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A PERSPECTIVE ON INDUSTRIAL

WASTE INCINERATION

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ECETOC Technical Report No.49

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EXPOSURE OF MAN TO DIOXINS: A PERSPECTIVE ON INDUSTRIAL WASTE INCINERATION SUMMARY AND CONCLUSIONS

The current knowledge of the occurrence, mechanism of formation and environmental fate of polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) is reviewed. Emissions of these materials originating from industrial waste incinerators (IWIs) are placed into perspective relative to their global emissions. Exposure levels, toxicological properties and human health impact are also addressed.

At present the major sources of atmospheric contamination by PCDDs and PCDFs are hospital and municipal waste incinerators and metallurgical processes. In contrast, industrial waste incineration (IWI) has lower emissions representing only about 1% of the total known sources. Atmospheric PCDD and PCDF emissions from industrial waste incinerators are strongly adsorbed on particles, i.e. dust and fly ash. The levels of PCDDs and PCDFs in soil and grass close to IWIs in the U.K. and Germany were found to be indistinguishable from urban background levels. The local contribution from an IWI is low with regard to PCDD and PCDF emissions when compared to other sources.

Atmospheric PCDDs and PCDFs emitted from all sources are deposited on soil, grass, vegetation and surface water. Subsequent absorption by animals and bioaccumulation make the food chain the most important route of exposure (>90%) for man; fish, dairy produce, meat products, and vegetables all being important. Global human exposure to PCDDs and PCDFs has been estimated in various European countries as an average daily intake of about 120pg/d, expressed as 2,3,7,8-TCDD toxic equivalents (TEQ). Conversion to an average body weight of 60kg results in a daily burden of 2pg/kgbw/d. Because of mothers milk contamination, a higher food intake in respect to body weight and the larger proportion of dairy products consumed by children, values up to 10pg TEQ/kgbw/d for their body burden may be expected.

Based on a combination of experimental toxicology and epidemiological data, an assessment was made of the health risk to man resulting from such exposure. The lowest effect level in animal studies is between 100 and 1,000pg TEQ/kgbw/d, and recent mechanistic studies support the existence of a threshold level below which there is no toxic effect. In man, the only clearly established toxic effect of these compounds is a severe form of acne called chloracne, which is observed after high accidental exposure to 2,3,7,8-TCDD. Data on other effects, including cancer, are inconclusive and remain controversial. By applying a safety factor of 100, which is commonly

used in Europe, it can be assumed that man would not be affected by a lifetime exposure to PCDDs and PCDFs corresponding to a daily intake of 1 to 10pg TEQ/kgbw/d.

Thus the total uptake of PCDDs and PCDFs from all known sources by man (adults and children) is within the range for this tolerable daily intake. Emphasis should be placed upon further minimisation or elimination of emissions from remaining major sources of contamination by PCDDs and PCDFs.

Although IWIs are a minor source of PCDD and PCDF emissions, further improvements are being sought. These include optimisation of waste preparation and loading rate, residence time, turbulence, temperature and waste/oxygen ratio. Better knowledge of the nature of the precursors and the mechanisms of formation of PCDDs and PCDFs are taken into consideration in designing modern IWIs, permitting the emissions of PCDDs and PCDFs to be reduced to 0.1-1ng TEQ/m^3. Even more advanced technologies which are presently being evaluated may reduce these emissions still further, but the technical effort and cost will be considerable for relatively little benefit.

Incineration avoids dangerous dumping of industrial waste, which may result in leachate and landfill gas problems for many years. The risk to human health from exposure to PCDDs and PCDFs originating from well studied industrial waste incineration processes using present state-of-the-art technology can be regarded as insignificant.

SECTION 1 INTRODUCTION

Halogenated tricyclic aromatics are members of a large family of substances with some congeners (Appendix A) notable for their toxic behaviour. Within this family two main groups exist, the polyhalogenated dibenzo-p-dioxins (PXDD) and the polyhalogenated dibenzofurans (PXDF). The chlorinated and brominated dioxins and furans are the most significant and have been the subject of much investigation. The major emphasis in this report will be on the chlorinated compounds as their presence is considered of most importance. This does not preclude the possible importance of other halogenated compounds, especially the brominated and mixed chlorinated and brominated compounds (Buser, 1987a, b).

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are known to be ubiquitous contaminants in ecosystems. In recent years considerable attention has been given to the formation of PCDDs and PCDFs as unwanted by-products in combustion processes involving chlorine-containing compounds and in some chemical manufacturing processes.

Public concern about risks to human health from exposure to PCDDs and PCDFs originating from industrial waste incinerators (IWIs) has led to many questions about the standards of performance of such incinerators regarding the control of PCDD and PCDF formation. The purpose of this report is therefore:-

- to review the knowledge of the formation and fate of dioxins and dibenzofurans during the incineration of chemical waste in relation to their occurrence from other sources;
- to review the approaches adopted for controlling emissions from industrial waste incineration;
- to review exposure to dioxins and dibenzofurans of toxicological significance and to assess the relevance of such exposure to man.

SECTION 2 BACKGROUND

PCDDs and PCDFs, popularly referred to as dioxins and furans, or even simply as "dioxins", are a group of closely related tricyclic aromatic chemical compounds. None of these compounds has any commercial use and they are not manufactured intentionally except on laboratory scale for research or analytical standards. Nevertheless, PCDDs and PCDFs are formed occasionally in extremely low concentration as by-products, or may be present as trace impurities in certain chemical processes involving chlorine and organic compounds. They are also produced in trace quantities in the combustion of organic materials whenever chlorine or chlorine compounds are present. The existence of these compounds as contaminants in products from a variety of processes and their consequent widespread distribution in the environment became evident as a result of advances in analytical techniques. PCDDs and PCDFs can now be detected below the parts per trillion (ppt) level.

2,3,7,8-Tetrachlorodibenzodioxin (2,3,7,8-TCDD) was the toxic contaminant in the defoliant Agent Orange used in the Vietnam war and was identified in emissions from chemical plant accidents, most notably the Seveso incident in 1976. Against this background, "dioxin" has been popularised as "the most dangerous substance known to man". When it became widely known that PCDDs and PCDFs could be produced in any combustion process where chlorine compounds as well as organic compounds were present, public concern about the environmental effects of waste incineration, and particularly industrial waste incineration, focused on the potential for these processes to generate and release PCDDs and PCDFs.

In recent years, largely in response to these concerns, regulatory authorities, research institutes and industry have studied the generation of PCDDs and PCDFs in combustion and manufacturing processes using organic compounds with chlorine or chlorine compounds. Numerous measurements have been made of the amounts of PCDDs and PCDFs emitted from such processes in flue gases, waste water discharges and solid wastes. Municipal and industrial waste incineration processes have been studied extensively in attempts to understand the mechanism of formation, and thereby methods for control, of PCDDs and PCDFs, both in combustion chambers and in flue gas cleaning systems. Surveys have been undertaken to determine the distribution of these substances in the immediate vicinity of known sources and in the environment generally.

A number of regulatory authorities and other organisations have used the available information on occurrence, distribution, environmental fate and toxicological properties of PCDDs and PCDFs to make assessments of the likely hazards to human health. Various proposals have been made about "acceptable daily intakes" and these have been used to assess hazards to public health associated with individual sources or types of sources.

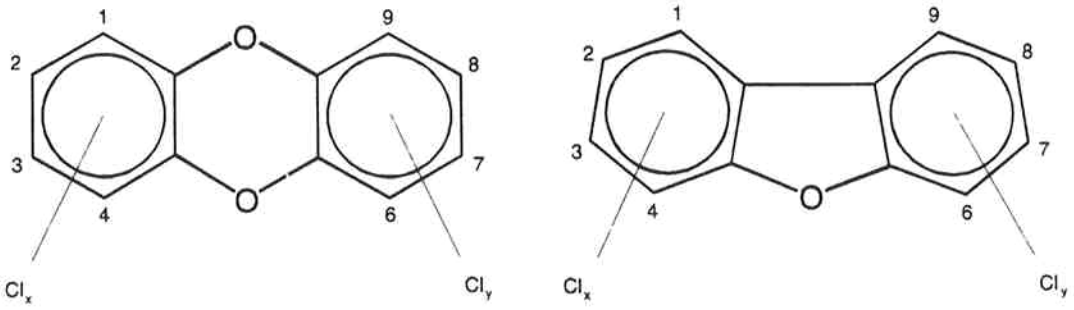
A comprehensive investigation of these compounds has been made including studies of their formation and evaluation of their ecological pathways from technical processes to the environment.

ECETOC has now carried out a critical review of the current knowledge of the occurrence, formation, distribution, environmental fate and toxicological properties of PCDDs and PCDFs. Available methods for estimating human exposure have been used and an assessment made of the relevance of such exposure to health. The approaches adopted by regulatory authorities and plant operators for controlling emissions from industrial waste incineration have also been examined. Plant performance criteria are recommended and a risk assessment approach put forward that can be used as the basis for the design and control of chemical waste incinerators in order to minimise discharges of PCDDs and PCDFs to the environment and to provide an adequate safeguard for public health.

SECTION 3 CHARACTERISATION OF PCDDs AND PCDFs

3.1. CHEMICAL IDENTITY

PCDDs and PCDFs are chlorinated tricyclic aromatic compounds. Each of these compounds has a triple ring structure consisting of two benzene rings inter-connected to each other by respectively two or one oxygen atoms. The number of chlorine atoms can vary between 1 and 8. The general formulae are:



Seventy five PCDD and 135 PCDF "congeners" are theoretically possible (cf Appendix A for definitions and abbreviations). Mixed halogenated substitution increases the number of congeners drastically, for example the number of congeners of chloro- and bromosubstituted dibenzo-p-dioxins and PCDFs is about 4,600 (Buser, 1987b).

The crystal structures of several chlorinated PCDDs have been reported (Boer *et al*, 1972; Cantrell *et al*, 1988). The molecules of the 2,7-dichloro, 2,3,7,8-tetrachloro and octachloro structures are all located on crystallographic centres of symmetry and are very nearly planar in each case (Cantrell *et al*, 1989). The correlation between the chemical structure of the compounds and their acute toxicity is shown in Table 1. The 17 PCDD and PCDF congeners substituted at least on all four positions 2,3,7 and 8 appear to be more toxic than the others by several orders of magnitude.

The toxic potential of each congener is expressed by a factor ("toxicity equivalency factor" or TEF) which relates the toxic potential of a given congener to the toxic potency of 2,3,7,8-TCDD as a reference (NATO/CCMS, 1988a, b, c; Kutz *et al*, 1990; Safe, 1990). These factors allow the toxic potential of each congener present in any source to be expressed as an equivalent concentration of 2,3,7,8-TCDD. The total toxic equivalent (TEQ) of a given environmental sample is calculated by multiplying the amount of each congener present in the sample by its specific "toxic equivalency factor" and by adding up the total (cf Section 5).

Table 1

Correlations between Chemical Structure and Acute Toxicity,
expressed as LD_{50} in Guinea Pigs†
(McKinney and McConnell, 1982; Schlatter, 1985)

Name of Compound	LD_{50} (µg/Kg)
2,3,7,8-Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD)	2
2,3,7,8-Tetrachlorodibenzofuran (TCDF)	7
3,3',4,4',5,5'-Hexachlorobiphenyl	223
2,3,7-Trichlorodibenzo-p-dioxin (TrCDD)	29,444
2,3,3',4,4',5,5'-Heptachlorobiphenyl	> 3,000
4,4'-Dichloro-3,3',5,5'-tetrafluorobiphenyl	> 3,000
3,3',4,4'-Tetrachlorobiphenyl	<552
3,3',4,4'-Tetrachlorobiphenyl ether	NDb
2,3,6,7-Tetrachloronaphthalene	> 3,000
1,2,4,7,8-Pentachlorodibenzo-p-dioxin (PeCDD)	1,125
1,2,3,7,8-Pentachlorodibenzo-p-dioxin (PeCDD)	3
2,3,4,7,8-Pentachlorodibenzofuran (PeCDF)	< 10
3,3',4,4',5-Pentachlorobiphenyl	NDc
2,3,7,8-Tetrabromodibenzo-p-dioxin (TBDD)	NDc
2,3,7,8-Tetrabromodibenzofuran (TBDF)	< 15
2,3,7-Tribromodibenzo-p-dioxin (TrBDD)	NDc
2,3,6,7-Tetrabromonaphthalene	242
1,2,4,6,7-Pentabromonaphthalene	200
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	73
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	70-100
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin (HxCDD)	60-100
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin(HpCDD)	> 600
Octachlorodibenzo-p-dioxin (OCDD)	NDb
2,3,4,6,7,8-Hexachlorodibenzofuran (HxCDF)	120
1,2,3,4,7,8-Hexabromonaphthalene	361
1,2,3,5,6,7-Hexabromonaphthalene	> 3,610
2,3,6,7-Tetrachlorofluoren-9-one	> 100

† Hartley strain guinea pigs given a single oral dose (gavage) and observed per 30 days.

NDb Not determined in guinea pig, but compound was inactive as inducer of cytochrome P-448 and associates enzymes in one or more biological test systems.

NDc Not determined in guinea pig, but compound was very active as inducer of cytochrome and associates enzymes in one or more biological test systems.

3.2. PHYSICO-CHEMICAL PROPERTIES

All PCDDs and PCDFs are organic solids with high melting and boiling points and low vapour pressures (Rordorf, 1985). They are characterised by extremely low water solubilities (Friesen *et al*, 1985; Shiu *et al*, 1988), and high n-octanol/water partition coefficients (Shiu *et al*, 1988). They have a tendency of being strongly adsorbed on surfaces of particulate matter (Walters and Guisleppe-Elle, 1988). The water solubility of PCDDs and PCDFs decreases and the solubility in organic solvents and fats increases with increasing chlorine content.

3.3. ANALYTICAL DETECTION OF PCDDs AND PCDFs

Details of the techniques used for the collection of samples from various environmental media together with state of the art of analytical techniques are fully discussed in Appendix B.

Results are usually expressed as TEQ. This necessitates the addition of at least 17 ^{13}C PCDDs and PCDFs as internal standards, and these materials are coincidentally used to provide quality assurance during the sampling and analysis procedure. In this report analytical results will be expressed in TEQ whenever possible.

The most important sampling points associated with IWIs are stack gases, water effluents, ash, slag and sludge from gas cleaning activities. The method for analysis of PCDDs and PCDFs is high resolution capillary gas chromatography interfaced with mass spectrometry, following sample clean-up by liquid chromatographic techniques. Detection limits of 0.01ng TEQ/m^3 in stack gases can be achieved, depending on sampling systems used. The accuracy largely depends on the sampling procedures, the efficiency of extraction and the instrumental analysis. Consequently, the results from different laboratories may differ by as much as one order of magnitude, particularly at low levels.

SECTION 4 ORIGIN, DISTRIBUTION AND FATE OF PCDDs AND PCDFs

4.1. ORIGIN

4.1.1. Natural

The known natural sources of PCDDs and PCDFs are related to fires and combustion processes. Thus, these toxic compounds may be produced from forest fires, by lightning or volcanic action. Bumb *et al* (1980) in their study on trace chemistry of fire have shown that hydrocarbon combustion in the presence of chlorine compounds can give rise to PCDDs and PCDFs in small amounts.

The pre-industrial existence of PCDDs and PCDFs has been demonstrated by analysis of ancient human Eskimo tissues (Schecter *et al*, 1988; Tong *et al*, 1990). Octachloro dibenzo-para-dioxin (OCDD) was found at concentrations of about 30pg/g with an analytical technique with a detection limit of less than 3pg/g and HeptaCDD (HpCDD) at a level of 10pg/g with a detection limit better than 1pg/g. The OCDD concentration is 25 times less than the levels found in adipose tissues of modern man (Schecter and Ryan, 1988). PCDDs and PCDFs were also found in 9 Chilean mummies (Ligon *et al*, 1989).

Hashimoto *et al*, (1990) confirmed that historical pollution by PCDDs and PCDFs occurred at least 6,100 years B.C. The total concentrations of PCDDs in sediment cores from Osaka Bay showed values of 8ng/g at a depth of 0.3m and 3ng/g dry sediment at a depth of 2.0m. They found PCDDs even at a soil depth of 8.75m on Harima-Nada with concentrations of 1,2,3,4,6,7,9-HpCDD at 0.05ng/g and OCDD at 0.3ng/g in dry sediment. The corresponding concentrations of PCDFs were at least one order of magnitude lower. The concentrations in historical sediments do not differ very much from those in a modern industrial landfill site (Stern *et al*, 1989). Similar levels of PCDDs and PCDFs compared to the values in historical sediments were also reported in urban soil samples from British cities in 1989 (Creaser *et al*, 1990):

PCDD (ng/g soil)		PCDF (ng/g soil)	
TCDD	0.07	TCDF	0.23
PeCDD	0.07	PeCDF	0.19
HxCDD	0.15	HxCDF	0.16
HpCDD	0.82	HpCDF	0.15
OCDD	10.0	OCDF	0.20

The natural or pre-industrial combustion sources produce PCDDs and PCDFs in low yields. The synthesis route is extremely complex and reaction kinetics are generally not favourable in open burning conditions. The generally low availability of halogens in the combustion of natural biomass will also limit the amount of PCDDs and PCDFs formed. Nevertheless, even at very low yields these primitive combustion processes could be significant sources of the compounds; a million tonnes of wood may be burned in a large forest fire.

4.1.2. Origin from Technological Sources

PCDDs and PCDFs have never been produced deliberately as commercial products. There are two main sources of PCDDs and PCDFs in the environment: industrial by-products and combustion processes.

Industrial Sources. The formation of PCDDs/PCDFs in a chemical reaction process is very dependent on the presence of oxygen, carbon, chlorine and heat. The presence or formation of precursor compounds with closely related molecular structures will greatly enhance the formation of these substances. The following sections of this section review a number of processes in which PCDDs and PCDFs can be produced as unwanted by-products.

Industrial Chemical Processes. Many manufacturing processes involving halogenated (particularly chlorinated and brominated) aromatic or aliphatic compounds are likely to involve side reactions leading to the production of halogenated polyaromatics. Processes involving chlorophenols, chlorobenzenes, chlorine substituted aliphatic compounds and chlorine containing catalysts are all potential sources of PCDDs, PCDFs and related compounds (Esposito *et al*, 1980; Heindl and Hutzinger, 1986). Other compounds are less likely to lead to dioxin formation because they would require an unusual combination of reaction steps to produce PCDDs (Esposito *et al*, 1980).

Hagenmaier and Brunner (1987) detected PCDDs and PCDFs as contaminants in pentachlorophenol (PCP) and sodium pentachlorphenate (PCP-Na) in amounts up to the mg/kg range. These compounds were widely used materials for pest control and wood preservation but their use is now restricted.

Another example of a process leading to significant production of PCDDs was the manufacture of Agent Orange, which achieved attention through its use as a defoliant in the Vietnam war. It was a 1:1 mixture of the n-butylesters of 2,4,5-T (2,4,5-

trichlorophenoxyacetic acid) and of 2,4-D (2,4-dichlorophenoxyacetic acid). Tetra- and higher chlorosubstituted dibenzodioxins were directly generated during the process via radical reactions of 2,4,5-T or the trichlorosodiumphenolate as intermediate compound. Analysis of 200 samples of Agent Orange (Kearney *et al*, 1973) showed a wide range of 2,3,7,8-TCDD content from 0.5mg/kg to 47mg/kg. It has been estimated that in the early 1970's manufacture of Agent Orange led to the formation of about 50kg of 2,3,7,8-TCDD per year.

2,4,5-T was also used as a herbicide in agriculture in the form of its esters or salts. For such products Smith and Pearce (1986) reported for 2,4,5-T average levels of 950ng/kg of 2,3,7,8-TCDD/kg in 1971. Subsequent improvements of the chemical processes brought a considerable reduction to a few ng 2,3,7,8-TCDD/kg. 2,4,5-T is no longer used in Western Europe and in the USA.

The potential PCDD and PCDF formation in certain manufacturing processes increases greatly if temperatures and pressures increase well beyond the normal operating conditions. Although such incidents are not common, Bruppacher and Reggiani (1986) have reported 16 accidents associated with the production of chlorophenols. Of these the best documented is the Seveso accident in 1976. The process in Seveso was the manufacture of 2,4,5-trichlorophenol (TCP), an intermediate for the production of the bactericide hexachlorphene. It was later estimated that some 300g of 2,3,7,8-TCDD had been discharged and spread over an area of about 2,2km^2. A further 600g were found in the remaining reactor mass and other parts of the plant (Kuenzi, 1982; Salomon, 1982; Sambeth, 1982; Krum, 1985; Hutzinger *et al*, 1985).

PCDDs and PCDFs were also detected as impurities in polychlorinated biphenyls and terphenyls used as dielectric fluids in transformers in the electricity supply network (UBA, 1984). They can also be formed during the production of halogenated diphenylethers and during the extrusion of thermoplastics containing such compounds (EEC, 1989; BASF, 1990).

Halogenated polyaromatics, in a wide range of chemical manufacturing processes, can give rise to PCDD and PCDF formation in waste streams, in recycled streams and as contaminants in intermediates and finished products.

Processes of the Pulp and Paper Industries. Manufacture of wood pulp and paper by processes using chlorine gives rise to PCDDs and PCDFs in products (Beck *et al*, 1989a) and in process sludge and waste water streams. It was demonstrated in a study of 104 paper mills in the USA (Whittemore *et al*, 1990) that the median values of

2,3,7,8-TCDD in sludge and waste water were 16 and 0.02ng/kg, respectively, but somewhat higher levels of 2,3,7,8-TCDF (82 and 0.061ng/kg) were observed in these media. Many other reports (Hutzinger *et al*, 1985; Clement *et al*, 1989; Beck *et al*, 1989a) describe the widespread occurrence of trace quantities of PCDDs and PCDFs from this industrial process but the mechanism of formation is not understood. Chloroaromatic compounds could be transformed into PCDDs by an enzymatic action (Wagner *et al*, 1990).

Metallurgical Processes. The production and emission of chlorinated aromatics especially PCDDs and PCDFs from metallurgical processes, such as scrap metal remelting, steel mills and metal chloride production processes have been reported (Tysklind *et al*, 1989; Heindl and Hutzinger, 1986).

Manufacture of Brominated Flame-retardants. Tribromo- and pentabromophenol, pentabromodiphenylether, decabromodiphenylether, hexabromobiphenyl and tetrabromobisphenol-A are used as flame-retardants (Larsen, 1980). Esposito *et al* (1980) reported on formation of tetra- and octabromodibenzo-para-dioxins from side reactions of their production processes.

Combustion Processes. There are many different combustion processes which produce PCDDs and PCDFs. These include municipal waste incinerators (MWIs), industrial waste incinerators, hospital waste incinerators, sewage sludge incineration, wood and coal combustion, oil combustion including motor vehicle engines and accidental fires.

The mechanisms for the formation of PCDDs and PCDFs in the gas phase are described (Bumb *et al*, 1980; Choudhry and Hutzinger, 1983). Burning and combustion conditions may vary extensively, and a large number of compounds with a wide range of chemical structures may be formed at detectable levels. In MWIs and IWIs these compounds are mainly formed as a result of heterogeneous reactions at 250 to 350°C within electrostatic precipitators and heat exchangers (Mariani *et al*, 1990; Nottrodt *et al*, 1989; Stieglitz *et al*, 1989). These heterogeneous reactions cause the formation of PCDDs and PCDFs from precursor molecules and organic or inorganic chlorine compounds (Vogg *et al*, 1987).

4.1.3. Regulatory perspective for controlling PCDD and PCDF emissions

The EEC "Proposal for a Council Directive on the Incineration of Hazardous Waste" provides for a specific obligation to minimise the emissions of PCDDs and PCDFs by the

most progressive techniques and to make every effort not to exceed a guide value of 0.1ng TEQ/m^3 by all average values measured over the sample period (EEC, 1992). Only three of its member countries regulate PCDDs and PCDFs explicitly, the emission standard in Germany and the Netherlands being 0.1ng TEQ/m^3. This dioxin criterion was tacitly accepted as a proposal by the conferees at "Dioxin 90" in Germany without significant comment. In the case of the UK, the standard is 1.0ng TEQ/m^3 with a goal of reaching 0.1ng/m^3. The US-EPA has a different approach for hazardous waste incineration. Rather than limiting these contaminants individually and quantitatively, EPA proposes controlling them by assuming good combustion as measured by stack emission control of carbon monoxide (CO) and total hydrocarbons (EPA, 1990).

Details of regulatory requirements for industrial waste incinerators in Europe are presented in Appendix E.

Municipal Waste Incinerators

Emission to the air. Total atmospheric emissions of PCDDs and PCDFs from MWIs are shown in Table 2. The emissions depend less on the nature of the waste, but more upon the combustion efficiency and the quality of the stack gas cleaning, and may depend on the design of the incinerator. Incinerators built in the 1960's or 1970's show levels of emissions up to 100ng TEQ/Nm^3 or more. When older incinerators are equipped with multistage stackgas cleaning procedures and electrostatic precipitators, the emission levels attain modern standards.

The goal for PCDD and PCDF emission levels of 0.1ng TEQ/Nm^3 will necessitate the development of new and costly technologies (BImSchV, 1991). It has been suggested that emissions at these low levels may be attained by optimisation of the combustion process and use of appropriate flue gas cleaning devices (Kilgroe *et al*, 1990). It has been shown on a pilot plant scale that the emission of PCDDs and PCDFs can be brought down to values of 0.03ng TEQ/Nm^3 (Eicken *et al*, 1990).

Emission via other routes. Flue gas, fly ash, slag and water emission streams may also contain PCDDs and PCDFs. The concentration of PCDDs/PCDFs in fly ash from MWIs ranges from 0.3 to 54ng TEQ/g. The concentrations in slags from MWIs fluctuate widely, with concentrations from 20pg/g to 250pg TEQ/g (cf Table 3).

Table 2

Emissions of PCDDs and PCDFs to Air from Municipal Waste Incinerators (MWI)

Location of MWI	Year of Install-ation	Emission TEQ ng/m^3	Literature	Remarks
KVA Josefstrasse, Zuerich		10	BUS-CH (1982)	TEF Schlatter
Rotterdam, NL	1963	92	SVA (1991)	
Muenchen Nord, Block 1, FRG	1964	1.4	AbfallwirtschaftsJournal (1991)	Municpal and Hospital waste mixed TEF BGA
Stuttgart-Muenster, FRG	1965	13	Schetter (1988)	TEF Eadon Stackgas upstream from the filter
Schweinfurt, FRG	1965(?)	2-4.5	AbfallwirtschaftsJournal (1991)	TEQ (NATO/CCMS)
Leiden, NL	1966-76	69	SVA (1991)	closed down
Den Haag, NL	1967-74	8.3	SVA (1991)	
Amsterdam, NL	1968	4.8	SVA (1991)	
Alkmaar, NL	1971	31	SVA (1991)	closed down
Dordrecht, NL	1972	62 ca. 17	SVA (1991)	After additional stack gas cleaning
Rijmond, NL	1972	53	SVA (1991)	
Schwabach, FRG	1972	0.8-0.9	AbfallwirtschaftsJournal (1991)	TEQ (NATO/CCMS)
Leenwaarden, NL	1973	100	SVA (1991)	closed down
Marchwood, UK	1973	3.8-7.6	Rechem International Ltd. (unpublished results 1992)	TEQ (NATO/CCMS)
Duiven, NL	1975	62 ca. 5	SVA (1991)	After additional stack gas cleaning
Ebenhausen, FRG	1976	3.1-6.9	AbfallwirtschaftsJournal (1991)	TEQ (NATO/CCMS)

Table 2 (ctd.)

Location of MWI	Year of Install-ation	Emission TEQ ng/m³	Literature	Remarks
Geiselbullach, FRG	1976	0.12-0.2	AbfallwirtschaftsJournal (1991)	Without Sorbalit TEQ (NATO/CCMS)
		0.04-0.08		With Sorbalit (limestone with 3% activated coke) TEQ (NATO/CCMS)
Havant, UK	1976	5-18	Rechem International Ltd. (unpublished results 1991)	TEQ (NATO/CCMS)
Roosendaal, NL	1976	14	SVA (1991)	
Zaanstad, NL	1976	239	SVA (1991)	closed down
Bamberg, FRG	1978	1.2-1.4	AbfallwirtschaftsJournal (1991)	TEQ (NATO/CCMS)
Wuerzburg, FRG	1984	1.76 0.62 0.37	Schetter (1988)	TEF Eadon Stackgas downstream from filter
Wuerzburg, Ofen 1, FRG	1984	0.04-0.08	AbfallwirtschaftsJournal (1991)	TEQ (NATO/CCMS)
Stockholm, Hoegdalen, S	1986	1.7	Schetter (1988)	TEF Eadon Stackgas upstream from filter
		< 0.3		Stackgas downstream
		1.8		Stackgas upstream from filter, 50% municipal waste, 50% wood chips
		0.03		Stackgas downstream, 50% municipal waste, 50% wood chips
Tulsa	1986	1.92 1.58 1.69	Schetter (1991)	TEF Eadon Stackgas downstream from filter
Coburg, FRG	1989	0.9	AbfallwirtschaftsJournal (1991)	Mixed Municipal and Hospital waste TEQ (NATO/CCMS)
Weust, NL	1990(?)	5.8	SVA (1991)	

Table 3

Occurance of PCDDs and PCDFs

Measured in	Total PCDD/PCDF	Reference
Urban Air	<5-10pg/m3	Jones and Bennet (1989)
	13.8pg/m3	Rappe at al, (1988)
	(70-120pg/m3 in industrial air)	
	0.1pg TEQ/m3	
	1.3-9.6pg/m3	Travis and Hattemer-Fry (1987)
Rural Air	<2pg/m3	Jones and Bennet (1989)
	1pg/m3	Rappe at al, (1988)
Urban Soil	12,000pg/g (mean)	Creaser *et al*, (1990)
	92.6ng/g	Yashura *et al*, (1987)
	6-460pg/g	Berlincioni and di Domenico (1987)
	51-9,100pg/g	Travis and Hattemer-Fry (1987)
Soil industrial area	< 135pg TEQ/g	Rappe and Kjeller (1987) de Jong *et al*, (1990)
Rural soil	452pg/g (mean)	DoE (1989)
	30-300pg/g	Fiedler *et al*. (1990b)
	5-7pg TEQ/g	Rappe and Kjeller (1987) de Jong *et al*, (1990)
Forest soil	100-200pg/g	Fiedler *et al*, (1990b)
UK soil	1-57pg TEQ/g	Rechem International Ltd (unpublished results 1991)
	< 0.5-1,400pg/g	DoE (1989)
Grass (UK)	1-43pg TEQ/g	Rechem International Ltd (unpublished results 1991)
Grass close to IWI (D)	0.7-8.8pg TEQ/g	Deister and Pommer (1991)
Soil close to IWI (D)	0.2-8.9pg TEQ/g	Deister and Pommer (1991)
Soil close to MWI	20-50pg TEQ/g	Rappe and Kjeller (1987) de Jong *et al*, (1990)
Ash from MWI	0.3-54ng TEQ/g	Hagenmaier (1987)
	24.4ng/g	Yasuhara *et al*, (1987)
	67-241ng TEQ/g (in landfill)	Stern *et al*, (1989)
Slag from MWI	20-250pg TEQ/g	Engelhardt (1985)
Ash from IWI	9-117pg TEQ/g	Rechem International Ltd (unpublished results 1991)
Slag from IWI	< 7pg TEQ/g	Brenner *et al*, (1986)
Water effluent	1.3-140pg TEQ/l	Rechem (1991)

Measured in	Total PCDD/PCDF	Reference
Potatoes	0.04pg TEQ/g	HMSO (1992)
Fish	ND-85pg/g (TCDD)	Travis and Hattemer-Frey (1987)
	18.1-43.4pg/g fat (TE BGA)	Beck *et al.* (1989b)
	0.48pg TEQ/g	HMSO (1992)
Meat	0.2-1.66pg/g fat (TE BGA)	Beck *et al.* (1989b)
	0.68pg TEQ/g	HMSO (1992)
Poultry	0.33pg TEQ/g	HMSO (1992)
Eggs	1.07pg/g fat (TE BGA)	Beck *et al.* (1989b)
	0.19pg TEQ/g	HMSO (1992)
Vegetable Oil	0.65pg TEG/g	HMSO (1992)
Vegetables	0.02pg TEQ/g	HMSO (1992)
Fruit	0.05pg TEQ/g fat	HMSO (1992)
Cow's milk (fat basis) Germany:		
Background	0.6-1.6pg/g (TE BGA)	Beck *et al.* (1990)
Industrial	0.9-3.8pg/g (TE BGA)	Beck *et al.* (1990)
Near metal reclamation plant	2.9-14pg/g (TE BGA)	Beck *et al.* (1990)
Netherlands		
Background	0.7-2.5pg TEQ/g	Liem *et al.* (1990)
MWI site	1.7-13.5pg TEQ/g	Liem *et al.* (1990)
United Kingdom		
Background	1.3pg TEQ/g	Startin *et al.* (1990)
Incinerator site	7.0pg TEQ/g	Startin *et al.* (1990)
Industrial area	5-7pg TEQ/g	Rechem (1991)
Derbyshire, industrial area	app. 70pg TEQ/g	HMSO (1992)
Human milk (fat basis)	195-1375pg/g	Jones and Bennett (1989)
	465-533pg/g (29.0-29.3pg TEQ/g)	DoE (1989)
	370-1600pg/g (25-41pg TEQ/g)	Beck *et al.* (1987)
Means of differant areas of Germany	375-715pg/g (30-36pg TEQ/g)	Beck *et al.* (1989c)
Human adipose tissue	850pg/g (22pg TEQ/g)	Byard (1987)
	96-014pg/g (in exposed 1560pg/g)	Jensen (1987)
	841-1460pg/g 75-1150pg/g 190-1374pg/g	Travis and Hattemer-Frey (1987)

Industrial Waste Incinerators. A general description of an IWI is given in Appendix D. Both the number of IWIs and the quantity of waste incinerated are substantially lower than for MWIs. IWIs are designed to provide maximum efficiency of waste destruction through ensuring that the following parameters are optimised:

- waste preparation and loading rate
- residence time
- turbulence
- temperature
- waste/oxygen ratio.

Ensuring very thorough mixing of the fuel/waste with oxygen is one of the most important factors (Acharya *et al*, 1991). Temperature itself is not the most important parameter, provided that a minimum temperature of 870°C is achieved. It is claimed that reaction kinetics of oxidation are not the limiting factor above such temperatures, and that the level of excess oxygen and temperature can only add small improvements once thorough waste/fuel and oxygen mixing have been achieved (Lee, 1988). It has been shown that the destruction efficiency of 2,4,8-trichlorodibenzofuran may be increased by a factor of 50 by raising the temperature to 870°C, or by increasing residence time by a factor of 4 (BUS-CH, 1986). IWIs commonly employ two-stage combustion with a secondary combustion chamber in order to provide greater residence time and improved turbulence through the physical design of this stage. The use of excess oxygen helps to overcome deficiencies in waste/fuel-oxygen mixing, although it has been demonstrated that increased oxygen raises the yield of PCDFs during combustion when other parameters are held constant (Dellinger *et al*, 1989, Song *et al*, 1992). In practice, however, attempts are made to optimise the advantages of all the above parameters.

Increased levels of PCDDs and PCDFs through reformation reactions, or so-called *de novo* synthesis, may be minimised when:-

- no secondary heat recovery is used
- rapid quenching such as by the wet process is used, and
- known precursors for PCDDs and PCDFs are not incinerated (Acharya *et al*, 1991).

Emission to the air. The atmospheric emissions of PCDDs and PCDFs from IWIs are shown in Table 4. Although the industrial wastes in IWIs normally contain more precursor substances than municipal waste, the concentrations of PCDDs and PCDFs

Table 4

Emissions of PCDDs and PCDFs to Air from Industrial Waste Incinerators (MWI)

Location of IWI	Year of Install-ation	Emission TEQ ng/m^3	Literature	Remarks
Schweinfurt, FRG	1965(?)	2-4.5	AbfallwirtschaftsJournal (1991)	TEQ (NATO/CCMS)
Schwabach, FRG	1972	0.8-0.9	AbfallwirtschaftsJournal (1991)	TEQ (NATO/CCMS)
Ciba-Geigy AG, Basel, CH	1974	0.018	Meister *et al*, (1988)	Pilot Plant, multistage washing procedure.
Nyborg, DK	1975	5.8	Demmich and Maurer (1987)	
Ebenhausen, FRG	1976	5.6-6.9 3.1	AbfallwirtschaftsJournal (1991)	1989 1990 TEQ (NATO/CCMS)
Biebesheim, FRG	1981	0.37 0.45	Demmich and Maurer (1987)	without PCB† charged. with addition 100kg PCB charged.
		0.5-1	Erbach and Schoener (1990)	without PCB charged.
BAYER AG, Brunsbuettel, FRG	1982	0.66	Mischer and Schnabel (1989)	trial with additional 8.4kg/h PCB and 63.0kg/h PCDM‡
		0.059	Mischer and Schnabel (1989)	trial with additional 0.34kg/h PCB and 18.7kg/h PCDM
Kumla, S	1983	15	Demmich and Maurer (1987)	outdated technology.
Netherlands 2 IWI	1987 -	1.2-2.3 27	Kleijn and van der Voet, (1991)	Older technology both TEQ (NATO/CCMS)
Rechem International Ltd., Pontypool, UK	1987	0.08	Rechem International Ltd., (unpublished results 1991)	approx. 4,000kg/d PCB combustion, new gas cleaning installed on existing incinerator (1987).
Rechem International Ltd., Fawley, UK	1990	0.06-0.15	Rechem International Ltd., (unpublished results 1991)	normal operation - no PCB combustion.

† PCB Polychlorobiphenyl

‡ PCDM Polychlorodiphenylmethane

emitted from IWIs are substantially lower than those from older MWIs, and generally still lower than those of the most advanced MWIs (cf Table 2). Notwithstanding, the total PCDD and PCDF emission from IWIs is estimated to range from 1.0 to 17% of that from MWIs (cf Tables 5, 6 and 7).

Emission via other routes. Slags and wash water have been examined for PCDD after burning chlorine-containing wastes in an IWI at Ludwigshafen (Brenner *et al*, 1986). The estimated levels of 2,3,7,8-TCDD were <5pg/g for slag and <1pg/g for wash water. The sum of the congeners from TCDD to OCDD was <7pg TEQ/g for slag and <2pg TEQ/g for wash water. Other reports of PCDDs and PCDFs in ash and water effluent (Table 3) range from 9 to 117pg TEQ/g and from 1.3 to 140pg TEQ/l respectively. It should be noted, however, that PCDDs and PCDFs present in slag, ash and waste water will generally have low mobility and tendency to disseminate into the environment. Studies in Japan (Yasuhara *et al*, 1987) indicated emission concentrations of OCDD of <0.01 and 0.73pg/g (ppt) in effluent water from MWIs and IWIs respectively.

Overview of Air Emissions from Different Combustion Sources. The total mass of PCDDs and PCDFs emitted from the main combustion sources in air in West Germany in 1990 was estimated by Fiedler and Hutzinger (1991) (cf Table 5). Hutzinger *et al*, (1990) estimated the total mass emitted per year for West Germany to be 1,700g TEQ of PXDDs and PXDFs including the bromo- and mixed bromo-chloro compounds.

The approximate amount of PCDDs and PCDFs emitted in the UK in 1989 is 3,870g TEQ (cf table 6), and the corresponding value for the Netherlands is 965g TEQ (Table 7). The differences between the figures for the total combustion emissions should not be considered as significant as the approaches used as well as the possible analytical errors may lead to differences of one order of magnitude.

Although not established on a common basis, the relative emissions of PCDDs and PCDFs from IWIs are compared to the total emissions from other combustion sources in the UK, the Netherlands and West Germany.

In Western European countries, where modern technology is widely used, the quantities of PCDDs and PCDFs released from IWIs are about 1% in relation to all other combustion sources (cf Table 8).

Table 5

Air emissions of PCDDs and PCDFs from combustion sources in West Germany in 1990 (Fiedler and Hutzinger, 1991).

Source	maximum g TEQ/year	%
Hazardous waste incineration	72	8
Municipal waste incineration	432	47
Non-iron metal plants	380	41
Steel mills	19	2
Car, leaded	7.2	0.8
Hospital waste incineration	5.4	0.6
Car, diesel	4.6	0.5
Sewage sludge	1.1	0.1
Home, oil	1.2	0.1
Home, coal + coke	1.1	0.1
Home, briq.	1.8	0.1
Car, unleaded, without Cat.	0.7	0.08
Car, unleaded, with Cat.	0.1	0.01
Cigarettes	0.01	0.001
Total	926	

Table 6

Emissions of PCDDs and PCDFs released into the atmosphere from combustion sources in the UK for 1989 (Based on DoE, 1981, but recalculated by using empirical comparison with a standard combustion pattern to express non-TEQ data in TEQ)

Source	g TEQ/year	%
Industrial waste incinerators	11	0.3
Municipal waste incinerators	1150	29.7
Vehicle exhaust	613	15.8
Other organic materials	559	14.4
Hospital incinerators	32	0.8
Industrial coal-fired plant	301	7.8
Coal-fired power stations	199	5.1
Domestic coal fires	989	25.6
Accidental fires	16	0.4
Total	3870	

Table 7

Air emissions of PCDDs and PCDFs from combustion sources in the Netherlands (Kleijn and van der Voet, 1991)

Source	maximum g TEQ/year	%
Hazardous waste incineration	12	1.2
Municipal waste incineration	790	82
Pesticides	50	5.2
Metal industries (total, including cable burning)	51	5.3
Hospital waste incineration	4	0.4
Chemical processes	5	0.5
Oil burning	2	0.2
Wood burning	16	1.6
Fires	9	0.9
Various	15	1.5
Total	965	

Table 8

Total and Relative Air Emissions from Industrial Waste Incinerators

Source	Country	Year	Total Amount of PCDDs and PCDFs emitted to the air from all IWIs in g TEQ/y	Relative emissions of PCDDs and PCDFs from IWIs compared to the emission from all combustion sources in %
Doe (1989)	UK	1989	11	0.3
Kleijn and van der Voet (1991)	NL	1991	12	1.2
Fiedler and Hutzinger (1991)	D	1990	0.5-72	1-8

4.2. ENVIRONMENTAL DISTRIBUTION AND FATE

4.2.1. Environmental Distribution

The compartments to which PCDDs and PCDFs may be released or to which they might transfer are determined by low volatility, low water solubility and a tendency to bind strongly to particulate matter of these compounds (Czuczwa and Hites, 1984; DoE, 1989;

cf Section 3). Consequently, when produced by combustion processes they are concentrated in the ash or fly ash. Particles emitted with stack gases are then distributed in the environment according to their size. The larger ones deposit on soil, water and vegetation in the vicinity of the stack; the smaller ones remain in the atmosphere much longer and, hence, are distributed over a much wider area (Anon, 1991). Eventually they are washed out by rainfall. Once in contact with the soil the PCDDs and PCDFs are strongly sorbed. Little migration occurs unless the soil itself is eroded (Young, 1987).

The PCDDs and PCDFs that fall into lakes, rivers or other aquatic systems tend to accumulate in sediments rather than in the aqueous phase although minor quantities may be volatilised (Podoll *et al*, 1986). Sediments from Lake Huron have trace (pg/g) amounts of PCDDs and PCDFs that have been present since the 19th century. Concentrations began to rise quite steeply from about 1940 onwards so that deposits in the 1980's range from 800 - 3,000pg/g. These values are expressed as total PCDDs and PCDFs. The major components are OCDD and HpCDF with only a small fraction being contributed by 2,3,7,8-TCDD. There is also some evidence to suggest that very little leaching or degradation of PCDDs and PCDFs takes place in the sediments (Czuczwa and Hites, 1984).

In the river Rhine, concentrations of 0.01ng - 310ng TEQ/l were measured in sediments originating mainly from industrial effluent rather than from atmospheric origin (Kleijn and van der Voet, 1991). Liquid effluents from incinerators provide another route for the distribution of PCDDs and PCDFs. Unpublished results by Rechem-UK indicate that typical emission levels for an IWI are a few pg TEQ/l.

Sanitary landfill and some dump sites which receive waste oils and chemicals may show several orders of magnitude higher concentrations (Goetz, 1986). Landfill operations may also be a source of airborne PCDD/PCDF emissions via dust generation.

4.2.2. Possible Environmental Transformations

Phototransformation. The main degradation process for dioxin is by reductive photolysis. This may take place in the atmosphere when PCDDs/PCDFs are adsorbed on airborne particles (Crosby, 1978). Molecules with lower chlorine contents are more readily phototransformed but the absolute and relative rates are unknown. Photolytic degradation of PCDDs and PCDFs involving an oxidation process is also known (Muto *et al*, 1991).

The photolytic half life of 2,3,7,8-TCDD vapour has been calculated to be about 200 hours (Podoll *et al*, 1986). In the case of OCDD, the half life was about 20 days (Choudhry and Webster, 1989).

Rate measurements showed that 2,3,7,8-TCDD is more rapidly photolysed in methanol than OCDD (Plimmer *et al*, 1973). The decomposition rates in solutions of 2,3,7,8-substituted congeners are faster than the rate of non-2,3,7,8-substituted congeners (Buser, 1976, 1979; Choudry and Webster, 1986; Tysklind and Rappe, 1991). The half life of 2,3,7,8-TCDD and 2,3,7,8-TCDF in extract solution were respectively 4.5 and 9.8 hours. The half-lives of the other 2,3,7,8-substituted congeners were respectively 17 - 39 and 2.1 - 10 hours (Tysklind and Rappe, 1991).

The rate of photolytic degradation may be lower in soil than in organic solutions. The half-life of 2,3,7,8-TCDD in soil, in the absence of UV light, has been estimated to be 10 years (Norris, 1981). In clear surface waters the half-life of 2,3,7,8-TCDD has been calculated as 118 hours in winter and 21 hours in summer. However, the strong adherence to particles and accumulation in sediments would seem to outweigh or delay photolytic processes (Podoll *et al*, 1986). Other evidence suggests photolysis is effective during initial dispersion but once binding to soil has taken place, the rate drops rapidly (Young, 1987).

OCDD photolyses to lower molecular weight products (Buser, 1976). A substantial difference exists in the photochemistry of OCDD at the soil surface, compared to that in solution. The formation of the more toxic 2,3,7,8 substituted congeners is more likely at the soil surface (Miller *et al*, 1989).

Biodegradation. Biodegradation of 2,3,7,8-TCDD has been demonstrated in the laboratory, albeit slowly. The white rot fungus (*Phanerochaete chrysosporium*) is capable of degrading 2,3,7,8-TCDD completely under ideal conditions (Hammel *et al*, 1986). It is not known whether this reaction occurs in the natural environment.

Laboratory studies have shown that highly chlorinated PCDDs and PCDFs are rather resistant to biodegradation. Di- and trichlorodioxins are, however, biodegradable (Bayer, 1989).

As PCDDs and PCDFs are strongly adsorbed to soil, their availability for biodegradation may be low. Laboratory studies in aqueous media and soil have shown that 2,3,7,8-TCDD is degraded very slowly, only 1% was transformed to the hydroxylated derivative after several months incubation (Huetter and Philippi, 1982).

4.2.3. Environmental Fate

All PCDDs and PCDFs are chemically and thermally stable under normal environmental conditions. Phototransformation and biotransformation reactions appear to be the only possible routes for environmental degradation.

Analysis of vegetation has demonstrated that when PCDDs and PCDFs are detected it is nearly always due to surface contamination rather than plant uptake (Jones and Bennet, 1989). Accumulation in animal tissue through ingestion of surface contaminated plants may be significant (cf Section 7).

In aquatic systems, PCDDs and PCDFs are mainly present in sediments and/or adsorbed on suspended solids. 2,3,7,8-TCDD has been shown to bioaccumulate in fish and other aquatic organisms from sediments and fly ash (Kuehl *et al*, 1985, 1987a, 1987b). The bioconcentration factors for 2,3,7,8-TCDD have been determined to be 66,000 for carp, 97,000 and 157,000 fathead minnow at 2 different exposure concentrations (NATO/CCMS, 1988c).

4.3. OCCURRENCE IN THE ENVIRONMENT

Many results have been published on the analysis of PCDDs and PCDFs in the environment, food products and samples of human origin. Table 3 gives an overview of the relevant data.

4.3.1. Levels in Soil and Grass - Impact of Nearby Incinerators

In forest areas concentrations of PCDD/PCDF of about 100 to 200pg/g have been found in upper soil layers corresponding to about 4 to 7µg/m² (Fiedler *et al*, 1990). Analysis of soils, not contaminated by point sources in Baden-Wuerttemberg, Germany, revealed concentrations of dioxins in the range of 30 to 300pg/g of soil. The amounts of furans were relatively low and comprised only 10 to 20% of the total concentrations (Fiedler *et al*, 1990). It was also shown in a study of the government of Northrhine-Westphalia that the levels of PCDDs and PCDFs are lower in summer than in Winter (NRW, 1991). The explanation could be that the contribution of the diffuse sources of home heating is much larger in winter.

The UBA (1990) reported the following maximum values for PCDDs for different matrices in upper soil layers:

Matrix	PCDD Concentration at max. (pg TEQ/g)
Soil	
Background	1
Immission area of:	
MWI	23
Cable smelter	29,000
Metal reclamation plant	7,930
Hospital waste incinerator	34
Roadside	261
Application of sewage sludge	260
Sewage sludge	1,000
Compost (from waste, single measurement)	50

Analysis of the top level of soil in the Netherlands and in Sweden resulted in an average of 5-7pg TEQ/g soil in rural areas, 20-50pg TEQ/g soil close to MWIs and up to 135pg TEQ/g soil in industrialised areas (Rappe and Kjeller, 1987; de Jong *et al*, 1990).

Total PCDD and PCDF levels in 78 random soil samples of the UK ranged from <0.5 to 1,400pg/g (DoE, 1989). In UK cities the levels in soil ranged from from 6 to 7,000pg/g. The mean level of 16 soil samples in Ireland, remote from industrial areas was 1.4pg/g with a standard deviation of 1.4 (Rechem, 1991).

Comparison of the levels of PCDDs and PCDFs in soil from sites close to an IWI in the UK, with both urban and suburban background levels can be made from the data in Table 9; the samples near the IWI exhibit no difference from background. More recent studies (Rechem, 1991) have shown that the levels of dioxins in soil and grass measured at 24 sites within 2 kilometres of an IWI, and at 90 background sites throughout South Wales, all fall within the range of 1 - 57pg TEQ/g and 1 - 43pg TEQ/g for soil and grass respectively. The area adjacent to the IWI is indistinguishable from other background areas with respect to concentrations of PCDDs and PCDFs.

Levels of 0.2 - 8.9pg TEQ/g in soil and 0.7 - 8.8pg TEQ/g in grass were reported from the vicinity of an IWI in Germany (Deister and Pommer, 1991). This indicates that the concentration ranges are beyond background levels of rural areas but are still below the levels in industrialised urban regions. There was no obvious correlation between the predicted immissions and PCDD/PCDF content determined in soil and grass. According to modelling studies (Deister and Pommer, 1991; Kleijn and van der Voet, 1991) the

maximum impact of PCDDs and PCDFs is predicted in the area about 500-750m of the chimney.

4.3.2. Levels in other media

PCDD and PCDF levels in air tend to be lower than 15pg/m^3 in urban locations, but may be higher in industrial areas for which values up to 120pg/m^3 have been reported. These levels are 1 to 2 orders of magnitude higher than remote rural locations, where <2pg/m^3 has been reported (cf Table 3).

While leafy vegetables might have been expected to show much higher levels than root vegetables or fruits, the very limited data reported did not support this. Reported values for cow's milk (on fat basis) are quite consistent and fall within the range of 0.7 - 7pg TEQ/g with peak values near point sources, i.e. up to 14pg TEQ/g near an MWI in the Netherlands. The highest value reported was approximately 70pg TEQ/g in an industrial area in the UK (cf Table 3). It is difficult to compare these values with human levels, but the observation that both human milk and adipose tissue have been reported levels considerably higher than those for cows milk is consistent with man being at the top of the food chain.

The solid wastes from incinerators are normally disposed of by landfill. Analyses of the top 15 cm of soil from a landfill site that had received bottom ash from a municipal incinerator without deliberate top soil addition showed PCDD and PCDF concentrations of 67-241pg TEQ/g (Stern *et al*, 1989).

Table 9

Comparison of Mean PCDD and PCDF Levels in Random Background Soil, Urban Soil and Soil near an IWI in Pontypool, UK (pg/g)

	Background UK†		Urban UK†		IWI UK‡	
TCDD	19	*36*	58	*42*	3.1	*2.5*
2,3,7,8,-TCDD	< 0.5	*1.0*			0.4	*0*
TCDF	48	*83*	140	*71*	14	*12*
PCDD	13	*22*	56	*50*	5.8	*6.7*
PCDF	48	*83*	120	*75*	7.5	*6.9*
HxCDD	63	*86*	160	*95*	15	*10*
HxCDF	68	*88*	120	*86*	9.6	*6.0*
HpCDD	98	*96*	350	*411*	123	*111*
HpCDF	43	*65*	130	*180*	30	*31*
OCDD	280	*290*	950	*1800*	680	*753*
OCDF	55	*100*	71	*110*	48	*48*

N.B. Numbers in *ITALICS* represent the Standard Deviation

† DoE (1989)

‡ Mundy *et al*, (1989)

SECTION 5 TOXICOLOGICAL PROFILE OF PCDDs AND PCDFs

5.1. INTRODUCTION

Four properties, common to certain PCDDs and PCDFs, influence their impact on biological systems:-

- a definite chemical configuration that favours a specific interaction with cellular targets ("receptors") is the basis of subsequent toxic action;
- very low water solubility combined with high lipophilicity leads to bioconcentration and biomagnification in the food chain and ultimately in man;
- a relative resistance to chemical and biological degradation;
- strong sorption capacities to solids and low volatility determining their dispersion into the biosphere adsorbed to particulates in air and sediments.

To evaluate the potential impact to human health three questions have to be considered:-

- which are the compounds which have these properties?
- which of the toxic effects are relevant to human health?
- at which dose level and by which route of exposure is there a risk to human health?

The purpose of this toxicological review is not to re-evaluate the large amount of data available since numerous overviews on the toxicity of PCDDs and related compounds were already published (US-EPA, 1985; Kimbrough and Jensen, 1989; WHO, 1989a; DoE, 1989; US-EPA/ATSDR, 1989; Skene *et al*, 1989; Rozman, 1989). The aim of this review is to summarise the most relevant information available in the context of the potential impact on human health. A section is devoted to the relevance of Toxicity Equivalency Factors (TEQ). Some details of the mechanisms of toxicological action are given in Appendix C.

Since oral intake is the major route (>90%) of human exposure (cf Section 7), this review is concerned mainly with toxicity data in relation to this route.

5.2. THE REASONS FOR THE FOCUS ON 2,3,7,8-TCDD

Although the environment is contaminated by complex mixtures of PCDDs and related compounds, one congener, 2,3,7,8-TCDD, was the particular subject of toxicological study.

This was because 2,3,7,8-TCDD was the first congener identified as specifically responsible in the past for a dermal disorder called "chloracne" observed occasionally among workers involved, particularly, in the production of trichlorophenol and trichlorophenoxy herbicides. When other congeners, including the PCDFs, were identified from several other sources (PCBs, waste incinerator effluents, other industrial processes, etc.) they were also toxicologically tested.

It was soon observed that the differences among congeners for the acute toxic effects were essentially quantitative. All active congeners elicited *in vivo* or *in vitro* toxic responses similar to those of 2,3,7,8-TCDD, this congener being the most toxic (McConnell *et al*, 1978; Nishizumi, 1978; Mason *et al*, 1985, 1986; Safe, 1987, 1990). It is for this reason that 2,3,7,8-TCDD has become the model congener for most studies.

Therefore the evaluation of the toxicological properties of all PCDDs and PCDFs, as well as their risk and hazard assessments, are essentially based on the data for 2,3,7,8-TCDD.

5.3. TOXICITY EQUIVALENCY FACTORS

5.3.1. Principle

In the early 1980s, there was a mandatory need to quantify the toxicological potential of mixtures of PCDDs and PCDFs detected in the environment, and scientists or regulatory agencies began to develop procedures for a quantitative evaluation based on the available toxicological data.

Although the dose level necessary to produce a toxic effect is different for each congener, the qualitative similarity of effects and the strong structure-activity relationship indicated a common mechanism of action and formed the basis of expressing the toxic potential of each congener by a factor ("toxicity equivalency factor" or TEF) (Safe 1987, 1990). This factor relates the toxic potency of a given congener to the toxic potency of 2,3,7,8-TCDD as a reference (NATO/CCMS, 1988a, b, c; Kutz *et al*, 1990; Safe, 1990). Initially, a series of different scales of TEF was suggested by various groups and authorities but at the initiative of the NATO/CCMS (1988a, b, c) a consensus was achieved on an internationally recognised scale of factors, the International Toxicity Equivilancy Factor (I-TEFs) (cf Table 10). Except where stated, the latter scale is used in this report.

Table 10

Toxicity Equivalence Factors

	BGA	Schlatter (1987)	Nordic equivalent Ahlborg, (1988)	Equivalent Factor NATO/CCMS (1988b)
2,3,7,8-TCDD	1	1	1	1
1,2,3,7,8 PeCDD	0.1	0.4	0.5	0.5
1,2,3,4,7,8 HxCDD	0.1	0.1	0.1	0.1
1,2,3,7,8,9 HxCDD	0.1	0.1	0.1	0.1
1,2,3,6,7,8 HxCDD	0.1	0.1	0.1	0.1
1,2,3,4,6,7,8HpCDD	0.01	0.01	0.01	0.01
OCDD	0.001	0.001	0.001	0.001
2,3,7,8-TCDF	0.1	0.1	0.1	0.1
2,3,4,7,8 PeCDF	0.1	0.4	0.5	0.5
1,2,3,7,8 PeCDF	0.1	0.01	0.01	0.01
1,2,3,4,7,8 HxCDF	0.1	0.1	0.1	0.1
1,2,3,7,8,9 HxCDF	0.1	0.1	0.1	0.1
1,2,3,6,7,8 HxCDF	0.1	0.1	0.1	0.1
2,3,4,6,7,8 HxCDF	0.1	0.1	0.1	0.1
1,2,3,4,6,7,8 HpCDF	0.01	0.01	0.1	0.01
1,2,3,4,7,8,9 HpCDF	0.01	0.01	0.01	0.01
OCDF	0.001	0.001	0.001	0.001

The total toxic equivalent (TEQ) of a given environmental sample is calculated by multiplying the amount of each congener present in the sample by its specific "toxic equivalency factor" and by adding up the total.

The advantage of this approach is to allow an approximate evaluation of the toxic potential of a source according to the pattern of congeners it emits. Such a scale also allows comparison between various sources, and helps communication among scientists and agencies on subjects of research, exposure levels, risk assessment and technology. However, it also suffers some important intrinsic limitations that will be discussed below.

5.3.2. Method

Each factor is defined according to the data available for the congener (Safe, 1990). The criteria used to assign the toxic equivalence factors are, when available:-

- information on the carcinogenic potency based on long-term animal studies;

- when this was not demonstrated or available, data on other effects such as reproductive or immunotoxic effects;

- acute toxicity data;

- otherwise, the results of *in vivo* and/or *in vitro* tests, with more weight given to data from *in vitro* receptor-binding tests and oxidative enzyme induction because of their better correlation with some *in vivo* toxicological end points such as immunotoxicity (thymic atrophy) or body weight loss.

In practice, for most congeners, no experimental data are presently available on carcinogenicity, reproductive effects or immunotoxicity. Therefore the TEFs are mostly based on limited information from acute toxicity and *in vitro* tests.

On such a basis a significant toxic potential was observed only for the 17 dibenzo-p-dioxin and dibenzofuran congeners with chlorine substitution at least on all four positions 2,3,7 and 8. For all other congeners, the toxicological potential was found to be more than 1,000 times less than that for 2,3,7,8-TCDD, and was by comparison considered to be negligible.

5.3.3. Limitations

The TEQ approach clearly suffers from some intrinsic limitations.

The basic assumption of the system is to consider that the effects of each congener are similar in nature, are dose-related and are simply additive. Although the effects for most congeners seem similar in nature, some data suggest that there can be some antagonism or synergism among the effects produced by different congeners (cf Sections 4 and 5).

A further limitation is that TEFs are based essentially on acute toxicity data or *in vitro* tests (receptor binding, enzyme induction, etc.). Although the relationship between structure and activity *in vitro* is rather good, the correlation between *in vitro* observations and toxicity observed *in vivo* is not well established. Moreover, it will be shown that the most critical potential toxic effects to human health are those resulting from chronic exposure to these compounds, such as reproductive effects, immunotoxicity or carcinogenicity. Therefore TEFs based mainly on acute and *in vitro* tests could over or underestimate the risk.

The TEFs of 2,3,7,8-substituted PeCDFs and HxCDFs, for example, based on *in vitro* tests and acute toxicity, are 0.1 and 0.01 respectively. However, results of subchronic toxicity studies in hairless mice gave evidence of tumour promoting properties and other toxic effects (splenic and thymic involution, liver hypertrophy, cell hyperplasia in the

glandular part of the stomach and body weight loss) at doses which would suggest a TEF of 2 - 4 fold higher for PeCDF and 8 - 16 fold higher for HxCDF (Hébert *et al*, 1990).

An additional limitation is that toxicokinetic parameters are not taken into account. The toxicokinetics of all congeners in all species are considered to be equivalent which, as shown below, is not the case.

The metabolism and elimination of congeners vary greatly. For example in the rat, the elimination half-life is 17 days for 2,3,7,8-TCDD, 30 days for 2,3,7,8-substituted PeCDDs, 110 days for 2,3,7,8-substituted HxCDDs and 270 days for 2,3,7,8-substituted HpCDDs. In man, the half-life of 2,3,7,8-TCDD is 100-fold longer than in the rat, i.e. 6 - 7 years (Poiger and Schlatter, 1986), but the liver concentration is only 20% of that observed in the rat and most is stored in the fat. If the human toxic dose is estimated on effects related to rat liver concentration, there is an overestimation by a factor 80 (Schlatter, 1990).

5.3.4. Evaluation of the Method

The present I-TEFs and TEQs do not give an accurate quantification of the toxic potential of PCDD and PCDF mixtures. From the scientific point of view it would be desirable that these factors would be regularly reassessed as new data become available, but for reasons of practicality this should only be done if major discrepancies become obvious, since legislation and guidelines are now frequently based on the I-TEF and TEQ approaches. Their replacement by a more appropriate method of toxicity evaluation is desirable, although not likely in the near future because of the multiplicity of compounds and the complexity of their mixtures. The US-EPA still considers the long-term toxicity assays as the ideal approach but a possibility is to use short-term *in vitro* or *in vivo* assays that could evaluate the effects of mixtures of PCDDs and related compounds as a whole (Safe, 1990).

In conclusion, it is essential not to give these factors and their applications more weight than is allowed by their limitations. They should be considered only as a temporary practical answer to the present need to quantify and rank the importance of the sources of PCDDs and related compounds in the environment.

5.4. ANIMAL TOXICITY OF 2,3,7,8-TCDD AND RELATED COMPOUNDS

5.4.1. Toxic Effects Resulting from Acute Exposure to PCDDs and PCDFs

The acute toxicity of 2,3,7,8-TCDD is extremely high to all animal species, and oral LD_{50} values range from 0.6µg/kgbw in guinea-pigs to 5,500µg/kgbw in hamsters. There are

large variations in susceptibility to 2,3,7,8-TCDD not only among animal species but also among strains and sexes. Death occurs after longer periods than normally observed in acute toxicity studies. The main effects resulting from acute exposure are skin disorders, a wasting syndrome, hepatotoxicity and effects on the reproductive and immune systems (WHO, 1989a).

The mechanisms of toxic effects are discussed in more detail in Appendix C. Many experts consider that most effects from exposure to PCDDs and PCDFs are receptor-mediated (Scheuplein, 1991; van der Heijden, 1991). This, however, is not a general consensus, and other possible mechanisms include interaction with the epidermal growth factor, oestrogen receptor, thyroid hormones and alteration of vitamin A metabolism.

5.4.2. Immunotoxic effects of PCDDs and PCDFs

2,3,7,8-TCDD. Immunodepressive and immunosuppressive effects of 2,3,7,8-TCDD have been observed in all animal species studied. These effects were recently summarised by Vos and Luster (1989). Thymic atrophy is one of the main characteristics of the acute and long-term effects of 2,3,7,8-TCDD, and the thymus is the main target organ in guinea-pig and mouse. Atrophy of the thymus particularly affects the cortical areas and the differentiation of T-lymphocytes. There is some correlation between thymic atrophy and effects on T-cell mediated immunity. All lymphoid organs (thymus but also lymph nodes, spleen) are affected by 2,3,7,8-TCDD over a wide range of doses. These specific effects vary between species and strains of animals and depend on the age of the animals with the guinea pig as the most sensitive species with a lowest effect level of 6ng/kgbw/wk.

Other PCDDs and PCDFs. Few studies have been performed with congeners other than 2,3,7,8-TCDD. Available data suggest that the effects on the immune system are similar to those reported for 2,3,7,8-TCDD (Vos and Luster, 1989). *in vitro*, a dose of 2,3,7,8-TCDF at least 10 to 30 fold higher than 2,3,7,8-TCDD is necessary to inhibit the antibody response (Vecchi *et al*, 1983).

in vivo, thymic atrophy has been noted in mice, rats, guinea-pigs and monkeys treated with 2,3,7,8-substituted PCDFs given as pure isomers or in mixture (Moore *et al*, 1979; Oishi *et al*, 1978; Pluess *et al*, 1989). Depressed IgG concentrations in mice and effects on cell-mediated immunity functions in guinea-pigs were observed by Luster *et al* (1979) only at a highly toxic dose of 2,3,7,8-TCDF (1µg/kgbw once a week for 6 weeks).

5.4.3. The Reproductive Toxicity of PCDDs and PCDFs

This question was extensively reviewed (Kimbrough, 1984; US-EPA, 1985; DoE, 1989; WHO, 1989a; Neubert, 1990). Even for 2,3,7,8-TCDD, a clear no-effect level has not been established for reproductive effects.

In mice, the lowest dose studied, 0.001µg/kgbw/d of 2,3,7,8-TCDD, produced an increased incidence of cleft palate although this was not observed at the next higher dose (Smith *et al*, 1976). The mouse is the only species with a well defined teratogenic effect. It is on this basis that a lowest observed effect level (LOEL) of 0.001µg/kgbw/d was suggested (Murray *et al*, 1979; Kimbrough, 1984).

In a three-generation rat study, foetotoxicity and reduced fertility were observed at 0.01µg/kgbw/d (Murray *et al*, 1979). While the authors considered the lower dose of 0.001µg/kgbw/d as a no-effect-level, Nisbet and Paxton (1982) re-examined the data and concluded that this latter level was a lowest-effect-level. Some aspects of the study protocol, however, were matter of discussion, and Kimbrough (1984) considered that this study could not be used for human risk assessment.

In non-human primates, the no-effect level for reproductive effects could even be lower than 1ng/kgbw/d but data are limited (Schantz *et al*, 1978; Allen *et al*, 1979; Barsotti *et al*, 1979). Postnatal behavioural changes, although without clinical toxic signs, were noted in one study at 0.12ng/kgbw/d (5ppt in diet) and body weight changes at 0.72ng/kgbw (25ppt in diet). Clinical effects were relatively well dose-related at 0.72ng/kgbw and above (Schantz *et al*, 1986; Bowman *et al*, 1989). Abortion was observed at 1ng/kgbw and possibly from 0.2ng/kgbw/d in a study by McNulty (1985). Palatal defects, stillbirths and abortions were noted at dose levels between 2 and 20ng/kgbw in a study that was reported in an abstract only (Zingeser, 1979).

These results indicate that foetotoxic effects occur in all animal species tested but teratogenicity is shown unequivocally only in some mouse strains. The evidence is thus insufficient to consider 2,3,7,8-TCDD as a human teratogen. The dose of 0.12ng/kgbw/d in the multigeneration rhesus monkey study can be considered as close to a no-effect level for the reproductive effects of 2,3,7,8-TCDD in non-human primates.

Recent data on 2,3,7,8-TCDD exposure of human palatal shelves in organ culture suggest that human embryonic palates are less sensitive than those of mice. A 1,000 times higher level of 2,3,7,8-TCDD would be required to elicit an effect on palatal differentiation (Abbott and Birnbaum, 1991).

With congeners other than 2,3,7,8-TCDD, higher dose levels are required to produce the same effects but some data (Birnbaum *et al*, 1987) suggest that the effects of exposure to several congeners can be more than additive.

5.4.4. Genotoxicity

The data on mutagenicity and genotoxicity studies of 2,3,7,8-TCDD were reviewed by many authors (Hay, 1982; Rogers *et al*, 1982; Giri, 1986; Zeiger, 1983; Shu *et al*, 1987) and several scientific panels (California Scientific Review Panel Discussions, 1985; US-EPA/ATSDR, 1989; WHO, 1989a).

Although Giri (1986) concluded that more testing was required, the conclusion of most other investigators on the basis of existing data is that PCDD and PCDF derivatives are non-genotoxic. There were very few positive responses in the mutagenicity studies and these results were either of limited quality or not confirmed by data in other laboratories.

5.4.5. Carcinogenicity of PCDDs and Related Compounds

Evidence of 2,3,7,8-TCDD carcinogenicity was demonstrated in various animal studies and particularly in the study by Kociba *et al* (1978) that served as the basis for human cancer risk calculations.

The US-EPA (1985) and the US-FDA performed a risk calculation with these data using a "linearised multistage model" (LMS). This model is usually considered suitable for genotoxic carcinogens, and assumes that there is no threshold dose for carcinogenic risk. The evaluation for 2,3,7,8-TCDD derived a risk of an increased frequency of 1 supplementary cancer for $1x10^6$ people exposed for their lifetime to a level as low as 0.006pg/kgbw/d (US-EPA, 1985) or 0.06pg/kgbw/d (US-FDA, 1983).

A re-examination of the liver sections of the female rat groups was performed recently by an expert group with the present histopathological criteria of classification of proliferative lesions in the rat liver. Because these criteria have significantly changed, this re-evaluation resulted in about two third fewer tumours than originally reported by Kociba *et al* (1978) with an increased incidence of hepatocellular carcinoma only in the high dose group and always in the presence of extensive hepatotoxicity. Furthermore, these tumours were diagnosed as "borderline" and equivocal and only 1 out of 4 was unanimously diagnosed as malignant by the expert group (Brown, 1991). On this basis, the No-Adverse-Effect-Level (NOAEL) was estimated 0.01µg/kgbw/d rather than 0.001µg/kgbw/day.

From these revised data and using the same approach as the EPA, Keenan *et al* (1991) calculated that the derived risk of increased frequency of cancer could be at least 10-fold lower than estimated initially.

Experts at a recent meeting concluded that the toxic effects of PCDDs and PCDFs were probably mediated through an hormone-like receptor mechanism. Such a mechanism of action indicates a minimal threshold level of effect (Scheuplein 1991; van der Heijden, 1991).

Therefore, since the mechanism of toxicity of 2,3,7,8-TCDD and related compounds is probably receptor-mediated and non-genotoxic by nature (cf 4.4 and Appendix C), it is considered, at least in Europe, that a chemical with a threshold effect level is inappropriate for evaluating the cancer risk. By neglecting a threshold effect an overestimation of the exposure related risk for human health could be made.

Furthermore, experimental data from several *in vivo* two-stage promotion tests, including the mouse skin and the rat liver (Cohen *et al*, 1979; Pitot *et al*, 1980; Di Giovanni *et al*, 1980) and the lack of genotoxicity, support the hypothesis that 2,3,7,8-TCDD acts as a tumour promoter. This action is usually characterised by a threshold dose.

Since the LMS model may not be appropriate for the evaluation of the carcinogenic properties of 2,3,7,8-TCDD, the US-EPA recently decided to re-evaluate the cancer risk.

On the basis of both the receptor-mediated model and carcinogenicity data, van der Heijden (199: WHO/Europe considered that a tolerable daily intake (TDI) for 2,3,7,8-TCDD could lie between 1 and 10pg/kgbw/day. This is much higher than the US-EPA or US-FDA values and is probably in better accordance with the experimental data on the toxic and carcinogenic effects of 2,3,7,8-TCDD and related compounds. It is also closer to other cancer risk assessments for dioxin compounds, such as WHO/EURO (1991), Canada, Japan and some European countries which established TDIs in the 1 to 10pg/kgbw/d range (US-EPA, 1988a, b).

5.5. HUMAN EFFECTS OF 2,3,7,8-TCDD AND RELATED COMPOUNDS

5.5.1. Acute Effects and Skin Effects

Death from acute exposure to 2,3,7,8-TCDD has never been observed in man, even after important accidental (Missouri, USA; Seveso, Italy) or occupational exposure (Vietnam herbicide sprayers, industrial accidental exposures at Monsanto, Boehringer or BASF) (WHO, 1989a). Chloracne is the most definite manifestation of human 2,3,7,8-TCDD

toxicity. This lesion is usually quite persistent and localised mainly on the face and upper body.

Other symptoms observed in man acutely exposed to chemicals contaminated with 2,3,7,8-TCDD were hyperpigmentation, hirsutism of the skin, hypertriglycideraemia, hypercholesterolaemia, aching muscles, digestive disorders, headaches, weight loss, loss of appetite, neuropathy, insomnia, loss of libido and sensory changes (Kimbrough and Grandjean, 1989). These were usually observed in workers with chloracne symptoms but the contribution of 2,3,7,8-TCDD to these symptoms is not known.

The main clinical symptomatology of people exposed to rice oil contaminated with PCBs and PCDFs ("Yusho" syndrome) included skin lesions (about 80% of exposed people), ocular manifestations (discharge, oedema and pigmentation of eyelids, 80% of exposed people), chronic bronchitis (40%), irregular menstrual cycles (60%), headaches, vomiting and diarrhoea (17-39%). Other symptoms included jaundice, stomatological alterations and hypertriglyceridaemia. Within the years that followed intoxication, symptoms such as skin lesions, ocular manifestations, stomatological alterations (except anomalies of teeth number and root shape), chronic bronchitis and hypertriglyceridaemia progressively decreased in severity and some returned to normal (Reggiani, 1980).

The symptomatology of a similar accident in Taiwan (Yu-Cheng disease in 1979) is comparable, with some additional symptoms such as visual disturbances, hearing difficulties and persistent bronchitis (WHO, 1989a).

These accidents allow the evaluation of the human body burden of PCDFs associated with toxic symptoms. Examination of the Yusho case (Ryan *et al*, 1990) showed that the mean uptake or body burden of 2,3,4,7,8-PeCDF associated with nausea and anorexia was 4.4µg/kgbw and that associated with chloracne to be 5.9µg/kgbw. For the Yu-Cheng case, blood measurements showed a similar body burden of 4µg/kgbw for chloracne symptoms. This latter value is equivalent to a 2,3,7,8-TCDD-TEQ amount of 2µg/kgbw or about 140µg for an adult person (Ryan *et al*, 1990), about 1,000 times the average current level of exposure in Europe (cf Section 7). Earlier, Wilson (1987) calculated from the rice oil consumed by the victims of the Yusho accident and from measurements of the oil composition that the median dose of total PCDFs causing "severe" symptoms was about 68µg/kgbw. Using the same estimates as those of Ryan *et al* (1990) about the congener pattern, the value of Wilson (1987) corresponds to a dose producing toxic symptoms of about 5.9µg PeCDF/kgbw. However in neither study, did the body burden evaluation take account of the presence of polychloroquaterphenyls at about 100 times greater

concentration than PCDFs and this could either increase or decrease the toxic response due to PCDFs by an unknown amount.

5.5.2. Immunological Effects

In the study performed on people exposed to PCDFs and PCBs in Taiwan (Yu-Cheng disease), it was shown that IgA and IgM, but not IgG, were decreased and that the number of positive response to skin tests for delayed-type hypersensitivity were significantly reduced and correlated with the severity of dermal lesions.

Several studies of human exposure to 2,3,7,8-TCDD after the Missouri episode, where TCDD-contaminated oil was spread on soil to reduce dust, reported reduced hypersensitivity responses, small effects on T-helper cells and cytotoxic lymphocytes as the only statistically significant difference from a control population. However, as observed by the authors themselves, most of these observations were artefacts and only the effect on T-helper-cells was confirmed by later studies. This effect is without known pathological consequence (Andrews, 1989).

The follow-up of the Seveso accident also failed to demonstrate immunological disorders in exposed people, such as abnormalities in serum immunoglobulin concentrations (Reggiani, 1980, 1989).

In conclusion, there is no evidence that the human species is particularly sensitive to the immunotoxic effects of PCDDs and PCDFs.

5.5.3. Reproductive effects

Epidemiological studies so far showed no evidence for a teratogenic potential of 2,3,7,8-TCDD in man (Neubert *et al*, 1973; Neubert 1990). No increase in the frequency of birth defects was observed among women directly exposed during the Seveso accident in 1976 (Mastroiacovo *et al*, 1988; Mocarelli *et al*, 1989; Neubert, 1990). There are limitations to this observation including the small population exposed and the number of induced abortions after the accident.

Some data on human reproductive effects, initially attributed to 2,3,7,8-TCDD alone, were later related to exposure to other chemicals such as 2,4,5-T contaminated by 2,3,7,8-TCDD. None of the reported associations unequivocally identify either 2,4,5-T or 2,3,7,8-TCDD as the causative agent (Hanify *et al*, 1981; Smith *et al*, 1982a, b; US-EPA, 1985, 1988a, b). Apart from the fact that the populations were exposed concomitantly to chemicals other than 2,3,7,8-TCDD, the main limitations were lack of quantitative data on

exposure of individuals and the size of the group did not allowed appropriate statistical evaluation. The characteristics and limitations of studies on reproductive toxicity of 2,3,7,8-TCDD in exposed populations (Reggiani, 1980; Thomas, 1980; Smith *et al*, 1982b) were similar to those discussed for immunotoxicity.

Although interferences of 2,3,7,8-TCDD with fertility in man were suspected and publicly debated in the past, no indication is available that such a relationship exists (Neubert, 1990) or that it induces paternal congenital abnormalities i.e. in Vietnam veterans (Fingerhut *et al*, 1987). This issue, however, remains controversed by veteran associations.

Finally, the relative similarity of the reproductive processes in the rhesus monkey and the human species and the similar half-life (about 6-7 years for 2,3,7,8-TCDD) in both species suggest that the data on monkeys are particularly relevant for human health risk evaluation.

From the results available on human exposure and particularly from the follow-up of the Seveso accident, it can be considered that human beings are at least no more sensitive to reproductive toxic effects than laboratory animals.

5.5.4. Carcinogenic effects

Several human groups have been exposed or are assumed to have been exposed to PCDDs and/or PCDFs. These include workers or populations occupationally or accidentally exposed during the production of chemicals such as trichloro- and pentachlorophenols or chlorophenoxy herbicides, and their use (i.e. herbicide sprayers).

The epidemiological studies on these groups suffer from a number of weaknesses. The major problem is that these people were always exposed to PCDDs and PCDFs only present as contaminants of other chemicals. Therefore, it is very difficult to determine whether the effects observed are due to the PCDDs or PCDFs themselves.

Another important problem encountered in the interpretation of most epidemiological studies so far published is that, until recently exposure estimates were always on a questionnaire basis because there was no analytical method to measure the true levels of exposition.

For example, in a Missouri village, people were exposed to the spraying of soil with a waste oil contaminated with PCDDs and PCDFs. The persons were initially classified by an exposure index based on such a questionnaire. However, 40 percent of those

considered as "highly exposed" had actually low exposure as shown recently by adipose and serum dioxin measurements (Houk, 1991). The same was observed for the US Vietnam veterans. Despite being classified as heavily exposed or little exposed by exposure indices, the distribution of serum dioxin levels measured later were for all virtually the same as in the unexposed controls. Even for the veterans who specifically served in the spraying of the herbicides (the "Ranch Hand") and had distinctly elevated serum dioxin levels, the exposure index based on the questionnaire showed to be not acceptable for exposure classification. As it was concluded by Houk (1991), the interpretation of the epidemiological studies based only on such index classification is clearly impossible.

A further problem with epidemiological studies on PCDDs and PCDFs is that the exposed groups were usually small and therefore these studies have a low power to detect an effect.

Several reviews can be found i.e. in US-EPA (1985) or in WHO (1989a). These studies are also regularly updated and completed and therefore this review focuses on the recent updates of the most significant of these studies (cf table 11).

The recent extensive epidemiological study from the US-NIOSH examined the possible linkage between professional exposure to 2,3,7,8-TCDD and cancer on 5,172 workers exposed between 1942 and 1984 (Fingerhut *et al*, 1991). The mortality rate in the overall cohort was 1.15 times higher than expected. A subgroup of 1,520 men with more than one year exposure and more than 20 years latency with 2,3,7,8-TCDD were found with blood levels about 60 times the normal population. The excess of mortality from cancer (expressed as standard mortality ratio (SMR) with a 95% confidence interval) in this subgroup was 1.46 for cancer of all sites, and statistically significant for cancer of the respiratory tract (SMR 1.42) and for soft tissue sarcoma (3 cases, SMR 9.22). The increased cancer rates observed in previous studies (stomach, liver, nasal cancer, Hodgkin disease and non-Hodgkin sarcoma) were not confirmed. The conclusions about the increased risk of mortality from respiratory tract cancer and soft tissue sarcoma were limited due to the possible contribution of other chemicals and of smoking.

Table 11

Continued Epidemiological Studies on Workers Occupationaly Exposed to TCDD

COHORT (exposure)	No. of workers	Mortality from malignent neoplasms		S M R (95% CI)	Reference Group	Reference
		Observed	Expected			
BOEHRINGER, (occupational, 1952-1984) Men, all neoplasms						
total cohort	1148	93	75.2	1.24 (1.00-1.52)	West Germany	Mantz *et al*, (1991)
		75	53.8	1.39 (1.10-1.75)	Gas workers	
high exposure	459	34	23.9	1.42 (0.98-1.99)	West Germany	
		29	16.3	1.78 (1.19-2.55)	Gas workers	
medium exposure	613	50	45.1	1,11 (0.82-1.46)	West Germany	
		39	32.6	1.20 (0.85-1.63)	Gas workers	
low exposure	76	9	6.2	1.45 (0.66-2.75)	West Germany	
		7	4.8	1.45 (0.95-2.99)	Gas workers	
employment >20y and high exposure	49	8	3.1	2.54 (1.10-5.00)	West Germany	
		7	2.3	3.07 (1.24-6.33)	Gas workers	
employment <20y and low exposure	69	8	5.6	1.42 (0.61-2.79)	West Germany	
		7	4.4	1.60 (0.64-3.29)	Gas workers	
NIOSH WORKERS FROM 12 PLANTS IN 11 US STATES (occupational, 1942-1984)						
total cohort	5172	265	229.9	1.15 (1.02-1.30)	US population	Fingerhut *et al*, (1991)
<1 year exposure	1516	48	46.8	1.02 (0.76-1.36)	)subgroup with >20	
>1 year exposure	1520	114	78.0	1.46 (1.21-1.76)	)years of latency	

Table 11 (ctd.)

COHORT (exposure)	No. of workers	Mortality from malignent neoplasms		S M R (95% CI)	Reference Group	Reference
		Observed	Expected			
BASF (1953)						
total cohort	247	23	19.68?	1.17 (0.80-1.66)	natioanl German mortality rates and 90% CI	Zober *et al*, (1990)
cohort >20 years employment		16	?	1.17 (0.80-1.66)		
basic cohort (all with chloracne)	69	9 8	6.94 3.37	1,31 (0.68-2.26) 2.38 (1.18-4.29)	second cohort (total) second cohort (>20yr exposure only	
second cohort (17 with chloracne)	84	11	6.45	1.71 (0.96-2.83)		
third cohort (9 with erythema)	94	3	6.29	0.49 (0.13-1.23)		
HERBICIDE SPRAYERS (total cohort 18910)						
TCDD exposure						
probable		236	215	1.05 (0.96-1.25)		
unlikely		279	293	0.95 (0.84-1.07)		
Years since first exposure						
>30		74	88	0.84 (0.66-1.05)		
20-29		171	159	1.07 (0.92-1.25)		
10-19		170	166	1.02 (0.87-1.19)		
0-9		100	94	1.06 (0.86-1.29)		
soft tissue sarcoma						
>30		0	0.24	0 (0-15.37)		
20-29		0	0.52	0 (0-7.09)		
10-19		4	0.66	6.06 (1.65-15.52)		
0-9		0	0.50	0 (0-7.38)		

In the most recent update of the mortality study in a group of workers exposed after an accidental release of 2,3,7,8-TCDD in 1953, an increased frequency of death from malignant neoplasm was observed in exposed people 20 or more years after the accident. The types of cancer observed were respiratory cancer and gastrointestinal cancer (Zober *et al*, 1990). In an evaluation of the same cohort, Rohleder (1990) considered non significantly exposed people were included in the cohort of exposed people and that this could have "diluted" the observed cancer incidence.

Another study recently published examined the mortality follow-up of 1,583 workers employed in a chemical plant that produced herbicides contaminated with 2,3,7,8-TCDD (>1,000ppb in 2,4,5-T and up to 60mg/kg in production waste) (Manz *et al*, 1991). Moderate, although statistically significant higher, mortality due to malignant cancer was observed among workers with 20 years or more employment and among men who began employment before an interruption of the production in 1954, due to an outbreak of chloracne. In a group of volunteers from the group classified as "highly exposed", the adipose tissue range averaged 296ng/kg 2,3,7,8-TCDD as compared to a median of 83ng/kg in volunteers of the medium and low exposure groups combined. An increased incidence of cancer was observed in many sites except for the colon but none was statistically significant (all SMR between 1 and 2 with 1 included in the 95% confidence interval).

The lower risk of mortality from cancer observed in the group employed after 1954 was attributed to a reduction of 2,3,7,8-TCDD contamination. The authors concluded a relationship, possibly dose-dependent, between duration of exposure and increased risk of mortality due to cancer. As for the other studies the influence of exposure to other carcinogenic chemicals could not be excluded.

Saracci *et al* (1991) reported the results of a historical cohort study of mortality among 18,910 workers from ten countries employed for production and spraying of chlorophenoxy herbicides, including those significantly contaminated by 2,3,7,8-TCDD. The total mortality was lower than expected on basis of national statistics and no excess of mortality related to most common epithelial cancer or soft tissue sarcoma were observed. Increased and also decreased risks were found for some uncommon cancer but the small numbers limited the interpretation. An excess of mortality due to soft tissue sarcoma was also claimed and discussed. However, there were only four cases and two of these were related to exposure to herbicides not specifically contaminated by 2,3,7,8-TCDD. Furthermore, the excess was significant simply because all the four cases were only in the particular subgroup with a latency period comprised specifically between 10 and 19 years but not longer while the study covered up to 30 years.

The results of this study obtained on a very large group of exposed workers did not confirm statistically the hypothetical correlation between exposure to 2,3,7,8-TCDD and soft tissue sarcoma or lymphoma suggested by some earlier more limited epidemiological case control studies (Hardell and Ericksson, 1981; 1988; Ericksson *et al*, 1981; 1991; WHO, 1989a).

Up to now the epidemiological studies on people exposed to 2,3,7,8-TCDD in Vietnam or in Seveso failed to reveal carcinogenic effects. In these cases, however, the follow-up periods after the exposure are too short to conclude, given the latency needed for cancer development, and some controversy remains. The long-term follow-up of the Seveso exposed population will probably be the only possibility to conclude about the carcinogenicity of 2,3,7,8-TCDD in man.

Meanwhile, the observation that a possible weak carcinogenic effect, if any, would only be observed in heavily exposed workers is important because the levels of exposure (see section 7) of the general human population are far below those to which these groups of workers were accidentally exposed and since there is probably a threshold level for the toxic effects of TCDD.

5.6. CONCLUSIONS: ESTIMATION OF A HUMAN ACCEPTABLE EXPOSURE LEVEL

The key experimental toxicity data relevant for an evaluation of a no-effect level are summarised below:-

Type of effect	Minimal or no effect level in the most sensitive species.	Reference
enzymatic induction	3ng/kg (rat, monkey)	Krueger *et al*, 1990
acute toxicity	4.86ng/kgbw (guinea pig)	de Caprio *et al*, 1986
chronic toxicity	1ng/kgbw (rat, liver toxicity)	Kociba *et al*, 1978
immunotoxicity	6ng/kgbw/d (guinea pig)	Vos *et al*, 1973
effects on reproduction	1-10ng/kgbw (rodents)	Murray *et al*, 1979
	0.72ng/kg/d (monkey, clinical) 0.12ng/kg/d (monkey, behavioural)	Bowman *et al*, 1989
carcinogenicity	100ng/kg/d (rat, carcinoma)	Brown, 1991
	10ng/kg/d (rat, adenoma)	re-evaluation of Kociba *et al*, 1978

Strong indications from human data and epidemiological studies provide evidence that, compared to experimental animals, the human species is not particularly sensitive to the toxic effects of 2,3,7,8-TCDD and other congeners, because:-

- there is no known case of death due to acute exposure to 2,3,7,8-TCDD although high exposure levels are known or have been measured: up to 18,000ng/kg in Missouri residents, up to 31,000ng/kg in workers (New Jersey plant), mean of 12,000ng/kg in 19 Seveso residents with up to 56,000ng/kg in one case (Needham *et al*, 1991);

- except for chloracne, there is no known human toxic effect attributed to 2,3,7,8-TCDD alone. Concerning the PCDFs, it was calculated from the contaminated oil episodes that the median dose causing severe symptoms in man was in the µg/kgbw range;

- there is no reported human case of immunotoxic effect or reproductive effect attributed to 2,3,7,8-TCDD, and the small increase of mortality from cancer in groups of heavily exposed workers cannot be unequivocally attributed to this compound alone.

On the basis of the No Observable Adverse Effect Level (NOAEL), a safety factor of 100, including a factor of 10 for species extrapolation and a factor of 10 for individual-to-population extrapolation can be considered as conservative enough for a risk assessment of the impact of the PCDDs and PCDFs in the human health.

The data in animal experiments, including the most sensitive species (see above), allow to conclude that this NOAEL is comprised between 100 and 1,000pg TEQ/kgbw/d. This takes into account the subclinical behavioural effects observed in monkeys although these are not clearly demonstrated and the fourfold accumulation of 2,3,7,8-TCDD observed in monkeys although this seems not to occur in human breast-fed infants (Neubert *et al*, 1991). By applying a safety factor of 100, it can be assumed that a lifetime dose of 1 to 10pg TEQ/kgbw/d would not affect human health, including that of children and could be considered as a tolerable daily intake.

Such range is comparable to that recently adopted by WHO-Europe and set in the European countries (UK, The Netherlands, Germany, Swiss Institute of Toxicology) where risk assessment on dioxins and related compounds were conducted and allowable daily intake based on no-adverse-effect-levels.

SECTION 6 PRESENT APPROACHES FOR CONTROLLING EMISSIONS FROM INDUSTRIAL WASTE INCINERATION

Incineration is an appropriate method of treatment for all types of wastes, especially for organic matter and for many kinds of hazardous wastes such as halogenated hydrocarbons (CHC) polychlorinated biphenyls (PCBs), process waste water containing organic materials, sewage sludges, contaminated solvents, soil etc. Incineration can simultaneously detoxify the waste and significantly reduce its volume. The end products of incineration can usually be disposed of in a safe and economical manner. Certain end products may need an after-treatment because of their content of toxic heavy metals as well as halogenated dioxins and furans, e.g. the pyrolysis of fly ash.

Appropriate incineration conditions depend on the nature of the waste to be treated, hence it is necessary to determine the chemical composition and physical properties of the main constituents of each waste. The nature of wastes to be incinerated determine the technical aspects of the incinerator and also the conditions to be applied. It is possible and necessary to optimise the incineration process especially in cases of highly stable substances for a complete burning-out of the waste, a minimisation of the concentration of organic, inorganic and metallic particulate or gaseous substances in the stack gas and in all other emission streams. Essential requirements of an IWI are given in Appendix D.

6.1. PRESENT PROCEDURES TO REDUCE PCDD AND PCDF EMISSIONS

The present state-of-the-art procedure for minimising dioxin emissions, consistent with the parameters described above and no energy is recovered, involves one of the two following processes (Acharya *et al*, 1991):

6.1.1. Semi-dry Process

In this procedure the hot combustion gases are quenched with water under controlled conditions so that significant quantities of water are not carried forward to the next stage of gas cleaning. Quenching is then followed by dry scrubbing in which the combustion effluent is intimately in contact with lime or other alkali, and the gases are finally passed to electro-static precipitators or a baghouse for particulate removal.

6.1.2. Wet Process

The combustion gases are quenched and partly scrubbed with a water deluge and then immediately passed to a wet alkaline scrubber system typically using sodium hydroxide.

Following scrubbing, the combustion gases are routed to electro-static precipitators, as wet gases are generally unsuitable for particulate polishing in a baghouse.

Using either of these methods, it has been demonstrated that, when conditions are favourable, a substantial decrease of emission may be achieved. It is common to observe emission levels of <0.1 to 5ng TEQ/Nm^3.

6.2. OTHER PROCEDURES TO REDUCE PCDD AND PCDF EMISSIONS

A number of other procedures are presently being evaluated for their suitability in reduction of PCDDs and PCDFs. Although offering no near-term solution to the emission of these species, brief descriptions are given below.

Experiments on IWI boilers by adding urea, ammonia, triethanolamine and other tertiary amines at temperatures of 400°C show that there is a decrease of the PCDD/PCDF concentration. Whether this will be sufficient to reach levels of about 0.1ng TEQ/Nm^3 is not clear and further measurements must be made.

Filters in both the crosscurrent in the countercurrent mode have been tested in IWI pilot plants. One type of IWI has been built using such a technique (RZR Herten, D).

In some cases, a dry absorption system is used to trap PCDDs and PCDFs. After conditioning the gases with water, lime or a mixture of lime and activated coke (2%) is injected. After separating most of the fly ash in a special settling chamber, the remainder is removed in bag filters (Schoeneiche, D).

To minimise NO_x concentrations of up to 400ng/Nm^3, selected catalytic reduction (SCR) is used in power plants. New investigations show, that an SCR unit in oxidising mode may also cause partial removal of PCDDs and PCDFs. In the case of IWIs, the lifetime of the catalyst would probably be shorter.

SECTION 7 PRESENT HUMAN EXPOSURE LEVELS TO PCDDs AND PCDFs

The assessment of the hazard of a substance to cause an adverse effect on man can be expressed by the comparison (ratio) of an overall no-effect concentration (NOEC) with the predicted exposure concentration. No-effect concentrations are discussed in Section 5. For PCDDs and PCDFs, exposure is expressed operationally according to the TEQ approach, using the international toxic equivalency factors (I-TEFs) method.

Quantitative exposure estimations to 2,3,7,8-TCDD and other congeners via various possible routes were recently discussed by Theelen (1991) and specifically for food intake by HMSO (1992). Representative data for the occurrence of these compounds in various media are discussed here and reported in Table 3.

7.1. ROUTES OF EXPOSURE

Exposure assessment can be made according to a pathway approach including the main exposure routes; inhalation, dermal contact and oral ingestion. A further distinction can be made between direct and indirect contact. For PCDD and PCDF exposures, direct contact is via particles of contaminated air and soil. Because of the lipophilicity of these compounds, the contamination via water is very limited and can be omitted. Indirect exposure include mainly the food chain.

7.1.1. Exposure via air.

The levels of 2,3,7,8-TCDD and related congeners in air and particulate were evaluated in various conditions. Range from 0.1 to 2pg TEQ/m^3 total air were detected in ambient air samples from West Germany and in close proximity of an incinerator (Christmann *et al*, 1989). Using these values, Theelen (1991) calculated a daily uptake of 2pg TEQ via inhalation and an additional uptake of approximately 1pg TEQ by the gastro-intestinal tract.

7.1.2. Exposure via soil

Analysis of the top layer of soil in The Netherlands and Sweden resulted in an average of 5 - 7pg TEQ/g soil in rural areas, 20 - 50pg TEQ/g soil close to municipal waste incinerator and up to 135pg/g soil in industrialised areas (Rappe and Kjeller, 1987; de Jong *et al*, 1990). From this basis and assuming a dermal application of 1mg of soil/cm^2 of skin, Theelen (1991) calculated an uptake of 0.15pg TEQ/d with an additional uptake of 0.1pg/d via ingestion of 15mg of soil.

It should be noted that these estimates represent the total intake and not actual uptake by the mammalian organism. Indeed, despite their high lipophilicity, these compounds are relatively highly adsorbed to airborne or soil particulate and only a fraction is absorbed by the mammalian organism. From various experimental studies, Theelen (1991) concluded that only about 2% of 2,3,7,8-TCDD intake from air and soil is really absorbed.

7.1.3. Exposure via the food chain

While there is little uptake by plants from the soil (Jones and Bennett, 1989), dust deposit on vegetation and subsequent absorption by animals is considered as the main source of human exposure to PCDDs and PCDFs. From analytical results in various food samples (vegetables, meat, chicken, eggs, dairy products, fish and others, cf Table 3) and from food consumption data, tentative estimates for the average dietary intake of PCDDs and PCDFs by various populations were done. The estimates for the Netherlands, U.K. and Germany are given in Table 12; details of the calculations can be found in the original references. These various estimates are strikingly similar and can be considered as representative of the situation in the European industrialised countries.

On this basis, the mean human daily intake can thus be estimated in the range of about 120pg per person or about 2pg/kgbw/d for a 60kg person. Somewhat different calculations can be made depending on whether the analytically undetected congeners were considered or not as being present at half the limit of detection.

Studies carried out in Canada (HMSO, 1992, Birmingham *et al*, 1989) and in Japan (Ono *et al*, 1986) gave a comparable estimated daily intake of 125 or 84pg TEQ/day, respectively (recalculated by Travis and Hattemer-Frey, 1989).

7.2. HUMAN TISSUE LEVELS

7.2.1. PCDD and PCDF levels in human tissues

From the various studies available (Schecter, 1991), a high geographical variation of PCDD and PCDF human tissue levels was evident. Relatively higher levels are found in industrialised countries. As observed by Schecter (1991), within a given country, tissue levels reflect food intake. Blood analyses of the general population were only recently analytically possible. On a lipid basis, the blood levels observed in samples from West Germany, Canada, Japan and the United States were about 40pg TEQ/g.

Table 12

Human Exposure to 2,3,7,8 TCDD and Related Congeners in Various Countries

Estimated Daily Exposure expressed in pg TEQ			
Route/Source	Netherlands	U.K.	West Germany†
Inhaled air	2		
Ingested air	1		
Soil via skin	0.15		
Soil ingested	0.1		
Total air and Soil	**3.2 (3%)**	-	-
Vegetables	1.8-7	15	3.7
Vegetable oil	14	19	0.3
Cereals		5.3	
Total vegetable	**18.5 (16%)**	**39.3 (31%)**	**4.0 (4.3%)**
Pork	4.2		
Beef	13		
Chicken	4.8 (with eggs)	5.6	
Eggs		4.6	4.2
Total meat & eggs	**22.0 (19%)**	**42.2 (34%)**	**27.7 (29.6%)**
Cow's milk	17	23	
Cheese & butter	26	12	
Total dairy product	**43.0 (36%)**	**35.0 (28%)**	**28.5 (30.5%)**
Seawater fish	14		
Freshwater fish	10		
Fish oil	7.2		
Total fish	**31.2 (26%)**	**7.7 (6%)**	**33.3 (35.6%)**
Total food	**118**	**125**	**93.5‡**
Reference	Theelen, (1991)	HMSO, (1992)	Beck *et al*, (1989b)

† calculated with German TEFs.

‡ equivalent to 203pg TEQ/d, recalculated with I-TEFs from German TEFs by HMSO (1992)

Adipose tissue levels of PCDDs and PCDFs were also measured in individuals from various countries and it was shown by the Center of Diseases Control in the U.S. that serum dioxin levels were highly correlated with adipose levels (Houk, 1990). The levels observed varied from 38pg/g in Japan to 69pg/g in Germany (Schecter, 1991) and the background adipose levels of 2,3,7,8-TCDD is lower than 25% of the total TEQ. Houk

(1990) calculated that a continuous daily dose of 1pg/kgbw is necessary to maintain the background level, a value in the range of that considered as Tolerable Daily Intake.

7.2.2. Human milk and exposure via breast feeding

Levels of 2,3,7,8-TCDD and congeners in human milk were measured within an international study program carried out by WHO (1989b). Levels varied between 3pg TEQ/g in Thailand and Cambodia, 20pg/g in USA and 26 - 27pg/g in Canada and Germany. Similar results were reported by other authors (cf Table 3). The corresponding intake of these compounds by breast feeding was estimated in the order of 100pg/kgbw/d (WHO, 1989a). This level is relatively high as compared to the TEQ daily intake of the adult. However, this intake occurs only during a limited part of the lifetime and WHO clearly considered that the benefits of breast feeding outweighed any risks from PCDDs and PCDFs exposure.

7.3. PHARMACOKINETIC ASPECTS

The pharmacokinetic parameter, although not easily taken into account in most risk assessments, is one of the most important determinants of the toxicity of chemicals in general and of PCDDs and PCDFs specifically. The elimination half-life of 2,3,7,8-TCDD in human was calculated to be 6 - 12 years (Poiger and Schlatter, 1986, Houk, 1990). This is approximately 100 times longer than in experimental animals at dose levels above the no-effect level (Schlatter, 1991). The half-life of hexa-, hepta- octa- and other 2,3,7,8-chlorosubstituted congeners is in the order of 15 - 50 years (Schlatter, 1991). Important differences with regard to organ distribution were also observed. In rat more than 60% of 2,3,7,8-TCDD and 90% of most of the 2,3,7,8-substituted congeners are stored in the liver whereas in man, more than 90% are in the adipose tissues (Schlatter, 1991). These observations could explain the difference in susceptibility to the toxic effects between some animal species and man (Needham *et al*, 1991).

Up to now, these kinetic and dynamic parameters were not really taken into account in the TEQ approach while the risk assessment of PCDDs and PCDFs is essentially based on animal data. As underlined by Neubert (1990; 1991), (cf Section 5), this is one of the main limitations for the validity of the TEQ approach.

7.4. COMPARISON OF ACTUAL EXPOSURE LEVELS WITH THE TOLERABLE DAILY INTAKE

On basis of the exposure data, it appears that the present level of exposure of the general population in industrialised countries is the highest and close to the estimated tolerable daily intake (TDI) of 1 to 10pg/kgbw (see Section 5). For a limited period of the lifetime, exposure

of young children could exceed the TDI because of their relatively high intake of milk products, including breast feeding. Other human groups with a high exposure to PCDDs and PCDFs possibly exceeding the TDI are those with a high fish consumption and those working or living in the close vicinity of an important source of PCDDs and PCDFs and eating dairy products mostly originating from this area. For these populations the safety margin between actual exposure levels and the lowest known no-effect levels is reduced. This is the reason why, since IWIs as sources of PCDDs and PCDFs present no significant health risk for the general human population, the further reduction of other sources of PCDDs and PCDFs remains a priority.

APPENDICES

APPENDIX A

GLOSSARY OF TERMS AND ABBREVIATIONS

microgram	µg,	10^{-6}g	ppm	1µg/g
nanogram	ng,	10^{-9}g	ppb	1ng/g
picogram	pg,	10^{-12}g	ppt	1pg/g
femtogram	fg,	10^{-15}g	ppq	1fg/g

ABBREVIATIONS

Chemicals

HpCDD	Heptachloro dibenzo-para-dioxin
HpCDF	Heptachloro dibenzofuran
HxCDD	Hexachloro dibenzo-para-dioxin
HxCDF	Hexachloro dibenzofuran
OCDD	Octachloro dibenzo-para-dioxin
OCDF	Octachloro dibenzofuran
PCDD	Polychlorinated dibenzo-para-dioxin
PeCDD	Pentachloro dibenzo-para-dioxin
PCDF	Polychlorinated dibenzofuran
PeCDF	Pentachloro dibenzofuran
PCDPE	Polychlorinated diphenylether
PCP	Pentachlorophenol
PXDD	Polyhalogenated dibenzo-p-dioxin
PXDF	Polyhalogenated dibenzofuran
TBDD	Tetrabromo dibenzo-para-dioxin
TBDF	Tetrabromo dibenzofuran
TCDD	Tetrachloro dibenzo-para-dioxin
TCDF	Tetrachloro dibenzofuran
TEF	Toxic Equivalence Factor
TEQ	Total toxic equivalent derived by multiplying the concentration of each congener by its TEF
TrBDD	Tribromo dibenzo-para-dioxin
TrCDD	Trichloro dibenzo-para-dioxin
XPA	Halogenated polyaromatic

Others

Congener	A specific member of a group of structurally related compounds
IWI	Industrial waste incinerator
MWI	Municipal waste incinerator
Flue gas	Gaseous/particulate emissions to atmosphere
Stack gas	Gaseous/particulate emissions to atmosphere

APPENDIX B

THE ANALYSIS OF PCDDs AND PCDFs

In the measurement of PCDDs and PCDFs it is essential to consider the sample collection and analytical techniques as a whole. It is important that internal and external standards are incorporated into the sampling protocol at the earliest opportunity.

B.1. SAMPLE COLLECTION

In order to collect a representative sample of PCDDs and PCDFs from an emission source or environmental medium, it is essential that the physical state of these species under the conditions of sampling is considered. For example, the collection of a sample from stack gases at elevated temperatures must take into account that the PCDDs and PCDFs will be distributed between the particulate and vapour phases. While suitable filtration may be used to recover material in the particulate phase, some means of vapour adsorption will be required to collect remaining material in the gas stream. Similarly, for the collection of these species from ambient atmospheres, it is generally desirable to include both particulate and vapour sampling, since the conditions of sampling may not preclude volatilisation of collected particulate material. In water, soil and grass samples, most PCDDs and PCDFs present will be adsorbed onto particulate matter.

Since it is normal practice to express the concentrations of PCDDs and PCDFs as Toxic Equivalents (TEQ), it is useful to utilise ^{13}C internal standards corresponding to each of the 17 isomers which are to be measured. It is usual to divide such internal standards into at least three groups, and to add them to the sample at appropriate stages during the sample collection and analysis.

B.1.1. Stack Gas Analysis

Sampling of stack gases may be carried out according to US EPA Modified Method 5, or some similar method which was derived from this procedure (US-EPA, 1971). Stack sampling utilises an isokinetic sampling probe heated to just above stack temperature, through which sample gases are drawn towards a filter which is typically maintained at stack temperature. Following filtration, the sample gases are usually cooled, and passed through a trap containing an adsorbent resin, or other carbon-based or inorganic material. In a variation of this method, a cold CO_2 trap is used in place of the resin adsorption. A new method of sampling of stack gases, the so-called LAB-VET-KISA system, has recently been developed by Marti *et al* (1991, 1992), which offers improved efficiency and ease of application. A dilution method (VDI Guideline 3499) and condensation methods

with or without cooled probe (Draft VDI Guideline 3499, parts 2-4) are also commonly used.

In stack sampling it is desirable that ^{13}C internal standards are placed upon the initial filter in the sampling train, in order to provide a measure of sampling efficiency and recovery. Further standards are added after sample collection, and at subsequent stages during analysis.

B.1.2. Water Effluents

Water effluents arise primarily as a result of gas cleaning processes in IWIs, measurements for PCDDs and PCDFs may additionally be made on water input to the facility, and on background water systems such as ponds lakes and rivers.

Because of their low water solubility a significant proportion of the PCDDs and PCDFs in water samples at normal temperatures is expected to be adsorbed on particulate material. It is nevertheless common practice to sample these materials from water systems using a combination of filtration and resin adsorption to avoid losses when large volumes of water are sampled. As in the case of stack sampling, it is usual to add a number of ^{13}C internal standards to the filter used for sampling. PCDDs and PCDFs may also be recovered from water by solvent extraction; for small water samples, this method is preferred.

B.1.3. Slag, Ash and Other Solids

While the physical sampling of solid materials, including slag, ash and soil would appear to be relatively straightforward, the task of obtaining a representative sample of the whole may be difficult. Where possible, the use of statistically acceptable sampling methods (ASTM, 1986) is preferred, although when collecting background soil samples it is more usual to utilise a simple random sampling pattern.

B.2. CHEMICAL ANALYSIS

The initial step in an analysis is to extract the collected sample or sampling medium with a suitable solvent, usually preceded by the addition of internal standards. Numerous procedures for solvent extraction have been described (NRCC, 1982; Ballschmitter *et al*, 1983; Hutzinger *et al*, 1986; Mundy *et al*, 1989; Brown *et al*, 1989; Pettit *et al*, 1990).

B.2.1. Sample Clean-up

The objective of the initial clean up is to remove those materials which might otherwise interfere with the analysis to be carried out. Interference may occur as a result of other chemical compounds with similar molecular weights to PCDDs and PCDFs, but any other

material with similar chromatographic characteristics may provide persistent interference in the mass spectrometer through suppression of ionisation. Sample clean-up is an important step in the analysis of PCDDs and PCDFs.

A variety of liquid chromatography separations have been used for sample clean-up, including silica, florisil, alumina, and various combinations of such columns (Norstrom *et al*, 1982, Tiernan, 1983; Lamparski and Nestrick, 1989; Mundy *et al*, 1989; Pettit *et al*, 1990).

High pressure liquid chromatography (HPLC) has also been used for sample clean-up (Lamparski *et al*, 1979, Tosine *et al*, 1983; Lamparski and Nestrick, 1989; Creaser *et al*, 1990). A very successful variation of the carbon column clean-up procedure described by Creaser *et al* is the low pressure carbon column procedure which can provide selective clean-up for PCDDs and PCDFs in grossly complex samples (Smith *et al*, 1984). Following sample clean-up it is common practice to add an additional number of ^{13}C internal standards in order to provide information on recovery during the clean-up step. At least 17 ^{13}C standards are required to be added at some stage during the sampling and analysis in order to provide for qualitative and quantitative analysis of all dioxin and dibenzofuran isomers which are required for calculation of a toxic equivalent, and also to provide information on the efficiency/recovery of each of the various steps in sampling and analysis.

B.2.2. Quantitative Analysis of Dioxins and Dibenzofurans

The identification and quantification of PCDDs and PCDFs can only be satisfactorily performed using combined gas chromatography and mass spectrometry techniques. While low resolution mass spectrometry has been successfully applied (see e.g. Creaser *et al*, 1989), more demanding and time consuming sample clean-up is required.

Gas chromatographic conditions employed have been described by a number of authors, although the methodology described by Pettit *et al* (1990) is representative of the technology. At the present time, no single column provides resolution of all congeners, and while polar columns give the best results, separation of some congeners is only achieved on an apolar column.

The mass spectrometry protocols developed by Tondeur *et al* (1989) and by Ambridge *et al* (1990) have generally been regarded as minimum acceptable criteria for high resolution instruments, and typical mass spectrometric operating conditions may be summarised. Maximum sensitivity has been attained by monitoring the PCDDs and PCDFs in five groups: tetra, penta, hexa, hepta and octa, employing "magnet switching" to minimise the

decrease in accelerating voltage, and using a "lock mass" for drift correction. A high resolution mass spectrometer is typically operated under the following minimum conditions:-

- 10,000 resolving power (static)
- single Ion Monitoring mode, with a total cycle time of less than one second
- calibration performed at the start of each analysis, and thereafter at 12 hour intervals, provided that satisfactory performance is obtained from standard mixtures of PCDDs and PCDFs which are alternated with samples and blanks during a normal automated analysis run
- a lock mass ion (e.g. perfluorokerosene,PFK) is monitored for each congener group.

For a peak to be regarded as a dioxin or dibenzofuran, the following criteria must be fulfilled:-

- the signal to noise ratio must be greater than 2.5 for a peak to be considered significant
- the retention time of the peak and the corresponding ^{13}C isomer must coincide within + or - 3 seconds
- both ion traces monitored for each congener must maximise within + or - 1 scan point
- the isotope ratios for the molecular ion peaks monitored must be within 15% of the theoretical expectation.

For gas chromatographic and high resolution mass spectrometric analysis, the following conditions should also be met:-

- each congener group must contain at least one ^{13}C isomer which is used for quantification and specific isomer identification within that group
- the congener elution windows from the gas chromatographic column must be periodically checked using (for example) the EPA column performance mixture containing the first and last isomers of each congener group
- it should be noted that the quantification of 2,3,7,8-TCDD can only be achieved if it is separated from the other 21 TCDD isomers. Since separation is frequently not

routinely achieved in many laboratories, this difficult separation should be demonstrated using (for example) the EPA column performance mixture of isomers before commencement of each analytical run, and thereafter every 12 hours (Pettit *et al*, 1990; Tondeur *et al*, 1989).

While mass spectrometry is undoubtedly the technique of choice for confirmation and analysis of PCDDs and PCDFs, a series of interfering compounds have been identified, and appropriate precautions are recommended for obviating such interference (Smith and Johnson,1983).

It should be noted that such analysis provides measurement of all congeners which are required for the expression of the analytical result in terms of a toxic equivalent, either e.g. German (BGA), Nordic, or NATO/CCMS (cf Table 10 of main text).

B.2.3. Detection Limits

While the detection limit for a single dioxin or dibenzofuran congener may be as low as 10fg (= 10^{-14}g), in practice there are many considerations which lead to a somewhat higher detection limit. As already emphasised above, in a measurement of PCDDs and PCDFs we are not so much concerned with individual congeners, but more with a determination of a Toxic Equivalent value (TEQ), which depends upon the individual measurement of 17 2,3,7,8-substituted compounds. Making the assumption that the instrument used for measurement is a high resolution mass spectrometer, coupled with capillary gas chromatography, if the final injection volume is 2µl (which might reasonably represent 10% of the final extract), then the following figures indicate the sensitivity which should be achievable in most high quality analytical laboratories. Under the best conditions, up to 1 order of magnitude better sensitivity may be achieved.

Medium	Sample Size	Detection Limit (TEQ)
Stack Gases	1-10m^3	0.01ng/m^3
Water	25-50l	1pg/l
Soil, Dairy Products, Foodstuffs	100g	0.5pg/g
Vegetables, Grasses	20g	2.0pg/g

In the case of stack emissions from chemical waste incinerators, it should be noted that the most stringent standard for new facilities is presently 0.1ng/Nm^3; analytical sensitivity

corresponding to this emission limit is just achievable under normal circumstances in a high quality analytical laboratory. The above detection limits for PCDDs and PCDFs in soils and grasses are approximately one to two orders of magnitude lower than typical background levels, and should be satisfactory for present environmental purposes.

B.2.4. Validity and Accuracy of Results

In addition of the various elements of quality assurance, an interlaboratory quality assurance programme may indicate a degree of confidence which may be expected from a sample analysis. It may also identify those analytical sequences which need further attention (Liem *et al*, 1989).

Even with adequate sampling procedures, it remains very difficult to assess accurately the efficiency of an extraction procedure (Ballschmitter *et al*, 1983).

Validation of the remaining analytical methods is more reliable since techniques involving spiking with radiolabelled PCDDs and PCDFs as internal standards have been used (O.Keefe *et al*, 1989). In some cases large discrepancies up to an order of magnitude were found (Tarkowski *et al*, 1987). Liem *et al* (1989) presented the results of an interlaboratory study on the determination of PCDDs and PCDFs (tetra to octa) in fly dust from a municipal incinerator. It was concluded that major differences in analytical results up to a factor 3 were introduced during the instrumental part of the analysis. Recently Marklund (1990) reported on parallel sampling using five different sampling techniques and analyses carried out in two different laboratories. The recoveries for all pre-sampling spikes were above 50%. Interlaboratory results did not differ by more than a factor of 2.7 for different sampling procedures, and less than 1.8 for the same procedure.

APPENDIX C

STUDIES ON THE MECHANISMS OF TOXICITY OF 2,3,7,8-TCDD AND RELATED COMPOUNDS

C.1. ENZYME INDUCTION AND BINDING TO RECEPTOR Ah

C.1.1. Induction of aryl hydrocarbon hydroxylase and Ah receptor binding

The enzymatic system responsible for the biotransformation of xenobiotics, and particularly the monooxygenase system (cytochrome P450 dependent) is known to be inducible. Nebert and Gielen (1972) showed that the induction of aryl hydrocarbon hydroxylase (AHH) activity was under genetic control at a specific locus designated as the Ah locus. 2,3,7,8-TCDD was shown to be the most potent AHH inducer with a potency about 30,000 times higher than that of 3-methyl-cholanthrene (Poland and Glover, 1973, 1974).

The ED_{50} (dose of 2,3,7,8-TCDD that produces half maximum induction of the AHH activity) is identical in several species including rat, mouse and hamster (Poland and Glover, 1975; Gaziewicz *et al*, 1986). 2,3,7,8-TCDD crosses the placental barrier and it was shown to induce the monooxygenase system in the foetus and the neonate (Lucier *et al*, 1975). *in vivo*, the induction of the foetic monooxygenase is observed above 3 ng 2,3,7,8-TCDD/kgbw (Krueger *et al*, 1990).

The inducibility is mediated through a cytosolic receptor which is the gene product of the Ah locus (Nebert *et al*, 1972). This receptor is a protein that interacts with polycyclic aromatic hydrocarbons or 2,3,7,8-TCDD and forms a complex that is translocated to the cell nucleus where it presumably initiates the transcription of a specific P450 mRNA.

Genetic studies demonstrated that inducibility is inherited as a autosomal dominant trait (Poland and Knutson, 1982). Responsive strains that express the gene are 10-fold more sensitive to 2,3,7,8-TCDD induction than non-responsive strains. The structural and regulatory genes necessary for enzymatic induction are probably also present in non-responsive mice.

Aitio and Parkki (1978) found that the monooxygenase system was induced in the liver, kidney, lung, small intestine and testes of male Wistar rats but not in the other tissues investigated. Enzyme induction by 2,3,7,8-TCDD was also reported in the rat mammary gland, prostate gland and testes.

C.1.2. Correlation between 2,3,7,8-TCDD Toxicity, Ah Locus and Binding to the Ah Receptor

Toxicology studies with genetically Ah responsive and Ah non-responsive mice suggested an association between the inducibility of the Ah locus and the susceptibility to the toxic effects induced by 2,3,7,8-TCDD, such as thymic atrophy or the wasting syndrome (Courtnay and Moore, 1971; Jones and Sweeney, 1980; Vecchi *et al*, 1983).

The association between the expression of the Ah locus and the toxic response to 2,3,7,8-TCDD was also suggested by the correlation observed in structure-activity relationships between the binding affinity of several congeners of 2,3,7,8-TCDD for the Ah receptor and their toxic potency.

Such an association, however, does not explain all aspects of 2,3,7,8-TCDD and related compounds toxicity. Species differences in toxic responses to 2,3,7,8-TCDD do not always strictly reflect differences in Ah receptor levels or in metabolic rates. For example, while the LD_{50} of 2,3,7,8-TCDD in guinea-pig and hamster differ by more than 1,000-fold, there are only minor differences in the 2,3,7,8-TCDD binding affinity for the Ah receptor (Gasiewicz and Rucci, 1984). Furthermore, 2,3,7,8-TCDD half-life in the hamster is only 3-6 fold longer than in the guinea-pig (Gaziewicz and Neal, 1979; Olsen *et al*, 1980). There are other examples of poor correlation between the concentration of the Ah receptor, 2,3,7,8-TCDD binding affinity and enzyme induction (Poland and Knutson, 1982, Gasiewicz and Rucci, 1984; Denison *et al*, 1986a, b) or liver toxicity (Greig *et al*, 1984).

Recently, Rozman (1989) summarised the evidence for and against association of toxic effects with the Ah receptor. For almost all parameters investigated there is evidence either supporting or against the association and the overall picture shows that the Ah receptor is not the only mechanism involved. It is possible that the toxic response is dependent on several gene batteries and is under the control of both the Ah and the hr gene loci (Knutson and Poland, 1982).

This is in opposition with the recent agreement reached by about 40 dioxin experts that most toxic effects of 2,3,7,8-TCDD and related compounds were mediated via the Ah receptor (Scheuplein, 1991; van der Heijden, 1991). There is, however no general consensus on this statement (Roberts, 1991).

There is little information on the Ah receptor and its role in the human species. In liver samples, the levels of Ah receptor reported in one study ranged from non-detectable to 10-fold lower than levels in responsive mice and half as much as levels in responsive or non-responsive rats (Roberts *et al*, 1985).

C.2. EFFECTS ON EPIDERMAL GROWTH FACTOR (EGF) AND OESTROGEN RECEPTOR

In keratinocytes, 2,3,7,8-TCDD directly regulates EGF receptors. This effect was also observed for other tumour promotors. However, the significance of this observation is unclear as is the physiological role of EGF. Recent data suggest that it could play a role in the stimulation of gluconeogenesis (Soley and Hallenberg, 1987) which could be important since hypoglycaemia and subsequent lethality induced by 2,3,7,8-TCDD has been associated with reduced gluconeogenesis (Gorski *et al*, 1988; Rozman, 1989).

While examining the reduced feed intake associated with the "wasting syndrome", it appears that, as for other factors affecting feed intake (adaptation to cold, dietary changes, hormone regulation) 2,3,7,8-TCDD could induce changes via changes in the intermediary metabolism. Gorski *et al* (1988) and Gorski and Rozman (1988) have shown that non-lethal 2,3,7,8-TCDD doses differentially modulates thyroxine and corticosterone levels and that thyroidectomy and adrenalectomy greatly alter the toxicity of 2,3,7,8-TCDD (Rozman *et al*, 1985, 1987). Moreover, as evidenced by studies with ^{14}C-labelled glucose and palmitic acid, the metabolic response of 2,3,7,8-TCDD treated rats to reduced feed intake is not similar to that of normal rats (Weber *et al*, 1985). It is thus possible that important toxic effects of 2,3,7,8-TCDD such as those leading to hypoglycaemia and death do not result from a single mechanism but rather from multiple actions on the regulation processes (hormone balances, energy regulation, metabolic pathways) controlling normal homeostasis.

Romkes *et al* (1987) also demonstrated the effects of 2,3,7,8-TCDD on oestrogen receptors in the uterus and liver of rats. These effects could be involved in some specific effects of 2,3,7,8-TCDD and related compounds but it remains unclear whether the effects observed are induced by 2,3,7,8-TCDD or are a secondary consequence of some other altered metabolism.

C.3. EFFECTS ON THYROID HORMONES AND VITAMIN A DEFICIENCY

The close relationship between the molecular structure of thyroxine and 2,3,7,8-TCDD (McKinney *et al*, 1985) suggested that some effects of 2,3,7,8-TCDD could be related to its role as agonist of thyroid hormones. On the other hand many of the effects of 2,3,7,8-TCDD are similar to those observed with vitamin A deficiency (Kimbrough, 1974; Brouwer, 1987). Both thyroid hormones and vitamin A modulates the toxicity of 2,3,7,8-TCDD and also vitamin A and 2,3,7,8-TCDD synergistically reduce serum levels of thyroxine (Thunberg, 1984; Rozman, 1989).

Studies on PCBs (compounds structurally similar to PCDDs) showed that a non-Ah receptor called transthyretin (TTR) was involved in the transport of both vitamin A via formation of a complex with retinol binding protein (RBP) and thyroid hormones in plasma (Brouwer and van den Berg, 1986). The binding of a hydroxylated metabolite of tetrachloro biphenyl (TCB) appeared to be competitive with thyroid hormone for the thyroid binding site on TTR. Exposure of rats, mice or marmoset monkeys to TCB resulted in a rapid and almost complete elimination of thyroxin from the plasma. Further study on the mechanism of plasma retinol reduction by PCBs revealed a concomitant reduction in plasma retinol binding protein (RBP) levels.

It is not established whether there is a direct correlation between 2,3,7,8-TCDD toxicity and the effects on thyroid hormone and vitamin A deficiency. Experimental data on seals exposed to PCBs (Brouwer *et al*, 1989) and herring gulls contaminated by 2,3,7,8-TCDD suggest, however, that alterations in vitamin A and thyroid hormone concentrations may serve as sensitive indicators for adverse effects resulting from exposure to halogenated aromatic hydrocarbons, including PCDDs and congeners.

The *in vivo* data of Lamb *et al* (1986) has demonstrated the influence of thyroid hormones on the mechanism of induction of cleft palate in the mouse.

C.4. MECHANISMS FOR IMMUNOTOXICITY

The mechanisms of 2,3,7,8-TCDD immunotoxicity remain largely unknown although various hypothesis have been tested.

Direct cytotoxicity seems unlikely since 2,3,7,8-TCDD is not toxic when added directly to several types of cultures (Vos and Moore, 1974; Kouri *et al*, 1974; Knutson and Poland, 1980). It is suggested that 2,3,7,8-TCDD affects the thymic epithelium. Clark *et al* (1983) and Nagarkatti *et al* (1984) showed that susceptibility to immunotoxic effects of 2,3,7,8-TCDD in some strains of mice segregates with expression of the genetic Ah locus. The limits of the Ah hypothesis, however, have already been discussed. Holsapple *et al* (1986) for example showed that although OctaCDD, a congener devoid of significant affinity for the Ah receptor, is also devoid of immunotoxic activity, 2,7-DCDD, which also has a low affinity for the Ah receptor, produces a number of decreased immune responses.

The toxicity of these compounds could also depend on totally independent toxic mechanisms (vitamin A deficiency, altered thyroid hormone metabolism, carbohydrate metabolism, etc.). This is important because the Ah inducibility and the affinity for the Ah receptor are, with acute toxicity data, the main criteria used to evaluate the toxic potency of 2,3,7,8-TCDD congeners.

APPENDIX D

DESCRIPTION OF INDUSTRIAL WASTE INCINERATION

The most common industrial waste incinerator with the broadest application is the rotary kiln incinerator which can be fed with miscible and non-miscible liquids, pastes, bulk solids or drums and even with gases via special equipment. For incineration of liquids and bulky solids, other types of hearths may be used.

A waste has to pass an entrance control following its description in accordance with the national laws concerning acceptance and transportation. Usually the waste must be stored in different ways for solids, slurries or liquids.

After establishing the daily menu for the incinerator, which is based on the technical equipment e.g. gas cleaning system, the waste is prepared for loading into the incinerator.

To achieve effective combustion a temperature of between 850°C and 1,200°C is necessary e.g. in the case of PCB- or PCP-combustion. The minimum residence time is usually 2 seconds, preferably longer.

For waste incineration of solids in rotary kilns with post-combustion chambers (PCC), an oxygen excess of 11% is usual, in other cases (liquid incineration with continuous feeding) 3% may be enough. For complete combustion, good turbulence in the PCC is necessary.

After leaving the PCC, the hot combustion gases may be cooled in a steam boiler, but present evidence suggests that this may lead to the formation of PCDDs and furans. To remove pollutants formed in the incinerator e.g. HCl, SO_x, NO_x, a gas cleaning system is installed. Such a system may be operated wet with water, dry with lime, or semi-dry with milk of lime. For dust-removal, electrostatic precipitators (dry, wet or with condensation) or a baghouse filter are used. The cleaned gases are then dispersed via a chimney.

D.1. WASTE PREPARATION

Incoming waste has to pass the entrance and control area with:-

- weighing (mass balance, authority, price),
- administrative control (check of documents depending on the existing regulations),
- laboratory control (identification, check on conformity, characterisation, screening).

Before waste can be accepted for incineration, the waste generator has to provide details of its analysis to the incineration plant which may in turn carry out analytical checks in accordance with the existing national regulations.

Analyses may include: flammability (flash-point), halogen content, sulphur, nitrogen, heavy metals, calorific value, pH-value, PCB and in special cases other toxic or thermostable components.

Usually the delivered waste cannot be loaded directly into the incinerator, so it is routed to the storage area. For some incinerator designs it is necessary to homogenise different solid waste fractions by mixing in the bunker, but small quantities of solids may be loaded directly in drums or placed upon the hearth of a fixed hearth incinerator.

D.2. COMBUSTION

For the incineration of chemical waste, there is no single best technology to achieve the required destruction. Because the oxidation processes occur in the gas phase, it is important to distinguish between design and operating requirements. In the case of incinerators operating with gases, liquids, or pumpable sludges, conventional burners with atomisers can be used. A more complex system is needed for the incineration of solids, which must be evaporated or broken down to form gaseous or volatile products which are then completely oxidised in the gas phase.

D.2.1. Combustion conditions

Effective combustion depends upon:-

- waste preparation and loading rate,
- temperature,
- waste/oxygen ratio,
- residence time,
- turbulence.

Incineration temperature is usually measured in the gas phase where the combustion takes place. Effective destruction of organic products, e.g. liquids containing the elements C, H, O, N, and S only, is usually possible at 850°C. In the case of thermally stable substances, e.g. PCBs, terphenyls, PCDDs or PCDFs, a higher temperature is normally used (1,100 - 1,250 °C, for example).

For the incineration of solids a lower temperature on the fixed hearth is possible, provided that the main combustion chamber and afterburner area are maintained at sufficient temperature and associated residence time. For complete oxidative degradation of organic compounds, correct waste/oxygen ratio and complete mixing are required. The incineration of solids or sludges is usually difficult due to the risk of non-stoichiometric proportions of waste and oxygen. This is so for drum feeding where an oxygen excess of 6%, and more frequently 11%, is required. If solely liquids are to be incinerated, it may be sufficient to operate with only 2-3% of excess oxygen because of the more uniform feed rate.

The residence time (in the gas phase) is the period in which the combustion products are maintained at the specified combustion temperature. Each incinerator has a more or less typical residence time, dictated by its design. Variations in the residence time are caused by variations in the feed rate and operating conditions for the induced draught fan. A minimum residence time of 0.5 seconds may be adequate in the case of simple thermally unstable liquids, but in most other cases a longer residence time is required. A minimum residence time of 2 seconds at a combustion temperature of 1,200°C is commonly used.

Clearly temperature and residence time are interrelated, thus if one of the parameters is increased, the other may be reduced.

D.2.2. Hearth

To meet the need for effective and quantitative destruction of waste, there are various types of hearths available, the choice depending upon the characteristics and nature of the waste. An afterburner chamber is important in order to achieve good destruction efficiency.

The most frequently installed rotary kiln combustion chamber is slightly inclined and rotates slowly. Besides the rotary kiln, there are various other types of hearths, e.g. static hearth, multi-hearth incinerators or fluidised bed reactors. Static and multi-hearth incinerators may be particularly useful for the incineration of large irregular solid objects.

D.3. ENERGY RECOVERY

The heat produced by the combustion process from an hazardous waste incinerator with a capacity of approximately 35,000t/a is between 15 and 50MW. The incinerator may be equipped with a boiler to produce steam and/or electric power for energy recovery. This unit must have high operational safety, and because of the relatively high dust content in the combustion gases, it must have an effective system to clean the heat exchanger during operation, in order to ensure good heat exchange. The temperature of the combustion gas

from the post-combustion chamber will decrease from about 1.000°C to 300°C, and the produced steam is usually super-heated (up to 350°C) at a pressure of up to 40 bar.

Investigations of different boilers associated with IWIs have shown that PCDDs and PCDFs may be formed through recombination of products of partial combustion, particularly at temperatures around 300°C. Therefore IWIs are not normally equipped with heat recovery systems in order to avoid the additional formation of PCDDs and PCDFs.

D.4. GAS CLEANING

During the gas phase oxidation process the organic compounds are destroyed, forming e.g. CO_2, CO, NO_x, SO_x, HX (X = Halogen), H_2O, and small amounts of organic compounds which are products of incomplete combustion (PICs). They must be reduced by best available technology in accordance with legal requirements.

There are three types of gas cleaning systems: dry, semi-dry and wet. The latter two types are now mostly used (cf Section 6 of main text).

The dry system involves the absorption of compounds, the semi-dry process utilises spray absorption with minimum use of water, and wet gas cleaning operations are carried out with large quantities of aqueous solution in scrubbers.

To reduce the acid compounds in a dry absorption reactor, water is injected to cool the gases to about 130°C. The gases are then passed through a multi-stage layer of slaked lime for removal of acid gases.

The semi-dry method operates with the injection of milk of lime or other alkaline solution, so that the water evaporates and the adsorbent droplets are dried. Usually a filtering device is connected to a spray absorber to reduce the particles in the gas. In both of the above cases (dry, semi-dry) a stoichiometric excess of the adsorbent is necessary (dry : factor 2 to 4, semi-dry; factor 1.5 to 2.5).

Wet gas cleaning usually starts with a quenching unit, where through injection of water the gas temperature is reduced in a very short time to 70° - 80°C. During such quenching, large quantities of acid gas such as HCl are effectively removed.

Further gas cleaning may be achieved by the addition of an alkali scrubber, and electrostatic precipitator (dry, wet, or with condensation), or through use of baghouse filters for removal of particulate matter.

D.5. CHIMNEY

The cleaned gases must be effectively dispersed by means of an adequately sized chimney. In the case of wet scrubbing, the plume is usually visible. If as in the UK, it is not permitted to produce a visible plume, then the stack gases must be directly heated through the use of heat exchangers or warmed make-up air must be added before final discharge.

D.6. SOLID RESIDUES

The incineration process may generate solid residues from unburnable inorganic materials. For example, slag/clinker from boilers, dust/sludge from electrostatic precipitators and gas cleaning systems, dust from baghouses, and various rubble from re-lining a kiln or during refractory repair. Depending on the temperature, inorganic material can leave the kiln as slag or dry ash, where it is deposited in a cooling and retrieval system, which may involve a water quench. The handling of the residues, which may be dusty, must prevent losses of material in windy conditions.

Sometimes it may be suitable to feed the ash back into the rotary kiln in order to get slag after re-melting. This may be more acceptable for disposal through landfill since slags are likely to have a much lower leachability. In any event, all solid materials and sludges which arise as wastes from the incinerator must be analysed in order to determine an appropriate disposal method.

D.7. AQUEOUS RESIDUES

Quenching and wet scrubber systems generally result in aqueous liquors and sludges which must be treated, for example, by neutralisation, followed by precipitation/flocculation to give a liquid effluent which meets local discharge requirements. The separated solid material may be discharged to landfill provided that levels of heavy metals and trace organics meet regulatory requirements.

APPENDIX E

REGULATORY REQUIREMENTS FOR EMISSIONS INTO THE AIR OF INDUSTRIAL WASTE INCINERATORS

Concerning PCDDs and PCDFs, general limit values only exist in Germany, in the Netherlands, the UK and, outside of the EEC, in Austria. At present the regulatory requirements vary widely among the countries of the EEC. Only few (e.g. Germany, the Netherlands, UK) have issued general legally binding limit values, others (e.g. France, Ireland) define such limits at the local level. The EEC has recently published a "Proposal for a Council Directive on the incineration of hazardous waste" (EEC, 1992) which, if adopted, will require member states to harmonise legal requirements before 30 June 1994. Details are given below.

EEC: (EEC, 1992)		
	Daily average (mg/Nm^3)	**Half hour average (ng/Nm^3)**
Dust	5	10
TOC	5	10
HF	1	2
SO_2	25	50
Cd, Tl (Total)	0.05	
Hg	0.05	
Sb, As, Pb, Cr, Co, Cn, Mn, Ni, V, Su (total)	0.5	
PCDDs and PCDFs (guide value)	0.1ng TEQ/Nm^3	

The EEC requires in its proposal that "the emission of dioxins and furans shall be minimized by the most progressive techniques" and define 0.1 ng/m^3 as a guide value which should not be exceeded by all average values measured over the sample period of 6 to 16 hrs.

BELGIUM	
The emission limits are defined for each individual plant in the operating permit. The only legally binding Limit Values are those for industrial combustion installations in general, which are for liquid fuels (EEC, 1989):	
Dust	150mg/Nm^3 (inside specially protected zones) = case 1 300mg/Nm^3 (outside specially protected zones) = case 2
SO_2	0.85g/Nm^3< 106 Kcal/h 3.2g/$Nm^3$106 - 206 Kcal/h, case 1 3.7g/Nm^3> 206 Kcal/h, case 1 4.7g/Nm^3case 2

DENMARK		
The values do not apply specifically for incineration of chemical waste, but they are supplemented by individual specific operation conditions and emission limits for the incineration of chemical waste and special hospital wastes (EEC, 1989)		
	Monthly Median Value	**Yearly Median Value**
Dust	40mg/Nm^3	
HCl	100mg/Nm^3 or 700g/t waste	
HF		2mg/Nm^3 or 14g/t waste
SO_2		300mg/Nm^3 or 2.1kg/t waste
Cd		100µg/Nm^3 or 700mg/t waste
Pb		1.4mg/Nm^3 or 11g/t waste
Hg		100µg/Nm^3 or 700mg/t waste
TOC		20mg/Nm^3

PORTUGAL	
Limit values are set at the local level by the licensing authorities. An example is given (EEC, 1989).	
	mg/Nm^3
Dust	100
CO	80
Cl (total)	150
F (total)	5

GERMANY	
Limit values (BImSchV - 17, 1990)	
	Daily averages (mg/Nm^3)
Dust	10
Co	50
Organic substances	10
SO_2	50
HF	1
NO_x	200
Hg	0.05
Cd ,Ti	0.05
Sb, as, Pb, Cr, Co, Cu, Mn, Ni, V, Sn	0.5
PCDDs and PCDFs	0.1ng TEQ/Nm^3

FRANCE	
Guidelines for establishing prefectorial decrees containing legally binding limit values for industrial waste incineration (EEC, 1989).	
Dust	150mg/Nm^3
Heavy metal (total)	5mg/Nm^3
Cl (total)	100mg/Nm^3
PCB-incineration destruction efficiency	99.9999%

THE NETHERLANDS	
The limit values apply directly to new installations. Existing incinerators must comply on 30 November 1993 at the latest (EEC, 1989).	
	Daily averages (mg/Nm^3)
Dust	5
HCl	10
F	1
CO	50
TOC	10
SO_x	40
NO_2	70
Sb, Pb, Cr, Cu, Mn, V, Sn, As, Co, No, Se, Te (total)	1
Cd	0.05
Hg	0.05
PCDDs and PCDFs	0.1ng TEQ/m^3 (NATO CCMS)

IRELAND	
Legally binding limit values are applied at the local level by the licensing authorities. The following is an example, related to an existing IWI (EEC, 1989).	
	Daily averages (mg/Nm^3)
Dust	100
SO_2	500
HCl	50
CO	100
TOC	20
NO_x as NO_2	500

UNITED KINGDOM		
Limit values (Environmental Protection Act, 1992)		
	Industrial Waste Incinerator (mg/Nm^3)	**Municipal Waste Incinerator (mg/Nm^3)**
Dust	20	30
As, Sb, Cr, Co, Pb, Mn, Ni, Sn, V	1	-
As, Cr, Cu, Pb, Mn, Ni, SN	-	1
Cd, Ti (total)	0.1	-
Cd	-	0.1
Hg	0.1	0.1
SO_2	50	300
HCl	10	30
HF	2	2
TOC	20	20
CO	50	150
NO_2	350	350
PCDDs and PCDFs†	1ng TEQ/m^3	1ng TEQ/m^3

† Goal to reduce PCDD and PCDF emissions to 0.1ng TEQ/m^3.

SPAIN
Regulations exist for solid waste incineration in general which limits particulates only depending on the capacity, divided in unpolluted and polluted areas (700mg to 250mg/Nm^3 or 350 to 150mg/Nm^3, respectively for new installations; EEC, 1989).
Smoke opacity: <20% (= No.1 on Ringelmann scale) for the average and <40% (= No.2 on Ringelmann scale) for periods of three minutes per hour.

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