codex alimentarius commission

FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS

WORLD HEALTH ORGANIZATION

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DISCUSSION PAPER ON DIOXINS

(PREPARED BY THE NETHERLANDS)

REQUEST FOR COMMENTS AND INFORMATION

Governments and interested international organizations wishing to submit comments on the following Discussion Paper on Dioxins are invited to do so **no later than 15 January 1999** as follows: Ms. S.P.J. Hagenstein, Netherlands Codex Contact Point, Ministry of Agriculture, Nature Management and Fisheries, P.O. Box 20401, 2500 EK The Hague, The Netherlands (Telefax: +31 70 378.6141; E-mail: s.p.j.hagenstein@mkg.agro.nl), with a copy to the Chief, Joint FAO/WHO Food Standards Programme, FAO, Via delle Terme di Caracalla, 00100 Rome, Italy.

BACKGROUND

1. The 30th Session of the Codex Committee on Food Additives and Contaminants accepted the offer of The Netherlands to prepare a discussion paper on dioxins for circulation, comment and consideration at its 31st meeting (ALINORM 99/12, para. 117).

2. Dioxins and PCBs are ubiquitously present as contaminants in the environment and in food. This presence has long been recognised as a risk to both human health and the environment due to similar toxic and persistent features of these compounds. Although high exposure to dioxins and PCBs may occur in occupational situations or as a result of accidents, dietary intake represents the common route of human exposure to dioxins and PCBs.

3. In line with the procedure as outlined in Annex II of the General Standard for Contaminants and Toxins in Foods and based on the information presented in the Discussion Papers at its 27th and 28th sessions, the Committee agreed that Codex maximum levels for dioxins and PCBs may eventually be developed. With respect to the gaps and uncertainties in the knowledge concerning the toxicological evaluation of dioxins and PCBs, the Committee agreed that, before Codex maximum levels will be elaborated, JECFA should evaluate both groups of contaminants. Therefore, the Committee agreed to maintain dioxins, dioxin-like PCBs and non-planar PCBs on the priority list for

JECFA evaluation. For the sake of clarity, the Committee agreed that risk assessment and risk management of PCBs and dioxins would be dealt with separately (ALINORM 95/12A, paras. 132 - 137).

4. The Discussion Paper is firstly adjusted according to the written and verbal comments presented at the 27th Session and comments submitted to the 28th CCFAC. Secondly, the risk assessment of dioxins and dioxin-like PCBs is carried out on basis of the re-evaluated TEFs by the WHO in 1997. Thirdly, the paper includes the recently published advice on the "Assessment of the health risk of dioxins: re-evaluation of the Tolerable Daily Intake (TDI)" held by the WHO-European Centre for Environment and Health (WHO-ECEH) and the International Programme on Chemical Safety (IPCS) in May 1998. This consultation was attended by 40 experts from Australia, Belgium , Canada, Denmark, Finland, Germany, Italy, Japan, The Netherlands, New Zealand, Spain, Sweden, United Kingdom, and USA. The participants discussed topics such as health risks for infants, cancer and non-cancer endpoints in humans and animals, mechanistic aspects, toxicokinetics, modelling, exposure, and the applicability of the TEF concept. Where possible, risk assessment and risk management are separated.

INTRODUCTION TO THE CONTAMINANTS

5. PCBs and dioxins have similar physical and chemical properties. Like dioxins, PCBs are lipophilic and persistent compounds that accumulate in the food chain. As a consequence, biologic and environmental samples often contain mixtures of both dioxin and PCB congeners.

Dioxins

6. The term ‘dioxins’ refers to a group of poly-chlorinated, almost planar aromatic compounds with similar structures, chemical and physical properties. This group of compounds consists of 75 dibenzo-p-dioxins (PCDD) and 135 dibenzofurans (PCDF). The most studied and toxic dioxins are 17 congeners with a 2,3,7,8-chlorosubstitution pattern, of which 2,3,7,8-tetraCDD (TCDD) is the most toxic and most studied congener.

7. Dioxins are lipophilic compounds that bind to sediment and organic matter in the environment and tend to be absorbed in animal and human fatty tissue. These compounds are extremely resistant towards chemical and biological transformation processes and, consequently, persist in the environment and accumulate in the food chain. Dioxins are not produced commercially and have no applications. They are formed during combustion processes in, for example, waste incinerators or as unwanted by-products of industrial processes. Evaporation from chlorophenol wood preservatives, emission by sinter industries, the use of defoliants, the preparation of herbicides, traffic and bleaching of paper pulp using chlorine are also known to contribute to environmental contamination with dioxins. Most of the Dioxins enter the environment mainly by emission to air, due to which deposition may occur both near and far away from the source. In industrialised countries dioxins have been identified in almost all environmental compartments. Relatively little is known about the fate of dioxins released into the environment, i.e. transport, distribution and transformation. Based on the available occurrence data, evidence has grown that environmental residence times are in the order of many years.

Polychlorinated biphenyls

8. The group of polychlorinated biphenyls (PCBs) consists of 209 congeners of which 130 are likely to occur in commercial products. The preferred conformation of all PCB congeners is a non-planar one. However, some PCB congeners may adopt a planar 'dioxin-like' chemical structure and have indeed been found to resemble TCDD in its biochemical and toxicological properties. PCBs have been produced commercially under various product names such as Aroclor, Clophen and Kanechlor. These products are mixtures of both coplanar en nonplanar PCBs and have been used in the past in both open and closed systems like transformers, capacitators, electric insulation and hydraulic fluids. PCBs enter the environment by spoils and leakage from these systems. Combustion processes contribute relatively little to the environmental pollution with PCBs.

Indications of potential health problems

9. A few well known accidents and incidents revealed human health problems probably related to exposure to high levels of dioxins and PCBs. In general, co-exposure to dioxins, PCBs and possible other contaminants can not be excluded. Therefore, the observed effects can not be exclusively associated with exposure to either dioxin or PCB congeners.

10. Cloracne is the only effect that correlates consistently with high exposure of humans to TCDD. Weak associations have been reported between exposure to dioxins and soft tissue carcinomas and lung cancer. Such associations have become apparent after a latency period of more than 5 years in accidentally highly exposed people in Seveso or after more than 20 years in occupational exposed people.

11. The earliest symptoms of high PCB exposure are reversible dermal and ocular effects. Respiratory complaints are persistent for some years. Recent data indicate that fetal exposure to dioxins and/or PCBs is possibly associated with cognitive deficits in infants and young children. These effects were measured in infants from mothers exposed to relatively high levels of PCBs due to a prolonged consumption of contaminated fish. In addition to the cognitive effects, an increase in tumour incidence and neurologic, endocrinic, hepatotoxic and immunotoxic effects were observed in populations in Japan and Taiwan that were accidentally exposed to cooking oil contaminated with high levels of PCBs and dioxins.

Methods for the analysis of dioxins and PCBs

12. For both risk assessment and risk management the availability of reliable, selective and sensitive methods of analysis is crucial. Regarding the high toxic potency and the low concentrations of dioxins and coplanar PCBs in biological and environmental samples (low picogram/gram range), highly sensitive and specific methods of analysis are required. These analytical methods determine levels of individual congeners. To facilitate risk assessment the concept of Toxic Equivalency Factors (TEFs) is used to express concentrations of mixtures of PCDDs, PCDFs and dioxin-like PCBs in equivalents of 2,3,7,8-TCDD (TEQs).

13. An extensive clean-up, using a number of different procedures successively, is necessary in order to prepare sufficiently concentrated and cleaned extracts for final quantitative analysis of samples. For dioxins and coplanar PCBs, gas chromatography combined with high resolution mass spectrometry (GC-HRMS) is at present the only suitable technique, combining sufficient sensitivity and selectivity. Nonplanar PCBs are normally analysed using gas chromatography with electron

capture detection. Such analysis of dioxins and PCBs is often laborious and very expensive, and as a result only limited monitoring can be performed.

14. In the last few years, promising bioassays, immunoassays and bioassays using recombinant DNA technology have been developed, currently tested and validated. They show improved sensitivity and selectivity to determine the total level of dioxin-like substances in environmental and biological samples. In some cases, observed TEQ levels appeared to be fairly well comparable with those determined by GC-HRMS methods. These techniques aim at further improvement of both sensitivity and selectivity of dioxin and PCB analyses.
Bioassays may therefore be a suitable alternative for the screening of large numbers of samples.
Future advantages of bioassays are may be a reduction of costs, the time required and the fact that the total TEQs of a sample can be estimated, without identification and quantification of all the single specific congeners contributing.

Toxic Equivalency Factors (TEFs)

15. TCDD is the most potent and most studied dioxin congener. Because environmental and biological samples in general contain complex mixtures of different dioxin congeners, the TEF-concept is developed. In this concept, toxic equivalency factors relate the toxicity of an individual dioxin congener to the toxicity of 2,3,7,8-TCDD. By doing this, the TEF-concept enables the transformation of analytical results into toxicological information. It is assumed that the individual toxic effects of dioxin congeners in a mixture are additive to Toxic Equivalents (TEQs).

16. In 1997 the WHO-consultation re-evaluated the TEFs for PCDDs, PCDFs and dioxin-like PCBS on the basis of an improved database of toxicological information. Revisions were made in the list of International TEFs for PCDDs and PCDFs issued by NATO/CCMS in 1988, and in the list of WHO-TEFs for dioxin-like PCBs as published in 1994. Using the revised TEFs in estimations of exposure and body burdens of the background population in various countries, it was found that TCDD generally accounts for only 10-20% of the PCDD/PCDF-TEQs. When dioxin-like PCBs are also included TCDD would contribute less than 5% to the total TEQ.
Recognising the inherent uncertainties in the TEF concept, the WHO-consultation for the re-evaluation of the TDI in May 1998 concluded that additional compounds can possess “dioxin-like” activity so that the use of TCDD alone as a measure of exposure to PCDDs, PCDFs and PCBs would severely underestimate the risk to humans from exposure to these compounds.
There it recommended that the new TEFs for PCDD/PCDF and dioxin-like PCBs derived by the WHO in 1997 should be used for future calculations of TEQs. This will result in an approximate 10% increase in TEQ calculations, compared to using I-TEFs and the initial 1994 WHO TEFs for dioxin-like PCBs. Furthermore, the TDI in humans of PCDDs, PCDFs, dioxin-like PCBs (*non-ortho* PCBs and *mono-ortho* PCBs) will be expressed in units of TCDD equivalents (TEQs) applying the in 1997 established WHO TEFs.

RISK ASSESSMENT OF DIETARY EXPOSURE

17. Combination of available data on toxicological evaluation, food consumption patterns and levels in food is essential for the assessment of the kind and magnitude of the risks to human health that may be involved with dietary exposure to contaminants. For both dioxins and PCBs, available data on the composing elements of the process of risk assessment of dietary exposure is presented. Because the re-evaluation of the TDI by the WHO consultation in 1998 is based on dioxins and dioxin-like PCBs, dioxins and PCBs are presented together.

DIOXINS AND DIOXIN-LIKE POLYCHLORINATED BIPHENYLS

Toxicological evaluation of dioxins and dioxin-like PCBs

18. Mechanism: Data on TCDD and dioxin-like compounds has shown the importance of the Ah receptor in mediating the biological and toxicological effects of dioxins. Although the precise chain of molecular events is not yet fully understood, alterations in key biochemical and cellular functions are expected to form the basis for dioxin toxicity. The activated receptor exerts two major types of functions: enhancement of transcription of a battery of genes (e.g. encoding drug-metabolising enzymes), and immediate activation of tyrosine kinases. Alteration of expression of other networks of genes may be directly or indirectly regulated by the Ah receptor. Activation of the Ah receptor can result in endocrine and paracrine disturbances and alterations in cell functions including growth and differentiation. Some of these effects have been observed both in humans and animals, indicating the existence of common mechanisms of action.

19. Kinetics: The toxicokinetic determinants of dioxin and related chemicals depend on three major properties: lipophilicity, metabolism, and binding to CYP1A2 in the liver. Lipophilicity controls absorption and tissue partitioning, metabolism is the rate-limiting step for elimination and induction of CYP1A2 leads to sequestration of dioxin.
There is a range of apparent half-lives for the various dioxin-like compounds. When background exposures are involved, an average half-life similar to that of TCDD may be used, but will underestimate daily exposure in short half-life chemicals and overestimate exposure for those with longer than average half-lives. In general, concentration in the target tissue would be the most appropriate dose metric. However, the body burden, which can be readily estimated in humans in animals, is highly correlated with tissue and serum concentration, and integrates the differential half-lives between species. Therefore, the WHO-consultation for the re-evaluation of the TDI in 1998 concluded that in order to compare risks between humans and animals, the body burden is the metric of choice.

20. Animal data: A variety of effects have been reported in animal studies following exposure to PCDDs, PCDFs and PCBs. Among the most sensitive endpoints are endometriosis, developmental neurobehavioral (cognitive) effects, developmental reproductive (sperm counts, female urogenital malformations) effects and immunotoxic effects. The lowest doses giving rise to statistically significant effects in these endpoints, have resulted in body burdens in the exposed animals of about 10 to 75 ng TCDD/kg bw. TCDD has been shown to be carcinogenic in several species at multiple sites. Short-term studies, however, have shown a lack of direct DNA-damaging effects, illustrating that TCDD is not an initiator of carcinogenesis. Tumour promotion studies in various animal species indicated a non-genotoxic mechanism. The ability of TCDD to enhance proliferation and inhibit apoptotic processes in focal hepatic lesions further supports an indirect mechanism of carcinogenicity. The no-observed adverse effect level of TCDD for hepatic adenomas of 1 ng/kg/day in the Kociba study corresponds with a body burden of about 60 ng/kg bw.

21. PCB mixtures are shown to induce cancer depending on congener composition. Rhesus monkeys are the most sensitive animal species for exposure to PCBs. After chronic exposure to various commercial PCB mixtures, immunotoxicity, increased mortality rate, growth retardation, dermal effects and embryotoxic effects were observed. For Aroclor 1242, a NOAEL of 0.04 mg/kg body weight/day could be established. In neonatal monkeys, in addition to these effects also neurological and immunological effects were found. In utero and lactational exposure caused neurological and immunological effects that are possibly related to behavioural effects in neonates and offspring of Rhesus monkeys.

22. Human data: Epidemiological evidence from the most highly 2,3,7,8,-TCDD exposed cohorts studied produces the strongest evidence of increased risks for all cancers combined, along with less strong evidence of increased risks for cancers of particular sites. It was noted, however, that the general population is exposed to levels of dioxins which are several orders of magnitude lower than those experienced by the exposed cohorts.
Noncancer endpoints were evaluated among groups exposed to dioxins, dioxin-like and non-dioxin-like polychlorinated aromatic compounds in a variety of exposure scenarios.
Among children exposed *in utero* to background levels, effects include subtle developmental delays and thyroid hormone alterations. Of the many effects evaluated in exposed adult populations, many were transient effects disappearing after the end of exposure. A few conditions appear to be in excess among the exposed cohorts when compared to unexposed referent groups including alterations in metabolic parameters, as well as mortality from cardiovascular and non-malignant liver disease.

23. Bioavailability in humans: The bioavailability of dioxins and dioxin-like PCBs in humans from food containing fat or oil is known to be higher than 75%. Distribution in the body is dose and congener dependent. In man, dioxins are known to accumulate in fatty tissue and in the liver. For dioxins metabolism and elimination in humans is relatively slow; the estimated half-life is seven years. For dioxin-like PCBs slow elimination rates are related to low metabolic rates; in humans half-lives of 8 to 10 months have been reported. Dioxins and dioxin-like PCBs can enter the mammalian fetus via the placenta.

Toxicological expert judgement

24. Several WHO meetings in the field of the health risk assessment of dioxins and related compounds have been convened. At a meeting in 1990, a tolerable daily intake (TDI) of 10 pg/kg b.w. for TCDD was established, based on liver toxicity, reproductive effects and immunotoxicity, and making use of kinetic data in humans and experimental animals. Since then new epidemiological and toxicological data have emerged, in particular with respect to neurodevelopmental and endocrinological effects.
In May 1998, a consultation convened by the WHO-ECEH and IPCS re-evaluated all available information and established a TDI range of 1-4 pg TEQs/kg body weight for Dioxins and Dioxin-like PCBs.

25. The consultation focused on the most sensitive effects which are considered adverse (hormonal, reproductive and developmental effects) seen at low doses in animal studies. These effects occur at body burdens in rats and monkeys in the range of 10-50 ng/kg bw.
Human daily intakes corresponding with body burdens similar to those associated with adverse effects in animals can be estimated to be in the range of 10-40 pg/kg bw/day.
Since body burdens have been used to scale doses across species, the WHO-consultation concluded that the use of an uncertainty factor to account for interspecies differences in toxicokinetics is not required. However, the estimated human intake was based on Lowest Observed adverse Effect Level (LOAELs) and not on No Observed Adverse Effect Level (NOAELs). Although for many parameters humans might be less sensitive than animals, still uncertainty remains regarding animal to human susceptibilities. Furthermore, differences exist in the half lives of elimination for the different components of a TEQ mixture. To account for all these uncertainties, a composite uncertainty factor of 10 was recommended.
The WHO-consultation recognised that subtle effects might already be occurring in the general population in developed countries at current background levels of exposure to dioxins and dioxin-like

compounds. It therefore recommended that every effort should be made to reduce exposure to the lower end of the advised range of 1-4 pg TEQs/kg body weight/day.

26. In several other countries Tolerable Daily Intakes (TDIs) have been established for TCDD ranging from 1 to 10 pg TCDD/kg body weight.

27. Also, several organisations calculated safe doses applying the Linear Multistage Model to liver tumour data observed in female rats after chronic exposure to TCDD. In this model TCDD is treated as a complete carcinogen, which implies that no threshold dose exists. The U.S. Environmental Protection Agency calculated a lowest safe dose of 0.006 pg TCDD/kg body weight/day, corresponding to an acceptable lifetime tumour risk of 10^{-6}. This risk assessment of dioxin is currently under re-evaluation by EPA.

Human exposure to dioxins and dioxin-like PCBs

28. The daily human exposure to dioxins and dioxin-like PCBs has been estimated in a number of industrialised countries such as Canada, Denmark, Germany, Italy, Japan, the Netherlands, New Zealand, Norway, Spain, United Kingdom and United States.
Over 90% of human exposure is estimated to occur through the diet, with foods from animal origin being the predominant sources. Non-food sources and pathways like air, soil, paper, smoking and drinking-water are of minor importance.

29. Dioxin and furan (PCDDs and PCDFs) contamination of food is primarily caused by deposition of emissions from various sources on farmland. Other sources may include contaminated feed for cow's, chicken and farmed fish, improper application of sewage sludge, flooding of pastures, waste effluents, food processing an immigration from chlorine-bleached packaging material. Information to evaluate the sources of dioxin-like PCBs is generally lacking. The present occurrence of PCBs in the environment and in foods for human consumption is probably the result of both former applications (residues of technical PCB formulations) and sources similar to those identified for PCDDs and PCDFs. In the 1970s many countries implemented regulatory measures to reduce the input of PCBs in the environment. Nevertheless it is estimated that there are still tons of PCBs annually released into the environment. Although the dioxin-like PCB fraction is only a few percents of total PCBs, this implies that there are still kilograms of dioxin-like PCBs annually emitted into air. Further studies are needed to identify the major sources that might be subject to additional regulation.

Levels of dioxins and dioxin-like PCBs in food

30. Levels of dioxins in food are strongly related to the levels of these contaminants in the environment. Due to their lipophilicity, dioxins accumulate in animal fatty tissue. Since the fat content of food of plant origin generally is low, levels of dioxins in food of plant origin have been reported to be near the current detection limit. In vegetable oils and fats, levels are comparable.

31. In several countries levels of dioxins in food of animal origin have been measured to range from 0.7 to 2.5 pg I-TEQ/g fat in dairy products (milk, butter, cheese) and 0.4 to 1.8 pg I-TEQ/g fat in beef, pork and chickenmeat. Concentrations in liver appear to be substantially higher than those in meat, varying with animal species.

32. In fish and fat of fish relative high levels of dioxins are found compared with other animal products. In Germany and the Netherlands 2.4 to 48.7 pg I-TEQ/g fat have been found. On product basis levels range from 0.5 to 5 pg I-TEQ/g fish. Also, raw fish oil usually contains high levels of

dioxins depending on geographical origin. Industrial refining processes usually more than decimate the levels of dioxins in fish oil. A Dutch survey of refined fish oils showed average dioxin levels of around 1 pg I-TEQ/g fish oil.

33. PCBs and dioxins have similar chemical and physical properties: Levels of PCBs are strongly related to the levels in the environment and PCBs accumulate in animal fatty tissue. Levels of PCBs in food of plant origin are usually very low and have been reported to be near the current detection limit and tend to be somewhat higher in vegetable oils and fats.

34. Total PCB levels measured in several countries in food of animal origin were 10 to 200 ng/g fat in diary products and 7 to 500 ng/g fat in meat products. Levels of coplanar PCBs have been determined in Dutch dairy products to be around 2 pg WHO-TEQ/g fat.

35. Relative high levels of PCBs are found in fat of fish compared with other animal products. PCB levels in fish vary from 10 to 200 ng/g. Also, raw fish oil usually contains high levels of PCBs. Industrial refining processes usually more than decimate the levels of PCBs in fish oil. The Dutch survey of refined fish oils revealed TEQ contributions from non-ortho PCBs comparable to those found for dioxins, i.e. around 1 pg WHO-TEQ/g fish oil.

Human dietary intake of dioxins and dioxin-like PCBs

36. The available information derived from food surveys in numerous industrialised countries indicates a daily intake of PCDDs and PCDFs in the order of 50-200 pg I-TEQ/person/day, or 1-3 pg I-TEQ/kg bw/day for a 60 kg adult.

37. If the dioxin-like PCBs (non-ortho and mono-ortho PCBs) are also considered, the daily TEQ intake may be greater by a factor of 2-3-fold. Food which caused the highest daily intake of (I)-TEQ were in Canada milk, eggs and beef; in Denmark fish (50%); in Finland fish (63%) and dairy products (33%); in Germany milk (30%), fish (30%) and meat (30%); in Japan fish, green vegetables, milk and milk products, meat and eggs, and cereals and potatoes; in the Netherlands milk (50%) and fish oil; in New Zealand meat; in Norway milk products; in United Kingdom milk, meat, oil, and fat; in United States milk and other dairy products (50-80%).
Recent studies from countries which started to implement measures to reduce dioxin emissions in the late 80s clearly show decreasing PCDD/PCDF and PCB levels in food and consequently a lower dietary intake of these compounds by almost a factor 2 within the past 7 years.
Compared to adults, the daily intake of PCDDs/PCDFs and PCBs for breast fed babies is 1-2 orders of magnitude higher. The latest WHO field study showed higher mean levels of PCDD/PCDF and PCB in human milk in industrialised areas (10-35 pg I-TEQ/g milk fat) and lower levels in developing countries (<10 pg I-TEQ/g milk fat). There is a clear evidence of a decrease in PCDD/PCDF levels in human milk between 1988 and 1993, with the highest rates of decrease in areas with the highest initial concentrations.
38. In 1990 JECFA estimated the daily dietary intake of total PCBs to range from 0.005 to 0.2 µg/kg body weight.

39. It can generally be concluded that food of animal origin is the predominant source of human dietary exposure to dioxins and PCBs. Environmental exposure to less toxic dioxin and PCB congeners occurs at higher concentrations, therefore less toxic congeners have a relative large contribution to the dietary exposure.

Risk characterisation of dioxins and dioxin-like PCBs in food

40. The average daily dietary intake of TCDD only, as well as the total intake of the 17 toxic dioxin congeners and the dioxins-like PCBs are compared to the TDI established by the WHO in 1998. It can be concluded that the current background intake are in the same range (2-6 pg TEQs/kg body weight) as the proposed TDI-range of 1-4 pg TEQs/kg body weight for Dioxins and Dioxin-like PCBs. This means that there are legitimate health concerns regarding the occurrence of these substances in food, especially when populations with an increased risk are taken into account.

POPULATIONS WITH AN INCREASED RISK

41. Local emission: In the above, data on the average daily dietary are used. The average daily dietary intake may be exceeded by consumers with consumption patterns deviating strongly from the average consumption pattern used above. This deviations can originate from cultural or regional differences as well as from preference for consumption of certain products. A higher daily dietary intake may e.g. occur due to consumption of products that contain relatively high levels of contamination. Special attention is needed for consumption of local products like farm products or products from vegetable gardens when these products contain high levels due to environmental contamination.

42. Consumption pattern: Since products of animal origin contribute most to the dietary intake of both dioxins and PCBs, people with a more than average consumption of such products possibly have an increased dietary intake. This might in particular be the case for people consuming large quantities of liver, regarding the relatively high levels in this tissue. Also people with a high consumption of fish or dairy can have an increased intake, especially if the product is derived from polluted (industrial) areas.

43. Intake in relation to age: In general, children show a relatively higher daily dietary intake, reciprocal with age, due to the relatively low bodyweight. In the Netherlands, the median daily dietary intake of dioxins of children is calculated to approach 3 to 4 pg I-TEQ/kg body weight maximum. When dioxin-like PCBs are taken into account the median daily dietary intake approaches a maximum of 6 to 8 pg I-TEQ/kg body weight.

44. Breast feeding: Relatively high intake of dioxins and PCBs has been reported for infants during breast feeding. The exposure will depend on the duration of the breast feeding period an the concentrations in the mother's milk. For breast fed infants the daily intake of dioxins has been estimated to range from 60 to 200 pg I-TEQ/kg body weight, based on average dioxin concentrations in breast milk of ranging from 16 to 40 pg I-TEQ/g fat. The intake of coplanar PCBs may be in the same order of magnitude, because levels of those compounds in human milk in TEQ are similar to dioxins.

45. Since the TDI concept is based on a lifetime intake, it is difficult to apply this concept for a risk characterisation for infants during breast feeding. In 1990 JECFA concluded that the advantages to the infant of breast-feeding outweigh any potential hazard due to PCB content of breast milk and anticipated no adverse health effects as a result of consuming breast milk for only a short period of the total life span. Concerning dioxins, in 1990 the WHO concluded that so far no adverse effects has been associated in infants with the levels of these chemicals now found in human milk.

RISK MANAGEMENT OF DIETARY EXPOSURE

46. Above, available information on risk assessment of dioxins and PCBs is described. Based on this information, several countries concluded that risk management measures were needed in order to reduce and/or control the risks of dietary exposure to dioxins and/or PCBs to acceptable risk levels.

Below, available information on risk management measures will be described including known social, technological and economic consequences of these measures.

47. Research and research coördination is essential for risk assessment and risk management. For risk assessment important fields of research are toxicology and epidemiology, occurrence assessment (monitoring and survey), consumption estimation and estimation of dietary exposure levels. For risk management monitoring of human and environmental exposure levels is important, as well as the development of (technological) methods to reduce production and emission of contaminants and of techniques for environmental and food decontamination. Information on and an overview of the situation in various countries is relatively scarce.

48. Risk management measures can be divided into two main categories, i.e. source related measures that aim to reduce environmental exposure and as a result the subsequent contamination of food with dioxins and PCBs and measures that prevent consumers from the consumption of food that may lead to exceeding acceptable risk levels.

Source related environmental measures

Dioxins

49. Reduction of the emission of dioxins by waste incinerators is the general policy measure in various countries to reduce environmental exposure. In several European countries, all waste incinerators must meet the legal emission standard of 0.1 ng I-TEQ/m^3. In the Netherlands, incinerators that did not respond to up to date demands for environmental hygiene have been shut down. Due to EU regulations, contributions of waste incinerators to the total emission of dioxins in Europe will substantially decrease. Ban of the use of pentachlorophenol and measures concerning the bleaching of paper are other important instruments to reduce exposure. Furthermore, monitoring programmes exist to check and control environmental exposure to dioxins.

50. In the Netherlands in 1990 combustion processes in waste incinerators contributed 80% of the total emission of dioxins. The main emission of dioxins was to the air (ca. 600 g I-TEQ/year), whereas emissions to water and soil together mounted to 7 g I-TEQ/year. The deposition of dioxins, important for the contamination of food, ranged from 2 to 25 ng I-TEQ/m^2/year. Airborne deposition of dioxins from sources from other countries contributed more than 35%; this relative contribution will increase with the results of source related measures in the Netherlands. Information on the emission of dioxins in other industrialised countries is relatively scarce, it is estimated that the emission of dioxins to the environment will be in the same order of magnitude.

51. To meet the new emission standards, waste incinerator companies in The Netherlands had to build new or adjust old installations/stack gas cleaning facilities. The costs for these operations varied between 65 and 90 guilders per ton of waste. The new installations produce and emit far less dioxins. In 1991 the total emission of dioxins in the Netherlands was reduced 50% compared with 1988. In 1998 the total emission of dioxins in the Netherlands was reduced 88% (87% for dioxins and PCBs) compared with 1991. It is estimated that till 2000 the emission of dioxins will decrease about 3% (4% for dioxins and PCBs) as compared to 1998.

Polychlorinated biphenyls

52. The most important regulatory measure to reduce environmental contamination with PCBs has been the OECD ban of the use of PCBs in open systems in the early 1970s and in new equipment

in the early 1980s. PCB exposure monitoring programmes exist in several countries. Since the OECD ban of PCBs, implemented by many countries, PCB levels in the environment have decreased in The Netherlands. In Scandinavian countries a similar trend was reported. In 1980 the Dutch Government has compensated 47 million guilders for the removal or destruction of transformers and capacitors.

Measures related to food production and food consumption

53. In various countries, consumer information and advice is aimed at pregnant and lactating women. In general, pregnant women are advised to avoid frequent consumption of products that contain relatively high levels of dioxins and PCBs. In addition, for lactating women dieting is not recommended as levels of dioxins and PCBs in human milk may increase under dieting conditions.

Dioxins

54. Due to the demands of several health authorities and subsequent efforts of the paper industry in reducing dioxin levels in bleached paper products, the migration of dioxins from bleached paper products into food like cows' milk, and with that the average daily intake of dioxins derived from food packaging material, has been substantially reduced.

55. In a few countries, in addition to source related measures the dietary intake of dioxins and PCBs is controlled by food quality standards for products that contribute most to the dietary intake. In The Netherlands a food quality standard for dioxins in cows' milk of 6 pg I-TEQ/g fat is established. In Germany and the UK a level for dioxins of respectively 5 pg I-TEQ/g milk fat and 17.5 pg I-TEQ/g milk fat (0.7 ng I-TEQ/kg whole milk) is used as action level.

56. In 1989, local contamination of cows' milk due to emissions of dioxins by waste incinerators in the Netherlands was followed by several regulatory measures, like prohibition on selling milk from farms within 2000 acres of the vicinity of the waste incinerators, prohibition on production of milk products, destruction of milk fat, prohibition of transport of grass outside contaminated areas, ear-marking cows, sheep and goats, canalisation of meat and destruction of organs and body fat and export certification of products suited for consumption. The Dutch government gave financial compensation to inflicted farmers. Due to effective source related environmental measures/emission reduction, in 1994 levels of dioxins in cows' milk from these areas were reduced to background levels and the above mentioned regulatory measures could be lifted.

Polychlorinated biphenyls

57. Several countries have established legal maximum levels for PCBs in food. These maximum levels differ as well in the level as in the way they are expressed (total PCBs or separate levels for chosen congeners). In the Netherlands food quality standards for certain PCB congeners are set for milk and fishery products, meat and eggs ranging from 0.5 to 1 mg total PCB/kg fat. In Germany similar maximum limits are set. The Swedish National Food Administration has recently notified a proposal to amend current maximum limits for PCBs in meat products, milk and milkproducts, eggs and fishery products.

CONCLUSIONS AND RECOMMENDATIONS

58. The information available on both risk assessment and risk management of dietary exposure to dioxins and PCBs is far from complete. Besides gaps and uncertainties in the information concerning risk assessment, information on technological, social and economic considerations is relatively

scarce. This complicates risk management as a decision-making process. Still, valuable conclusions and recommendations are possible.

59. Although dioxins, furans- and dioxin-like PCB induce similar effects, the mechanisms leading to the various toxic effects of dioxins and PCBs have not been fully elucidated. Further research and expert judgement may contribute to the clarification of these mechanisms. In spite of these and other uncertainties, including non-additive interactions, differences in shape of the dose-response curve, and in species responsiveness, it can be concluded that the TEF concept is still the most plausible and feasible for risk assessment and risk management of exposure to dioxins, furans- and dioxin-like PCBs.

60. The more recent epidemiological and toxicological data, in particular with respect to neurodevelopmental and endocrinological effects show that dioxins and dioxin-like PCBs have a broader impact on health-problems than previously assumed. This indicates that attention for the hazard characterisation of these compounds should be continued. Furthermore there are indications of potential health problems related to dietary exposure to both dioxins and PCBs. The average exposure already is at the same level as the TDI. These potential problems are related to exceptionally high dietary exposure that strongly deviate from average exposure, due to contamination of food commodities (e.g. fish) and to consumption patterns deviating from average patterns. The presently available information regarding the dietary intake in relation to the toxicological advice (TDI) clearly leads to the conclusion that there are potential health problems associated with dietary exposure to dioxins and PCBs.

61. In a few countries, different food quality standards have been established for dioxins and PCBs. In relation to dioxin contamination, consignments of dairy and meat products have been refused entry before certification for suitability for consumption. Furthermore food-, and feedstuffs incidentally contaminated with dioxins can also be refused and therefore create financial, trade and confidential problems for the countries which import and export. In relation to PCBs international trade has in some cases been hampered by contamination of food. Thus, there are potential (and in some cases there have been real) trade problems.

62. Thus for both health and trade reasons it is desirable that risk management measures should be taken to reduce contamination of food with dioxins and PCBs and that further Codex maximum levels are developed for foods moving in international trade. Proposals for international (Codex) levels for dioxins and PCBs in relevant foods should be developed by the Codex Committee on Food Additives and Contaminants according to the procedure outlined in the General Standard on Contaminants and Toxins in Food.

63. Various risk management measures can, and in fact do, reduce the levels of dioxins and PCBs in foodstuffs. Since dioxins and PCBs are persistent in the environment, so that their levels tend to increase as a result of continuous release, prerequisite for the reduction of exposure to dioxins and PCBs are source directed measures that are wholly or partially the responsibility of other Codex Committees or national authorities and international organisations:

a) reduction of emission of dioxins by combustion processes of chemical and household waste, sludge, wood, cables, fuels for traffic and oil by technological approvement of the incinerator installations.
b) reduction of emission of dioxins by industrial production processes by technological improvements of industrial processes and installations;

c) reduction of the production and use of chlorine containing chemicals like certain pesticides and wood preservatives in combination with reduction of the contamination of these products with dioxins and PCBs;
d) reduction or ban of the use of PCBs in open systems, combined with the collection, removal or destruction of old transformers and capacitators.

64. In order to ensure that levels of dioxins and PCBs in food are as low as reasonably achievable, it is recommended that appropriate technology is developed and applied to reduce and control contaminant levels in the production, processing, transport, packaging and storage of food. Also, the development of codes of practices and quality control programmes like HACCP can contribute substantially to the reduction of contaminant levels in food. Regular monitoring of the dietary exposure and of trends in the levels of dioxins in PCBs in relevant indicator foods is important to assess the effects of regulation and control measures. Therefore we propose to develop a Code of Practice within the CCFAC especially related to the control of dioxins and PCBs in foodstuffs.

65. Furthermore we propose to conclude that based on the presently available information, it is advisable to start with the development of Codex MLs for dioxins and PCBs in foods. Enough toxicological information and advice is available at present to justify starting ML-development now, and further toxicological advice will probably be ready when final decisions about MLs have to be made.

66. As stated before, new epidemiological and toxicological data, in particular with respect to neurodevelopmental and endocrinological effects in the risk assessment of dioxins and PCBs are available. In the near future, more results of toxicological research are expected to contribute to the clarification of the mechanisms leading to this effects. Therefore, CCFAC is advised to maintain dioxins and dioxin-like PCBs on the priority list for JECFA evaluation. In addition, it is advised that CCFAC decides to add non-planar PCBs on the priority list as well.

67. Risk management of contaminants entails deciding which measures should be taken, and to which extent, to reduce and/or control the risks of dietary exposure to these compounds to acceptable risk levels. To meet the objectives of the Codex Alimentarius, in the decision making process benefits and costs of the food contamination and risk management measures chosen must be balanced. In order to achieve international acceptance of Codex maximum levels for dioxins and PCBs in food, it is necessary that in this balancing process all relevant information is incorporated.

68. Therefore, to contribute to the development of Codex maximum levels for dioxins and PCBs in accordance with the criteria for the establishment of maximum levels in food as mentioned in Annex 1 of the GSCTF governments are invited to provide of <u>additional</u> information on:

a) Identify those food products commonly in international trade, that are of main interest for dietary exposure to dioxins.
b) methods of analysis and ranges of current levels (in TEQ) of dioxins and PCBs in food, preferably for specific dioxin and PCB congeners;
c) contribution of different foods to the dietary intake of the contaminants;
d) both average consumption patterns and regional and cultural deviations for exposure evaluations;
e) sources of contamination of food with dioxins and PCBs related to food production, handling and consumption;
f) maximum levels, guideline levels or action levels for dioxins and/or PCBs in food and the basic assumptions for these standards, like acceptable risk levels etc;

g) a description of (the results and costs of) risk management measures taken to avoid, reduce and control contamination of the environment and food with dioxins and PCBs, other than maximum, guideline or action levels;
h) technological, social and economic consequences of contamination and risk management measures.