles from Lake Ketel. In table 4.5 the data for the sediments studied have been summarized. For levels in water organisms (fishery products) reference is made to section 4.5.

4.3.1 Rhine basin

In samples of settled Rhine sediment, dating from the period 1984-1985 and collected at various measuring points along the route between Augst (Switzerland) through the Hollands Diep, Evers et al. (1988) reported dioxin levels ranging from 0.01 ng I-TEQ.kg⁻¹ d.m. upstream to 310 ng I-TEQ.kg⁻¹ d.m. in the industrial downstream area. In the Dutch part of the Rhine the levels ranged from 12 to 220 ng I-TEQ.kg⁻¹ d.m. (table 4.5). At various locations the proposed rehabilitation value of 100 ng I-TEQ.kg⁻¹ d.m. was exceeded (Van Zorge, 1987). Dioxin contamination is mainly caused by local industrial processes and by
incomplete incineration of PCB-containing waste in the Ruhr area. In addition it has been derived from the data that a considerable amount of the dioxins in the Dutch aquatic environment originates from transfrontier dioxin contaminated loads (Evers et al., 1988). Turkstra and Pols (1986) calculated for the Rhine a transfrontier load of 50-70 g I-TEQ.year\(^{-1}\) at Lobith.

Data on the Ketel lake sediment (Beurskens and Van de Guchte, 1992) show that the dioxin levels in the more recently sedimented layers originating from the Rhine, are lower than those from former periods. From this it may be concluded that the dioxin levels in the suspended sludge from the Rhine show a downward trend over the past decades. This downward trend may apply to other Dutch sediments as well.

4.3.2 Other rivers and canals

With regard to the other rivers and canals samples collected near potential sources in particular have been studied. The measuring data show that at several locations in the Noordzeekanaal, the Apeldoornskanaal and in the Hollandse IJssel the proposed rehabilitation value is exceeded. The samples were collected near potential sources (rubbish tips, industry). The Noordhollands Kanaal was sampled because of the possible effects from a waste incinerator (Turkstra and Pols, 1986). The concentrations in the sediments from the Meuse and the Scheldt were relatively low. The influx through the transfrontier load for the Meuse amounts to an estimated 5-10 g I-TEQ.year\(^{-1}\) (Turkstra and Pols, 1986).

4.3.3 Harbours and stagnant water

Elevated to highly elevated dioxin levels were found (50-4,000 ng I-TEQ.kg\(^{-1}\) d.m.) in sediments from several heavily loaded harbours. The elevated dioxin level in the sediment from the Derde Petroleumhaven may be ascribed to the presence of an industrial incinerator, although the congener pattern also shows some similarity to that of pentachlorophenol. Two waste incinerators and a production unit discharge on the Eerste Petroleumhaven. In the Chemiehaven eight discharge points were located, the most significant discharge being that from a vinylchloride production unit. Because of its extremely high dioxin levels, sludge from the Chemiehaven will constitute a problem area for quite some time, despite the fact that a purification installation was recently put up that area. In the near vicinity of the
Laurenshaven there is a waste incinerator mainly for domestic waste. The congener pattern deviates considerably from that originating from the Chemiehaven (Evers et al., 1991).

**Table 4.5** An overview of the dioxin levels (in ng I-TEQ kg⁻¹ dry matter) found in Dutch sediments. Rounded figures, extracted from Turkstra and Pols (1986, 1987 and 1989), Evers et al. (1988 and 1991), CCRX (1991) and Beurskens et al. (1991)

<table>
<thead>
<tr>
<th>Rhine basin:</th>
<th>Year</th>
<th>Number of samples</th>
<th>Concentration in ng I-TEQ per kg dry matter</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Tolkamer (frontier)</td>
<td>1984</td>
<td>1</td>
<td>78</td>
</tr>
<tr>
<td>- Lobith</td>
<td>1984</td>
<td>1</td>
<td>22</td>
</tr>
<tr>
<td>- Lobith (river forelands)</td>
<td>1984</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>- Tiel</td>
<td>1984</td>
<td>1</td>
<td>12</td>
</tr>
<tr>
<td>- Dalem</td>
<td>1984</td>
<td>1</td>
<td>220</td>
</tr>
<tr>
<td>- Hollands Diep</td>
<td>1984</td>
<td>2</td>
<td>17</td>
</tr>
<tr>
<td>- New Meuse</td>
<td>1984</td>
<td>2</td>
<td>78</td>
</tr>
<tr>
<td>- Nieuwe Waterweg</td>
<td>1990</td>
<td>2</td>
<td>48</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Other rivers and canals:</th>
<th>Year</th>
<th>Number of samples</th>
<th>Concentration in ng I-TEQ per kg dry matter</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Noordzeekanaal</td>
<td>1980</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>- Apeldoornskaal</td>
<td>1980</td>
<td>1</td>
<td>260</td>
</tr>
<tr>
<td>- Meuse at Eijsden</td>
<td>1985</td>
<td>1</td>
<td>12</td>
</tr>
<tr>
<td>- Meuse at Roermond</td>
<td>1984</td>
<td>1</td>
<td>82</td>
</tr>
<tr>
<td>- Meuse at Grave</td>
<td>1984</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>- Hollands Diep-Moerdijk</td>
<td>1984</td>
<td>1</td>
<td>25</td>
</tr>
<tr>
<td>- Hollandse IJssel</td>
<td>1984</td>
<td>3</td>
<td>42</td>
</tr>
<tr>
<td>- West-Scheldt</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Schaar v.Ouden Doel</td>
<td>1984</td>
<td>1</td>
<td>28</td>
</tr>
<tr>
<td>- N.-Holl.kanaal - Alkmaar</td>
<td>1984</td>
<td>1</td>
<td>12</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Harbours and stagnant waters:</th>
<th>Year</th>
<th>Number of samples</th>
<th>Concentration in ng I-TEQ per kg dry matter</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Eerste Petroleumhaven</td>
<td>1983</td>
<td>1</td>
<td>250</td>
</tr>
<tr>
<td>- Derde Petroleumhaven</td>
<td>1983</td>
<td>1</td>
<td>23</td>
</tr>
<tr>
<td>- Chemiehaven</td>
<td>1983</td>
<td>1</td>
<td>4,000</td>
</tr>
<tr>
<td>- Laurenshaven</td>
<td>1983</td>
<td>1</td>
<td>2,600</td>
</tr>
<tr>
<td>- Lake IJssel - Den Oever</td>
<td>1984</td>
<td>1</td>
<td>91</td>
</tr>
<tr>
<td>- Lake Veluwe</td>
<td>1984</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>- Uimmerput</td>
<td>1984</td>
<td>1</td>
<td>120</td>
</tr>
<tr>
<td>- Haringvliet H12</td>
<td>1984</td>
<td>1</td>
<td>120</td>
</tr>
<tr>
<td>- Lake Ketel</td>
<td>1945</td>
<td>1</td>
<td>59</td>
</tr>
<tr>
<td>- Lake Ketel</td>
<td>1965</td>
<td>1</td>
<td>650</td>
</tr>
<tr>
<td>- Lake Ketel</td>
<td>1985</td>
<td>1</td>
<td>79</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Estuaries and seas:</th>
<th>Year</th>
<th>Number of samples</th>
<th>Concentration in ng I-TEQ per kg dry matter</th>
</tr>
</thead>
<tbody>
<tr>
<td>- West-Scheldt</td>
<td>1990</td>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td>- Waddenzee</td>
<td>1987 &amp; 1990</td>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td>- Eems Dollard</td>
<td>1990</td>
<td>1</td>
<td>10</td>
</tr>
</tbody>
</table>
The dioxin levels in sediment samples from stagnant waters exceed the proposed rehabilitation value (appr. 120 ng I-TEQ.kg\(^{-1}\) d.m.) for the IJmeerput and the Haringvliet. The generally low levels in Lake Veluwe (appr. 10 ng I-TEQ.kg\(^{-1}\) d.m.) may be considered as present background levels in Dutch sediments. Geochronological studies into the occurrence of dioxins in the Ketel lake sediment (Beurskens et al., 1991) show that the highest 2,3,7,8-TCDD values occurred from 1955 to 1975. This may be connected with the production of 2,4,5-T (Beurskens et al., 1991). After 1965 the dioxin levels have decreased, the 2,3,7,8-TCDD ones relatively fastest.

4.3.4 Estuaries and seas

Dioxin levels in sediments from estuaries and seas (range: 8-21 ng I-TEQ.kg\(^{-1}\) d.m.) are low as compared to those found in freshwater sediments. As a result of mixing with marine material the levels in estuary sediments will decrease with decreasing distance from the sea. The sediment from the Oestergronden, an important spawning area for fish such as plaice and cod, and approximately 100 to 150 km northwest off the coast of Terschelling, contains only a slightly lower level than that from the western Wadden Sea. This is remarkable since the Wadden Sea is more affected by the contamination of the Rhine than areas in the open sea.

4.4 OCCURRENCE IN AIR

Dioxin air concentration and deposition measurements in the Netherlands are not available, except for occasional observations (Olie et al., 1983). Mid 1991 a measuring programme was started. The first data from this measuring programme have recently become available (Bolt and De Jong, 1992), however, they are mainly concern a validation of the measuring method (sampling and analysis). For the following considerations therefore data from abroad have been used, together with model calculations. The model calculations have been carried out with the OPS-model (Van Jaarsveld, 1989). The distribution and deposition of particulate contamination is in this model indicated as a function of the particle size. The calculations are based on emissions measured in 1990 at twelve waste incinerators (Slob et al., 1992). In addition the particle size distribution of the emitted compound has been determined at a
number of WIs. Other emissions in the Netherlands are based on Bremmer (1991). Estimates of emissions abroad are based on published data along with estimates for countries and emission categories for which data have not been reported (Schutter and Van Jaarsveld, 1991). In the calculations it is assumed that all source emitted dioxins are particle bound, which is not fully justified as is apparent from section 3.3. Since gaseous dioxins deposit considerably less rapidly than particulate dioxins, the calculated deposition may possibly be an overestimation. This overestimation will on theoretical grounds probably be 25% and on the basis of measurements 50% at the most. However, no indications have been found which point at an actual overestimation.

4.4.1 Indoor air

Concentrations in the occupational environment
Measurements of dioxin air concentrations in the occupational environment are not available. Higher concentrations are to be expected in the production of compounds contaminated with dioxins, such as those in the production of pentachlorophenol. Data concerning this production are not available.

Concentrations outside of the occupational environment
The use of dioxin contaminated materials or compounds in building materials may lead to elevated concentrations of these compounds in indoor air. Eckrich (1987) carried out measurements in 70 day care centres in West Germany. In these day care centres pentachlorophenol had been used as a wood preservative. The concentrations ranged from 0.004 to 2.1 pg.m\(^{-3}\) TCDD equivalents (BGA TEFs, see table 1.4), with a median value of 0.1 pg.m\(^{-3}\). The measurements also indicated that a rise in temperature of 10\(^{\circ}\)C resulted in an increase of the PCDD/PCDF levels by a factor 4 to 10. Measures to decrease the dust concentration greatly influenced the PCDD/PCDF concentrations.

4.4.2 Outdoor air

Concentrations and depositions national/regional
Kirschmer (1987) reports an average value of 4.5 pg.m\(^{-3}\) PCDD and 0.65 pg.m\(^{-3}\) PCDF (together appr. 0.013 pg I-TEQ.m\(^{-3}\)) at two locations in an area not polluted by local sources
(Eifel) during a triple three-days' measuring period. Over a period of 37 days for a rural area 130 km southwest of Stockholm, Broman et al. (1991) report an average of 0.23 pg.m\(^{-3}\) PCDD and 0.19 pg.m\(^{-3}\) PCDF (together 0.0044 pg I-TEQ.m\(^{-3}\)). For an open coastal area 80 km northeast of Stockholm they report values that are about a factor 2 higher. Eitzer and Hites (1989) found 0.24 pg.m\(^{-3}\) PCDD and 0.18 pg.m\(^{-3}\) PCDF during a two-weeks' measuring period in a rural location in Indiana (USA).

Figure 4.2 indicates the distribution of the annual average (of the total of dry and wet) dioxin deposition as calculated on the basis of emissions in the Netherlands for the situation at the end of 1990 (Bremmer, 1991) and emissions abroad (Schutter and Van Jaarveld, 1991). The average dioxin concentration is calculated at 0.025 pg I-TEQ.m\(^{-3}\) and the average deposition at 8 ng I-TEQ.m\(^{-2}\).year\(^{-1}\). In rural areas the annual average concentration amounts to 0.01-0.04 pg I-TEQ.m\(^{-3}\) (rural western Netherlands) and the deposition 2-25 ng I-TEQ.m\(^{-2}\).year\(^{-1}\). The dry deposition rate at a rural scale amounts to appr. 0.005 m.s\(^{-1}\).

**Urban/local**

In outskirts of Hamburg average concentrations were found of 1.9 pg.m\(^{-3}\) PCDD and 1.2 pg.m\(^{-3}\) PCDF (appr. 0.09 pg I-TEQ.m\(^{-3}\))(Bruckmann and Hackhe, 1987). The same authors report 14 pg.m\(^{-3}\) PCDD and 14 pg.m\(^{-3}\) PCDF in a traffic tunnel (50,000 vehicles per day). Broman et al. (1991) found over five days an average concentration in the centre of Stockholm of 0.69 pg.m\(^{-3}\) PCDD and 0.70 pg.m\(^{-3}\) PCDF (together 0.024 pg I-TEQ.m\(^{-3}\)). In outskirts of Stockholm over 47 days 0.61 pg.m\(^{-3}\) PCDD and 0.44 pg.m\(^{-3}\) PCDF were found (together 0.013 pg I-TEQ.m\(^{-3}\)). In an urban/industrial area such as the Rhine-Ruhr area (6 measuring locations) Kirschmer (1987) reported average values of 3.2 pg.m\(^{-3}\) PCDD and 5.5 pg.m\(^{-3}\) PCDF (together appr. 0.12 pg I-TEQ.m\(^{-3}\)). The values found by Hunt and Maisel (1990) in the urban coastal area in the northeast of the USA in winter are in the same order of magnitude, that is 4.4 pg.m\(^{-3}\) PCDD and 0.6 pg.m\(^{-3}\) PCDF (together appr. 0.09 pg I-TEQ.m\(^{-3}\)). The first data from the Dutch measuring programme started in 1991 indicate similar concentrations in an urban/industrial area, ranging from 0.015 pg I-TEQ.m\(^{-3}\) (local background) to immission levels exceeding 0.080 pg I-TEQ.m\(^{-3}\) (Bolt and De Jong, 1992). It is as yet difficult to establish whether the elevations observed as compared to the background concentrations may be due to for example industrial or traffic-related sources.
Figure 4.2  Calculated deposition distribution over the Netherlands including contributions from abroad for the situation at the end of 1990 (Schutter and Van Jaarsveld, 1991). Depositions in ng 1-TEQ.m\(^2\).year\(^{-1}\)

Concentrations in rainwater

Eitzer and Hites (1986) reported dioxin concentrations in rainwater. These measurements have been carried out in Bloomington, Indiana, a town with appr. 50,000 inhabitants. Apart from rainwater concentrations, air concentrations have also been measured both in the gas and the particulate phase. The concentrations in rainwater were on average 81 pg.l\(^{-1}\) PCDD and 13 pg.l\(^{-1}\) PCDF, whereas the corresponding air concentrations amounted to 1.1 and 0.78 pg.m\(^{-3}\), respectively.
Breakdown into congeners shows a marked correlation with particulate concentrations in the air of the same congeners, however not with concentrations in the gas phase. This confirms the statement made in section 3.3 that gaseous PCDDs and PCDFs are hardly removed by wet deposition. The total deposition calculated by the authors mentioned amounted to 530 ng.m\(^{-2}\).year\(^{-1}\) PCDD/F, assuming a dry deposition rate of 0.01 m.s\(^{-1}\). Assuming the concentration of total PCDD/F to be 60 times higher than the I-TEQ-value, the deposition will be 9 ng I-TEQ.m\(^{-2}\).year\(^{-1}\). Wet deposition in this (urban) area constitutes only 18% of the total deposition.

Near point sources

Waste incinicators (WIs) and metal smelters may be considered to be the most significant categories of point sources. Measurements in the near vicinity of a copper smelter in Austria (Brixlegg) show a maximum concentration of 34 pg.m\(^{-3}\) PCDD and 65 pg.m\(^{-3}\) PCDF (appr. 1.2 pg I-TEQ.m\(^{-3}\))(Riss et al., 1990). These maximums were found at appr. 250 m from the stack and were determined as an average over 4 periods of 11 to 15 days.

Bruckman and Hacke (1987) reported in Hamburg at appr. 1500 m leeward of 2 WIs over a three-days’ measuring period concentrations of 23 pg.m\(^{-3}\) PCDD and 40 pg.m\(^{-3}\) PCDF (0.74 pg I-TEQ.m\(^{-3}\)). Calculated annual average dioxin concentrations in the near vicinity of WIs in the Netherlands do not exceed appr. 0.1 pg I-TEQ.m\(^{-3}\) and the deposition does not exceed appr. 200 ng I-TEQ.m\(^{-2}\).year\(^{-1}\). However, higher annual average maximums may be found near sources in the Netherlands, for instance when the emission height is extremely low.

In figure 4.3 the calculated deposition near an arbitrary waste incinicator is given. This deposition is based on the average data as found in emission determinations recently carried out. The deposition shows a maximum value at several hundred meters from the source. This maximum consists mainly of wet deposition. Depending on the source height and heat contents of the flue gases there is a submaximum at 500 - 3000 m from the source, which is caused by dry deposition. Both maximums are found northeast of the source. The direction is mainly determined by the southwesterly winds predominant in the Netherlands, which in addition bring the most precipitation. The dry deposition rate near point sources is highly influenced by the size distribution of the particles in the emitted dust. Installations with relatively high emission factors appear to emit a relatively high number of coarse particles. The dry deposition rate for this kind of installations amounts to appr. 0.02 m.s\(^{-1}\).
Figure 4.3  Calculated deposition distribution at a typical WI. Depositions are expressed in ng I-TEQ m⁻² year⁻¹

The most direct way of assessing the calculated deposition based on measurements, is comparing it with the dioxin level accumulated in the soil (De Jong et al., 1990). In figure 4.4 the result of this assessment is indicated. The "measured" deposition consists of the total of dioxins found in the topmost 10 cm of the soil in the vicinity of a WI, assuming that this deposition was formed over a period of 20 years. The figure shows that the dioxin accumulation found in the soil is indeed explained by the model. The good (spatial) correlation may reveal that the soil accumulation is mainly caused by the concerning WI. A good correlation was also found between calculated deposition and dioxin concentration in cow's milk.
Figure 4.4  Comparison of deposition derived from measurements in soil samples with calculated deposition based on emission. Depositions in ng 1-TEQ·m$^{-2}$·year$^{-1}$
4.4.3 Contribution from abroad to concentrations and depositions in the Netherlands

From section 3.3 it is apparent that the range of small particles in particular is extremely great, which means that apart from a load caused by local sources a considerable contribution may occur from distant sources (abroad). The calculated contribution from abroad to air concentrations in the Netherlands amounts to an average of appr. 45% and to depositions appr. 35% (Schutter and Van Jaarsveld, 1991).

Table 4.6 gives an overview of upper limits of air concentrations such as measured at various locations in Europe for the various spatial scales. As a comparison calculated concentrations and depositions for the Netherlands have also been incorporated.

<table>
<thead>
<tr>
<th></th>
<th>Measured conc.</th>
<th>Calculated conc.</th>
<th>Calculated deposition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pg 1-TEQ.m⁻³</td>
<td>pg 1-TEQ.m⁻³</td>
<td>ng 1-TEQ.m⁻³.y</td>
</tr>
<tr>
<td>Near point sources</td>
<td>&lt;1.2</td>
<td>&lt;0.15</td>
<td>&lt;200</td>
</tr>
<tr>
<td>Urban area</td>
<td>&lt;0.10</td>
<td>&lt;0.05</td>
<td>&lt;40</td>
</tr>
<tr>
<td>Industrial areas</td>
<td>&lt;0.12</td>
<td>&lt;0.05</td>
<td>&lt;40</td>
</tr>
<tr>
<td>Rural area</td>
<td>&lt;0.015</td>
<td>0.01-0.04</td>
<td>2-25</td>
</tr>
</tbody>
</table>

4.5 OCCURRENCE IN FOOD AND DRINKING WATER

Since the dioxin concentrations in food and drinking water are in general extremely low, they can only be detected in a limited number of analytical laboratories. Moreover, the costs involved are extremely high. As a result data are only available for a very limited number of foodstuffs and all date from the last few years. Therefore, there is no insight into possible dioxin concentrations in food and drinking water in the past. Furthermore most analyses are limited to foodstuffs from areas suspected to be polluted.
4.5.1 Drinking water

With a theoretical physico-chemical model Travis and Hattemer-Frey (1987) calculated 2,3,7,8-TCDD concentrations of 5 fg.l\(^{-1}\) in drinking water. Jobb et al. (1990) reported non-detectable concentrations of 2,3,7,8-chlorine substituted dioxins and furans in Canadian drinking water, whereby the detection limit amounted to approximately 10 fg.l\(^{-1}\). Only OCDD could be detected up to concentrations of 40 pg.l\(^{-1}\).

4.5.2 Foodstuffs of vegetable origin

The concentrations I-TEQ in fat and waxlayers, such as in the skin of fruits, are higher than in other parts of plants (Wipf et al., 1982). In vegetable foodstuffs the I-TEQ level generally approximates the current detection limit of 0.1-0.5 pg.g\(^{-1}\). Beck et al. (1989) and Fürst et al. (1990) report concentrations of less than 0.4 pg I-TEQ.g\(^{-1}\) vegetable oil. In various industrially purified vegetable oils Liem et al. (1991) found concentrations of non-detectable levels of up to 0.05 pg I-TEQ.g\(^{-1}\). In fourteen samples of kale originating from various Dutch locations levels have been found of 0.08 to 0.2 pg I-TEQ.g\(^{-1}\) wet weight (Liem et al., 1992).

4.5.3 Meat and animal products

Meat- and milkfat

Results from dioxin analyses in a large number of Dutch foodstuffs are listed in table 4.7. Levels in cow’s milk in cartons amounted to appr. 1.5 pg I-TEQ.g\(^{-1}\) milkfat and are comparable to those from earlier analyses (De Jong et al., 1990a), amounting to 0.8 to 2.5 pg I-TEQ.g\(^{-1}\), and to those found in milktanks of individual dairy farmers in non-suspected areas (Kootstra et al., 1990). However, as a result of local pollution in the vicinity of a waste incinerator, considerably higher dioxin concentrations were found in milkfat amounting to 1.3 to 12.9 pg I-TEQ.g\(^{-1}\), with an average of 5.7 pg I-TEQ.g\(^{-1}\) fat (Liem et al., 1989a and 1991). Concentrations in cheese from a dairy farm located in a polluted area amounted to 4.8 to 11.5 pg I-TEQ.g\(^{-1}\) fat (Liem et al., 1990). Elevated levels of 6.1 to 18 pg I-TEQ.g\(^{-1}\) fat were also found in meat from beef cattle in these loaded areas (De Jong et al., 1990b).
Table 4.7  Dioxin levels in various categories of Dutch food products. The levels have been determined from the analyses of two (unless stated otherwise) separately composed national mixed samples of the products indicated (Liem et al., 1991.* = Liem, pers. comm.)

<table>
<thead>
<tr>
<th>Category</th>
<th>Level (pg I-TEQ.g⁻¹ on a fat basis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>beef</td>
<td>1.8</td>
</tr>
<tr>
<td>beef liver</td>
<td>5.7</td>
</tr>
<tr>
<td>pork</td>
<td>0.43</td>
</tr>
<tr>
<td>pig liver</td>
<td>15</td>
</tr>
<tr>
<td>chicken</td>
<td>1.7</td>
</tr>
<tr>
<td>chicken liver</td>
<td>3.3</td>
</tr>
<tr>
<td>mutton</td>
<td>1.9</td>
</tr>
<tr>
<td>sheep liver</td>
<td>30</td>
</tr>
<tr>
<td>horsemeat</td>
<td>14</td>
</tr>
<tr>
<td>horse liver</td>
<td>61</td>
</tr>
<tr>
<td>goat</td>
<td>4.2</td>
</tr>
<tr>
<td>goat liver</td>
<td>42</td>
</tr>
<tr>
<td>meat products</td>
<td>0.67</td>
</tr>
<tr>
<td>cow's milk</td>
<td>1.5 (n=3)</td>
</tr>
<tr>
<td>butter</td>
<td>1.8</td>
</tr>
<tr>
<td>cheese</td>
<td>1.4</td>
</tr>
<tr>
<td>eggs</td>
<td>2.0</td>
</tr>
<tr>
<td>fat marine fish</td>
<td>6.8</td>
</tr>
<tr>
<td>lean marine fish</td>
<td>49</td>
</tr>
<tr>
<td>eel</td>
<td>28 (n=6)</td>
</tr>
<tr>
<td>freshwater fish</td>
<td>2.4</td>
</tr>
<tr>
<td>domesticated rabbit*</td>
<td>12</td>
</tr>
<tr>
<td>wild rabbit *</td>
<td>22</td>
</tr>
<tr>
<td>wheat flour *</td>
<td>0.40 (n=1)</td>
</tr>
<tr>
<td>rye *</td>
<td>0.32 (n=1)</td>
</tr>
<tr>
<td>wheat *</td>
<td>0.31 (n=1)</td>
</tr>
<tr>
<td>fish oil</td>
<td>2.2 (n=10)</td>
</tr>
<tr>
<td>nuts</td>
<td>0.20</td>
</tr>
<tr>
<td>soybean oil **</td>
<td>0.05 (n=4)</td>
</tr>
<tr>
<td>rape seed oil **</td>
<td>0.01</td>
</tr>
<tr>
<td>palm oil **</td>
<td>0.03</td>
</tr>
<tr>
<td>sunflower oil **</td>
<td>0.01 (n=4)</td>
</tr>
<tr>
<td>coconut fat **</td>
<td>0.02</td>
</tr>
<tr>
<td>palm fat **</td>
<td>0.02</td>
</tr>
</tbody>
</table>

N.B.  The above-mentioned average values are based on two (or more) measuring data with a variation coefficient of appr. 100% for vegetable oils and fats (**), of appr. 5% for fish oils and of appr. 20% for the other products (Liem et al., 1991).

Fish and fish oil

I-TEQ levels found in fish are in general considerably higher than those found in other animal fats. Great differences in concentrations are also found when comparing species of freshwater and marine fish. As is apparent from table 4.7, differences are greatest between fat and lean species of fish. However, when the concentrations per g fish fat are converted to levels on a product basis, practically all differences are levelled out to the range of 0.5 to
5 pg I-TEQ.g\(^{-1}\) fish. These levels have also been found in crustaceans (Hagel, 1990). Dioxin levels in processed fish oil, such as is used in the food processing industry, vary greatly, ranging from less than 0.24 pg to 4.4 pg I-TEQ.g\(^{-1}\) oil (Liem et al., 1991). Crude oil contains considerably higher concentrations of up to 80 I-TEQ.g\(^{-1}\) oil. Considering the great differences in I-TEQ levels in fat, as measured in the various fish species, this variation is not remarkable. Industrial purification processes appear to effectively reduce the dioxin levels, whereby a decrease in concentration by a factor ten is not exceptional (Rappe et al., 1989; Brevik et al., 1990).

4.5.4 Fat in the food processing industry

Concentrations of dioxin and related compounds in foodstuffs from the food processing industry are determined by the percentage of fat in the product, and the type of fat used. According to MVO (1988) about 80% of these industrial fats are from a vegetable origin, whereas the remaining 20% consist of fish oil. Therefore, Liem et al. (1991) estimate a proportional average of 0.02 pg I-TEQ.g\(^{-1}\) industrially used vegetable oil and 0.4 I-TEQ.g\(^{-1}\) for the mixed vegetable and animal oil in the products from the food processing industry.

4.6 FLUXES AND BACKGROUND CONCENTRATIONS IN THE ENVIRONMENT

The calculation of fluxes of PCDDs and PCDFs in the Dutch environment is carried out on a I-TEQ basis. The fluxes are based on a schematized representation of the Dutch environment, the air over the Netherlands, the surface water and the soil (including groundwater) being considered as an ideally mixed system. Obviously this oversimplifies the reality; calculations only serve to get an impression of the PCDD- and PCDF fluxes in the Dutch environment. In the calculation of the fluxes in the Netherlands the total surface is assumed to be 45,750 km\(^2\) (88% soil, 12% water; Slooff et al., 1991).

The calculation of the fluxes is based on the values as mentioned in chapters 2-4. The compartmental and intercompartmental fluxes have been summarized in table 4.8 and figure 4.5. Table 4.9 shows an overview of the background concentrations to be expected in soil, water and air.
Surface water
- The load which through suspended sludge is carried into the Netherlands at Lobith via the Rhine amounts to 50-70 g I-TEQ.year$^{-1}$ (section 4.3). The influx through the transfrontier load in the Meuse amounts to appr. 5-10 g I-TEQ.year$^{-1}$ (section 4.3). According to estimates made by Van Egmond et al. (1991) more than 60 g I-TEQ.year$^{-1}$ ends up in the waters of southern Holland via suspended sludge from the Meuse and the Rhine. Most of it is deposited in the southernmost part of the northern

Table 4.8  Compartmental and intercompartmental PCDD- and PCDF fluxes, expressed in grammes I-TEQ per year

<table>
<thead>
<tr>
<th></th>
<th>Influx</th>
<th>Outflux</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Surface water</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>influx:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Rhine</td>
<td>50-70</td>
<td></td>
</tr>
<tr>
<td>- Meuse</td>
<td>5-10</td>
<td></td>
</tr>
<tr>
<td>outflux (via suspended sludge to sea)</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>emission to water</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>deposition</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>dredged material</td>
<td>&lt;35</td>
<td></td>
</tr>
<tr>
<td>(dumped outside of compartment) accumulation (influx-outflux)</td>
<td>&gt;65-90</td>
<td></td>
</tr>
<tr>
<td><strong>Air</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>influx</td>
<td>500</td>
<td></td>
</tr>
<tr>
<td>emission to air</td>
<td>600</td>
<td></td>
</tr>
<tr>
<td>deposition (outflux from compartment)</td>
<td>370</td>
<td></td>
</tr>
<tr>
<td>outflux (balancing figure)</td>
<td>730</td>
<td></td>
</tr>
<tr>
<td><strong>Soil</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>emission to soil</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>deposition</td>
<td>325</td>
<td></td>
</tr>
<tr>
<td>application sewage sludge</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>diffuse application of dredges</td>
<td>0.3</td>
<td></td>
</tr>
<tr>
<td>removal by grazing cattle</td>
<td>&lt;3</td>
<td></td>
</tr>
<tr>
<td>accumulation (influx-outflux)</td>
<td></td>
<td>330</td>
</tr>
</tbody>
</table>

Table 4.9  Estimated background concentrations in compartments and dioxin fluxes in the Netherlands

<table>
<thead>
<tr>
<th>Compartment-flux</th>
<th>Average concentr.</th>
<th>Range</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soil (rural area)</td>
<td>2-5 ng I-TEQ.kg$^{-1}$ d.m.</td>
<td></td>
<td>section 4.2.1</td>
</tr>
<tr>
<td>Water (sediment)</td>
<td>10 ng I-TEQ.kg$^{-1}$ d.m.</td>
<td></td>
<td>section 4.3.5</td>
</tr>
<tr>
<td>Air (rural area)</td>
<td>0.025 pg I-TEQ.m$^{-3}$</td>
<td>0.01-0.04</td>
<td>section 4.4.2</td>
</tr>
<tr>
<td>Deposition (incl. air contr. abroad)</td>
<td>8 ng I-TEQ.m$^{-2}$.year$^{-1}$</td>
<td>2-25</td>
<td>section 4.4.2</td>
</tr>
</tbody>
</table>
Delta basin (accumulation), whereas a small part will be carried to the sea through the Haringvliet sluices and the Nieuwe Waterweg (appr. 5 g I-TEQ.year\(^{-1}\); Van Egmond et al., 1991) and a small part is deposited in the Rotterdam harbours. In the western part of the Rotterdam harbours the sludge is mixed with "clean" seasludge (pers. comm. Rijkswaterstaat, Directie Zuid-Holland) (Ministry of Transport and Public Works, South Holland).

- Approximately 26 million tonnes of "maintenance sludge" is dredged from the various South Holland waterways annually. Since measuring data are lacking, the amount of dioxins which is removed from the environment is not exactly known. It is estimated that about half of the amount of the deposited suspended material is dredged again (Van der Voet et al., 1989). Assuming the amount of dioxins carried through the rivers to be evenly distributed over the dredged material and a negligible amount via point sources, this would mean a removal of about 35 g I-TEQ.year\(^{-1}\) (Van Egmond et al., 1991). However, about half of the dredged material involves "clean" (sea)sludge and is mainly dumped into the sea again (communication Rijkswaterstaat, Directie Zuid-Holland). Based on this information the removal of 35 g I-TEQ.year\(^{-1}\) mentioned by Van Egmond is probably an overestimation and the dioxin outflow via dredged material will be lower.

- The emissions to surface water amount to appr. 4 g I-TEQ annually (section 2.2, table 2.5) and as a result of deposition appr. 45 g I-TEQ per year ends up in the surface water (average deposition 8 ng I-TEQ.m\(^{-2}\).year\(^{-1}\), section 4.4.2; 12% of
45,750 km\(^{-2}\).

- It is assumed that the remaining dioxins are eventually deposited along with the suspended sludge. Evaporation from the water phase and any possible transformations are considered relatively negligible. Thus accumulation in the sediment amounts to 65-90 I-TEQ.year\(^{-1}\). Since the outflow through dredged material is overestimated, this amount will be higher.

**Air**

- Based on the situation at the end of 1990 appr. 600 g I-TEQ per year will be emitted to the air (section 2.3, table 2.5).

- The amount carried into the Netherlands through the atmosphere (via emissions from abroad and influx from the atmosphere) is estimated to be appr. 500 g I-TEQ.year\(^{-1}\) (based on a contribution of 45% to the Dutch air concentration, see section 4.4.4).

- The amount flowing to the water and soil compartments through deposition (compartment influx) can be calculated from the average deposition of 8 ng I-TEQ.m\(^{-2}\).year\(^{-1}\) and the total surface of the Netherlands of 45,750 km\(^2\) and amounts to 370 g I-TEQ.year\(^{-1}\).

- The amount remaining after influx, emission and deposition is considered to flow out of the country (balancing figure), and it amounts to appr. 730 g I-TEQ.year\(^{-1}\). The contribution of photochemical oxidation and photolysis is considered negligible.

**Soil**

- Of the total average deposition appr. 325 g I-TEQ (88% of 370 g) per year ends up diffusely on the soil.

- Through emissions appr. 3 g I-TEQ.year\(^{-1}\) (section 2.2, table 2.6) ends up diffusely on the soil.

- Through the application of sewage sludge in agriculture (22% of the total production, with an average concentration of 70 pg I-TEQ.g\(^{-1}\) d.m., see 2.2) appr. 7 g I-TEQ.year\(^{-1}\) ends up on the soil, locally or diffusely.

- The removal from the soil by grazing cows can be calculated from the number of animals (appr. 5 million cows; CBS, 1991), consumption data for grass and soil (15 kg dry matter per cow per day and 225 g per cow per day, respectively; Slob et al., 1992), a dioxin level in soil of between 2 and 5 ng I-TEQ.kg\(^{-1}\) d.m. (see 4.2.1), a
dioxin level in grass of appr. 1 pg I-TEQ.g⁻¹ d.m. (Linders et al., in preparation) and a bioavailability of dioxin to grass/soil in the cow of less than 10% (Olling et al., in preparation). From the above-mentioned data a nett removal from the soil by grazing cattle of less than 3 g I-TEQ. year⁻¹ may be calculated.

From the above-mentioned measuring data an accumulation in the soil (balancing figure) of appr. 330 g I-TEQ. year⁻¹ can be calculated. The contribution of evaporation, soil erosion, photochemical and (micro)biological transformation is considered negligible.

4.7 EXPOSURE LEVELS

4.7.1 Sources

Due to the lipophilic properties accumulation occurs in the food chain, and animal fats will be the main source of total exposure. Based on models Travis and Hattemer-Frey (1987) estimate a contribution of 95% via food. This estimate is confirmed in several studies on the contribution of various exposure routes to the exposure of humans to dioxins. Birmingham et al. (1989) estimate a daily contribution of 5 pg I-TEQ per person from soil, water and air and one of 140 pg I-TEQ from food in Canada. Theelen arrived at a contribution of 4 pg I-TEQ from direct exposure to soil, water and air and 116 pg I-TEQ from food in 1989 for the Netherlands.

4.7.2 Food

The daily exposure via food for the total population of Europe, Canada, US and Japan has been estimated in various studies, by multiplying the I-TEQ levels in foodstuffs by the average consumption data in the various countries. The results range from 50 to 170 pg I-TEQ per day (NEPB, 1988; Beck et al., 1989; Theelen, 1989; Birmingham et al., 1989; Beck et al., 1990; Fürst et al., 1990; Faeden, 1991). In the recent food study in the Netherlands (Liem et al., 1991) dioxin levels in foodstuffs were measured, which subsequently were combined with data from about 6,000 individual consumption patterns. The median daily intake per person amounted to 35 to 70 pg I-TEQ for children up to 20
years of age and 70 pg I-TEQ for adults. Based on the individual body weight that corresponds with a median intake of 1 pg I-TEQ per kg body weight per day for adults older than twenty years of age. Young persons of up to twenty years of age showed a higher intake, inversely proportional with age, as a result of the lower body weight. The individual variation for the total intake as well as per kg body weight showed a lognormal distribution. These results are indicated in figures 4.6 and 4.7 in relation to age and body weight, together with the main percentiles.

Figure 4.6 Intake of PCDDs and PCDFs via food in pg I-TEQ per day, put against age
(Source: Liem et al., 1991)
Figure 4.7  Intake of PCDDs and PCDFs via food in pg I-TEQ per kg body weight per day, put against age [Source: Liem et al., 1991]

In addition this study shows that half of the total exposure of I-TEQ via food over seventy years originates from foodstuffs with milk, butter, cheese and associated beef fat (Olling et al., 1990), as is shown in figure 4.8.

Figure 4.8  Contribution of various food categories to the total dioxin intake by the Dutch population (Liem et al., 1991)
Monitoring the dioxin levels in cow's milkfat, as occurs in the framework of the Commodities Act is therefore very effective in the control of the exposure of the total population. The remaining 20% of the exposure originates from fats from industrially processed foodstuffs, for which fish oil in particular is responsible. However, in view of the great variance in levels of dioxin and related compounds in fish oil, this contribution may be assumed to vary considerably. Therefore, a better understanding is required of dioxin levels in fish oil from various geographical origin as well as from various industrial processing techniques and of the flows and products containing fish oil.

4.7.3 Non food

Exposure to non-food products is mainly possible through paper. Contribution from smoking cigarettes appears to be negligible. A study by Liem et al. (1991) shows that the contribution from milkcartons is negligible in relation to the I-TEQ level in milk fat. Average daily exposure through paper tissues and various packaging materials was estimated at 9 pg (EPA)-TEQ per person (EPA, 1990).

4.7.4 Infants

Dioxins present in human milk constitute a source of exposure for breast fed infants. In pooled samples from ten different regions in the Netherlands (each composed of 9 to 13 individual samples) dioxin levels have been determined ranging from 31 to 40, with an average of 34 pg I-TEQ.g\(^{-1}\) fat (Liem et al., 1989b). At an individual level a greater variation could be born in mind, in the order of 10-100 pg I-TEQ.g\(^{-1}\) fat (see e.g. Pluim et al., 1992). Assuming an average daily intake of 150 ml mother's milk per kg body weight for infants, the exposure will be appr. 150 pg I-TEQ per kg body weight per day, based on a fat content of 3% and a dioxin content of 34 pg I-TEQ.g\(^{-1}\) fat.
4.8 SUMMARY AND CONCLUSIONS

In the near future the reliability and comparability of measuring results will be improved nationally as well as internationally by standardization of measurements and quality assurance/quality control of dioxin measurements. However, a number of Dutch laboratories is at the present time able to carry out these measurements in a reliable way.

Data on the occurrence in the soil are in general scarce or comparable to a limited extent. The occurrence is mainly restricted to the top 10 cm of the soil. Based on currently available regional and local data a background level varying from 2 to 5 ng I-TEQ.kg⁻¹ dry matter is expected, whereas near sources (WIs) as well as industrial and urban areas higher dioxin levels may be expected (local determinations to appr. 100 µg I-TEQ.kg⁻¹ d.m.). The levels are comparable to those from abroad.

Levels in Dutch sediments suggest a background concentration of appr. 10 ng I-TEQ.kg⁻¹ dry matter. This value can be exceeded locally under the influence of potential sources such as incinerators and production processes. The relatively high dioxin levels in heavily loaded harbours may constitute a source for the aquatic environment for a considerable period of time.

Measurements in air or rainwater for the Netherlands are hardly available or not at all. Therefore, in the estimations of the current air concentrations data from abroad have been used, supplemented with model calculations. Using these an average air concentration of 0.025 pg I-TEQ.m⁻³ has been calculated, and an average deposition of 8 ng I-TEQ.m⁻².year⁻¹. As the first results from a measuring programme concerning dioxins in ambient air started in 1991 suggest, higher concentrations (higher than 0.080 pg I-TEQ.m⁻³) are found in urban and industrial areas.

Food contributes for appr. 95% to the total dioxin exposure of people in the Netherlands. Based on a recent study into the occurrence of dioxins in food categories a median daily intake has been calculated of 35 to 70 pg I-TEQ for children up to 20 years of age and 70 pg I-TEQ for adults. Based on the individual body weight this corresponded with a median intake of 1 pg I-TEQ per kg body weight per day for adults older than 20 years of age. Individuals younger than 20 years of age showed a higher intake, inversely proportional with age, as a result of lower body weight. The highest contribution (appr. 45%) to the intake via food originates from milk and milk products, such as butter and cheese. Fats (mainly fish oils) in products from an industrial origin contribute for appr. 22%, whereas meat products
(including beef and pork) are mainly responsible for the remaining 33%. The exposure of breast fed infants is, based on an average dioxin level of 34 pg I-TEQ.g\(^{-1}\) human milk fat, estimated at appr. 150 pg I-TEQ per kg body weight per day.

Data on the occurrence in the Dutch groundwater, as well as in surface water (with the exception of sediments), ambient air and plants/crops are extremely scarce and can be estimated for only several items with great uncertainty.
5. **EFFECTS**

5.1 **HUMAN TOXICITY**

This evaluation is mainly based on the Banbury-meeting held recently (Gallo et al., 1991), where international consensus was reached on the mechanism of action of 2,3,7,8-TCDD and on the WHO/EURO meeting (Bilthoven, 1991), where a tolerable daily intake for 2,3,7,8-TCDD was determined. In addition, in describing the toxic effects of PCDDs and PCDFs the literature used includes the following review reports and articles: ATSDR & EPA (1989), EPA (1985, 1988a and 1988b), IARC (1982, 1987a, 1987b), IPCS (1989) and Van der Heijden et al. (1982). No claim was made to be exhaustive, in view of the considerable amount of available and evaluated data from 2,3,7,8-TCDD in particular (a compound often used as a model compound) this was not considered advisable.

Only for the most toxic congener, 2,3,7,8-TCDD, has a recommended value been determined. The limited data concerning the other congeners have been incorporated in the so-called toxicity equivalency factors (TEFs). These are comparison factors used for expressing the toxicity of the congeners concerned in relation to that of 2,3,7,8-TCDD. A TEF of for instance 0.01 means that the compound is 100 times less toxic than 2,3,7,8-TCDD. TEFs have been developed for the purpose of estimating the toxicity of PCDD- and PCDF-mixtures.

5.1.1 **Kinetics and metabolism**

Experimental animal studies show that following oral exposure PCDDs and PCDFs are absorbed from the gastrointestinal tract. The availability of the compounds is highly dependent on the matrix. The absorption of 2,3,7,8-TCDD in an oily substance amounts to 30% to 100%; when bound to non-biological material (such as soil particles) the degree of absorption is considerably lower. In experimental animals a slight uptake of 2,3,7,8-TCDD through the skin can be found. Concerning inhalation no data are available. Following absorption PCDDs and PCDFs accumulate in tissues with a high fat content. In most animal species the liver is the principal organ depot. PCDDs and PCDFs pass the placenta and can be excreted through milk.

Biotransformation of 2,3,7,8-TCDD takes place at a slow rate, the more polar compounds
are eliminated through urine or bile. However, part of the dosis taken up is deposited in an unmetabolised state or excreted through faeces or milk. The elimination half-lives of 2,3,7,8-TCDD in experimental animals vary from 11 days in hamsters to >1 year in monkeys. Kinetic data from the other PCDDs and PCDFs are very scarce.

Quantitative data on the absorption of 2,3,7,8-TCDD in humans following oral exposure are lacking. In humans exposed to background levels (approximately 1 pg TEQ kg\(^{-1}\) body weight) the concentration in adipose tissue is 5 to 10 times higher than in the liver. For a group of Vietnam veterans exposed to dioxins the half-life for 2,3,7,8-TCDD is estimated at 7.1 years (with a 90% confidence interval of 5.8-9.6 years) (Michalek et al., 1992).

The general population is mainly exposed through food. Frequent analyses carried out on tissues of mammals and humans show that a selective retention of 2,3,7,8-substituted PCDDs and PCDFs occurs. The animal fat in our food therefore mainly contains these laterally substituted compounds. Vegetable food also contains other congeners. Some of the laterally substituted compounds, 2,3,7,8-PeCDF in particular, have a long half-life in experimental animals (108 days). Studies on Yusho patients (exposed to PCDF contaminated PCBs) also show the elimination of this compound to be slow (Van Zorge et al., 1989).

**Mechanism of action**

With regard to the mechanism of action of 2,3,7,8-TCDD various models have been postulated: for example, the effects of 2,3,7,8-TCDD are said to be linked with vitamin A deficiency, an increase of lipid peroxidase or with an imbalance in the endocrine regulation. These models, however, can only partly explain the effects of 2,3,7,8-TCDD.

At the Banbury-meeting in 1990 it was concluded that before 2,3,7,8-TCDD could cause any effects it should bind to an intracellular receptor first, the so-called Ah-receptor (Aromatic hydrocarbon receptor). Following binding to the "dioxin-receptor", through mechanisms as yet unknown, the induction to P450 IA1 occurs as the first observed effect. The other effects caused by TCDD are considered to be indirectly related to this induction.
5.1.2 Toxicity

Effects in experimental animals

A wide spectrum of effects is reported in experimental animals following oral exposure to 2,3,7,8-TCDD, depending on for instance animal species, strain and age. The main effects are loss of weight (the so-called wasting syndrome), thymus atrophy, liver damage and effects on the skin. Additionally, effects have been reported on bone marrow and vitamin A balance. There are marked differences in the susceptibility of various animal species for 2,3,7,8-TCDD; oral LD50-values vary from 0.6 µg.kg⁻¹ body weight for guinea pigs to 5051 µg.kg⁻¹ body weight for hamsters. Characteristic for the acute toxicity of 2,3,7,8-TCDD in all species is the time interval between exposure and the occurrence of the first effects (2 weeks up to 2 months). The great difference in susceptibility for 2,3,7,8-TCDD can only partly be explained by differences in toxicokinetics and by differences in cellular contents of certain receptors to which 2,3,7,8-TCDD is supposed to bind (see section 5.1.1, mechanism of action).

Long-term oral exposure to 2,3,7,8-TCDD in experimental animals causes mainly liver- and immune toxicity, effects on the reproduction and teratogenicity, as well as carcinogenicity (see section 5.1.4).

Effects on the liver

In (sub)chronic studies on rodents following exposure to 2,3,7,8-TCDD the liver appears to be one of the target organs. Morphological changes (including hyperplasia and hypertrophy) and associated impaired liver function and porphyry occurred in all species studied to a varying degree. In addition at lower dosages an increase in the activity of microsomal liver enzymes occurred (P450 IA1 in particular); enzyme induction is the most sensitive effect of 2,3,7,8-TCDD. For rats a no (adverse) effect dose of 0.001 µg.kg⁻¹ body weight per day was reported (Kociba et al., 1978).

Effects on reproduction and teratogenicity:

2,3,7,8-TCDD appeared to be teratogenic in mice (e.g. cleft palate) at dosages of 0.1 µg.kg⁻¹ body weight. In rats the effects are mainly embryotoxic (a decrease in birth weight and an increased mortality). The teratogenic and embryotoxic effects occurred at dosages which were
toxic to the female parent. A three-generation reproduction study with rats by Murray et al. (1979) shows a no (adverse) effect dose of 0.001 \( \mu \text{g.kg}^{-1} \) body weight per day. In limited studies with monkeys exposure to levels from 0.0015 \( \mu \text{g} \) 2,3,7,8-TCDD.\( \text{kg}^{-1} \) body weight per day resulted in an increasing number of abortions.

**Immunotoxicity:**
The effects of 2,3,7,8-TCDD on the immune system vary among various animal species and strains and are influenced by age (function thymus decreases with age). 2,3,7,8-TCDD causes a considerable atrophy of the thymus and the spleen and, to a lesser degree, of the peripheral lymph nodes. Other immunotoxic effects (such as changes in the T-cell function) are particularly thymus related. From (limited) studies with monkeys it appears that in this animal species the immune system is not the principal target-organ; adverse effects on the skin and reproduction occur earlier.

**In vitro toxicity parameters:**
PCDDs and PCDFs have been tested extensively in various in vitro cell systems. In vitro studies are a fast, inexpensive and simple way of carrying out comparative research with PCDDs and PCDFs. The results of in vitro studies can be used for the determination of TEF (see 5.1.5) and can contribute to a further understanding of the mechanism of action.

The parameters studied most frequently are the induction of AHH (aryl hydrocarbon hydroxylase) and EROD (7-ethoxyresurofin-o-diethylase) activity in hepatocytes and the induction of cell hyperkeratosis (excessive keratinization of the skin) in keratinocytes (epidermis cells).

**Effects in humans**

A great number of studies has been carried out on the effects of PCDDs on humans. These studies were partly carried out among occupationally exposed groups (such as persons engaged in the production of trichlorophenol) and partly among population groups exposed as a result of for instance industrial accidents (for instance the population of Seveso in North Italy, 1976) or the application of herbicides. An instance of the group mentioned last is the application of the defoliant "Agent Orange" (a mixture of 2,4-dichloro- and 2,4,5-trichlorophenoxy-acetic acid; containing up to almost 50 mg.\( \text{kg}^{-1} \) 2,3,7,8-TCDD) in Vietnam in the
period from 1960 to about 1969.

Chloracne is the only effect in all of these studies that is consistently related to 2,3,7,8-TCDD exposure. This skin disorder is not only a local effect, but is considered to be a systemic effect. It is characterized by the occurrence of cysts and comedones particularly in the face, followed by inflammatory reactions, melanosis and hyperkeratosis. In rather severe cases of chloracne these symptoms may persist for years and healing of these skin lesions usually results in deeply pitted scars.

Apart from chloracne various other effects have been reported among persons acutely exposed to mixtures of compounds contaminated with 2,3,7,8-TCDD. Several effects are gastro-intestinal disorders, effects on the liver (such as elevated serum enzyme levels and porphyria cutanea tarda), neurological (e.g. headache) and psychiatric effects (such as sleep disorder, depression). However, in further studies a relation between exposure to 2,3,7,8-TCDD and the occurrence of the above-mentioned effects was not confirmed (ATSDR & EPA, 1989; IPCS, 1989).

With respect to the correlation between exposure to 2,3,7,8-TCDD and reproduction toxicity in humans, positive (elevated number of spontaneous abortions and birth defects) as well as negative studies are available. The positive studies always involved mixed and not sufficiently quantified exposure. The negative data from Seveso are not completely reliable, due to induced abortion during the accident. However, for the time being there is insufficient evidence to consider 2,3,7,8-TCDD embryotoxic or teratogenic in humans (EPA, 1985; IPCS, 1989; Skene et al., 1989).

Of the various human data those from Seveso are the most significant, although it involves data following short-term, mainly dermal (and/or oral, through dust), exposure. Due to the extreme toxicity of the compound an extensive medical research programme was started following the explosion in the ICMESA factory. Between 1976 and 1985 inhabitants of more or less contaminated areas were medically examined. The most striking fact was the increased incidence of chloracne in exposed subjects, occurring 15 to 20 days following the exposure. Afterwards two other effects could neither be confirmed (decrease in motor nerve conduction velocity) nor be assessed due to the lack of individual data (an increase in glucaric acid levels in the urine indicating an elevated microsomal enzyme activity)(IPCS, 1989). The degree of exposure of the Seveso population is not exactly known. However, serum samples taken at the time from exposed as well as from non-exposed subjects have been stored. The 2,3,7,8-TCDD levels in a number of these samples have recently been
measured and compared with the medical history of the patients. It involved 10 subjects from the so-called A-zone (in the nearest vicinity of the plant) with severe chloracne, 10 subjects from the A-zone without chloracne and 10 subjects from a non-contaminated zone as a control group. In all exposed subjects elevated 2,3,7,8-TCDD levels were measured in the serum. The serum levels in the subjects from the A-zone with severe chloracne varied between 828 to 56,000 ng.kg$^{-1}$. With the exception of one case this group consisted of children. Serum levels of subjects from the A-zone without chloracne varied from 1770 to 10,400 ng.kg$^{-1}$. These subjects, with the exception of one, were all adults. The data suggest that children develop chloracne at lower serum levels than adults do. The authors concluded that chloracne is a marker for 2,3,7,8-TCDD exposure, however, they found it not to be dose related. Impaired liver function occurred in children as well as in adults. A dose response relation did not exist. In addition, impaired liver function was also observed in one of the subjects of the control group (Mocarelli et al., 1991).

**PCDFs**

With respect to the effects of PCDFs in humans two large-scale food poisoning incidents are known from Japan (1968) and Taiwan (1979). These poisonings were called "Yusho" and "Yu-cheng" (oil-disease), respectively, and were caused by the use, over several months, of consumption oil contaminated with PCDFs, chlorinated biphenyls (PCBs) and chlorinated quarter phenyls (PCQs). The number of cases amounted to a total of approximately 4,000. The clinical symptoms partly resembled those of PCDD poisoning; chloracne, abnormal skin pigmentation and hypersecretion of sebum by the sebaceous glands. The average daily exposure is estimated at 0.1 to 0.2 $\mu$g.kg$^{-1}$ body weight of 2,3,7,8-substitued PCDFs. Due to the simultaneous exposure to PCBs and PCQs the role of PCDFs is difficult to assess, but these compounds may possibly have been mainly responsible for the occurring effects.

5.1.3 Genotoxicity

The majority of the *in vitro* and *in vivo* genotoxicity studies with 2,3,7,8-TCDD are consistently assessed as negative in the literature. In a small number of studies an equivocal or weakly positive response is reported. This concerns several gen-mutation tests in procaryotes and eucaryotes, a host-mediated assay with yeast cells and a test on chromosome aberrations in bone marrow cells from rats *in vivo*. Part of these positive results are found
at doses which are distinctly cytotoxic and at which the water solubility of 2,3,7,8-TCDD (maximally 0.2 µg.l⁻¹) will prove to be problematic. In addition part of the results appeared to be non-reproducible. There are no indications for a direct interaction with DNA. Studies on chromosome aberrations in exposed humans are contradictory. A study among workers exposed to for instance 2,4,5-trichlorophenoxyethanol (contaminated with 2,3,7,8-TCDD) showed an increase in the frequency of chromatid-type and unstable chromosome aberrations in periferal lymphocytes. The exposure levels of the individual workers were not given, hampering an interpretation (Czeizel and Kiraly, 1976, evaluated by EPA, 1985). In persons exposed to "Agent Orange" and in individuals involved in the Seveso accident no chromosome aberrations were observed. The fact that simultaneous exposure occurred to other (possibly active) compounds constitutes a problem in all these studies, in addition to the inability of determining the degree of 2,3,7,8-TCDD exposure quantitatively. Based on these results it is concluded that insufficient evidence exists to consider 2,3,7,8-TCDD genotoxic.

5.1.4 Carcinogenicity

Long-term oral administration of 2,3,7,8-TCDD to mice and rats resulted in liver and thyroid tumours. Furthermore carcinomas of nasal and oral cavity, and lungs were reported. Of the carcinogenicity studies the one by Kociba et al. (1978) is generally considered a key-study (lowest no-effect level and relevant exposure route). In this two year study groups of rats were fed 0.001, 0.01 and 0.1 µg 2,3,7,8-TCDD per kg body weight per day through food. In the highest dose group a statistically significant increase was found in liver cell carcinomas and squamous cell carcinomas of nasal and oral cavity, and lungs as well as various toxic effects (increased mortality, growth retardation, clinico-chemical and pathological changes in liver, lungs, bloodvessels and lymphoid organs). In the 0.01-group a significant increase in hepatocellular nodules occurred. The no-effect level was 0.001 µg.kg⁻¹ body weight. The other studies support the findings of Kociba et al. Therefore sufficient evidence exists for the carcinogenicity of 2,3,7,8-TCDD in experimental animals (IARC, 1987b).

Apart from 2,3,7,8-TCDD only a (1:2) mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-HxCDD has been tested on carcinogenicity. In two-year gavage studies, groups of mice (m) and rats (m/f) were given doses of 0, 1.25, 2.5 or 5 µg.kg⁻¹ body weight per week and female mice doses
of 0, 2.5, 5 or 10 μg.kg⁻¹ body weight per week. Liver tumours and neoplastic nodules were found in both the treated and the control groups. A significant increase in liver carcinomas and/or adenomas was found in the highest dose group of mice (m/f) and of female rats and in the mid and high dose group in female rats. Liver toxicity occurred in all treated animals (NTP, 1980a). Long-term dermal application of the same mixture did not result in increased tumour incidence in mice and rats (NTP, 1980b). Based on the above-mentioned data there is sufficient evidence for carcinogenicity of the 1,2,3,6,7,8- and 1,2,3,7,8,9-HxCDD mixture in experimental animals.

**Epidemiological studies**

In a number of epidemiological studies an increased incidence has been reported of soft-tissue sarcomas and Hodgkin’s lymphomas and non-Hodgkin’s lymphomas in groups exposed to 2,3,7,8-TCDD contaminated pesticides such as phenoxy acetic acids and chlorophenols. It should be noted that increased incidence of soft-tissue sarcomas, nose tumours and non-Hodgkin’s lymphomas is also found in workers exposed to chlorophenoxy herbicides without TCDD. In a number of more recent studies on occupationally exposed persons and groups exposed as a result of accidents no or no distinct correlation was found. In some of the cohorts studied (such as the population of Seveso) the latency period is (still) too short to find a (possibly) increased tumour incidence. In a study with a 34-year follow-up period on 247 workers of BASF, exposed in 1953 as a result of an accident, no conclusive evidence was found of any increased tumour incidence either (Zober et al., 1990). Fingerhut et al. carried out a retrospective cohort study among 5,172 persons occupationally exposed to 2,3,7,8-TCDD contaminated pesticides. In this study no increased risk to any of the above-mentioned tumours was found for the entire cohort. However, the mortality as a result of all types of cancer (combined) was slightly increased. Furthermore in one subcohort (with >1 year exposure and >20 year latency period) a significant increase in soft-tissue sarcomas was found (SMR=922) as well as a slightly significant increase in cancer of the respiratory stract (SMR=142). The significance of the increased mortality as a result of soft-tissue sarcomas is limited due to the small number of cases and through the fact that this type of cancer is difficult to classify. With respect to the slightly increased mortality (as a result of all cancers and cancers of the respiratory stract) the authors noted that this could not be completely explained by smoking being the disturbing variable. On the other hand there was no dose-
response relationship (Fingerhut et al., 1991). The lack of recent exposure data and the occurrence of mixed exposure generally renders difficult the assessment of the results. Based on these results it is for the time being concluded that insufficient evidence exists for the carcinogenicity of 2,3,7,8-TCDD for humans.

A number of epidemiological studies is still in progress and these studies will in future yield additional data. Furthermore an international registration of persons occupationally exposed to herbicides (contaminated with 2,3,7,8-TCDD) has been started by institutions such as IARC in order to study the effects of this exposure. For the purpose of this registration various cohorts of exposed persons have been combined (a total of approximately 19,000 persons) (Huff et al., 1991).

Initiation/promotion studies

From initiation/promotion studies 2,3,7,8-TCDD appears to possess strong promotor properties in the liver of rats, whereas initiating activity was not observed. In the skin of mice 2,3,7,8-TCDD showed no promotor- or initiator activity.

5.1.5 Toxicity equivalency factors (TEFs)

Since 2,3,7,8-substituted PCDDs and PCDFs are considerably more toxic than the non-laterally substituted congeners, the toxicity of PCDD and PCDF mixtures are only assessed for the presence of 2,3,7,8-substituted congeners. With the exception of 2,3,7,8-TCDD toxicological data from these PCDDs and PCDFs are very limited. Therefore, in 1977 Grant attached weighing factors (TEF) to the various compounds based on the toxicological data available at the time. TEF would form the resultant of an overall comparison of the toxicity of the various compounds in relation to the most toxic congener, 2,3,7,8-TCDD.

By multiplying the concentration of the individual compounds by the corresponding weighing factors and by adding up the results, the toxicity of the mixture is calculated as a comparable amount of 2,3,7,8-TCDD. Although the concept originally formulated by Grant has been adapted over the years, its starting points were increasingly supported. The first starting point being that all 2,3,7,8-substituted congeners have a similar mechanism of action, and the second that mixtures of 2,3,7,8-substituted congeners show an additive toxicity.

Subsequently many institutions have developed their own TEF-system. The weighing factors
are a resultant of the assessment of in vivo and in vitro experimental animal studies. Due to the differences in the assessment of these studies the various TEF-systems show differences. Instances have been indicated in table 1.4. This has led to a confusing situation where all calculations are not comparable any longer due to deviating TEFs. In this document the proposal made in the framework of an agreement between the Netherlands and the NATO-CCMS (NATO-CCMS, 1988a, 1988b; Van Zorge et al., 1989), has been adopted. In annex 1 an overview is given of the comparative activities of 2,3,7,8-congeners in relation to the "standard" 2,3,7,8-TCDD. Based on these data the I-TEFs indicated in table 5.1 (see also table 1.4) have been calculated. The parameters incorporated include acute toxicity (LD50-values), (sub)chronic toxicity, carcinogenic and teratogenic potential and the activity in in vitro test systems. In determining the TEFs the working group assumed that 1) chronic data outweigh subchronic ones and these again outweigh acute data, 2) mammalian data are more important than those from other animals, 3) results from in vivo studies outweigh those from in vitro studies.

For further consideration in relation to the determination of various factors reference is made to the publication by Van Zorge et al. (1989).

The TEF concept should be seen as a pragmatic approach. It facilitates the estimation of the toxicity of PCDD and PCDF mixtures, despite the limited amount of available toxicological information on the 2,3,7,8-substituted PCDDs and PCDFs. The proposed TEF factors may in future be further adjusted when more toxicological data become available. In addition the current concept, based on additivity, may in future be less useful. If synergism and antagonism appear to be significant in the exposure to mixtures of PCDDs and PCDFs, a considerably more complicated TEF model is required (Van Zorge et al., 1989).

**Table 5.1** An overview of TEF factors (International TEFs or I-TEFs), as drawn up in the framework of agreements between the Netherlands and the NATO-CCMS

<table>
<thead>
<tr>
<th>2,3,7,8-TCDD</th>
<th>1</th>
<th>2,3,7,8-TCDF</th>
<th>0.1</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,2,3,7,8-PeCDD</td>
<td>0.5</td>
<td>1,2,3,7,8-PeCDF</td>
<td>0.05</td>
</tr>
<tr>
<td>1,2,3,4,7,8-HxCDD</td>
<td>0.1</td>
<td>1,2,3,4,7,8-HxCDF</td>
<td>0.1</td>
</tr>
<tr>
<td>1,2,3,6,7,8-HxCDD</td>
<td>0.1</td>
<td>1,2,3,6,7,8-HxCDF</td>
<td>0.1</td>
</tr>
<tr>
<td>1,2,3,7,8,9-HxCDD</td>
<td>0.1</td>
<td>1,2,3,7,8,9-HxCDF</td>
<td>0.1</td>
</tr>
<tr>
<td>1,2,3,4,6,7,8-HpCDD</td>
<td>0.01</td>
<td>1,2,3,4,6,7,8-HpCDF</td>
<td>0.01</td>
</tr>
<tr>
<td>1,2,3,4,6,7,8,9-OCDD</td>
<td>0.001</td>
<td>1,2,3,4,6,7,8,9-OCDF</td>
<td>0.001</td>
</tr>
<tr>
<td>Other PCDDs</td>
<td>0</td>
<td>Other PCDFs</td>
<td>0</td>
</tr>
</tbody>
</table>
The following sections 5.2 through 5.4 are summaries of a background report written for the purpose of this basic document on the possible ecotoxicological effects of dioxins and on the effects of these compounds on farm animals (Oekerman et al., 1992). Only a limited number of references is given in these sections, which facilitates recovery of important data from the background report and from the annexes 2a-c containing basic data concerning the aquatic ecotoxicity. Other references may be found in the background report.

5.2 ECOTOXICITY - AQUATIC ORGANISMS

5.2.1 Accumulation

The (potential) degree of accumulation of a compound is usually given as a bioconcentration factor (BCF). The BCF may be calculated from the ratio between the concentration in the organism and the concentration in the medium in which the organism is exposed: 

\[ \text{BCF} = \frac{C_{\text{organism}}}{C_{\text{medium}}} \]

However, this method results in an underestimation of the BCF when an equilibrium is not yet reached. The BCF may also be calculated from the ratio between the uptake rate constant \( (k_1) \) and the elimination rate constant \( (k_2) \): 

\[ \text{BCF} = \frac{k_1}{k_2} \]

In the latter case it automatically concerns an "equilibrium" BCF. For aquatic organisms the concentration in the organism is usually related to the concentration in water and sometimes, for benthic organisms (organisms living on or in sediment), to the concentration in sediment. The BCF values mentioned in the following are, unless stated otherwise, related to the concentration in water. All BCF values indicated in the following (including the values indicated as "BCFs," that are related to the concentration in the sediment) are, as far as known, based on the fresh weight of the organisms, whereby the whole organism has been analyzed (whole body BCF).

The data on the accumulation of dioxins (PCDDs and PCDFs) in aquatic organisms are mainly based on experiments with freshwater fish. For marine organisms only one bioaccumulation study has been carried out, in a water-sediment system (see corresponding section).

Uptake routes

Several experiments with fish showed that various media (water, sediment, food) may play a role in the uptake of dioxins. Considering the limited data, the differences in congeners tested and/or the differences in study protocol no unequivocal conclusions can be drawn.
concerning specific congeners. Thus no significant accumulation of OCDD was found at exposure through water in one out of two tests with guppies (*Poecilia reticulata*), whereas this was the case in the other test. Considering the high degree of adsorption of dioxins to organic material and sediment particles the "direct" uptake from the water phase will be hampered by the presence of suspended sludge; the "indirect" (oral) uptake, however, partly through food, will increase.

**Exposure in water (BCF-values)**

Accumulation studies with fish have resulted in equilibrium BCF-values (calculated from $k_1$ and $k_2$) of approximately 100 to 86,000 for various dioxins; these data are mainly based on studies with PCDDs. The highest equilibrium BCF-values by far, 7,900 to 86,000, were found for 2,3,7,8-TCDD. Based on the Mehrle et al. study (1988), for this compound a value of appr. 40,000 is considered to be the most reliable, due to the test conditions used. From two studies with fish, where the "2,3,7,8-TCDD" content was determined with two different analyzing methods (scintillation counting of $^{14}$C or $^3$H following oxidative destruction of the fish and GC-MS analysis of 2,3,7,8-TCDD following extraction of 2,3,7,8-TCDD) it appeared that the greatest part by far of the "2,3,7,8-TCDD" content measured in fish consisted of non-metabolized 2,3,7,8-TCDD. For the remaining dioxins studied the equilibrium BCF-values are usually between 500 and 2,500 to 5,000. These values are usually considerably lower than the equilibrium BCF-values calculated on the basis of (log) Kow, even for 2,3,7,8-TCDD. For instance, equilibrium PCB-values of 3,000-68,000 (5 out of 6 values: approximately 30,000 or higher) have been calculated for 2,3,7,8-TCDD in EPA (1984) based on a measured log Kow of 6.15 and of 7,000-330,000 (5 out of 6 values: approximately 100,000 or higher) based on an estimated log Kow of 6.8. These calculations are based on the relations between (log)BCF and (log)Kow reported by various authors, for instance $\text{BCF} = 0.048 \times \text{Kow}$. In accumulation studies with fish BCF-values for 2,3,7,8-TCDD and 2,3,7,8-TCDF have also been calculated from $C_{\text{organism}}$ and $C_{\text{water}}$; these values are 5,800-28,700 for 2,3,7,8-TCDD and 2,450 - 6,050 for 2,3,7,8-TCDF. A comparison between experimentally determined BCF-values for dioxins and those for PCBs (polychlorobiphenyls) with comparable (log) Kow values shows that those for dioxins, except for 2,3,7,8-TCDD, are considerably lower than those for PCBs. The above-mentioned data indicate that the calculation methods for BCF-values used for lipophilic compounds (based
on the relation between BCF and Kow) overestimate the dioxin accumulation in aquatic organisms.

In a multiple species experiment BCF-values were calculated for 2,3,7,8-TCDD (from $C_{\text{organism}}$ and $C_{\text{water}}$) of 1,800-7,100 in water fleas and of 740-3,700 in snails. At exposure of fish through water elimination half-lives were found for dioxins varying from 1 up to 58 days. The elimination process of 2,3,7,8-TCDD, 1,2,3,4,7,8-HxCDD and 1,2,3,4,6,7,8-HpCDD was slowest (half-lives usually 15 to 20 days). For the remaining dioxins studied (including lower chlorinated compounds as well as OCDD and OCDF) half-lives between 0.5 and 9 days were found.

It is noted that the BCF-values mentioned as well as the half-lives are based on various tests with varying conditions such as exposure time and exposure concentrations, which may have led to differences that are not realistic.

**Exposure in water-sediment systems**

In an eight-week experiment where carps (Cyprinus carpio) were exposed to dioxins through contaminated sediment, accumulation of 2,3,7,8-substituted congeners, in particular, was observed. The other congeners were found in the sediment, however, not in the fish (detection limit: 1 ng.kg$^{-1}$ wet weight). BCF$_s$-values varied from 0.001 to 0.1 for congeners with at least 6 chlorine atoms (present in the sediment in the highest concentrations) and from 0.1 to 1.9 for congeners with 4 or 5 chlorine atoms (present in the sediment in the lowest concentrations). In another experiment it was observed that the accumulation of 2,3,7,8-TCDD in freshwater fish decreased with increasing organic carbon content of the sediment. A bioaccumulation study with marine worms, mussels and shrimps resulted in BCF$_s$-values $<1$ for 2,3,7,8-TCDD (content in sediment: 655 ng.kg$^{-1}$) and 2,3,7,8-TCDF (content in sediment 335 ng.kg$^{-1}$). In the mussels the equilibrium was reached after 7 weeks, whereas in worms this was not yet the case after 15 weeks. The elimination of these compounds was slow in the worms, while in the mussels a very rapid elimination was found.

**Exposure through feed**

In experiments where fish were exposed to dioxins through feed (single or repeated exposure)
an uptake-efficiency\(^1\) of 2\%-53\% was calculated for the congeners studied. For comparison, in the case of PCBs an uptake-efficiency of approximately 70\% was reported. For 2,3,7,8-TCDD, based on a comparison between the contents in fish and those in feed, an uptake-efficiency of 42\% and 51\% was estimated. The elimination half-lives determined in these experiments varied from 2 to 105 days. The half-lives determined in the feed experiments with fish are at least a factor 2 higher than the corresponding half-lives determined in studies where fish were exposed through water. For instance, the half-life of 105 days for 2,3,7,8-TCDD determined in a feed experiment with the rainbow trout *Salmo gairdneri* is a factor 2-7 higher than the half-lives of 15-58 days determined in the studies where *S. gairdneri* was exposed to 2,3,7,8-TCDD through water. In particular for 2,3,7,8-TCDD, 2,3,4,7,8-PeCDF and (to a lesser degree) 1,2,3,4,7,8,-HxCDD, a relatively high uptake-efficiency was found combined with a relatively long half-life. For OCDD and OCDF a relatively low uptake-efficiency was found combined with a relatively short half-life.

**Distribution and metabolism in organisms**

In fish the highest dioxin contents were especially found in liver, bile and adipose tissue. The contents in muscular tissue are considerably lower, however, due to its great mass this tissue contains the greatest absolute amounts. These data are mainly based on data concerning 2,3,7,8-TCDD.

In studies with fish metabolism of certain specific dioxins has been observed. Injection of 2,3,7,8-TCDD in fish resulted in the formation of several metabolites, including a glucoronideconjugate. Injection of 1,2,3,7-TCDD and 1,2,3,4,7-PeCDD resulted in the formation of non-extractable residues, whereas 1,2,3,4,7,8-HxCDD and 1,2,3,4,6,7,8-HpCDD are scarcely transformed. In a study where fish were exposed to a fly ash extract in water in the absence of piperonylbutoxide (PBO, an inhibitor of the mono-oxygenase activity) accumulation of only laterally substituted dioxins was observed, whereas in the presence of PBO accumulation of non-laterally substituted dioxins was also found. The latter study indicates metabolism of the latter dioxins.

\(^{1}\) Uptake-efficiency: \( C_{\text{fish}} \times k_2 \times (1 - e^{k_2 t}) / f \times C_{\text{food}} \)

\( f\): food uptake rate; \( k_2\): elimination rate constant
Conclusions accumulation

Most data concern the accumulation of PCDDs in freshwater fish. Dioxin uptake may occur in various mediums (water, sediment, food). Accumulation studies with fish have resulted in equilibrium BCF-values (calculated from the uptake and elimination rate constant) of approximately 100 to 86,000 for various dioxins. The highest equilibrium BCF-values by far were those determined for 2,3,7,8-TCDD; for this compound a value of 40,000 was considered to be the most reliable. For the other compounds studied the equilibrium BCF-values usually vary from 500 to 2,500-5,000. For 2,3,7,8-TCDD, in accumulation studies, several BCF-values have also been calculated from $C_{\text{organism}}$ and $C_{\text{water}}$, resulting in the following values: 740-3,700 in snails, 1,800-7,100 in water fleas and 5,800-28,700 in fish; for 2,3,7,8-TCDF BCF-values of 2,450-6,050 in fish were calculated in a similar way. From the above-mentioned BCF-values it appears that dioxins can be accumulated (concentrated) from the water phase to a considerable or high degree. However, the experimentally determined equilibrium BCF-values are usually much lower than the values calculated on the basis of (log)Kow and also lower (with the exception of the values for 2,3,7,8-TCDD) than those for PCBs with a corresponding (log)Kow. For various dioxins, in sediment-water systems with contaminated sediment, BCF$_s$-values have also been calculated ($C_{\text{organism}}$ divided by $C_{\text{sediment}}$); these values are usually <1 (invertebrates; fish).

Irrespective of the exposure route the highest accumulation was found for certain specific congeners with 4 to 6 chlorine atoms. The degree in which congeners from this group are accumulated depends partly on the position of the chlorine atoms. In fish exposed through feed longer half-lives have been observed than in fish exposed through water, indicating that the latter exposure route may contribute considerably to the accumulation. In feeding experiments, for 2,3,7,8-TCDD and 2,3,4,7,8-PeCDD in particular, a relatively high uptake-efficiency was found combined with a relatively long half-life.

5.2.2 Toxicity

Toxicity related to the external concentration

I. Exposure in water

All data mentioned in this section are based on single-species tests with freshwater organisms. These tests are summarized in the Annexes 2a-c of this chapter. Most tests were
performed with early-life stages of fish (so-called ELS-tests: early-life stage test or embryo-larval test). Since the exposure times in most tests with fish were relatively short (4-28 days) in relation to the generation time of these organisms, no distinction has been made in the annexes between short-term exposure (acute toxicity) and long-term exposure (chronic exposure). It should be noted that the exposure period in most studies was followed by a purification period in clean water, so that delayed effects (characteristic for dioxins) were also incorporated in the assessment of the toxicity. The NOEC-, NOLC- and LC50-values determined may therefore reflect the chronic toxicity reasonably well. It should be noted, however, that in these tests the maximal concentration was not yet reached in the fish (no equilibrium yet).

2,3,7,8-TCDD (annex 2a)
A number of the tests with fish, following the actual exposure period, were continued for a specific time in order to study the lag effects. From these tests, for instance the very extensive tests by Helder (1980, 1981) and Van Mehrle et al. (1988), the delayed occurrence of the mortality is evident, which means that mortality often only occurs after the actual exposure has been finalized for a considerable time (weeks). This lag effect is especially found during exposure to relatively low concentrations. Exposure of young fish larvae (yolk sac fry) to 2,3,7,8-TCDD results in a number of specific pathological abnormalities, of which severe forms of haemorrhages and (pericardial) oedema in particular are to a high degree correlated to mortality. At exposure from the embryo stage, even before or during hatching of the eggs, these effects may lead to mortality, however, at exposure to relatively low concentrations the highest mortality is found in yolk sac fry, probably due to the fact that during the absorption of the yolk sac a relatively great amount of 2,3,7,8-TCDD (high Kow) is taken up. In addition to the above-mentioned effects morphological (structural) abnormalities such pugheadedness are found, and usually temporary growth retardation. When the exposure has already started in the embryo stage, the latter effect will very probably be connected with an effect on the embryonic development, resulting in smaller larvae.

Tests with fish embryos and/or young fish have resulted in 14-21 day LC50-values of 1.7 to 13 ng.l⁻¹. The lowest LC50, 0.046 ng.l⁻¹, based on measured concentrations, was found in a continuous flow study with rainbow trout Salmo gairdneri (Mehrle et al., 1988), in a 56-day study (28 days exposure, followed by 28 days in clean water). Most NOEC- and NOLC-
values for fish are approximately 1 ng.1⁻¹. However, in a number of tests, at concentrations varying from 0.038 ng.1⁻¹ (S. gairdneri, Mehrle et al., 1988) to 0.1 ng.1⁻¹ (various studies), effects were still found, including morphological and pathological abnormalities, mortality and growth retardation. The NOEC- and NOLC-values for invertebrates vary from (minimally) 10 to appr. 1000 ng.1⁻¹. Based on this, despite the limited number of data for the latter group, it is concluded that invertebrates are much less susceptible to 2,3,7,8-TCDD than are fish.

Other PCDDs (annex 2b) and PCDFs (annex 2c)

With respect to the other dioxins considerably less data are available, "all" originating from tests with fish. In the comparative study by Wisk and Cooper (1990a), where, in addition to the toxicity of 2,3,7,8-TCDD, also that of a number of other PCDDs and of 2,3,7,8-, TCDF was studied, with regard to the major effects (severe pathological abnormalities and mortality) NOEC-values were found varying from 2 ng.1⁻¹ for 2,3,7,8-TCDF to 10 - (at least) 50,000 for the PCDDs. The NOEC for 2,3,7,8-TCDF was identical to that for 2,3,7,8-TDCD. In this study the same effects were repeatedly found, irrespective of the congener studied, however, the concentrations at which the effects occurred differed. In the study by Mehrle et al. (1988) the toxicity found for 2,3,7,8-TCDF was at least ten times lower than that for 2,3,7,8-TCDD. In the Wisk and Cooper study (1990a) the exposure time may have been too short to indicate an actual difference between the toxicity of 2,3,7,8-TCDD and 2,3,7,8-TCDF.

Combination toxicity

In tests with young larvae of the rainbow trout S. gairdneri the combination toxicity of PCDDs, PCDFs and PCBs was studied. Following the determination of the toxicity equivalency factors (TEFs)² for the individual compounds (see also annexes 2a-c, Bol et al., 1989) tests were carried out with (in most cases binary) mixtures, where, in theory, the TEF-value of the mixtures amounted to 1. Tests with various mixtures of 2,3,7,8-substituted dioxins resulted in TEF-values of 0.96-1.08, indicating an additive action. Tests with binary mixtures of 2,3,7,8-TCDD with 1,3,6,8-TCDF, 3,4,3',4'-TCB or 2,4,5,2',4',5'-HxCB, and

² TEF: toxicity equivalency factor in relation to 2,3,7,8-TCDD, the most toxic congener (TEF=1).

The lower the TEF, the lower the toxicity.
with binary mixtures of 2,3,4,7,8-PeCDF and 3,4,3',4'-TCB resulted in TEF-values of 1.30-1.77, according to Bol et al. (1989) clearly indicating synergism. It is noted that the difference between the TEF-values for the various mixtures was small, smaller than a factor 2, thus an additive action seems the most likely.

Conclusions toxicity
The available data clearly show that 2,3,7,8-TCDD is the most toxic congener, followed by other 2,3,7,8-substituted congeners, particularly tetra- and penta-PCDDs and PCDFs. This is in agreement with the data on accumulation in aquatic organisms, and with the toxicity data for mammals (experimental animal studies). In a number of tests with fish, at 2,3,7,8-TCDD concentrations varying from 0.038 to 0.1 ng.l⁻¹, effects were still observed, including mortality, morphological and pathological abnormalities, and growth retardation. Exposure of fish to other dioxins resulted in similar effects, however, the concentrations at which the effects occurred differed. Tests with mixtures of 2,3,7,8-substituted dioxins indicate an additive action.

Toxicity related to the external concentration
II. Exposure in sediment-water systems

The toxicity of 2,3,7,8-TCDD was studied in recirculating aquatic model ecosystems with sediment (38% sand, 49% silt, 12% clay, 1.5% organic matter). Test organisms were algae (Oedogonium cardiacum), water fleas (Daphnia sp.), snails (Helisoma sp.) and, in a screened off compartment, fish (Gambusia affinis). A total of 3 treated systems and 3 controls was used. In all of the 3 treated systems a benzene solution of ¹⁴C-2,3,7,8-TCDD was added to the sediment, subsequently the sediment was dried; only one concentration was tested. The 2,3,7,8-TCDD level in the treated sediment slowly decreased from 118,000 ng.kg⁻¹ on day 30 to 97,000 on day 180; the concentrations in water varied from 2.4 to 4.2 ng.l⁻¹. Fish were put into the treated systems twice, both times all fish died within 15 days. No adverse effects were found in the other test organisms for 32 weeks (parameters: food uptake, growth and/or reproduction).

In another study the carp C. carpio was exposed for 12 weeks through sediment contaminated with various dioxins (levels varying from non-detectable for 2,3,7,8-TCDD to 4,600 ng.kg⁻¹ for 1,2,3,4,6,7,8-HpCDF). Following 6 weeks of exposure a maximal induction of the cyto-
chrome P-450 and EROD activity was found, clearly correlated to the PCDF- and PCDD-
level in the liver, whereby no distinction is made between the various congeners.

Toxicity related to the internal concentration (i.c.dose)

I. Oral exposure/exposure via feed

In a feeding experiment rainbow trout (*S. gairdneri*) were exposed to 500 ng 2,3,7,8-
TCDD.kg⁻¹ feed for 13 weeks and subsequently during a similar period were fed a TCDD-
free powder. The maximal level found was 12 ng per fish, corresponding to an internal dose
of 250 ng.kg⁻¹ body weight. During the entire period no effects on growth were found, and
no necrosis was observed.

The carp *C. carpio* was fed PCDFs dissolved in gel, oil or sediment for 7 days. The fish
were treated with 2,3,4,7,8-PCDF and 1,2,3,6,7,8-HxCDF (1800 and 1000 ng.kg⁻¹ body
weight, respectively) or 2,3,4,7,8-PCDF and 1,2,3,6,7,8-HxCDF (1900 and 2750 ng.kg⁻¹
body weight) (6 treated groups in total). After 2 weeks the oral administrations of PCDFs
resulted in increased enzyme activity and relative liver weight, especially with solutions with
gel or oil and to a lesser degree with contaminated sediment.

Toxicity related to the internal concentration (i.c.dose)

II. Exposure via injection

In a number of studies, particularly with fish, the toxicity of 2,3,7,8-TCDD was studied
following a single administration via injection. For various freshwater fish species LD50-
values of 3000 to 16,000 ng.kg⁻¹ body weight were found, calculated 12 weeks following
application. The internal dose at which mortality no longer occurred, was appr. 1000 ng.kg⁻¹
body weight. At a dose of appr. 500 ng.kg⁻¹ body weight, however, effects were observed
in the liver (also following an exposure of 12 weeks), that is histopathological abnormalities
and enzyme induction (EROD activity). In larvae and adults of the Mexican bullfrog exposed
to internal doses of up to 1,000,000 ng.kg⁻¹ (1 mg.kg⁻¹) body weight no effects of 2,3,7,8-
TCDD on the larval development (metamorphosis) and the survival were observed, neither
were morphological or histological abnormalities. In an experiment with the marine fish
species *Stenotomus chrysops* injection of 2,3,7,8-TCDF resulted in enzyme induction
(cytochrome P-450E and EROD), at an internal dose of 3100 ng.kg⁻¹ body weight.
5.3 ECOTOXICITY - TERRESTRIAL ORGANISMS

Data on terrestrial organisms are limited, both with respect to accumulation and toxicity. The available information contains results from a number of field studies which are often difficult to interpret, partly due to the vague exposure (great dispersion in reported levels in soil and/or food).

5.3.1 Accumulation

In the following the extent of accumulation is, where possible, indicated as the concentration factor (CF = \( C_{\text{organism}} \) divided by \( C_{\text{soil}} \)). The term concentration factor (CF) is used here to distinguish from the accumulation from water, for which in section 5.2 the term bioconcentration factor (BCF) has been used. Both terms have the same meaning, however, the medium to which the BCF and the CF are related is different.

Plants

Very limited data with respect to 2,3,7,8-TCDD indicate a slight uptake from the soil and a small degree of transport (translocation) from the root to the emergent parts. Thus in pot experiments with maize and beans no increase of the 2,3,7,8-TCDD level in the emergent parts was found at soil levels of 1 to 750 ng kg\(^{-1}\). In another experiment 0.15% of the amount of 2,3,7,8-TCDD added to the soil was found in the emergent parts of agricultural crops (soy bean, oats), however, when harvested no 2,3,7,8-TCDD was indicated either in the grain or in the beans. Several experiments with 2,3,7,8-TCDD also indicate adsorption on emergent parts, at evaporation from the soil or at deposition from the air. The significant contribution from deposition to the level found "in" the emergent parts, is illustrated by the fact that in aboveground crops higher levels are found than in underground crops, and by the occurrence of relatively high levels in large-leaved crops. A small degree of uptake and translocation of 2,3,7,8-TCDD and other dioxins is also expected based on the physicochemical properties of these compounds, such as the high (log)Kow.

At various locations in Great Britain background levels of dioxins were determined in the emergent parts of the vegetation. For TCDDs, PeCDDs, HxCDDs, HpCDDs and OCDD average levels were found of 7.7, 6.2, 6.2, 8.8 and 16.1 ng kg\(^{-1}\) dry weight, respectively.
The group TCDDs mainly consisted of 1,3,6,8- and 1,3,7,9-TCDD; the maximal 2,3,7,8-TCDD level in the vegetation was estimated to be <0.1 ng.kg\(^{-1}\) dry weight. For TCDFs, PeCDFs, HxCDFs, HpCDFs and OCDF the average levels amounted to 11, 6.6, 4.0, 2.7 and 1.4 ng.kg\(^{-1}\) dry weight. No correlation was found between the concentrations of the various congeners, neither a geographical trend.

**Soil organisms**

In a long-term laboratory study with earthworms *Allolobophora caliginosa*, at exposures of non-lethal 2,3,7,8-TCDD levels of 0.05\(\times\)10\(^6\), 0.5\(\times\)10\(^6\), 1.5\(\times\)10\(^6\) and 5\(\times\)10\(^6\) ng.kg\(^{-1}\) (0.05-5 mg.kg\(^{-1}\)) soil CF-values were reported of 4-9, 0.6-1.1, 0.4-0.9 and 0.15-0.44, respectively, depending on the exposure time. The highest CF per exposure group was found after 7 or 20 days; at longer exposure times the CF was lower. Based on the nominal levels in the soil and the corresponding CF-values, the levels in live worms varied from 0.2-0.45 mg.kg\(^{-1}\) fresh weight at the lowest concentration to 0.75-2.2 mg.kg\(^{-1}\) fresh weight at the highest non-lethal concentration. In the dead worms, exposed to a lethal concentration of 10 mg 2,3,7,8-TCDD.kg\(^{-1}\) soil, levels were found up to 1.8-3 mg.kg\(^{-1}\) fresh weight. This study showed that the CF decreased with increasing exposure concentration. The study was carried out in sandy loam with 5% organic matter (OM) and a pH of 6.7. The 2,3,7,8-TCDD level in worms and soil was determined with gaschromatography (GC). Additional tests where the earthworms *A. caliginosa* and *Lumbricus rubellus* were exposed to 2,3,7,8-TCDD treated filter paper suggested that this compound is hardly taken up by the skin of these organisms (Reinecke and Nash, 1984). In a laboratory study on the distribution of pesticides in experimental micro-ecosystems ('microagro-ecosystem chambers') 2,3,7,8-TCDD levels were also determined in earthworms and soil. Based on the reported data CF-values of 1 and 0.8 were calculated. These calculations were based on the non-metabolized 2,3,7,8-TCDD concentration in the worms (0.4 and 0.7 ng.kg\(^{-1}\) fresh weight, respectively, determined with GC) and the average level in the topmost 13 cm of the soil (0.4 and 0.9 ng.kg\(^{-1}\), respectively). The soil used in the micro-ecosystems contained 5% OM and had a pH of 6.7. The earthworm species analyzed were not specified (Nash and Beall, 1980). From these studies CF-values may also be calculated that are considerably higher than 1, depending on the analytical method used for the determination of the levels in worms and depending on the choice of the soil profile to which the level in worms is related (the level in the soil highly decreases with increasing
depth). Thus, Heida et al. (1986) reported CF-values of 65-80, based on the results published by Nash and Beall. The latter did not report any CF-values.

In several field experiments in contaminated areas (including Seveso-Italy and the Volgermeer polder-The Netherlands) the accumulation of dioxins in invertebrates and/or vertebrates was studied.

In Seveso the accumulation of 2,3,7,8-TCDD in earthworms (Allobophora rosea, A. caliginosa and A. chlorotica) was determined, resulting in CF-values of 5-43, with an average value of 15. A linear correlation was indicated between the 2,3,7,8-TCDD concentration in the soil and that in earthworms. It is not clear whether these data are based on the dry weight or the wet weight of the worms. The concentration in soil was not mentioned in the secondary publication used, neither were data on soil properties.

In the Volgermeer polder (former rubbish tip) the accumulation of 2,3,7,8-TCDD and a number of PCDFs (TCDFs, PeCDFs and HxCDFs) was determined in earthworms (L. rubellus, Dendrobaena rubia and A. rosea) and in the liver of mice (the herbivorous wood mouse Apodemus sylvaticus and the carnivorous shrew Sorex araneus). At the 'VA' location (soil with 20% OM, 9% water and a pH of 7.5) 2,3,7,8-TCDD levels of 6,840 and 16,760 ng.kg⁻¹ dry weight were found in the worms and in the liver of mice, respectively, at a concentration in the topmost 10 cm of the soil of 1930 ng.kg⁻¹ dry weight, resulting in CF-values of 3.5 for worms and 8.5 for mice. No distinction was made between the various species of worms and mice. At the 'VC' location (soil with 80% OM, 66% water and a pH of 4.9) these levels were 115 and 1725 ng.kg⁻¹ dry weight, respectively, at a level in the topmost 10 cm of the soil of 315 ng.kg⁻¹, resulting in CF-values of 0.35 for worms and 5.5 for mice. Based on fresh weight the CF-values for the worms are 0.7 (VA) and 0.07 (VC); in the calculation a standard factor 5 has been used, in accordance with Romijn et al. (1991b). For PCDFs CF-values for worms and mice were found of 0.02 - 12 and 0.06 - 185, respectively, based on dry weights. For the worms these values correspond with fresh weight CF-values of 0.004 - 2.4. The soil levels of individual PCDFs varied from undetectable to over 7000 ng.kg⁻¹. In almost all instances the CF-values at the VC-location were lower than those at the VA-location, for worms as well as for mice, indicating a lower availability at a high organic matter content. The results of these studies indicate a selective accumulation of specific higher chlorinated dioxins in mice, resulting in biomagnification (accumulation in the food chain). This has been found for instance for 2,3,7,8-TCDD, 2,3,7,8-TCDF and
2,3,4,7,8-PeCDF. For a number of PCDFs inconsistent results were found for one single compound. The above-mentioned levels were determined with GC-MS (Heida and Olie, 1985; Heida et al., 1986).

In a field experiment abroad in an area contaminated with industrial sludge (11 ng 2,3,7,8-TCDD.kg\(^{-1}\) soil and 110 ng 2,3,7,8-TCDF.kg\(^{-1}\) soil) a 2,3,7,8-TCDD level of 36 ng.kg\(^{-1}\) fresh weight was found in worms, resulting in a CF of 3.3. The 2,3,7,8-TCDF level in the worms was not determined. In mice (Peromyscus maniculata) levels of 15 ng 2,3,7,8-TCDD.kg\(^{-1}\) fresh weight and 7.3 ng 2,3,7,8-TCDF.kg\(^{-1}\) fresh weight were reported, resulting in CF-values of 1.4 and 0.07, respectively.

**Birds**

Near the Lake IJssel from 1980 to 1985 dioxin levels were measured in the liver of birds preying on fish (cormorant Phalacrocorax carbo, grey heron Ardea cinerea, grebe Podiceps crisatus) and in eel (Anguilla anguilla), a major food source for the cormorant. In the liver of the birds only 2,3,7,8-substituted PCDDs and PCDFs were identified. The highest levels by far (1000-2700 ng.kg\(^{-1}\) fresh weight) were found for 1,2,3,4,6,7,8-HpCDD, 2,3,4,7,8-PeCDF and 1,2,3,4,6,7,8,-HpCDF, in cormorants. In the grebe (one sample only) and in the herons the levels of these congeners were much lower, with a maximum of 260 ng.kg\(^{-1}\) fresh weight for 2,3,4,7,8-PeCDF in herons. Levels of other congeners in the liver of the birds varied from non-detectable to 600 ng.kg\(^{-1}\) fresh weight. The levels in eel varied from 0.4-6.4 ng.kg\(^{-1}\) fresh weight, a factor 10 to 340 lower than the level of the corresponding congener in the liver of the cormorants. In eel and in cormorants the authors reported qualitatively the same congener pattern. In a study near Lake Ontario the same phenomenon was observed: accumulation only of 2,3,7,8-substituted PCDDs and PCDFs in fish (herring Alosa pseudoharengus) and fish-eating birds (gull). In the latter study the level of the corresponding congener in the fish was found to be 4-18 times lower than in the liver of the birds and 7-32 times lower than in the whole bird. In both studies considerably higher 2,3,7,8-TCDD levels were found in the liver of birds than in fish (factor 7-18), whereas this was not the case for 2,3,7,8-TCDF.
Conclusions accumulation

Dioxins present in the soil are very likely to be taken up by plants to a small degree only, and when this uptake via the roots does occur, transported to the emergent parts to a small degree. For crops with detectable levels 'in' the emergent parts these are very probably caused by adsorption, deposition from the air and evaporation from the soil playing a great part. Reliable concentration factors (CF-values) for the accumulation of 2,3,7,8-TCDD in earthworms vary from 0.07 to 9.4; most of these values are appr. 1 or < 1. These values are based on the level of unmetabolized 2,3,7,8-TCDD in the worms (on a fresh weight basis) Standardization of these experimentally determined values to a soil with 10% organic matter results in CF_{st}-values of 0.2-4.7, with a geometric mean value of 0.7. These data suggest that 2,3,7,8-TCDD is hardly or not at all concentrated from the soil by earthworms. However, at high 2,3,7,8-TCDD levels in the soil correspondingly high levels in the earthworms may be found, also in view of the insusceptibility of the earthworms to this compound. Thus in live earthworms 2,3,7,8-TCDD levels may be indicated of 0.75-2.2 mg kg\(^{-1}\) fresh weight, when exposed to a soil level of 5 mg kg\(^{-1}\), the highest non-lethal level. For other dioxins no or hardly any CF-values are available. Only for a number of PCDFs in one field study were CF-values for the accumulation of these compounds in earthworms determined, resulting in values of 0.004-2.4.

Data from various field studies suggest that in higher animal species, fish-eating birds as well as (worm-eating) mammals, selective accumulation occurs of specific 2,3,7,8-substituted PCDDs and PCDFs with 4 to 8 chlorine atoms. For a number of such dioxins biomagnification, accumulation in the food chain, has been demonstrated. This, for instance, is true for 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 2,3,7,8-TCDF and 2,3,4,7,8-PeCDF, the most toxic dioxins. However, the results with respect to biomagnification are not unequivocal. The differences observed may partly be explained by species specific differences, related to behaviour (food) and kinetics.

5.3.2 Toxicity

Microbial processes

In 7-week laboratory tests several micro-organisms and the microbial activity in three
contaminated soils were compared to those in a control soil. The 2,3,7,8-TCDD levels in the contaminated soils were $8 \times 10^3$, $1100 \times 10^3$ and $2400 \times 10^3$ ng.kg$^{-1}$ (8 - 2400 µg.kg$^{-1}$). No adverse effects of 2,3,7,8-TCDD were observed with respect to various eutrophic bacteria, actinomycetes and fungi, and with respect to enzyme activities and soil respiration (measured as CO$_2$-production). The number of oligotrophic bacteria in all contaminated soils was lower than that in the control soil, however, the number increased with increasing 2,3,7,8-TCDD content. The contaminated soils (originating from various locations) and the control soil were loamy soils with more or less comparable properties (2%-6% OM, 4%-12% clay, pH 6.5-7.7). In 4-week laboratory tests no effects on the respiration were found when 0.005 - 50 ng 2,3,7,8-TCDD.kg$^{-1}$ dry weight was added to the soil (6% OM, pH 5.6) and when 0.013 - 130 ng 2,3,7,8-TCDD.kg$^{-1}$ dry weight was added to the humus layer (80% OM, pH 6.2). Higher levels were not studied.

**Plants**

Toxicity studies with plants are not available. In the pot experiments with maize and beans mentioned in section 5.3.1 germination and "maximal development" occurred at 2,3,7,8-TCDD levels of 1-750 ng.kg$^{-1}$ soil, however, germination, growth and yield were not considered quantitatively in this accumulation study.

**Soil organisms**

In a 85-day laboratory test with earthworms *A. caliginosa* no mortality occurred at 2,3,7,8-TCDD levels in the soil of up to $5 \times 10^6$ ng.kg$^{-1}$. A level of $10 \times 10^6$ ng.kg$^{-1}$ caused 100% mortality following 30 days of exposure (see also 5.3.1).

Experimental animal studies with guinea pigs and rats suggest that the "biological availability" of 2,3,7,8-TCDD in soil is lower than that of 2,3,7,8-TCDD in oily solvents. The biological availability is estimated based on tissue levels and occurring effects. The availability is influenced by soil properties such as the carbon content (decreasing availability with increasing carbon content), the nature of the contamination (for instance increased availability in the presence of oil) and the residence time in the soil (decreasing availability with increasing residence time). All of these factors influence the adsorption on soil particles and therefore the resorption in the gastrointestinal tract.
In a long-term field study (Young et al., 1987) in a 2,3,7,8-TCDD contaminated area a significantly (p<0.01) increased liver weight was found in pregnant mice; this effect was observed over a number of years. In these mice no histopathological effects were found, neither in the liver nor in any other organs. The numbers of fetuses per litter, an average of 3.1 and 3.4 in the contaminated area and the control area, respectively, were in the same range. In the contaminated area the 2,3,7,8-TCDD level in the soil removed by the mice for nest building was 75-285 ng.kg\(^{-1}\). In this long-term field study an inventory was also made of a great number of animal species, including insects, reptiles, birds and small and large mammals, and of a great number of plant species. Based on this it was concluded that no significant ecological effects occur at 2,3,7,8-TCDD levels of up to 1500 ng.kg\(^{-1}\) soil. It should be noted that the results of this study are difficult to interpret, since in this article no details on the inventory were reported and the 2,3,7,8-TCDD levels in the soil of the experimental area, measured in the 0-15 cm toplayer, varied considerably: from <10 to 1500 ng.kg\(^{-1}\). Per subarea concentrations were measured that also varied considerably, with average values of 325 (<10 - 1500), 115 (<10 - 470) and 30 (<10 - 150) ng.kg\(^{-1}\). The levels were measured over a period of five consecutive years. The area concerned mainly consisted of sandy soil (0.2% organic matter, 4% clay, pH 5.6).

In another field study no toxic effects were observed in worms, mice and birds originating from an area contaminated with industrial sludge, with 2,3,7,8-TCDD and 2,3,7,8-TCDF levels of appr. 11 and 110 ng.kg\(^{-1}\), respectively, in the soil. Comparison with a control area shows no effects on the reproduction. It was remarkable that the bio-diversity in the treated area had increased, possibly by application of the organic waste (increased amount of food available).

**Birds**

In a field study in British Colombia a possible effect of 2,3,7,8-TCDD was observed on the hatching index of herons. One year when all eggs in a specific colony broke during the breeding season (due to a considerably thinner eggshell than usual), an average 2,3,7,8-TCDD level of 210 ng.kg\(^{-1}\) wet weight was found in the eggs, a factor 3 higher than in the preceding year. The levels of other compounds examined in the study (other dioxins, PCBs, pesticides, heavy metals) were not elevated.
Conclusions toxicity

Useful toxicity data are available for 2,3,7,8-TCDD only, however, the number of data for this compound is limited.

In two laboratory studies no effects were observed on microbial activity (including soil respiration) at 2,3,7,8-TCDD levels of 0.005 - 50 ng.kg\(^{-1}\) dry weight and 8\(\times\)10\(^3\) - 2400\(\times\)10\(^3\) ng.kg\(^{-1}\), respectively, in the soil. Reliable toxicity data for plants are not available, however, in pot experiments with maize and beans germination and "maximal development" occurred at 2,3,7,8-TCDD levels of 1 - 750 ng.kg\(^{-1}\). A long-term study with the earthworm \textit{A. caliginosa} resulted in a NOLC of 5\(\times\)10\(^6\) ng.kg\(^{-1}\); twice that concentration resulted in a 100% mortality.

In a long-term field study, based on an inventory of a great number of various animal species (including insects, reptiles, birds and mammals) and plant species, it was concluded that no significant ecological effects occur at 2,3,7,8-TCDD levels of 1500 ng.kg\(^{-1}\) in the soil. It is noted that the dispersion of the 2,3,7,8-TCDD levels in the area was great, varying from <10 to 1500 ng.kg\(^{-1}\), with average levels in three different subareas of 325, 115 and 30 ng.kg\(^{-1}\), respectively. In gestating mice (\textit{Peromyscus polionotus}) originating from this area the liver weight was significantly increased at 2,3,7,8-TCDD levels of 75-285 ng.kg\(^{-1}\) in the soil, however, no histopathological effects were observed, neither any effects on the reproduction (number of fetuses per gestation).

5.4 TOXICITY FOR FARM ANIMALS

5.4.1 Accumulation

Most data on accumulation and other aspects concerning the kinetics of dioxins in farm animals are based on oral exposure studies with cows. Therefore, the following data are limited to the results from these studies; the very limited data from studies with other farm animals have not been taken into account. The available data mainly concern 2,3,7,8-substituted dioxins, because of the high degree of accumulation of these persistent compounds.

The degree of uptake in the gastrointestinal tract as well as the further kinetics of dioxins
depend for instance on the substitution pattern (degree of chlorination).

Estimates for the biological availability (the percentage of the dose resorbed in the gastrointestinal tract) vary from 1%\,-\,3\% for hepta- and octacongeners to appr. 30\%\,-\,50\% for tetra-, penta-, and hexacongeners (with the exception of 2,3,7,8-TCDF the availability of which is estimated at 1\%-2\%). These estimates are based on studies where the compounds were directly added to the feed, for instance in olive oil. In substrates such as soil and fly ash the availability will be lower (thus in 7.3.1 an availability is estimated of 0.5\%-10\%, for the total group of dioxins, when taken up from the ground).

Following uptake accumulation mainly takes place in adipose tissue and in the liver. In milk fat and body fat of lactating cows exposed for 10 weeks to technical-pentachlorophenol (t-PCP) contaminated with PCDDs and PCDFs levels were found 1000 times higher than those found in blood. In lactating animals excretion in the milk may be more significant than the metabolic clearance. This is evident partly from the elimination half-lives in body fat of non-lactating cows (appr. 110\,-\,280 days) that are considerably longer than those in body fat of lactating cows (25\,-\,40 days). The distribution to adipose tissue is slow and may take several tens of days, thus shortly after exposure accumulation will mainly take place in the liver. Elimination half-lives for (2,3,7,8-substituted) dioxins in milk fat vary from appr. 30 to 110 days. However, for 2,3,7,8-TCDF in milk fat an extremely short half-life of appr. 1 day was found, indicating that this compound is readily metabolized. In one of the studies where half-lives in milk fat were determined (Olling et al., 1990; Derks et al., 1991), half-lives for most 2,3,7,8-substituted dioxins following long-term exposure (via soil and crops) were found to be longer than those following single exposure (intraruminal application).

In a study with lactating cows exposed to 500 ng 2,3,7,8-TCDD kg\(^{-1}\) feed, concentration factors were calculated of 0.18 for milk and 1.56 for cream.

5.4.2 Toxicity

Cattle

In cows following a single oral dose of 50 ng 2,3,7,8-TCDD kg\(^{-1}\) body weight enzyme induction was found (increased AHH- and EROD- levels).

Data concerning the toxicity of dioxins at repeated or long-term exposure mainly originate from studies where animals were exposed to "technical"-PCP (tPCP). Thus in a feeding study
with young cattle given a dose of 15-20 mg t-PCP.kg\(^{-1}\) body weight per day growth retardation was found after 3 weeks of exposure. The effect did not occur in animals exposed to a similar dose of "analytical"-PCP (a-PCP) containing considerably fewer dioxins and other contaminants. In other experiments with cattle at long-term exposure to comparable doses a great number of effects was found, including effects on the body weight, organ weights, liver function (increased AHH activity; effects on cytochrome P-450) and on haematological parameters (anaemia). In one of such studies with calves exposed for 6 weeks to 1 mg t-PCP.kg\(^{-1}\) body weight per day an increased liver weight and a decreased thymus weight was still found. For more details on these studies reference is made to the integrated criteria document Chlorophenols (Slooff et al., 1990) and the corresponding appendix "Effects" (Janus et al., 1990).

**Poultry**

In the United States many millions of chickens died in 1957 as a result of PCDD-contaminated feed. (Pericardial) oedema was the most characteristic symptom, that is why this disease has become known as "chicken oedema disease". Mixtures of tri- and tetraCDDs in feed at a dose as low as 10 ng.kg\(^{-1}\) (corresponding to appr. 1.8 ng PCDD.kg\(^{-1}\) body weight per day) lead to the above-mentioned symptom and to mortality. For chicken embryos LD50-values have been reported of 7 ng per egg for 2,3,7,8-TCDD and 3 ng per egg for 1,2,3,7,8-PeCDD, corresponding with 130 and 60 ng.kg\(^{-1}\) per egg, respectively. At a total-dioxin level of 0.9 ng per egg (17 ng.kg\(^{-1}\)) the final percentage and growth reduction in chicks has been demonstrated to be reduced by 50%.

### 5.5 SUMMARY AND TOXICOLOGICAL RECOMMENDED LEVELS

#### 5.5.1 Humans

In mammals and humans selective retention occurs of 2,3,7,8-substituted PCDDs and PCDFs. Since these compounds have very long half-lives they accumulate in body fat. There is great variance in susceptibility of various experimental animals for 2,3,7,8-TCDD, which may only partly be explained by differences in toxicokinetics and variations in cellular levels
of Ah-receptors (to which 2,3,7,8-TCDD binds). The most significant effects following long-term exposure are liver- and immunotoxicity and effects on the reproduction and teratogenicity in experimental animals.

2,3,7,8-TCDD is carcinogenic in experimental animals, however, insufficient evidence exists to prove its carcinogenicity in humans. It has also been shown to possess a strong promotor action in the liver of rats, whereas no initiating action was demonstrated. There is insufficient evidence to consider 2,3,7,8-TCDD to be genotoxic. With respect to the mechanism of action of 2,3,7,8-TCDD it is internationally concluded that in order for 2,3,7,8-TCDD to cause any effects it must first bind to an intracellular receptor, the Ah-receptor. Following binding to this "dioxin-receptor" the induction of P450 IA1 occurs as the first observable effect, via (as yet) unknown mechanisms. The other effects are assumed to be related with this induction. In the determination of a toxicological recommended value based on these data a threshold value is assumed to exist.

In a great number of epidemiological studies among occupationally exposed persons as well as among population groups exposed due to industrial accidents, chloracne was the only effect consistently related to dioxin exposure. In all of these studies mixed and not properly quantified exposure was involved. Of all the human data those concerning Seveso are the most important. However, as the exposure there could not be properly quantified also, these data are not suitable for the determination of a recommended value.

At the WHO/EURO meeting (1990) the following method was used to determine the tolerable daily intake (TDI) for 2,3,7,8-TCDD. Data from Seveso and those from animal experiments showed that serum levels in humans which could not be associated with effects, did cause effects in experimental animals. From this it could be safely concluded that humans are not more susceptible than are experimental animals. In further calculations, to be on the safe side, the WHO assumed the susceptibility of humans and experimental animals to be the same. This holds that at a similar internal concentration (in a specific target organ) the effects in humans and in experimental animals will be similar. Since the liver is a primary target organ, the internal concentration in the liver was taken as a starting point. By means of pharmaco-kinetic calculations, based on a specific intake by experimental animals, the resulting internal concentration in the liver may be calculated, and subsequently the intake by humans resulting in similar internal concentrations. Hence the no effect dose in experimental animals can be converted into a no effect dose in humans.
Chronic studies with rats show a no effect dose of 1 ng.kg\(^{-1}\) body weight per day for liver toxicity as well as for effects on the reproduction. Via the pharmacokinetic approach, based on a steady-state situation, it was determined that a daily exposure to 1 ng.kg\(^{-1}\) body weight in rats over 2 years results in a liver concentration which in humans will be reached at a daily intake of 100 pg.kg\(^{-1}\) body weight over 70 years. Thus the no effect dose for humans is 100 pg.kg\(^{-1}\) body weight per day. For effects on the production it may not be assumed that humans are not more susceptible, due to the unreliable data from Seveso. Furthermore the data from Seveso show a great variability in susceptibility for dioxin exposure within the population. Therefore the WHO recommended a tolerable daily intake in 1990 of 10 pg 2,3,7,8-TCDD per kg body weight per day, using the no effect dose of 100 ng 2,3,7,8-TCDD per kg body weight, with a safety factor of 10 (WHO, 1991; Theelen et al., 1991). This WHO-recommendation was supported by the RIVM (note Knaap, 1991).

Assessment of mixtures

With regard to 2,3,7,8-TCDD related compounds the WHO has (as yet) not made any recommendations. According to the WHO the TEF principle for the determination of the risk is useful, although according to them the risk will be slightly overestimated. Since no data are available to quantify the degree of overestimation, the RIVM for the time being recommends in the assessment of mixtures to interpret the recommended value as 10 pg TEQ per kg body weight per day. Hereby the TEF values recommended by the TEF working group need to be taken up (see table 5.1).

A number of PCBs (the planar and the mono-ortho planar PCBs) cause effects corresponding with those of the 2,3,7,8-PCDDs and PCDFs. Therefore, in a number of articles, these compounds are considered to be dioxin related (McKinney et al., 1985; Leece et al., 1985; Safe, 1987). Since humans and animals are exposed to PCBs as well as to PCDDs and PCDFs, Safe et al. (1990) and the Dutch TEF working group have proposed TEF-factors for these compounds as well (see table 5.2).

In a RIVM study recently carried out (Liem et al., 1991), levels of PCB have also been determined in foodstuffs, in addition to those of 2,3,7,8-substituted PCDDs and PCDFs. Based on these data the median intake of planar PCBs by adults and children is calculated. Based on the TEF-factors of the Dutch TEF working group the median intake for adults amounted to 1 pg TEQ.kg\(^{-1}\) body weight per day. For children and adolescents (up to 20 years) the daily intake was slightly higher (due to a lower body weight). These figures mean
that the median exposure to TEQ is doubled when the TEQ is considered as the sum of TEQ as a result of 2,3,7,8-substituted PCDDs and PCDFs, and TEQ as a result of planar PCBs.

**Table 5.2** Proposal for TEF-factors for planar and mono-ortho planar PCBs

<table>
<thead>
<tr>
<th>PCB-IUPAC-no.</th>
<th>Structure</th>
<th>TEF-factors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Safe (1990)</td>
</tr>
<tr>
<td>planar</td>
<td></td>
<td></td>
</tr>
<tr>
<td>77</td>
<td>3,4,3',4'-TCB</td>
<td>0.01</td>
</tr>
<tr>
<td>126</td>
<td>3,4,5,3',4'-PeCB</td>
<td>0.1</td>
</tr>
<tr>
<td>169</td>
<td>3,4,5,3',4',5'-HxCB</td>
<td>0.05</td>
</tr>
<tr>
<td>mono-ortho planar</td>
<td></td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>2,3,4,4'-TCB</td>
<td>-</td>
</tr>
<tr>
<td>74</td>
<td>2,4,5,4'-TCB</td>
<td>-</td>
</tr>
<tr>
<td>105</td>
<td>2,3,4,3',4'-PeCB</td>
<td>0.001</td>
</tr>
<tr>
<td>114</td>
<td>2,3,4,5,4'-PeCB</td>
<td>0.001</td>
</tr>
<tr>
<td>118</td>
<td>2,4,5,3',4'-PeCB</td>
<td>0.001</td>
</tr>
<tr>
<td>123</td>
<td>3,4,5,2',4'-PeCB</td>
<td>0.001</td>
</tr>
<tr>
<td>156</td>
<td>2,3,4,5,3',4'-HxCB</td>
<td>0.001</td>
</tr>
<tr>
<td>157</td>
<td>2,3,4,3',4',5'-HxCB</td>
<td>0.001</td>
</tr>
<tr>
<td>167</td>
<td>2,4,5,3',4',5'-HxCB</td>
<td>0.001^a</td>
</tr>
</tbody>
</table>

* no data available

Nationally as well as internationally discussions are currently being held on the TEF-values to be used for the PCBs mentioned. More results are to be expected from a WHO-consultation planned for early 1993. For the interpretation of the results from the above-mentioned food study the RIVM (note Knaap) recommended in 1991 to incorporate the planar PCBs in the assessment of the total load of TEQ of the population through food. However, due to the lacking international consensus on the level of the TEF-values and the as yet lacking data concerning the mono-ortho planar PCBs, in the revision of the Commodities Act standard for cow's milk, for which the food study served as a base, it could not yet be decided to incorporate the PCBs. However, it was recommended to perform further research in order to obtain a better understanding of the exposure levels of these compounds. A possible international consensus, to be reached at the next WHO-consultation in 1993, might offer further support to arrive at a new revision of starting-points concerning the assessment of mixtures of dioxins and related compounds.
5.5.2 Aquatic and terrestrial environment

In the determination of ecotoxicological recommended values (maximally tolerable risk levels, MTRs) it was departed from the RIVM guidance document (Slooff, 1992). In the current report it is indicated which extrapolation methods are currently used within the RIVM. If the data allow, two MTRs are determined per environmental compartment, one based on the results of single species toxicity data, for the protection of aquatic ecosystems (MTR_{ecosystem}) and one on food-chain poisoning, for the protection of predators (MTR_{predator}). In both cases different extrapolation methods are available, a more advanced refined effect assessment method (used to calculate a 95% protection level based on the distribution of chronic NOEC values or chronic NO(A)EL-values) and a less advanced preliminary effect assessment method, whereby fixed extrapolation factors are used. For the application of the refined effect assessment method for the determination of a MTR_{ecosystem} and a MTR_{predator}, a minimum of 4 chronic NOEC-values (for different species) and 4 chronic NO(A)EL-values (for different mammalian species), respectively, are required. In the determination of MTRs for dioxins, due to the limited number of reliable toxicity data, only the preliminary effect assessment method can be applied, based on the data for 2,3,7,8-TCDD. The MTRs determined are therefore considered as indicative values. For a further general elucidation of the extrapolation methods reference is made to Slooff et al. (1992). An overview of the MTRs determined and the data on which they are based is given in table 5.3.

Fresh water

2,3,7,8-TCDD

Based on single species toxicity data a MTR_{ecosystem} is determined of 0.0012 ng.l\(^{-1}\), being the lowest result of the extrapolation based on the lowest acute LC50 and the lowest chronic NOEC, respectively. This MTR_{ecosystem} is based on a test in a continuous-flow system where faeces and excess food were removed daily and therefore considered as dissolved fraction. Departing from a partition coefficient (Kp\(^3\)) of 315,000 l.kg\(^{-1}\) and a suspended sludge content of 30 mg.l\(^{-1}\) this MTR_{ecosystem} corresponds with a total concentration in water of

\[ Kp = Kow \times foc = 6310000 \times 0.05 = 315000 \text{ l.kg}^{-1} \]

(Slooff et al., 1992; foc=fraction organic carbon)
0.012 ng.l\(^{-1}\) (dissolved and particulate fraction). The MTR\(_{ecosystem}\) is based on studies with fish; invertebrates are considerably less susceptible. Based on the NO(A)EL\(_{predator}\) a MTR\(_{predator}\) of 0.00005 ng.l\(^{-1}\), dissolved fraction, is determined corresponding with a total concentration of 0.00005 ng.l\(^{-1}\). The MTR\(_{predator}\) is the quotient of the NO(A)EL\(_{predator}\) and the BCF\(_{fish}\): MTR = NO(A)EL/BCF. Field studies that can be used to test the above-mentioned MTRs are not available.

Other PCDDs and PCDFs

For the other dioxins maximally only one NOEC and/or NOLC is available, based on studies with fish. With the preliminary effect assessment method an indicative MTR can in principle be determined for any compound for which a NOEC or NOLC is available, however, due to the limited data -quantitative as well as qualitative- the result of this method is very unreliable. Based on the corresponding effects of the various PCDDs and PCDFs in fish and based on the results from the study by Bol et al. (1989) in which the action of a combination of dioxins was studied, PCDDs and PCDFs are assumed to possess an additive action. From the compounds studied 2,3,7,8-TCDD is the most toxic. For these reasons it is suggested to interpret the MTRs determined for 2,3,7,8-TCDD as "toxic equivalent" concentrations (expressed in equivalents TEQ.l\(^{-1}\)). In the risk assessment of mixtures of PCDDs and PCDFs toxicity equivalency factors are then used for the various compounds. The TEF for 2,3,7,8-TCDD is hereby put at 1; the one for the other dioxins is lower, due to their lower toxicity. The TEF concept is similar to the human-toxicological approach.

TEFs for various dioxins have been calculated from several studies with fish (Mehrl et al., 1988; Bol et al., 1989; Wisk and Cooper, 1990a (annexes 2a-c); Helder et al. [unpublished results, see Van Zorge et al., 1989]). Most of the TEFs calculated from these studies are easily comparable with the so-called I-TEFs (table 5.1) which are mainly based on studies with mammals. Only for 2,3,7,8-substituted PCDDs and PCDFs with minimally 6 chlorine atoms it is true that the TEFs determined on the basis of the aquatic studies are usually lower (factor 2.5 to 250) than the I-TEFs used for these compounds. Considering the small number of aquatic studies and the extremely poor water solubility of the higher chlorinated dioxins it may however be questioned to what extent the differences found are realistic. For these reasons, until more reliable aquatic toxicity data are available, it is as yet proposed to also
use the TEFs in the assessment of mixtures of PCDDs and PCDFs in surface water.
In the conversion of the MTRs based on the dissolved fraction to that based on the total concentration, dissolved and particulate, it should be taken into account that the partition coefficients (Kp) for the various dioxins differ, depending on the Kow. Also in view of the uncertainties concerning the determination of the above-mentioned MTRs it is, however, proposed to use only one Kp for the total group of dioxins, which is the one for 2,3,7,8-TCDD:315000 l. kg\(^{-1}\).

**Seawater**

Due to the lack of toxicity data for marine organisms, the same MTRs are proposed for seawater as for fresh water (MTR\(_{\text{ecosystem}}\):0.0012 ng I-TEQ.l\(^{-1}\), dissolved fraction; MTR\(_{\text{predator}}\):0.00005 ng I-TEQ.l\(^{-1}\), dissolved fraction).

**Sediment**

Toxicity data for benthic organisms are not available. For this reason a MTR can only be determined with the equilibrium partition method, as follows: MTR-sediment = MTR-surface water (dissolved fraction) \(*\) Kp. Based on the MTR\(_{\text{ecosystem}}\) this results in a MTR-sediment of 378 ng I-TEQ.kg\(^{-1}\) dry matter. Based on the MTR\(_{\text{predator}}\) this results in a MTR-sediment of 15 ng I-TEQ.kg\(^{-1}\) dry matter.

**Soil**

**2,3,7,8-TCDD**

Based on single species toxicity data a MTR\(_{\text{ecosystem}}\) has been determined of 500,000 ng.kg\(^{-1}\). It should be noted that this value is exclusively based on a study with earthworms; for soil arthropodes and for plants, two other important groups of soil organisms, no toxicity data are available. Microbial tests have resulted in NOEC-values ranging from >50 ng.kg\(^{-1}\) to >2,400 \(\mu\)g.kg\(^{-1}\). Based on the NO(A)EL\(_{\text{predator}}\) a MTR\(_{\text{predator}}\) has been determined of 3 ng.kg\(^{-1}\). The MTR\(_{\text{predator}}\) is the quotient of the NO(A)EL\(_{\text{predator}}\) and the BCF\(_{\text{worm}}\), as follows: MTR = NO(A)EL/BCF.

In a long-term field study where over the years a great number of species (including plants,
insects, reptiles, birds and mammals) was studied, no significant ecological effects were observed at 2,3,7,8-TCDD levels in the soil up to 1500 ng.kg\(^{-1}\). Per subarea average levels of 30 to 325 ng.kg\(^{-1}\) were measured. It is not known whether in this field study a survey of worm-eating predators has taken place.

**Other PCDDs and PCDFs**

For the other dioxins no toxicity data are available. Similar to the approach followed for surface water the MTRs determined for 2,3,7,8-TCDD are considered as equivalents I-TEQ, resulting in a MTR\(_{\text{ecosystem}}\) of 500,000 ng I-TEQ.kg\(^{-1}\) and a MTR\(_{\text{predator}}\) of 3 ng.kg\(^{-1}\).

**Concluding remarks ecotoxicological recommendations**

With respect to all of the above-mentioned MTRs (surface water, sediment, soil) it is once again emphasized that, due to a number of uncertainties, it concerns indicative values. One of the uncertainties is the use of fixed extrapolation factors, such as the use of a factor 10 at the lowest NOEC\(_{\text{fish}}\) and the lowest NO(A)EL\(_{\text{rat}}\). Comparisons of MTR\(_{\text{ecosystem}}\)-values for a great number of compounds, which in the past few years have been assessed by the RIVM, show that the use of a preliminary effect assessment-method usually leads to a lower MTR than the use of more advanced refined effect assessment-methods with which the 95%-protection level is calculated. Further uncertainties concerning the above-mentioned MTRs partly concern the determination of a lowest NOEC\(_{\text{fish}}\) from effect concentrations (MTR\(_{\text{ecosystem}}\)-surface water), the possible effects on the BCF\(_{\text{fish}}\) through exposure to a concentration which eventually led to an elevated mortality percentage (MTR\(_{\text{predator}}\)-surface water), the partition coefficient used (MTR-sediment) and the use of toxicity equivalency factors for the total group of dioxins. Despite the uncertainties concerning the determined MTRs, the conclusion may be justified that food-chain poisoning as a result of biomagnification is the most crucial effect of 2,3,7,8-TCDD (and other persistent dioxins), both in the aquatic and the terrestrial environment.
Table 5.3  Determination indicative MTRs for 2,3,7,8-TCDD in surface water and soil

<table>
<thead>
<tr>
<th>Data input</th>
<th>Extrapolation factor</th>
<th>Bioconcentration factor</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Surface water - determination MTR\textsubscript{ecosystem}</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lowest acute L(E)C50\textsuperscript{1}</td>
<td>EF</td>
<td>1000</td>
<td>0.003 ng.l\textsuperscript{-1}</td>
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<tr>
<td>3 ng.l\textsuperscript{-1}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lowest chronic NOEC\textsuperscript{2}</td>
<td>EF</td>
<td>10</td>
<td>0.0012 ng.l\textsuperscript{-1} MTR\textsubscript{ecosystem} (dissolved fraction)</td>
</tr>
<tr>
<td>0.012 ng.l\textsuperscript{-1}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Surface water - determination MTR\textsubscript{predator}</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NO(A)EL\textsubscript{predator} \textsuperscript{3}</td>
<td>BCF\textsubscript{fish} \textsuperscript{4}</td>
<td>40,000</td>
<td>0.00005 ng.l\textsuperscript{-1} MTR\textsubscript{predator} (dissolved fraction)</td>
</tr>
<tr>
<td>2 ng.kg\textsuperscript{-1} feed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Soil - determination MTR\textsubscript{ecosystem}</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lowest chronic NOEC\textsuperscript{5}</td>
<td>EF</td>
<td>10</td>
<td>500,000 ng.kg\textsuperscript{-1} MTR\textsubscript{ecosystem}</td>
</tr>
<tr>
<td>5000000 ng.kg\textsuperscript{-1} soil</td>
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<tr>
<td><strong>Soil - determination MTR\textsubscript{predator}</strong></td>
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<td></td>
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<tr>
<td>NO(A)EL\textsubscript{predator} \textsuperscript{3}</td>
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<td>3 ng.kg\textsuperscript{-1} MTR\textsubscript{predator}</td>
</tr>
<tr>
<td>2 ng.kg\textsuperscript{-1} feed</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Legenda:
1 Acute LC50 for the rainbow trout *S. gairdneri* (Bob et al., 1989). Other acute LC50-values are not available; see annex 2a.
2 *S. gairdneri* (Mehrle et al., 1988). This is the only study where a relatively long test time (a total of 56 days: 28 days exposure and 28 days purification) was combined with exposure in a continuous-flow system and with analyses of the actual exposure concentrations. In this study with larvae of *S. gairdneri* effects were still observed at the lowest test concentration of 0.038 ng.l\textsuperscript{-1}, eventually resulting in 45% mortality (versus 7% in the control). This effect was statistically significant (p < 0.05). The concentration mentioned is lower than the concentrations (whether or not resulting in a NOEC) which where used in the other tests; see annex 2a. For these reasons a 56-d LC10 has been estimated from the graph of the concentration-effect relation found in this study; this LC10 is used as a NOEC. In this test at the end of the exposure time a statistically significant effect on the growth was also observed at all concentrations, however a 56-d EC10 for growth could not be determined for the reason that at the higher concentrations the weight was not determined at the end of the study (high mortality percentage).
3 In the draft Integrated document (Working document March 1992) a LC10 was calculated from the Mehrle et al. study with a logistic calculation method according to Haanstra et al. (1985), resulting in a LC10 of 0.0046 ng.l\textsuperscript{-1}. The 56-d LC50 of 0.016 ng.l\textsuperscript{-1} calculated with this model is, however, a factor 3 lower than the 56-d LC50 (0.046 ng.l\textsuperscript{-1}) calculated by Mehrle et al. and the 56-d LC50 (0.045 ng.l\textsuperscript{-1}) calculated with the trimmed Spearman-Karber method. For this reason this calculation method has not been used.
4 The NO(A)EL\textsubscript{predator} is based on the long-term feeding studies with rats also mentioned in section 5.1 (Kociba et al., 1978; Murray et al., 1979). In these studies at doses of 10 ng 2,3,7,8-TCDD kg\textsuperscript{-1} body weight per day (200 ng.kg\textsuperscript{-1} feed) effects were observed on the reproduction, the viability of the offspring and/or the body weight. Both studies resulted in a NO(A)EL, no-observed-(adverse)-effect-level, of 1 ng.kg\textsuperscript{-1} body weight per day (20 ng.kg\textsuperscript{-1} feed). Since no useful NO(A)EL-values are available for other mammal
species, the above-mentioned NO(A)EL of 20 ng kg\(^{-1}\) feed is divided by an extrapolation factor 10 (in accordance with Romijn et al., 1991a), resulting in a NO(A)EL predator of 2 ng kg\(^{-1}\) feed.

In the food-chain poisoning method the same calculation can be made for fish-eating birds or for the combined group of birds and mammals, however, in this case no NO(A)EL-values or other useful toxicity data are available for birds.

The BCF\(_{\text{fish}}\) is based on the Mehrle et al. study (1988) with \(S. \text{gairdneri}\) mentioned before where at the lowest test concentration of 0.038 ng l\(^{-1}\) an equilibrium BCF was calculated of 39,000 and at the other concentrations equilibrium BCF-values of 38,000, 86,000 and 37,000, respectively. In addition to these values two equilibrium BCF\(_{\text{fish}}\)-values are available, one of 9,300 (\(S. \text{gairdneri}\)) and one of 7,900 (\(P. \text{promelas}\)), originating from a test in a statistical system and a semi-statistical system, respectively (see table 5.2 in Okkerman et al., 1992). All values mentioned are whole-body BCF-values, based on the fresh weight of the fish.

In the food-chain poisoning method a geometric mean BCF and a maximal BCF are used in the calculation, the result based on the geometric mean BCF being considered as MTR\(_{\text{predator}}\). For 2,3,7,8-TCDD this would result in a geometric mean BCF\(_{\text{fish}}\) of 16,000 [being the geometric mean of the value for \(P. \text{promelas}\) (7,900) and the geometric mean of the 5 values for \(S. \text{gairdneri}\) (33,500)]. However, in fact none of the available BCF-values meet all of the criteria laid down by Romijn et al. (1991a), since at all test concentrations an increased mortality percentage was observed, either during the exposure time, or during the purification period in clean water (Whether this may have led to an overestimation or an underestimation of the BCF-values, is not known). The BCF amounting to 39,000, determined at the lowest test concentration, is therefore considered to be the most reliable (also considering the fact that a continuous-flow system was used in the study). This value, rounded to 40,000, is used as BCF\(_{\text{fish}}\). In addition calculation of the geometric mean would lead to an underestimation of the BCF, due to the fact that it is highly influenced by the only (low) BCF for \(P. \text{promelas}\).

It is finally noted that the corresponding results from the two different analytical methods applied in the Mehrle et al. study show that the major part of the level measured in fish consisted of non-metabolized 2,3,7,8-TCDD, both during the 28-day exposure period and the 28-day purification period. The analytical methods used were scintillation count of \(^{3}H\) following total destruction of the fish and GC-MS analysis following extraction of \(^{3}H\)-2,3,7,8-TCDD.

NOLC for the earthworm species \(A. \text{caliginosa}\) (Reinecke and Nash, 1984). No other reliable NOEC-values nor acute L(E)C50-values are available.

The BCF\(_{\text{worm}}\) used is the geometric mean value of the CF\(_{\text{st}}\)-values. CF\(_{\text{st}}\)-values are the CF-values that are standardized to a soil with 10% organic matter:

\[
\text{CF}_{\text{st}} = \text{CF}_{\text{exp}} \times \% \text{OM/10} \quad \text{(Romijn et al., 1991b) }
\]

The experimentally determined CF-values (and therefore also the standardized CF-values) are based on the level non-metabolized 2,3,7,8-TCDD in worms (on a fresh-weight basis), determined with GC or GC-MS. The CF-values originate or are determined from the studies by Nash and Beall (1980), Reinecke and Nash (1984) and Heida et al. (1986); both the CF\(_{\text{exp}}\)- and the CF\(_{\text{st}}\)-values are given in table 5.7 in Okkerman et al. (1992).
**Annex 1**  Comparative activities of 2,3,7,8-substituted dibenzo-p-dioxins and dibenzofurans in relation to the standard 2,3,7,8-TCDD (Van Zorge et al., 1989)

<table>
<thead>
<tr>
<th>Compound</th>
<th>Terat.</th>
<th>Carc.</th>
<th>ED50</th>
<th>DES50</th>
<th>ED50</th>
<th>LD50</th>
<th>LD50</th>
<th>LD50</th>
<th>ED50</th>
<th>ED50</th>
<th>AHH</th>
<th>EROD</th>
<th>Cell</th>
</tr>
</thead>
<tbody>
<tr>
<td>2,3,7,8-TCDD</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>1,2,3,7,8-PeCDD</td>
<td>0.4</td>
<td>0.081</td>
<td>0.529</td>
<td>0.64</td>
<td>0.59</td>
<td>0.31</td>
<td>0.129</td>
<td>0.007</td>
<td>0.011</td>
<td>0.5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1,2,3,4,7,8-HxCDD</td>
<td>0.031</td>
<td>0.084</td>
<td>0.03</td>
<td>0.24</td>
<td>0.036</td>
<td>0.133</td>
<td>0.034</td>
<td>0.045</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1,2,3,6,7,8-HxCDD</td>
<td>0.03</td>
<td>0.16</td>
<td>0.0006</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>1,2,3,7,8,9-HxCDD</td>
<td>&lt;0.14</td>
<td></td>
<td>0.0023</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
<td>0.005</td>
<td></td>
</tr>
<tr>
<td>1,2,3,4,6,7,8-HpCDD</td>
<td>&lt;0.003</td>
<td></td>
<td>&lt;0.0001</td>
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<td>&lt;0.001</td>
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<td>OCDD</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2,3,7,8-TCDF</td>
<td>0.049</td>
<td>0.016</td>
<td>0.025</td>
<td>0.3</td>
<td>&lt;0.03</td>
<td>&lt;0.03</td>
<td>0.1-0.01</td>
<td>0.006</td>
<td>0.006</td>
<td>0.018</td>
<td>0.092</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>1,2,3,7,8-PeCDF</td>
<td>0.026</td>
<td>0.01</td>
<td>0.019</td>
<td>0.051</td>
<td></td>
<td>0.04</td>
<td>0.003</td>
<td>0.002</td>
<td>0.028</td>
<td>0.060</td>
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<tr>
<td>2,3,4,7,8-PeCDF</td>
<td>0.095</td>
<td>0.4</td>
<td>0.048</td>
<td>0.429</td>
<td>&gt;0.2</td>
<td>0.38</td>
<td>0.108</td>
<td>0.167</td>
<td>0.282</td>
<td>1.381</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1,2,3,4,7,8-HxCDF</td>
<td>0.010</td>
<td>0.038</td>
<td>0.180</td>
<td></td>
<td></td>
<td>0.031</td>
<td>0.014</td>
<td>0.012</td>
<td>0.203</td>
<td>0.488</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1,2,3,6,7,8-HxCDF</td>
<td>0.1</td>
<td>0.016</td>
<td>0.097</td>
<td></td>
<td></td>
<td>0.04</td>
<td>0.012</td>
<td>0.014</td>
<td>0.049</td>
<td>0.149</td>
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</tr>
<tr>
<td>1,2,3,7,8,9-HxCDF</td>
<td>0.0004</td>
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</tr>
<tr>
<td>2,3,4,6,7,8-HxCDF</td>
<td>0.018</td>
<td>0.097</td>
<td>0.02</td>
<td></td>
<td>0.0071</td>
<td>0.015</td>
<td>0.014</td>
<td>0.105</td>
<td>0.322</td>
<td></td>
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</tr>
<tr>
<td>1,2,3,4,6,7,8-HxCDF</td>
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</tr>
<tr>
<td>1,2,3,4,7,8,9-HpCDF</td>
<td></td>
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<tr>
<td>OCDF</td>
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</tr>
</tbody>
</table>

(1) Teratogenicity mouse; (2) Carcinogenicity rat; (3) ED50 13-week rat; (4) ED50 weight loss rat; (5) Thymus atrophy rat; (6) LD50 guinea pig; (7) LD50 mouse; (8) LD50 rat; (9) LD50 embryo rainbow trout; (10) ED50 induction BaP hydroxylase rat; (11) ED50 4-CH hydroxylase; (12) AHH induction in vitro; (13) EROD induction in vitro; (14) cell keratinization.
### Annex 2a: Toxicity tests with 2,3,7,8-TCDD (freshwater organisms)

<table>
<thead>
<tr>
<th>Species (Order/Family)</th>
<th>A/N</th>
<th>Test type</th>
<th>Test compound</th>
<th>Test water</th>
<th>pH</th>
<th>Hardness (mg CaCO₃/l)</th>
<th>Exposure/recovery (d)</th>
<th>Results (ng/l)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oligochaeta (Annelida)</td>
<td>N</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>spring water, sol. in acetone (0.3 ml/l)</td>
<td>6.9</td>
<td>64</td>
<td>55/0</td>
<td>NOEC₉₅</td>
<td>200</td>
</tr>
<tr>
<td>Mollusca (Mollusca)</td>
<td>N</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>spring water, sol. in acetone (0.3 ml/l)</td>
<td>6.9</td>
<td>64</td>
<td>36/12</td>
<td>NOEC₉₅</td>
<td>200</td>
</tr>
<tr>
<td>Ceratostoma (Hexapoda, Arthropoda)</td>
<td>A</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>spring water, sol. in acetone (&lt;0.1 ml/l)</td>
<td>6.8-7.7</td>
<td>110</td>
<td>2/7</td>
<td>NOLC &gt;1.030</td>
<td>Adema, 1988 [8]</td>
</tr>
<tr>
<td>Daphnia magna (3 age groups)</td>
<td>A</td>
<td>R (3 d)</td>
<td>2,3,7,8-TCDD</td>
<td>DSW, sol. in DMSO</td>
<td>8.2</td>
<td>210</td>
<td>21/0</td>
<td>NOEC &gt;10</td>
<td>Adema, n.p. [9,10]</td>
</tr>
<tr>
<td>Aedes aegypti (larvae)</td>
<td>N</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>spring water, sol. in acetone (0.3 ml/l)</td>
<td>6.9</td>
<td>64</td>
<td>17/23</td>
<td>NOEC₉₅ &gt;200</td>
<td>Miller, 1973 [1]</td>
</tr>
<tr>
<td>Fishes (Pisces, Vertebrata)</td>
<td>N</td>
<td>R (4 d)</td>
<td>2,3,7,8-TCDD</td>
<td>tape water, sol. DMSO+acetone</td>
<td>6.9</td>
<td>94-102</td>
<td>4/19</td>
<td>NOEC₉₅</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Jordanella floridana (egg -&gt; larvae)</td>
<td>A</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>DSW, sol. in hexane+DMSO</td>
<td>8.2</td>
<td>210</td>
<td>4/100</td>
<td>NOEC₉₅&lt;0.1</td>
<td>Adema, n.p. [9]</td>
</tr>
<tr>
<td>Jordanella floridana (egg -&gt; larvae)</td>
<td>A</td>
<td>R (1 d)</td>
<td>2,3,7,8-TCDD</td>
<td>DSW, sol. in hexane+DMSO</td>
<td>8.2</td>
<td>210</td>
<td>4/100</td>
<td>NOEC₉₅&lt;0.1</td>
<td>Adema, n.p. [9]</td>
</tr>
<tr>
<td>Daphnia magna (35 d)</td>
<td>A</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>DSW, sol. in hexane+DMSO</td>
<td>8.2</td>
<td>210</td>
<td>4/100</td>
<td>NOEC₉₅&lt;1</td>
<td>Adema, n.p. [9]</td>
</tr>
<tr>
<td>Onorhynchus kiusutch (larvae)</td>
<td>N</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>spring water, sol. in acetone (0.3 ml/l)</td>
<td>6.9</td>
<td>64</td>
<td>4/65</td>
<td>NOLC &lt;0.058</td>
<td>Miller, 1973 [2,19]</td>
</tr>
<tr>
<td>Oryzias latipes (egg -&gt; larvae)</td>
<td>N</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>artificial water sol. in nonane + acetone</td>
<td>-</td>
<td>185</td>
<td>14/0</td>
<td>NOEC₉₅ &gt;2</td>
<td>Wask, 1990a [12]</td>
</tr>
<tr>
<td>Oryzias latipes (egg -&gt; larvae)</td>
<td>A</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>artificial water sol. in nonane + acetone</td>
<td>-</td>
<td>185</td>
<td>14/0</td>
<td>NOEC₉₅&lt;0.4</td>
<td>Wask, 1990a [8,12,13,17]</td>
</tr>
<tr>
<td>Oryzias latipes (egg)</td>
<td>A</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>artificial water sol. in nonane + acetone</td>
<td>-</td>
<td>185</td>
<td>11/0</td>
<td>NOEC₉₅&lt;0.5</td>
<td>Wask, 1990b [8,12,13,18]</td>
</tr>
<tr>
<td>Pimephales promelas (larvae)</td>
<td>A</td>
<td>R (4 d)</td>
<td>2,3,7,8-TCDD</td>
<td>spring water, sol. in acetone (&lt;0.1 ml/l)</td>
<td>7.3</td>
<td>110</td>
<td>4/60</td>
<td>NOLC 0.7</td>
<td>Adema, 1988 [2,8]</td>
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<tr>
<td>Pimephales promelas (juvenile)</td>
<td>A</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>spring water, sol. in acetone (&lt;0.1 ml/l)</td>
<td>7.3</td>
<td>110</td>
<td>28/0</td>
<td>LC₅₀ 1.7</td>
<td>Adema, 1988 [2,8]</td>
</tr>
<tr>
<td>Poecilia reticulata (9 - 40 mm)</td>
<td>N</td>
<td>S</td>
<td>2,3,7,8-TCDD</td>
<td>spring water, sol. in chloroform + acetone</td>
<td>-</td>
<td>-</td>
<td>5/32</td>
<td>LC₅₀ 100</td>
<td>Norris, 1974 [3]</td>
</tr>
<tr>
<td>Salmo gairdneri</td>
<td>N</td>
<td>R (1 d)</td>
<td>2,3,7,8-TCDD</td>
<td>tape water, sol. in acetone</td>
<td>7.5-7.9</td>
<td>50-87</td>
<td>4/17</td>
<td>NOLC 1</td>
<td>Bol, 1989 [2,5]</td>
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<tr>
<td>Species</td>
<td>(larva)</td>
<td>(eggs)</td>
<td>Concentration</td>
<td>溶剂</td>
<td>pH</td>
<td>LC50</td>
<td>NOEC</td>
<td>NOECd</td>
<td>NOECsm</td>
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</tr>
<tr>
<td>Salmo gairdneri</td>
<td>N</td>
<td>R (1 d)</td>
<td>2,3,7,8-TCDD (≥98%)</td>
<td>tape water, in DMSO + acetone</td>
<td>7.4-7.5</td>
<td>96-105</td>
<td>4/164</td>
<td>NOECg &lt;0.1</td>
<td>NOECsm 0.1</td>
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<tr>
<td>Salmo gairdneri</td>
<td>N</td>
<td>R (1 d)</td>
<td>2,3,7,8-TCDD (98.6%)</td>
<td>tape water, in DMSO + acetone</td>
<td>7.4-7.5</td>
<td>96-105</td>
<td>4/133</td>
<td>NOECg &lt;1</td>
<td>NOECsm &lt;1</td>
</tr>
<tr>
<td>Salmo gairdneri</td>
<td>N</td>
<td>R ?</td>
<td>2,3,7,8-TCDD (99.5%)</td>
<td>dechlorinated tape water sol. in DMSO + acetone</td>
<td>7.4-7.5</td>
<td>96-105</td>
<td>4/16</td>
<td>NOECg 1.6</td>
<td>NOECsm 1.0</td>
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<tr>
<td>Salmo gairdneri</td>
<td>A</td>
<td>F</td>
<td>2,3,7,8-TCDD (≥99%)</td>
<td>fresh water, sol. in acetone ((≤0.05 \text{ml} l^{-1}))</td>
<td>7.7</td>
<td>153</td>
<td>28/28</td>
<td>NOECg &lt;0.038</td>
<td>NOECsm 0.046</td>
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### Annex 2b  
Toxicity tests with PCDDs (freshwater organisms)

<table>
<thead>
<tr>
<th>Species (Phyto, Vertebrata)</th>
<th>A/N</th>
<th>Test type</th>
<th>Test compound</th>
<th>Test water</th>
<th>pH</th>
<th>Hardness (mg CaCO₃/l)</th>
<th>Exposure Criteria (days)</th>
<th>Concentration (ng/l)</th>
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Explanations Annexes 2a-c

**Organism (1st column)**
- *: Life stage exposed
- P --> F: 2-Generation study (parent generation -- > filial generation); effects studied on (e.g. reproduction)

**Analysis test concentrations (2nd column)**
- A: Analysis of exposure concentration (analysed/actual concentration)
- N: No analysis of exposure concentration (nominal concentration)

**Test type (3rd column)**
- F: Flow-through/continuous-flow system
- R: Semi-static test system where the test medium is frequently renewed (see interval indicated) (renewal system)
- S: Static test system

**Test water (5th column)**
- DSW: Dutch Standard Water
- Art. w: Artificial water

**Expo-/depuration (8th column)**
- Exposure time (days) at exposure concentration indicated followed by depuration time (days) in clean water (exposure time / depuration time). Effects studied to end of depuration time.

**Criteria (9th column)**
- LC50: Concentration, calculated from concentration-effect relation, to cause mortality in 50% of the organisms exposed to this concentration (median lethal concentration, i.e. that concentration which is calculated [from a number of test concentrations] to cause mortality of 50% of the number of organisms exposed to that concentration).
- EC50: Concentration, calculated from concentration-effect relation, to cause the effect indicated in 50% of the organisms exposed to this concentration (median effect concentration, i.e. that concentration which is calculated [from a number of test concentrations] to cause a particular response in 50% of the number of organisms exposed to that concentration).
- NOEC: Highest concentration in the test range used to cause no effect in relation to the effect parameter indicated (no-observed-effect-concentration).
- NOLC: Highest concentration in the test range used to cause no mortality (no-observed-lethal-concentration).
- <: Concentration indicated is the lowest in the range used.
- >=: Concentration indicated is the highest in the range used.

**Effect parameters:**
- d = developmental rate embryo’s
- g = growth
- h = % hatching
- l = severe, life-threatening lesions including vascular hemorrhaging and pericardial edema
- m = morphological anomalies which are not life-threatening, such as pugheadedness and protruding eyes, and other anomalies not described.
- p = total or rate of pupation
- r = reproduction
- s = survival

**Additional:**
- n.p. = not published

**Footnotes:**
1. Only one concentration tested.
2. Depuration phase in continuous-flow system with clean water.
3. Depuration phase in renewal system with clean water.
4. For 1,2,3,7,8-PeCDD; 2,3,4,7,8-PeCDF and 1,2,3,7,8,9-HxCDF TEF-values (in relation to 2,3,7,8-TCDD) of 0.77, 0.35 and 0.006, respectively, are determined by Bol et al. (1988) based on the parameter 'survival'. The NOLC values indicated for these compounds are calculated from the NOLC of 2,3,7,8-TCDD for *S. gairdneri* (Bol et al., 1989, annex 2a) and the TEF values indicated, as follows: NOLC compound "X" is NOLC 2,3,7,8-TCDD divided by TEF compound "X".
5. Exposure of yolk-sac fry.
7. In this test a consistent effect of reproduction was observed, which, however (through the great variance in the replicas) was not statistically significant.
9. Concentrations indicated are estimated based on analysis of the highest test concentration (about a factor 6 lower than the nominal concentrations).
10. Test medium renewed at days 3, 6, and 9, however, not at the remaining 12 days of exposure.
11. Exposure of the eggs during the entire embryonic phase and subsequently of the larvae up to 14 days after hatching. At concentrations from 0.1 ng.l$^{-1}$ the growth of the larvae was significantly retarded through day 21, but at day 23 only at 10 ng.l$^{-1}$.
12. Exposure of the eggs during the entire embryonic phase and subsequently of the larvae until 3 days after hatching.
13. Only initial concentrations measured.
14. Exposure of recently fertilized eggs. Developmental rate embryos not affected, however, immediately following hatching the larvae were smaller and less pigmented than the controls. The growth of the fishes was significantly retarded at day 72, however, not so in the following period.
15. Weekly (radiometric) analysis of all test concentrations; the background concentration in the water used was 1 pg.l$^{-1}$. Of the nominal concentrations only 33% to 43% was recovered. The cumulative mortality at the end of the purification period was 7% (control), 45%*, 83%* and 95%* (* = P < 0.05), at concentrations of 0, 0.038, 0.079 and 0.176 ng.l$^{-1}$, respectively. The cumulative mortality at the two highest concentrations was extremely high during the exposure period (73%* and 85%* at concentrations of 0.382 and 0.798 ng.l$^{-1}$, respectively), therefore these exposed groups were discarded before the purification period. At the end of the purification period the percentage of growth retardation in the lowest exposure group was 49% (P < 0.05); at this stage the growth was not measured in the remaining exposure groups.
16. Weekly (GC-MS) analysis of all test concentrations; the background concentration in the water used was <0.02 ng.l$^{-1}$. Of the nominal concentrations only 32% to 41% was recovered.
17. Marginal effect (10%, 1 of the 10 exposed embryos, versus 0% in the controls) with respect to parameters "l" (life-threatening lesions) and "s" (survival) at 0.4 ng.l$^{-1}$. Distinct effects (50% versus 10% in the controls) with respect to the parameter "m" (morphological anomalies, not life-threatening) at 0.4 ng.l$^{-1}$.
18. In this test no distinction was made between the parameters "l" and "m".
19. The cumulative mortality at the end of the purification period was 2% (control), 12%, 14%, 55% and 100% at exposure concentrations of 0, 0.056, 0.56, 5.6 and 56 ng.l$^{-1}$, respectively. The average survival time decreased significantly (P<0.01) with increasing exposure concentrations.
6. EMISSION REDUCTION MEASURES

Measures concerning reduction of dioxin emission to air, water and soil will be discussed in this chapter. The same classification of processes will be used as in chapter 2.

Studies on the influence of the process conditions during waste incineration and literature studies have shown that through optimization of the process conditions and improvement of the management the dioxin emission may be reduced (Sein et al., 1989). Such a reduction is not only possible for new installations, but also for existing installations, following possible technical adjustments. For existing installations the conditions from the Guideline Incineration 1989 are still being applied. No limit values for dioxins are mentioned in this guideline. Based on the better understanding of the incineration process and the state of the art of technology concerning the purification of flue gases the emission limit values of the existing guideline were made more stringent in 1989, and for a number of other compounds, including dioxins, limit values were also determined. The limit value for dioxins in the Guideline Incineration 1989 is put at 0.1 ng TEQ.m$^{-3}$ flue gas (und, 11% oxygen) (Nijpels, 1989). The Guideline Incineration 1989 (GI89) has immediately come into effect for new installations. For existing installations the required limit values are to be met as per 30 November 1993. An implementing regulation is in preparation. The GI89 is applied for the incineration of municipal and similar industrial waste. This guideline should be applied by the licensing authorities for the incineration of other relevant waste combinations or categories, such as incineration of chemical waste, purification sludge, cable waste, contaminated soil, that need to be thermally processed.

In the NER (DEG), de Nederlandse Emissie Richtlijn (the Dutch Emission Guideline), dioxins are considered as 'extremely hazardous compounds', for which minimalization is required. For compounds like these a zero emission should be aimed at. For as long as the emissions are not eliminated the relevant emission requirements of the DEG will remain effective.

In the production of pig iron 0.1 ng TEQ.m$^{-3}$ und, (16% O$_2$) needs to be considered as a guideline for the sinter machines. In wire reclamation incinerators and similar installations, for which the Guideline Incineration 1989 does not apply, the emission requirement of 0.1 ng TEQ.m$^{-3}$ und (6% O$_2$) holds. For installations for thermal purification of the soil the same emission requirement applies, however in this case at 11% oxygen. For iron/steel foundries/ smelters the raw materials should contain as little as possible chlorine compounds
to prevent dioxin formation.
In a considerable number of processes other than WIs the extent of dioxin emission is currently being studied. The choice of the processes to be studied is made based on data mentioned in chapter 2. These studies will show to what extent emission reduction measures are required for these processes.
Since the developments concerning the implementation of emission reduction measures are in a very early stage, no distinction has been made between autonomous developments and additional measures. Only the Guideline Incineration 1989 and the increased use of unleaded petrol may be considered as autonomous development. In each section it is mentioned to what extent emission control should take place when the Guideline Incineration 1989 comes into effect.

6.1 EMISSION CONTROL BY SOURCE

6.1.1 Waste incinerators

The annual emission to air in the operational waste incinerators (WIs) in 1990 amounted to appr. 400 g I-TEQ. If, after the GI89 has come into effect, the same amount of waste is incinerated, the annual emission to air should be reduced to less than 2 g I-TEQ.
The amount of waste is ever increasing. The policy is aimed at reducing the growth, by prevention and reusage. Since only a limited space is available for the dumping of waste, the waste still to be removed should mainly be incinerated. The amount of waste to be incinerated in the year 2000 is expected to increase to appr. 7 million tonnes (RIVM, 1991), resulting in a dioxin emission of maximally 4 g I-TEQ per year, being only 1% of the current emission. In order for these low emissions to be realized a great deal of effort will be required. In addition to lower emissions to air, lower dioxin levels in the solid purification residues should also be aimed at.
The measures to be taken will be aimed at:
- Reduction of dioxin formation.
- Control of dioxin emission to air by using additional flue gas purification systems.
- Processing of purification residues with the aim of destroying or removing the dioxins present.
Reduction of dioxin formation

The most probable mechanism of dioxin formation is de-novo synthesis, where the hydrochloric acid originating from various chlorine sources reacts catalytically with products from incomplete incineration, see 2.1.1. This implies that the combustion process needs to take place under optimal conditions, in order to keep the concentration of incompletely incinerated products as low as possible. In accordance with the GI89 the flue gas temperature in the furnace should be at least 850°C at a minimum oxygen content of 6% and a minimum residence time of the flue gases of 2 seconds. The distribution of oxygen over the fire and the mixing of combustion gases with air are of great significance so that at all sites in the furnace complete combustion may take place. The furnace design, the state of maintenance and especially also the process control play a major role. Dramatic adjustments of the existing furnace will sometimes be required to optimize the combustion process. In new installations, built using the most recent technologies, the dioxin formation in the furnace may even be reduced to 1 ng I-TEQ.m^{-3} flue gas (Hagenmaier, 1990).

Reduction of the hydrochloric acid level in flue gas, by removing a chlorine source in waste, should lead to a decreased dioxin formation. The amount of chlorine present in municipal waste is on average about a million times higher than that required for the formation of dioxins. With 50-70% PVC is the major chlorine source in municipal waste, in addition much chlorine occurs in vegetable waste (vegetable-, fruit- and garden waste). By reducing one or more chlorine sources in waste the hydrochloric acid emission may be reduced, however, this does not mean that insufficient hydrochloric acid would be present for the formation of dioxins. Practical experiments with low PVC concentrations in waste are difficult to carry out. However, in order to demonstrate a possible relation between the amount of hydrochloric acid and dioxin emission, experiments have been carried out with a chlorine content increased by PVC addition.

In the 'Study emission incinerator furnaces' (Sein et al., 1989), at a double amount of PVC in waste, a higher chlorophenol as well as an increased dioxin concentration was observed. A quantitative relation between the hydrochloric acid concentration and dioxin emission has not been found. Similar results are mentioned in the literature (Visally, 1987; Sierig, 1989; Caroll, 1988; Vikelsoe, 1989). Some scientists consider the increased concentrations not to be significant. At the university of Leyden laboratory studies have been carried out with dried and ground fractions of municipal waste. These experiments showed chlorophenol to
be an intermediary for dioxin formation in relation to the hydrochloric acid concentration (Kanters et al., 1992). This study also shows there is hardly any influence on the chlorophenol concentration at higher or lower PVC amounts in the waste. Only when the oxygen concentration becomes lower than appr. 0.15 g.m\(^{-3}\), that is 20% of the average level in a WI with no additional flue gas purification, does the chlorophenol concentration begin to decrease. The decreasing amount of common salt in municipal waste, through the separate collection of vegetable waste (vegetable-, fruit- and garden waste), will for the same reasons only slightly affect the dioxin emission. However the drier residue fraction is expected to make possible a more regular combustion. Injection of lime, ammonia or amines before the E-filter is applied to bind the hydrochloric acid formed, and to inactivate the fly ash. In a number of Dutch WIs lime injection is applied. A limited decrease of hydrochloric acid occurs, however, insufficient to result in a lower dioxin concentration. Since dioxin formation is optimal at 200-450\(^\circ\)C the formation reaction may also be influenced by a decrease in the temperature before the E-filter. This is applied in the WIR (Waste Incinerator Rijnmond) by constructing a low pressure boiler, resulting in a decrease of the temperature at the E-filter from appr. 300\(^\circ\)C to appr. 190\(^\circ\)C. The possibility of separating the dust at temperatures between 400\(^\circ\)C and 500\(^\circ\)C is also studied.

**Reduction of emissions to air**

In the case of dioxins being formed the emission to air may be reduced by the application of additional flue gas purification systems. In this field many developments have taken place over the last few years. Since dioxins are mainly bound to fly ash, a proper dust trapping is very important. In the GI89 a standard for dust emission of 5 mg.m\(^{-3}\) is included, which for some WIs is a factor 20 lower than the amount currently emitted. Studies are also carried out on purification systems where dioxins are decomposed.

The most important purification systems that are applied or studied, in addition to an E-filter or a fabric filter, are:

- wet or dry gas washing with lime,
- adsorption on active carbon or coke,
- removal with an electrodynamic venturi (will be used in Amsterdam)
- oxidation on catalysts combined with selective katalytic reduction for denitrification (NOx-prevention),
Chemical oxidation (being studied), for instance by means of injection of hydrogen peroxide in the flue gases,
- chemosorption on silicon-aluminum sorbents (also in an experimental stage)

Studios are carried out in WIs on the economical and technical aspects in the application of these systems or combinations thereof. In these studies attention is not only being paid to the reduction of dioxin emissions, but also to the other compounds the emission of which should be reduced according to the GI89.

Processing of purification residues

As long as dioxins are formed and not decomposed, advanced technologies may limit the emission, however, the problem of contaminated residues will remain. It is estimated that in E-filter ash appr. four times as much dioxins will be removed than in flue gases (see 2.2.1). In case dioxin formation can be limited the concentration in the residues will also decrease.

In order to improve the applicability of slag and E-filter ash as secondary raw materials or to prevent the risks of long-term monitoring of waste tips, the possible processing of these residues is being studied. Positive results have already been obtained abroad. The most important items to be studied are:
- dechlorination, whereby E-filter ash is treated at higher temperatures
- combustion, by feeding the E-filter ash in pellets to the furnace again, together with the waste
- combustion following extraction, whereby the acid water of the wet purification is used for the extraction of heavy metals, subsequently the remaining filtercake is pelleted and combusted,
- vitrification in an electric smelter furnace at 1300°C, whereby dioxins are decomposed and metal vapours are trapped; the glass-like residual product can be used in road construction.

6.1.2 Incineration of chemical waste

The Guideline Incineration 1989 applies to the incineration of chemical waste. In the year 2000 an estimated amount of 270,000 tonnes of chemical waste will be removed by companies and incinerated by others (RIVM, 1991). A number of companies apply on-site
incineration of chemical waste. Legally, for instance in the Chemical Waste Act, these compounds are not considered as waste. Strictly speaking these companies do not have to meet the Guideline Incineration 1989. It is assumed that for these installations in the course of the 90s in another framework, for instance via the Nuisance Act, requirements will have to be put up similar to the Guideline Incineration 1989.

In industry there are plans for the near future for on-site incineration of chemical or industrial waste. This appeared from the many applications for permissions (Bremmer and Hesseling, 1991). The amount of chemical waste which will be incinerated on-site by the companies in the year 2000 is estimated at 100,000 tonnes per year (RIVM, 1991). Assuming a dioxin emission of 0.1 ng I-TEQ.m\(^{-3}\) and an annual amount of 370,000 tonnes of chemical waste, which is incinerated on-site by the companies or by others, a dioxin emission of appr. 0.2 g I-TEQ per year is expected for the year 2000.

In order to meet the Guideline Incineration 1989 additional flue gas purification will be required as in WIs. When wet gas washing is being applied (particularly to reduce emission of dust and/or hydrochloric acid) a slight emission to water may occur.

For E-filter ash, as in WIs, a considerable decrease in the dioxin concentrations per weight may occur for the same reasons as described for WIs.

6.1.3 Rubbish tip gas

It is estimated that by the year 2000 an annual amount of 260,000 tonnes of purification sludge will be incinerated (RIVM, 1991). Incinerators for purification sludge need to meet to the Guideline Incineration 1989. The maximal dioxin emission of 0.1 ng I-TEQ.m\(^{-3}\) und flue gas results in an emission of 0.13 g I-TEQ per year.

The survey (Bremmer and Hesseling, 1991) showed that for 5 - 10 rubbish tips there are plans to capture the rubbish tip gas. Assuming a doubling of the combustion of rubbish tip gas or similar gas in the year 2000 and an emission of 0.1 ng I-TEQ.m\(^{-3}\) und flue gas, which under optimal combustion conditions in the gas engine seems feasible, an emission of 0.1 g I-TEQ per year may be expected.

The amount I-TEQ in municipal purification sludge, of which it is assumed that it is mainly caused by atmospheric deposition, will as a result of emission reduction decrease from 20 g I-TEQ in 1990 to an estimated 7 g I-TEQ in 2000, and the municipal discharges from appr. 13 to appr. 5 g I-TEQ (at an equal contribution from abroad).
As a result of the additional measures concerning the sewer system (including restriction overflow) the municipal discharges will decrease to appr. 3 g I-TEQ.

6.1.4 Pesticides

In the long-range plan crop protection (Gabor, 1991), it is indicated that the use of agricultural pesticides in the year 2000 needs to be reduced by 50% in relation to that in 1985. The dioxin contamination in pesticides is assumed to further decrease. It is estimated that in 2000 pesticides will be contaminated with a total of 0.2 g I-TEQ.

The amount of PCP-preserved wood will among other things decrease through demolition of houses. Importation of PCP-preserved wood and wood products will decrease since the use of PCP as a wood preservative will be prohibited in more and more countries.

The total amount of PCP accumulated in wood reached an all-time high by 1985 (Slooff et al., 1990; Haskoning, 1989). The emission of PCP from wood also shows a decrease (Haskoning, 1989). Based on physico-chemical properties the evaporation of dioxins from preserved wood is expected to occur more slowly than that of PCP. It may even be questioned whether the evaporation of dioxins already shows a decrease. Therefore, the evaporation of dioxins from PCP-preserved wood from 1990 to 2000 may only slightly decrease. The evaporation for 2000 is estimated at an annual 42 g I-TEQ.

The application of PCP as a fungicide in pesticides and PCP-laureate as a fungicide and antiprutification in textiles and sponges will probably be prohibited very shortly. In Germany the use of PCP-laureate as a fungicide and as antiprutification is not allowed any longer.

6.1.5 Metal industry

At Hoogovens (blast furnaces) extensive studies are currently being carried out on dioxin emission. The flue gas purification in the sinter plant has been improved. The dioxin emission will decrease considerably because of these improvements.

In the non-ferro industry the dioxin emission will be reduced, by using raw materials with a lower chlorine content, as well as by adjusting the process conditions, and by installing a better flue gas purification. It is estimated that the dioxin emission in 2000 by the metal industry will amount to appr. 7 g I-TEQ/year.
6.1.6 Cable burning

Wire reclamation incinerators need to meet the Guideline Incineration 1989 as per 30 November 1993. Since adjustments of the existing installations will probably involve very high costs these will not be cost-effective for most of these installations.

Due to high emissions of lead, copper and PAHs, the Province of Gelderland started a procedure by the end of 1990 for closing down two wire reclamation incinerators. It is to be expected that these installations shortly have to meet the Guideline Incineration 1989 or will have to be closed down. For another Gelder wire reclamation incinerator the contract for the processing of cables had expired as from 1 January 1992. At present no cables are being burned off here.

The wire reclamation incinerators in Gelderland have since the beginning of 1991 only used a small part of their capacity. It is estimated that in the first half of 1991 appr. one third of the amount of cable was burnt off compared as to the amount in the same period in 1990. In the survey (Bremmer and Hesseling, 1991) one company appeared to be able to shred underground cables with electric insulation of paper, jute and bitumen instead of burning them off. Shredding of underground cables is already applied in practice. The bituminous mass, which in addition to the metals lead and copper is released, is dumped. This bituminous mass, based on its contents of PAHs, may probably be chemical waste. Within the Ministry of VROM (Housing, Regional Development and the Environment) a policy is currently being developed concerning the removal of cable waste. The shredding of underground cable will probably be applied more widely. One or several companies may remain using the process of burning off. Assuming a dioxin emission of 0.1 ng I-TEQ.m\(^{-3}\) und flue gas, an emission of less than 0.1 g I-TEQ per year is expected.

Illegal burning off of cables will probably decrease. In the years to come companies will need to be more careful in the selling of cable waste. The government will be able to better control the flows of cable waste and the import and export of it. The dioxin emission in the illegal burning off of cables is estimated at 2 g I-TEQ per year for the year 2000.

6.1.7 Traffic

For the year 2000 road traffic is estimated to cover appr. 80*10\(^9\) km with unleaded petrol, 2*10\(^9\) km with leaded petrol and appr. 19*10\(^9\) km with diesel (RIVM, 1991). Assuming the
dioxin emission per km to be as estimated in chapter 2 for the current situation, this means an emission of appr. 2 g I-TEQ per year in 2000.

6.1.8 Hospital waste incinerators

The survey (Bremmer and Hesseling, 1991) showed a clear tendency to close down installations and to have specific hospital waste burnt somewhere else. At the end of 1991 an installation was put into use at the site of the Dordrecht WI for the purpose of incinerating specific hospital waste (de ZAVIN= de Ziekenhuis Afval Verbrandings Installatie Nederland) (the NHWI= the Netherlands Hospital Waste Incinerator). It is expected that in 2000 all small installations will be closed down and specific waste will be incinerated in one or more specialized installations, which should meet the Guideline Incineration 1989. The dioxin emission is lower than 0.1 g I-TEQ per year.

6.1.9 Chemical production processes

In chemical production processes where dioxin emission may (possibly) occur, studies are or will be carried out in order to obtain a better understanding of the emission. Studies are mainly carried out in big companies. It is assumed that for processes with a considerable dioxin emission, emission reduction measures will be taken. The dioxin emission in 2000 is expected to amount to appr. 3 g I-TEQ per year.

6.1.10 Incineration of waste oil

At present no more permissions are granted for the internal use of non-processed waste oil for the heating of working accommodations. Therefore the incineration of waste oil will decrease. No statement can be made on the use of processed waste oil in the near future. It is, for the time being, assumed that the dioxin emission through the incineration of waste oil will remain at the same level. Thus the dioxin emission in the year 2000 will remain at the level of 2 g I-TEQ per year.
6.1.11 Incineration of wood

In view of the limited data only a rough estimation can be made for the dioxin emission caused by the incineration of wood. Further studies on the extent of emissions is required. Emission reduction might take place by providing information to companies and private individuals on compounds that should not be used as fuel and on the combustion conditions. The dioxin emission in the year 2000 is assumed to amount to 10 g I-TEQ per year.

6.1.12 Fires

There is no reason to assume that the dioxin emission in fires will change in the near future. Concerning open fires where chlorine containing materials such as PVC are incinerated further studies on dioxin emissions are required. Open fires, particularly in rural areas by private individuals and companies, may be reduced by provided information and by better controls. It is assumed that the dioxin emission in the year 2000 caused by fires will decrease to appr. 7 g I-TEQ per year.

6.1.13 Various processes

In a number of the 'various processes' where dioxin emission is expected, studies are or will be carried out to get a better understanding of the emission. In processes with considerable dioxin emission it is assumed that emission control measures are or will be taken. Assuming the dioxin emission in various processes in the year 2000 to be about half that in 1990, the dioxin emission will be estimated to be appr. 8 g I-TEQ per year.

6.2 EMISSIONS TO WATER IN 1985 AND 1995

In the framework of the Rhine and North Sea Action Programmes (RAP/NAP) it is agreed by the participating countries, including the Netherlands, to reduce the input of dioxin in the Rhine and the North Sea by 70% or more in the period of 1985-1995. In order to be able to judge whether the agreements in the framework of the Rhine and North Sea Action Programmes are observed, the emissions to water in the years 1985 and 1995 can be estimated, based on the estimates of the dioxin emission to water for the years 1990 and
2000.

In tables 6.1 and 6.2 estimates of the emissions and surface water loads are given as calculated with the STRAVERA model (Quarles van Ufford and Ross, 1991). For a comparison the emissions to soil and air are also indicated in these tables. For 1985 the same emissions have been used as in 1990, with the exception of the emission from WIs, for which an emission is used of 637 g I-TEQ (Matthijsen and Scheffer, 1992). Although the data are uncertain, an additional emission of 2 g I-TEQ is used for the emission to water for the situation in 1985. This partly originated from a few companies in the chemical industry. These emissions have meanwhile either been reduced or terminated (background data: Coppoolse and Kersten, 1992).

### Table 6.1
**Estimated dioxin emission to soil, water (gross discharges for purification in waste water treatment plant) and air for 1985, situation end 1990, and in 1995 and 2000 with measures implemented and additional measures (in g I-TEQ per year)**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>soil</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>0.2</td>
</tr>
<tr>
<td>water</td>
<td>6</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>air</td>
<td>840</td>
<td>610</td>
<td>123</td>
<td>125</td>
<td>100</td>
</tr>
</tbody>
</table>

### Table 6.2
**Estimated load of the fresh surface water (appr. 3700 km²) with dioxin (g I-TEQ year⁻¹) for 1985, end 1990, and the situation to be expected in 1995 and 2000 with measures implemented and with additional measures in the Netherlands (2000+) and 70% reduction air emissions abroad in 2000 (2000 max.)**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct discharges</td>
<td>6</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Deposition</td>
<td>37</td>
<td>30</td>
<td>14</td>
<td>14</td>
<td>13</td>
</tr>
<tr>
<td>Municipal</td>
<td>18</td>
<td>13</td>
<td>4</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>61</strong></td>
<td><strong>47</strong></td>
<td><strong>22</strong></td>
<td><strong>23</strong></td>
<td><strong>20</strong></td>
</tr>
<tr>
<td>In sludge waste</td>
<td>21</td>
<td>20</td>
<td>7</td>
<td>7</td>
<td>4</td>
</tr>
</tbody>
</table>
### Table 6.3

**Estimated dioxin emissions to air (situation end 1990) and emissions to be expected in 2000 with measures implemented and additional measures (in g I-TEQ per year)**

<table>
<thead>
<tr>
<th></th>
<th>Situation end 1990</th>
<th>In 2000 with implemented measures</th>
<th>In 2000 with additional measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incineration of urban waste</td>
<td>410</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Incineration of chemical waste</td>
<td>43</td>
<td>6</td>
<td>0.1</td>
</tr>
<tr>
<td>Incineration rubbish tip gas, biogas, sludge</td>
<td>0.4</td>
<td>0.5</td>
<td>0.2</td>
</tr>
<tr>
<td>Application pesticides</td>
<td>50</td>
<td>42</td>
<td>42</td>
</tr>
<tr>
<td>Metal industry</td>
<td>45</td>
<td>20</td>
<td>7</td>
</tr>
<tr>
<td>Cable burning</td>
<td>6</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Traffic</td>
<td>6</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Hospital waste incinerators</td>
<td>4</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Chemical processes</td>
<td>5</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Incineration of waste oil</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Incineration of wood</td>
<td>16</td>
<td>16</td>
<td>10</td>
</tr>
<tr>
<td>Fires, flares</td>
<td>9</td>
<td>9</td>
<td>7</td>
</tr>
<tr>
<td>Various processes</td>
<td>15</td>
<td>15</td>
<td>8</td>
</tr>
</tbody>
</table>

### 6.3 SUMMARY AND CONCLUSIONS

In the Guideline Incineration 1989 it is stipulated that in the incineration of waste the dioxin emission as from 1 December 1993 should be lower than 0.1 I-TEQ.m$^{-3}$. In the NER (Dutch emission guideline) guidelines are given concerning dioxin emissions for the sinter machines in the production of pig iron and emission requirements for wire reclamation incinerators and the thermal purification of soil. In table 6.3 estimates are given of the dioxin emissions to air for 1990 and the emissions to air to be expected in 2000 with measures implemented and additional measures.

'Measures implemented' only include emission reduction measures concerning those processes to which the Guideline Incineration 1989 applies and measures which are already put into effect. All of the other measures are ranged under 'additional measures'. In the table calculated values are given, however, this suggests too great a certainty. The total amount of dioxins emitted annually (situation end 1990) is estimated at appr. 600 g I-TEQ/year. The dioxin emissions to air to be expected with measures already put into effect, will be appr. 125 g I-TEQ/year in 2000 and with additional measures appr. 100 g I-TEQ/year.

In the next decade slight dioxin emissions to water may occur near WIs, in the incineration of chemical waste and hospital waste when wet gas washing is applied as additional flue gas purification. The current emissions to water will probably decrease. It is assumed that the
increase and the decrease will be in balance, meaning that in the year 2000 the emission to
water will amount to appr. 4 g I-TEQ/year. With additional measures the emissions to soil
in this period are expected to decrease from 3 to 0.2 g I-TEQ/year.
7. EVALUATION

7.1 EXCEEDING OF CURRENT STANDARDS AND GUIDELINES

Emission
The (scanty) measurements carried out in the various WIs show that the emission limit value of 0.1 ng I-TEQ.m$^{-3}$ und (which will come into effect per 30 November 1993) laid down in the Guideline Incineration 1989 (Nijpels, 1989), will not be met as yet. The dioxin emission to air from WIs amounted to an average of appr. 35 ng I-TEQ.m$^{-3}$ flue gas (situation end 1990, see table 2.1), being a factor 350 higher than the limit value mentioned above.

Soil and sediment
In comparing the dioxin levels in soil and sediment with the remediation values as proposed by Van Zorge (1987; see 1.3), the following may be concluded.

For residential areas a remediation value is proposed of 1000 ng I-TEQ.kg$^{-1}$ dry matter. Levels exceeding this proposed value are observed only locally (former rubbish tips or industrial sites, illegal incineration sites).

For grazing areas a level higher than 10 ng I-TEQ.kg$^{-1}$ dry matter is considered undesirable (Van Zorge, 1987). In grazing lands near WIs and other sources levels have been detected approximating or higher than (ranging from 2 to 250 ng I-TEQ.kg$^{-1}$ d.m.) the proposed remediation value (ranging from 2 to 250 ng I-TEQ.kg. For soil in grazing lands situated in rural areas (outside of highly localized deposition areas and the Randstad (section 4.2)) a dioxin content is expected between 2 and 5 ng I-TEQ.kg$^{-1}$ dry matter.

For sediment Van Zorge (1987) proposed a remediation value of 100 ng I-TEQ.kg$^{-1}$ dry matter. In the basin of rivers and canals levels have been detected that were either lower or higher than this proposed value (between 10 and 200 ng I-TEQ.kg$^{-1}$ dry matter). Exceeding may in most cases be ascribed to local discharges, although an influence by hydrological phenomenons on the site may often not be excluded. Highly elevated dioxin levels exceeding the proposed remediation value (up to a factor 40), have been detected in sediment samples from heavily loaded harbours. Available data suggest a background value for sediments of appr. 10 ng I-TEQ.kg$^{-1}$ dry matter.
Food

At some locations dioxin levels in cow’s milk exceed the Commodities Act standard of 6 pg I-TEQ.g⁻¹ milk fat. Exceeding is observed in the vicinity of the WIs in the Rijnmond area (Lickebaert, frequently in the period from May 1989 to the present), in Duiven (from January-March 1990), Zaandam (from September 1989 through June 1990) and near the wire reclamation incinerator at Culemborg (September 1989 through March 1990). Dioxin levels in milk from dairy farms may strongly fluctuate under the influence of weather conditions, management and fluctuations in emissions from neighbouring sources. The measures as laid down in July 1989 (revised in 1991: Staatscourant, 1991) in the framework of the Commodities Act Dioxin in Milk, are because of the exceeding of the Commodities Act standard for milk fat still in effect for several dairy farms in the Lickebaert area.

7.2 EXCEEDING OF TDI AND MTRs

7.2.1 Risks for humans

Sufficient toxicological data for the determination of a recommended value are only available for the most toxic congener 2,3,7,8-TCDD. The limited data concerning the other congeners have been incorporated in the so-called toxicity equivalency factors (TEFs).

2,3,7,8-TCDD is carcinogenic in experimental animals, however, there is no sufficient evidence to prove its carcinogenicity for humans. In the determination of the recommended value, based on data from genotoxicity studies, mechanistic studies and the mechanism of action, a threshold value was assumed to exist. At the WHO/EURO meeting (1990) a tolerable daily intake (TDI) of 10 pg.kg⁻¹ body weight was determined for 2,3,7,8-TCDD via a pharmacokinetic approach. This WHO recommendation is endorsed. Concerning 2,3,7,8-TCDD related compounds WHO made no recommendation. It was mentioned, however, that the TEF principle is useful in the assessment of risk, although the risk will be slightly overestimated. In the assessment of mixtures it is proposed to interpret the recommended value as being 10 pg I-TEQ.kg⁻¹ body weight, since no data are available to quantify this overestimation. Hereby the TEF factors indicated in table 5.1 need to be used.

Children and adults

For the general population food is the main source of exposure. For adults the contribution
through food is estimated at appr. minimally 90% to 95% (see 4.7).
The average intake amounts to 35 to 70 pg I-TEQ for children and adolescents up to 20 years of age and 70 pg I-TEQ per day for adults (see 4.7). Based on the individual body weight this means a median intake of 1 pg I-TEQ.kg\(^{-1}\) body weight for adults. In children and adolescents (breast-fed infants are considered separately) the intake per kg body weight is higher, as a result of a lower body weight. Thus, the tolerable daily intake of 10 pg I-TEQ per kg body weight for PCDDs and PCDFs for children and adults is not exceeded. Thus at the current exposure levels of PCDDs and PCDFs, through food as well as through other sources, no adverse effects are to be expected either in children or in adults. [However, if three planar PCBs are also considered in the derivation of the total TEQ-content, 1% of the children under the age of six years will exceed the value of 10 pg TEQ per kg body weight per day (Liem et al., 1991)].

**Infants**

In breast-fed infants exceeding of the recommended value occurs; the average daily intake during that period is estimated at 150 pg I-TEQ.kg\(^{-1}\) body weight. However, according to the WHO (1991) the recommendation for the TDI for 2,3,7,8-TCDD applies for an exposure time of 70 years and as such cannot be applied for infants. Furthermore it was stated by the WHO that in breast-fed infants the fat content increased to such an extent during the breast-feeding period, that at an increasing dose no proportional increase in the dioxin concentration in fat occurs (the so-called 'diluting effect').

The available information did not show breast-fed infants to be adversely affected by the intake of dioxins through mother’s milk, whereas in epidemiological studies the positive effects of mother’s milk on infants have been indicated (Howie et al., 1990; Van den Boogaard, 1990). It should be noted, however, that possible adverse effects, which are supposed to occur first following exposure to dioxins, are not to be measured non-invasively in infants. From the above-mentioned it is concluded that at present insufficient data are available to be able to assess the effects of dioxin exposure through mother’s milk. The committee mother’s milk of the Health Council has come to the same conclusion in 1991, however, they saw no reason to advise against breast-feeding. The committee considered further epidemiological research essential, particularly on effects of dioxins on infants. Such studies have by now been carried out. Pluim et al. (1992) have compared two groups of infants exposed to relatively low and high dioxin levels through mother’s milk (an average
of 19 and 38 ng I-TEQ.kg\(^{-1}\) milk fat, respectively). No significant difference was found between the two groups of infants concerning parameters such as growth and neurological development. The group exposed to the higher levels showed a slight but significant increase in thyroid hormones and thyroid regulating hormones in the blood in relation to the group exposed to the lower levels. However, the statistics used were unusual since no use was made of for instance individual data. A further statistic analysis of the data is considered desirable.

In the coming years more results will become available from studies on possible effects of dioxins in mother’s milk on infants. Based on these data a more reliable estimation of possible risks will become possible. In addition with pharmacokinetic models, which are being developed by the RIVM, a better understanding will be obtained of the internal loading of and possible effects on infants by exposure through mother’s milk. Based on this, concentrations in the target organs of infants can be estimated, which might contribute to a more reliable risk assessment.

*Risks for people living in the neighbourhood of WIs*

In the vicinity of WIs additional exposure may take place via the intake of dust or via dermal contact with such dust. Assuming the dioxin content of the dust to be appr. 10 ng I-TEQ.kg\(^{-1}\) dry matter, this additional exposure, locally near waste incinerators, was estimated at appr. 1 pg I-TEQ per person per day (Matthijssen et al., 1991). The additional exposure via direct inhalation of dust in the vicinity of waste incinerators is considered negligible (Theelen, 1989). However, as a result of the markedly elevated dioxin contents in cow’s milk fat through local deposition of fly ash, an additional exposure to cow’s milk and derived products with concentrations higher than 6 pg I-TEQ.g\(^{-1}\) milk fat, will result in exceeding of the TDI (Liem et al., 1989; Liem et al., 1991). In the framework of the Commodities Act measures are being taken in the case of the dioxin content in the milk fat (or derived products) being higher than 6 pg I-TEQ.g\(^{-1}\) fat. Therefore exceeding of the TDI via this route is considered unlikely.

7.2.2 *Risks for ecosystems*

In table 7.1 an overview is given of the indicative maximally tolerable risk levels for soil, sediments and surface water derived in this document. In accordance with the RIVM
Guidance document (Slooff, 1992) two MTRs for soil have been determined, one for the protection of the organisms living in the compartment concerned (MTR_{ecosystem}) and one for the protection of predators preying on organisms from the compartment concerned (MTR_{predator}). Using the equilibrium partition method this has resulted in two MTRs for the sediment. Because of the incidicative character of the MTRs several effect levels and no-effect levels have also been given in table 7.1.

**Table 7.1** An overview of indicative maximally tolerable risk levels (MTRs), effect and no-effect levels for dioxins in water and sediments (in 1-TEQ), based on data for 2,3,7,8-TCDD

<table>
<thead>
<tr>
<th>Concentration</th>
<th>MTR or (No) Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soil</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>ng.kg^{-1} d.m</td>
</tr>
<tr>
<td>75-285*</td>
<td>ng.kg^{-1} d.m.</td>
</tr>
<tr>
<td>&lt;10-1500**</td>
<td>ng.kg^{-1} d.m.</td>
</tr>
<tr>
<td>(30-325)*</td>
<td>ng.kg^{-1} d.m.</td>
</tr>
<tr>
<td>50,0000</td>
<td>ng.kg^{-1} d.m.</td>
</tr>
<tr>
<td>10,000,000</td>
<td>ng.kg^{-1} d.m.</td>
</tr>
<tr>
<td>50 - 2400000</td>
<td>ng.kg^{-1} d.m.</td>
</tr>
<tr>
<td>Sediment and water</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>ng.kg^{-1} d.m.</td>
</tr>
<tr>
<td>(0.00005</td>
<td>ng.l^{-1}; diss. fraction)</td>
</tr>
<tr>
<td>378</td>
<td>ng.kg^{-1} d.m.</td>
</tr>
<tr>
<td>(0.0012</td>
<td>ng.l^{-1}; diss. fraction)</td>
</tr>
<tr>
<td>11,970</td>
<td>ng.kg^{-1} d.m.</td>
</tr>
<tr>
<td>(0.038</td>
<td>ng.l^{-1}; diss. fraction)</td>
</tr>
</tbody>
</table>

* Range of average concentrations
** Total range of concentrations (minimum-maximum)
*** Exclusively based on a test with earthworms

Concerning the risk assessment mentioned below it is noted that the MTR_{predator}-values are determined from a NO(A)EL (no-observed-(adverse)-effect-level) for mammals. A NO(A)EL for birds is not available, therefore no statement can be made on the risk for worm-eating or fish-eating birds. However, studies with chickens showed birds to be at least as susceptible as mammals. For these reasons the term predators mentioned below may mean mammals as well as birds.
Terrestrial environment

For soil the following recommendations have been determined: a MTR_{ecosystem} of 500,000 ng I-TEQ.kg\(^{-1}\) dry matter and a MTR_{predator} of 3 ng I-TEQ.kg\(^{-1}\) dry matter. These MTRs are considered as levels based on dry matter, although this is not clear in view of the data mentioned below.

The MTR_{ecosystem} is not known to be exceeded anywhere in the Netherlands.

The MTR_{predator} corresponds with the background level measured in the reference location Bergambacht (ranging from 1 to 9 ng I-TEQ.kg\(^{-1}\) dry matter in the 0-10 cm top layer) and with the background levels measured abroad. The average background level is probably between 2 and 5 ng I-TEQ.kg\(^{-1}\) dry matter. In the vicinity of WIs in the Netherlands maximal levels were measured in 1990 of appr.25-250 ng I-TEQ.kg\(^{-1}\) dry matter in the 0-10 cm top layer. At such levels, 8-80 times higher than the MTR_{predator}, effects may occur in worm-eating predators, also in view of the fact that earthworms are not susceptible to such levels (NOLC: 5,000 \(\mu g\) 2,3,7,8-TCDD.kg\(^{-1}\)). It should be noted that the risk for predators is lower than may be determined from the above-mentioned range, since worms move in a greater layer than the 0-10 cm top layer, resulting in a lower average exposure level. At the levels measured in the vicinity of WIs the possibility of effects occurring on microbial processes is considered to be small to extremely small, considering the reported NOEC-values of >50 ng 2,3,7,8-TCDD.kg\(^{-1}\) dry matter to >2400 \(\mu g\) 2,3,7,8-TCDD.kg\(^{-1}\). In an extensive long-term field study no significant ecological effects were observed at 2,3,7,8-TCDD levels up to 1500 ng.kg\(^{-1}\); the average level in sub-areas ranged from 30 to 325 ng.kg\(^{-1}\). The survey carried out in this study included plants, insects, amphibians, reptiles, birds and mammals; it is not known whether a survey of worm-eating predators was also carried out. Locally, near illegal incineration sites of cables and cars, and in the Volgermeer, levels have been detected of up to maximally 1-100 \(\mu g\) I-TEQ.kg\(^{-1}\) dry matter. At such levels, more than 300 times higher than the MTR_{predator}, effects on worm-eating predators very probably occur.

Based on the above-mentioned data it is concluded that the risk for soil ecosystems at a national and a regional scale is limited. At a regional scale (in the vicinity of WIs) and a local scale (at illegal incineration sites, in particular) there is a possibility and a strong probability, respectively, of an intolerable risk for worm-eating predators occurring. The same may apply to other carnivorous mammals and birds. However, the uncertainties in the MTR_{predator} (see table 5.5.2) and the fact that heavily polluted locations are usually relatively
small in relation to the entire feeding ground of predators should be taken into account.

For groundwater neither ecotoxicological data nor measuring data are available. A risk assessment is therefore not possible. Considering the immobile nature of PCDDs and PCDFs in the soil it is improbable that groundwater organisms form a group at risk.

Aquatic organisms

For surface water the following recommendations have been determined: a $MTR_{ecosystem}$ of 0.0012 ng I-TEQ.l$^{-1}$ (dissolved fraction) and a $MTR_{predator}$ of 0.00005 ng I-TEQ.l$^{-1}$ (dissolved fraction). Due to the lack of measuring data in water a direct risk assessment is not possible.

For sediment measuring data are available, however, since no toxicological data are available for benthic organisms no direct risk assessment is possible in this case either. For sediment MTRs are determined via the equilibrium method amounting to 378 ng I-TEQ.kg$^{-1}$ dry matter (based on the $MTR_{ecosystem}$) and 15 ng I-TEQ.kg$^{-1}$ dry matter (based on the $MTR_{predator}$).

The $MTR_{ecosystem}$ is generally not exceeded in the Netherlands, with the exception of several heavily loaded harbours, such as the Laurenshaven and the Chemiehaven (2600 and 4000 ng I-TEQ.kg$^{-1}$ dry matter, respectively, situation 1983, see table 4.3).

From 1983 to 1985 levels of 4 to 250 ng I-TEQ.kg$^{-1}$ dry matter (see table 4.3) were found in most sediments of big rivers, canals and lakes. In practically all of these waters the $MTR_{predator}$ of 15 ng I-TEQ.kg$^{-1}$ dry matter was exceeded, in some cases several tens of times (in the above-mentioned harbours more than 150 times). In estuaries and the marine environment the levels (1987-1990) approximate the $MTR_{predator}$.

Based on the above-mentioned data it is concluded that the risk for aquatic ecosystems is in general limited, with the exception of several heavily loaded locations (harbours). At a national and a local scale there may be a possible and a probable, respectively, intolerable risk for fish-eating predators. However, the uncertainties in the $MTR_{predator}$ (see 5.5.2) and the fact that highly polluted locations are usually relatively small in relation to the entire feeding ground of predators, should, again, be taken into account.
7.3 IMPORTANCE OF POLICIES IMPLEMENTED AND INTENDED

**Guideline Incineration 1989**

The emission limit value of 0.1 ng I-TEQ.m\(^{-3}\) und from the Guideline Incineration 1989 is based on the principle of the best available technologies and therefore not determined from toxicological limit values. Based on data from the emission incinerator furnace study (Sein et al., 1989) and on statements by suppliers of flue gas purification devices, the emission reduction required is considered to be technically feasible for newly constructed as well as for modernized WIs. Following implementation of emission control measures the emission from individual WIs will not be higher than 0.5 g I-TEQ per year (assuming a maximal annual capacity of 10\(^6\) tonnes of waste per year per WI). Assuming the dioxin emissions from other sources and from the sources abroad to remain unchanged and using an atmospheric transportation model (OPS-model; Van Jaarsveld, 1989), an average deposition can be estimated for that situation of appr. 4-5 ng I-TEQ.m\(^{-2}\).year\(^{-1}\), with regional values up to 12 ng I-TEQ.m\(^{-2}\).year\(^{-1}\) maximally.

Calculations with the chain model show that at a deposition of 12 ng I-TEQ.m\(^{-2}\).year\(^{-1}\) the contribution from WI-emitted dioxins to cow's milk will be negligible (maximally 1 pg I-TEQ.g\(^{-1}\) milk fat; Slob, 1993). However, in areas where in the past cumulation of dioxins in the soil took place, the dioxin content in milk will remain higher than the national average level, probably amounting to 3-4 pg TEQ.g\(^{-1}\) milk fat, maximally. Based on these calculations it is concluded that the implemented measures in the framework of the Guideline Incineration 1989 will lead to a situation where humans, even at a local scale, are being sufficiently protected.

Calculations also show that at a deposition of 12 ng TEQ.m\(^{-2}\).year\(^{-1}\) the concentration in the soil will annually increase by appr. 0.1 ng TEQ.kg\(^{-1}\) dry matter. At an average background level of 2 to 5 ng I-TEQ.kg\(^{-1}\) dry matter and considering the determined MTR for soil ecosystems (500,000 ng I-TEQ.kg\(^{-1}\) dry matter), such an accumulation will hardly influence soil ecosystems. The MTR\(_{\text{predator}}\) is already at the background level. Therefore, any increase in the soil concentration will lead to an increased risk for worm-eating predators.

With the implementation of the Guideline Incineration 1989 the dioxin problem, with respect to the emission to air, will have moved into realm of maintenance. However, the influence of the emission control measures on the dioxin levels in solid residues, still needs to be studied. It is not known whether the residual compounds can be applied without everlasting
monitoring or need to be dumped in accordance with IBC criteria. The Guideline Incineration 1989 is also effective for the emissions occurring during incineration of other relevant waste categories, however, not for the other sources. Through the implementation of emission reduction measures, the emission from other sources will decrease, however, less spectacular so than that from WIs (table 6.1). The calculation based on the emission to be expected following implementation of the Guideline Incineration 1989 shows that, also at a constant emission from other sources, regionally no exceeding is to be expected of the Commodities Act standard for the dioxin level in milk. The other sources are widely scattered over the whole of the Netherlands. To what extent high depositions may locally occur in the near vicinity of a source is dependent on the emission and on the nature of that source.

Rhine- and North Sea Action Programmes
In the framework of the Rhine- and North Sea Action Programmes (RAP/NAP) the participating countries, including the Netherlands, have agreed to reduce the dioxin input into the Rhine and the North Sea via all routes by 70% or more from 1985 to 1995. From the scenario calculations in chapter 6 (table 6.2) it may be concluded that through the implementation of the proposed measures the emissions to air from 1985-1995 will be reduced by 85% and that the surface water load in the Netherlands (at a constant contribution from abroad to the atmospheric deposition) will be reduced by appr. 64%. When in the countries surrounding the Netherlands the emission reduction will also be reached, the load of the Dutch surface water will be reduced by more than 70%. The consequences of these reductions for the water quality of the North Sea have not been assessed.

7.4 CURRENT AND INTENDED STUDIES - PROPOSALS FOR FUTURE STUDIES

Most of the policy relevant dioxin studies were finalized in 1992. This provided a good understanding of the dioxin problem in the Netherlands. With respect to a number of subareas there are, however, still some voids. A number of studies is still in progress or in preparation. An overview of this research is given in section 7.4.1. In order to address these technology gaps as much as possible several recommendations have been made for future
7.4.1 Current and intended studies

The following studies are either in progress or will shortly be initiated. The overview has partly been extracted from Slob et al. (1992).

**Analytical methods**

- Nationally as well as internationally standardization takes place of methods and techniques for sampling and measuring PCDDs and PCDFs in flue gases from stationary sources.
- At the University of Amsterdam and the Dutch Cancer Institute an immunoassay is being developed for the determination of PCDDs and PCDFs in biological samples, aimed at the development of, in relation to GCMS-based methods, less costly methods for the determination of PCDDs and PCDFs in mother’s milk or similar matrices.
- Studies on the development of adequate determination methods for mono-ortho-substituted planar PCBs, as well as for brominated and brominated-chlorinated compounds in samples from a biotic as well as from an abiotic origin.

**Formation, sources and emissions**

- Besides in waste incineration, dioxins are also formed in other processes (Bremmer, 1991). Studies on the emissions in a number of processes has been performed in the project *"meten overige bronnen"* (MOB) (measuring other sources, MOS) and is reported in early 1993.
- Attention has as yet been strongly focused on emission and diffuse distribution of dioxins. In absolute terms a far greater amount of dioxins is found in E-filter ash. Dioxins also occur in slag. In new or modernized installations part of the dioxins will be concentrated in flue gas purification residues. In 1992 the dioxin concentrations in these solid waste flows were measured. The data will be reported in 1993.
- Slag and E-filter ash are applied as secondary basic materials in for instance road construction. It is of importance to understand whether in this application dioxins are leached out, as a result of which they would end up in the groundwater. End 1992 a study was started on the leaching out behaviour of dioxins from slag and ash.
Research was initiated in WIs in order to be able to meet the Guideline Incineration 1989. Thus analyses were carried out of dioxins in separately collected vegetable waste (vegetable-, fruit- and garden waste). In addition a study is carried out on necessary adjustments to the furnace/boiler combination. Furthermore a study is in preparation on a high-temperature gas cleaning, a demonstration project for the reduction of NO$_x$-emission with various systems, also aimed at a possible decomposition of dioxins and the injection of active carbon powder in the flue gas flow.

**Occurrence**

- Measuring of dioxin concentrations in soil is currently being continued in order to obtain a better insight into background levels. Measuring of dioxin concentrations in air will be continued in 1993.

- No systematic research was as yet performed on dioxin levels in grass. By the end of 1992 a study was commenced on the levels in grass and soil in relation to precipitation. In this study several other compounds will also be measured, particularly PCBs and several metals. Since grass constitutes the major exposure source for cows, the results are significant. In addition this information will be used as a further basis for the chain model. The study will be reported early in 1993.

- In 1992 a work group was set up which will study to what degree further research is required on the occurrence of dioxins in the soil of riverforeland locations.

- The food studies will be continued with a study on seasonal dependency and geographical variation of the TEQ-level in milk for human consumption and on the variation of the TEQ-level in fish-oil flows.

- In preparation for a 5-year study on mother’s milk concerning PCDDs and PCDFs which will be carried in 1993-1994 for the second consecutive time, an indicative study will be performed at the University of Utrecht on the presence of other dioxin related compounds in mother’s milk (planar and mono-ortho substituted PCBs).

**Effects**

- The toxicokinetic study was continued in 1992. The study was also aimed at obtaining data on the uptake of dioxins in humans. A toxicokinetic model for humans will be developed, which may lead to a better risk assessment of dioxins in humans.
The Sophia Child Hospital in Rotterdam, the University of Groningen and the University of Agriculture of Wageningen carry a study on the possible effects of dioxins and PCBs occurring in mother’s milk on infants. The results from this study will be available within one or two years.

- Nothing is as yet known about the risks of dust loads during transport and processing of E-filter ash. Research in cooperation with a processing company is in preparation.
- In 1991 an integrated field ecological and environmental chemical study was commenced by the Department of Tidal Waters on effects and occurrence of dioxins and planar PCBs in the common tern.

7.4.2 Proposals for future studies

From the above section it may be concluded that current studies and studies already initiated or in preparation, may fill most of the gaps in the research on the dioxin problems in the Netherlands. In addition this document shows that studies are carried out both nationally and internationally. However, there are still some areas in which future research should provide a better understanding. It concerns the following aspects:

- Even when the Guideline Incineration 1989 comes into effect, it is to be expected that the amount of dioxins remaining in the form of flue gas purification residues will still be considerable. It is recommended to control the amounts of dioxins associated with these residual compounds, by means of periodic measurements. Although these dioxins occur in an almost immobilized form and risks of distribution during controlled processing may be considered small, technologies need to be developed in order to further control the amount of bound dioxins.
- The contributions from abroad need to be further studied by means of international periodic measuring campaigns.
- Research aimed at the possible effects for exosystems needs to be continued. Current ecotoxicological data are extremely limited and still show a high degree of uncertainty. Ecotoxicological studies need to be aimed at possible effects on (top) predators in particular, due to the accumulation of persistent dioxins in the food chain and due to the high susceptibility of mammals and (possibly) birds to these compounds and related compounds such as PCBs.
- The periodic research on dioxin levels in mother’s milk needs to be continued, in
order to be able to follow trends in the human exposure to dioxins. The research needs to be coupled with research on the pharmacokinetics of dioxins in infants, in order to improve the risk assessment of the exposure of infants through breastfeeding.

Studies on other dioxin related compounds (PCBs, bromine/chlorine compounds) need to be carried out in connection with the current lacking knowledge of formation, sources, exposure levels and effects on humans and the environment.
7.5 CONCLUSIONS AND RECOMMENDATIONS

Exceeding of current standards and recommended values
The emission limit value as laid down in the framework of the Guideline Incineration 1989 (date of effect 30 November 1993) is at present not (yet) met by WIs. Exceeding of the Commodities Act standard for cow’s milk is presently only observed in the Lickebaert area.

Risks for humans
At the current exposure levels of PCDDs and PCDFs, through food as well as via other (local) sources, no adverse effects are to be expected in children and adults. With respect to exposure of breast-fed infants through mother’s milk insufficient data are currently available to arrive at a proper risk assessment.

Risks for ecosystems
At the current exposure levels the risks for aquatic and terrestrial ecosystems are considered limited. However, locally, in heavily loaded harbours in particular, effects are to be expected on aquatic organisms or the aquatic ecosystem. Food-chain poisoning (effects in the top of food-chains) is considered to be the most critical effect of dioxins. With respect to this effect, effects on worm-eating predators may occur at a regional scale (in the vicinity of WIs) and most probably occurs at a local scale (at illegal burning sites, in particular). Furthermore an intolerable risk for fish-eating predators may exist at a national scale and probably exists at a local scale. In these conclusions it is again pointed at the uncertainties in the determined maximally tolerable risk levels for predators and at the fact that heavily polluted locations are usually relatively small as compared to the entire feeding area of predators.

Importance of policies implemented or intended
The required emission control for new as well as for modernized WIs, in order to be able to meet, as from November 1993, the emission limit value of 0.1 ng I-TEQ.m\(^{-3}\) und as laid down in the Guideline Incineration 1989, is considered technically feasible. In case the dioxin emissions from other sources and sources abroad remain unchanged, it is calculated that in that situation the contribution of WI-emitted dioxins to the level in cow’s milk will be negligible. Even in those areas where in the past a cumulation of dioxins took place, the dioxin levels in cow’s milk in the Netherlands will remain below the Commodities Act
standard of 6 pg I-TEQ.g\textsuperscript{-1} milk fat. This would create a situation where humans, even at a local scale, are sufficiently protected. The annual increase of the dioxin level in the soil (appr. 0.1 ng I-TEQ.kg\textsuperscript{-1}) will hardly be of any influence on soil ecosystems.

Future studies
From current and intended studies it is to be expected that, by means of resulting understanding, a considerable part of the technology gaps in the Netherlands will be addressed. In addition technological developments were initiated in order to minimalize, as from December 1993, the major emissions to air from Incinerators. Additional studies are required to address several gaps. This concerns studies including those on the future fate of solid purification residues, other sources and contributions from abroad, possible effects in (top)predators, the assessment of risk of exposure of infants to dioxins via breast-feeding and, finally, the exposure levels and the associated risks of other dioxin related compounds for humans and the environment.
8. **AFTERWORD**

The estimates of the dioxin emissions as described in this report are based on measurements at WIs and on a survey of processes in the Netherlands where dioxins may be formed. Based on dioxin measurements carried out in several installations the dioxin emission was estimated for all processes of the same type. For most process types the dioxin emission was estimated using literature data. In 1991 and 1992 additional measurements were carried out at dioxin sources not yet measured before. The selection of installations was based on a survey of the sources. Per process type one or more companies or installations were selected which served as a model for the process type considered. Several measurements were carried out in process types expected to contribute most. In this way a more reliable picture of the total dioxin emission in the Netherlands will be obtained than from earlier estimates. The results from this study will be reported in the first half of 1993 (RIVM-TNO report, RIVM report no. 770501003, in preparation).

As far as is presently known, the total emission in 1991 amounted to appr. 500 g I-TEQ. This is about 100 g I-TEQ less than estimated for the year 1990. The difference may be partly explained by the emission control measures already implemented and partly by too high estimates of a number of emission factors for the situation in 1990.

The main part of the total emission estimated for 1991 was still emitted by WIs, that is appr. 80%. The remainder of the annual emission is divided over 16 process types, of which sinter processes (with 5.5% of the total), the former use of wood preservatives (with 5%) and the incineration of chemical waste (with appr. 3.5%) are the most important. Since insufficient data are available no estimation can be made of the emission resulting from fires. Further studies are required on the conditions whereby dioxins are formed during fires in order to get an understanding of the significance of this source. Additional data are available on the use of PCP-containing wood preservatives. This resulted in an estimated dioxin emission which was only half of the amount as estimated for 1990.

Earlier the dioxin emission in the incineration of coal was assumed to be negligible. In a coal-fired E-power plant a low emission concentration was indeed measured. The great coal consumption of the power plant results in a considerable flue gas output. As a result the dioxin emission of 0.7% of the total is not negligible. For the combustion of wood in homes only an indicative estimate can be made since insufficient data are available on the amounts of contaminated wood being combusted. With appr. 2.5% of the total emission the relative
emission of this source is at present the same as in 1990. Further studies are recommended on the emissions in combustion of wood in fireplaces and wood-burning stoves.

The Guideline Incineration 1989, in which an emission limit value of 0.1 ng I-TEQ·m\(^{-3}\) is included, was substituted by the resolution air emissions waste incineration (Staatsblad 1993 nr 36). The resolution concerns the incineration of municipal and similar industrial waste in WIs. As from 1 January 1995 existing installations need to meet the emission limit values stipulated for these installations. For other processes emission requirements will be laid down in the Nederlandse Emissie Richtlijnen Lucht (Dutch Emission Guidelines Air), which are based on the GI89. The specific nature of these installations will be taken into account.

Based on these resolutions and the implemented measures the dioxin emission is expected to decrease to appr. 60 g I-TEQ in the year 2000. The emission as a result of the incineration of municipal waste will have decreased to maximally 4 g I-TEQ (depending on the amount to be incinerated). The former use of pesticides will then be the major source. In contrast with what is indicated in table 6.1, the emission from this source in the year 2000 is expected to amount to not more than 20 g I-TEQ. This also explains the main part of the difference in the current estimate of 60 g I-TEQ with the estimates for the year 2000 indicated in table 6.1. Since the use of PCP-containing wood preservatives is prohibited, this source will slowly become less significant by the replacement of wood.

Model calculations show that dioxin emission from the processes where additional measurements were carried out will not lead to exceeding of the standard for cow’s milk. By further reducing the emissions this will also in the future not be the case.
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