codex alimentarius commission

FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS

WORLD HEALTH ORGANIZATION

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JOINT OFFICE: Viale delle Terme di Caracalla 00153 ROME Tel: 39 06 57051 www.codexalimentarius.net Email: codex@fao.org Facsimile: 39 06 5705 4593

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Second Session
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DOCUMENT FOR INFORMATION AND USE IN DISCUSSIONS RELATED TO CONTAMINANTS AND TOXINS OF THE GSCTF

(Prepared by Japan and the Netherlands)

Background

1. This document has been prepared by Japan and the Netherlands in accordance with the recommendation endorsed by the 38th Session of the Codex Committee on Food Additives and Contaminants (CCFAC)[1] and on the basis of the Document CX/CF 07/1/6 published for the First Session of the Committee on Contaminants in Foods (CCCF) held in April 2007. It incorporates all the decisions made at the First CCCF and subsequently adopted by the 30th Codex Alimentarius Commission in 2007: newly adopted maximum levels for tin and a code of practice; deleted standard; and new categorization of contaminants (metals, mycotoxins, other chemicals and radionuclides)[2]. Where reference was made to "CCFAC", it was updated as appropriate. A new text was inserted on the background of the Working Document. In addition, a new text of information on aflatoxins and ochratoxin A evaluations by the 68th JECFA in 2007 was added.

2. In order to assist consideration of maximum levels in various steps, issues arising from previous discussions of maximum levels for a contaminant/toxin are surrounded by broken lines while information on the nature and toxicity is surrounded by solid lines in the list.

3. The list of maximum levels for contaminants and toxins in foods and Schedule I are attached to this document (starting from page 2 and page 56 respectively).

[1] ALINORM 06/29/12 para. 116
[2] ALINORM 07/30/41, para. 46 and ALINORM 07/30/REP, para. 191

Working Document for Information and Use in Discussions related to contaminants and toxins of the GSCTF

Introduction

This working document presents contaminants and toxins that are or have been dealt with in the CCFAC and CCCF. It does not only encompass the contaminants and toxins for which Codex standards exist or are being developed, but also those for which further information is sought or about which a Codex decision has been taken.

The Working Document has the purpose of providing an overview of the situation regarding Codex decisions about this subject and guidance about further actions required. Therefore also relevant information and references are added to the list.

The list of maximum levels / guideline levels is thus active, which needs regular update.

The situation regarding contaminants and toxins is very complex and many substances are or have been the subject of scientific research and discussion regarding their occurrence in foods and their significance for human and animal health. On a national level, there are many activities, sometimes implying legal measures which may affect international trade in foods and feeds. It is obviously important for the CCCF to take note of the developments in this field and to consider the necessity of actions. In order to obtain an overview of the situation, the CCCF shall develop and maintain a working document in which more comprehensive information regarding contaminants and toxins in foods is presented in a summary form.

The Working Document has two parts: *Part 1* containing maximum and guideline levels developed by CCFAC/CCCF and contaminant provisions included in commodity standards; and *Part 2* containing maximum levels developed for copper, iron and zinc which are regarded as quality factors as opposed to safety factors. Part 1 also contains those levels still at various Steps of the Codex elaboration procedure for the facilitation of consideration of proposed maximum levels by the CCCF.

INDEX OF CONTAMINANTS IN ALPHABETICAL ORDER

EXPLANATORY NOTES

Background of the Working Document

The Working Document was established in its current form when the 36th CCFAC agreed to integrate the Annotated List of Contaminants and Toxins in Foods (Annex IV to the Preamble of the GSCTF, Part 1 and Part 2) into a separate document "Working document for information and use in discussions on the GSCTF" (ALINORM 04/27/12, para. 119). Annex IV had the purpose of providing an overview of the situation regarding Codex decisions about contaminants and toxins and guidance about further actions required. It was originally included in the GSCTF as an introduction text without the lists of contaminants and toxins (ALINORM 97/12, para. 68). All is now included in the Working Document.

It was agreed that the Working Document (Annex IV) would:

- contain information not only for contaminants and toxins for which Codex standards exist or are being developed, but also those for which further information is sought or about which a Codex decision has been taken, and that relevant information and references are added in order to give guidance about further actions required (ALINORM 04/27/12, para. 116 and APPENDIX XIII);
- include references to validated methods of analysis as well as references to information on toxicological guidance, if available (ALINORM 95/12A, para. 99);
- exclude references to revoked standards (ALINORM 04/27/12, para. 116);
- include maximum levels for quality-related parameters such as copper, zinc, iron, etc. as a record of the complete range of contaminants in the Codex system (ALINORM 04/27/12, para. 120).

The format of the Working Document is that of Schedule I. This results from the agreement of the 32nd CCFAC to create a new Schedule I to the GSCTF, for which a working document was created in its format, and under the name of Schedule I. It was noted that Schedule I would not be added to the GSCTF until the relevant levels were adopted by the Commission (ALINORM 01/12, para. 79).

In the following Sessions of the Committee it was agreed that this Schedule I:

- should include all current maximum and guideline levels for contaminants in food and those under elaboration by the Committee, as well as current maximum and guideline levels contained in Codex Commodity Standards, with an indication of their step status (ALINORM 01/12A, para. 118).
- would contain two lists, i.e. List 1 with MLs for contaminants and toxins already adopted as final texts and List 2 with MLs for contaminant and toxins under discussion at different steps of the Codex procedure (ALINORM 03/12, para. 104).
- would be used as a working document during the Working Group and the plenary sessions (ALINORM 03/12, para. 104).

In this Schedule I as prepared for the 36th CCFAC, it was identified that List 2 was in fact Annex IV, and was renamed accordingly to distinguish it clearly from Schedule I, the list of adopted Standards (CX/FAC 04/36/16). The Committee endorsed the recommendation to include Schedule I (List 1) in the GSCTF (ALINORM 04/27/12, para. 117). The Committee noted that Annex IV was useful in providing an overview of the situation regarding Codex decisions about contaminants and toxins, and to give guidance about further actions required by CCFAC. The Committee agreed with the recommendation that such information should be part of a working document to be updated yearly and presented at each Session of the Committee, and requested the delegations of the Netherlands and Japan to perform this task (ALINORM 04/27/12, paras 118 and 119). The current Working Document is the subsequent result.

Clarification of presented information

Reference to JECFA:	References to JECFA meeting in which the contaminant was evaluated and the year of that meeting
Toxicological guidance value:	Toxicological advice about the tolerable intake level of the contaminant for humans, expressed in milligrammes (mg) per kg body weight (bw). The year of recommendations and additional explanation are included.
Residue definition:	Definition of the contaminant in the form of which the ML applies or which may or should be analyzed in commodities.
Synonyms:	Symbols, synonyms abbreviations, scientific descriptions and identification codes used to define the contaminant.
Related code of practice:	Name of any code(s) of practice related to the contaminant and its (their) reference number(s).
Commodity code:	The code for food commodities are according to the food categorization system as contained in Annex IV of the GSCTF or the Codex Classification of Foods and Animal Feeds as contained in Volume 2 of the Codex Alimentarius. The food/feed categorization system also specifies the part of commodity which should be analyzed and to which the ML applies, unless a specific commodity definition is provided as an annex to the ML. For those maximum levels contained in Codex commodity standards, the relevant standard numbers are referred, if the code numbers are not readily available for these commodities.
Type:	Indicates whether the value is Codex maximum level (ML) or Codex guideline level (GL). See also the definitions of these terms in the Preamble of the GSCTF.
Step:	Step of the Codex Elaboration Procedure at which each maximum level is (at the time of the publication of this paper). See the Codex Procedural Manual. The term "Adopted" is used for an adopted MLs and Codex Standards.
Reference or adoption year:	Reference number of the commodity standard in which the maximum level is established or the year of adoption of the maximum level following the recommendation of the Codex Committee on Food Additives and Contaminants (up to 2006).

Qualification of MLs

C:	In canned products only

Definitions of some toxicological terms

PMTDI:	*(Provisional Maximum Tolerable Daily Intake)* The endpoint used for contaminants with no cumulative properties. Its value represents permissible human exposure as a result of the natural occurrence of the substance in food and in drinking-water. In the case of trace elements that are both essential nutrients and unavoidable constituents of food, a range is expressed, the lower value representing the level of essentiality and the upper value the PMTDI.
PTWI:	*(Provisional Tolerable Weekly Intake)* An endpoint used for food contaminants such as heavy metals with cumulative properties. Its value represents permissible human weekly exposure to those contaminants unavoidably associated with the consumption of otherwise wholesome and nutritious foods.
PTMI:	*(Provisional Tolerable Monthly Intake)* An endpoint used for a food contaminant with cumulative properties that has a very long half-life in the human body. Its value represents permissible human monthly exposure to a contaminant unavoidably associated with otherwise wholesome and nutritious foods

Ref: http://jecfa.ilsi.org/section1.htm#52

Metals

Aluminium

Reference to JECFA: 67 (2006)
Toxicological guidance value: PTWI 1 mg/kg bw (2006, for all aluminium compounds in food, including additives)
Synonyms: Al
Related code of practice: Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

The WHO Representative clarified that exposure through food contact utensils and containers had been considered during the evaluation by JECFA and that it was concluded that they were not main contributors fro human exposure to aluminium. (ALINORM 07/30/41, para. 31)

Aluminium occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements, such as sodium and fluorine, and as complexes with organic matter. Aluminium is a major component of the earth's crust. It is released to the environment both by natural processes and from anthropogenic sources, whereby natural processes far outweigh the contribution of anthropogenic sources. Mobilization of aluminium through human actions is mostly indirect and occurs as a result of emission of acidifying substances to the atmosphere. Aluminium is highly concentrated in soil-derived dusts from natural processes, coal combustion, and activities as mining and agriculture. In addition, aluminium finds use in a wide variety of applications including structural materials in construction, automobiles and aircraft, packaging materials, various containers and kitchen utensils and pharmaceuticals (Environmental health criteria for aluminium; International Programme on Chemical Safety (IPCS); 1997).

Non-occupational human exposure to aluminium is primarily through ingestion of food and water. Food being the principal contributor, as aluminium is naturally present in varying amounts in most foodstuffs consumed. The intake of aluminium can be increased greatly through the use of aluminium-containing pharmaceutical products (especially antacids). (Environmental health criteria for aluminium; International Programme on Chemical Safety (IPCS); 1997).

Aluminium and its compounds appear to be poorly absorbed in humans; the mechanism of gastrointestinal absorption has not yet been fully elucidated. Variability results from the chemical properties of the element and the formation of various chemical species, which is dependent upon the pH, ionic strength, presence of competing elements and complexing agents within the gastrointestinal tract. The urine is the most important route of aluminium excretion. Aluminium has a long half-life. (Environmental health criteria for aluminium; International Programme on Chemical Safety (IPCS); 1997).

The 67th JECFA established a PTWI for Al of 1 mg/kg bw for all aluminium compounds in food, including additives; previously established ADIs and PTWI for aluminium compounds were withdrawn. The JECFA concluded that aluminium compounds have the potential to affect the reproductive system and developing nervous system at doses lower than those used in establishing the previous PTWI.

The evaluation of the PTWI was based on the combined evidence from several studies: the studies conducted with dietary administration of aluminium compounds were considered most appropriate. The lowest LOELs for Al of different studies in mice, rats and dogs were in the range of 50-75 mg/kg bw per day. An uncertainty factor of 100 was applied (to 50 mg/kg bw per day) to allow for inter- and intraspecies differences. An additional uncertainty factor of 3 was applied to cover deficiencies in the database (absence of NOELs in majority of studies and absence of long-term studies on relevant toxicological endpoints). Also, deficiencies are counterbalanced by the probable lower bioavailability of the less soluble aluminium compounds present in food. Because of the potential for bioaccumulation the JECFA confirmed that the resulting health-based guidance value should be expressed as a PTWI.

Metals

Aluminium

The JECFA noted that the PTWI is likely to be exceeded to a large extent by some population groups, particularly children, who regularly consume foods that include aluminium-containing additives. The JECFA also noted that dietary exposure to Al is expected to be very high for infants fed on soya-based formula.

The 67th JECFA recommended: Further data on the bioavailability of different aluminium-containing food additives are required; There is a need for an appropriate study of developmental toxicity and a multigeneration study incorporating neurobehavioral end-points, to be conducted on a relevant aluminium compound(s); Studies to identify the forms of aluminium present in soya formulae, and their bioavailability, are needed before an evaluation of the potential risk for infants fed on soya formulae can be considered.

Metals

Arsenic

Reference to JECFA: 5 (1960), 10 (1967), 27 (1983), 33 (1988)
Toxicological guidance value: PTWI 0.015 mg/kg bw (1988, for inorganic arsenic)
Residue definition: Arsenic: total when not otherwise mentioned or inorganic arsenic; or other specification
Synonyms: As
Related code of practice: Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Edible fats and oils	0.1	ML	Adopted	CS 19-1981	FO	Edible fats and oils not covered by individual standards	1)
	Fat spreads and blended spreads	0.1	ML	Adopted	CS 256-2007	FO		1)
	Named animal fats	0.1	ML	Adopted	CS 211-1999	FO	Lard, rendered pork fat, premier jus and edible tallow.	1)
OR 0305	Olive oil, refined	0.1	ML	Adopted	CS 33-1981	FO		
OC 0305	Olive oil, virgin	0.1	ML	Adopted	CS 33-1981	FO		
OR 5330	Olive, residue oil	0.1	ML	Adopted	CS 33-1981	FO	Olive pomace oil	
OC 0172	Vegetable oils, Crude	0.1	ML	Adopted	CS 210-1999	FO	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein.	
OR 0172	Vegetable oils, Edible	0.1	ML	Adopted	CS 210-1999	FO	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein.	1)
	Natural mineral water	0.01	ML	Adopted	CS 108-1981	NMW	Expressed in total As mg/l	Changed from 0.05 mg/l in 2001.
	Salt, food grade	0.5	ML	Adopted	CS 150-1985	NFSDU		

1) The revised Standards for oils and fats contain the following wording for the mentioned contaminant MLs: "The products covered by the provisions of this Standard shall comply with MLs being established by the CAC but in the meantime the following limits will apply." CS for Edible Fats and Oils Not Covered by Individual Standards contains the same contaminant provision as the other recent Standards for oils and fats (only applying to Pb and As).

A position document CX/FAC 99/22 on arsenic discussed in the 31st CCFAC in 1999 noted that several countries have established MLs for arsenic in food commodities and some of these were stringent regarding seafoods, so trade problems might occur. The present range of Codex MLs for arsenic in some commodities do not cover all national MLs. The document concluded however that in general there are no indications that specific Codex MLs for arsenic in food commodities would be necessary. Also, at present there is no sufficient basis to decide about the establishment of Codex MLs for arsenic, due to the uncertainties mentioned about the levels of naturally occurring arsenic species in foods, about

Metals

Arsenic

their toxicity and about the availability of suitable analytical methods. It was acknowledged that at present especially the ML for arsenic in drinking water and in mineral water is relevant. The CCFAC agreed that a finalized position paper would form the basis for future work until such time as routine methodology became available to determine toxic arsenic compounds in food. (ALINORM 99/12A, para. 137)

Arsenic is a metalloid element which is normally occurring in mineral bound form in the earth's crust and which can become more easily available by natural sources such as volcanic activity and weathering of minerals, and by anthropogenic activity causing emissions in the environment, such as ore smelting, burning of coal and specific uses, such as arsenic-based wood preservatives, pesticides or veterinary or human medicinal drugs. As a result of naturally occurring metabolic processes in the biosphere arsenic occurs as a large number of organic or inorganic chemical forms in food (species). Especially in the marine environment arsenic is often found in high concentrations of organic forms, up to 50 mg/kg of arsenic on a wet weight basis in some seafood including seaweed, fish, shellfish and crustaceans. In fresh water and in the terrestrial environments arsenic is normally found in much lower levels (typically 0-20 ug/kg) in crop plants and in livestock. Higher levels may be found in rice, mushrooms and sometimes in poultry which is fed fish meal containing arsenic. Levels of arsenic in drinking water are of concern in many countries; levels exceeding 200 mg/l have been reported, which can adversely affect the health of consumers. The most toxic forms of arsenic are the inorganic arsenic (III) and (V) compounds; the inorganic arsenic trioxide is well known as a rat poison, which was also sometimes used for homicide. Methylated forms of arsenic have a low acute toxicity; arsenobetaine which is the principal arsenic form in fish and crustaceans is considered non-toxic. In shellfish, molluscs and seaweed dimethylarsinylriboside derivatives occur ("arsenosugars"), the possible toxicity of which is not known in detail. Only a few percent of the total arsenic in fish is present in inorganic form, which is the only form about which a PTWI has been developed by JECFA. The human epidemiological data used for this risk assessment is based on exposure to inorganic arsenic in drinking water. IARC has classified inorganic arsenic as a human carcinogen, and the estimated lifetime risk for arsenic-induced skin cancer which may be caused by drinking water at or in excess of the WHO guideline for arsenic in drinking water is estimated at 6x 10-4.

The analysis of total arsenic in food has up to date suffered from difficulties with respect to accuracy and precision. Furthermore, specified data for arsenic are strongly needed because of the large differences in toxicity to humans of the various forms of arsenic.

The intake of total arsenic in the human diet is usually dominated by organic arsenic derived from seafood. The available data about the possible human exposure to inorganic arsenic (often using the assumption that non-seafood commodities contain only inorganic arsenic) suggest that the PTWI will normally not be exceeded, unless there is a large contribution from drinking water. Further research is needed about the fate of organic arsenicals and the possibility that they might be converted to more toxic inorganic forms of arsenic, whether by processing or by metabolism in animals or humans.

Metals

Cadmium

Reference to JECFA: 16 (1972), 33 (1988), 41 (1993), 55 (2000), 61 (2003), 64 (2005)
Toxicological guidance value: PTWI 0.007 mg/kg bw (1988, maintained in 2000 & 2003)
Residue definition: Cadmium, total
Synonyms: Cd
Related code of practice: Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
VB 0040	Brassica vegetables	0.05	ML	Adopted	2005	FAC		
VA 0035	Bulb vegetables	0.05	ML	Adopted	2005	FAC		
VC 0045	Fruiting vegetables, cucurbits	0.05	ML	Adopted	2005	FAC		
VO 0050	Fruiting vegetables, other than cucurbits	0.05	ML	Adopted	2005	FAC	Excluding tomatoes and edible fungi.	
VL 0053	Leafy vegetables	0.2	ML	Adopted	2005	FAC		
VP 0060	Legume vegetables	0.1	ML	Adopted	2001	FAC		
VR 0589	Potato	0.1	ML	Adopted	2005	FAC	Peeled	
VD 0070	Pulses	0.1	ML	Adopted	2001	FAC	Excluding soya bean (dry)	
VR 0075	Root and tuber vegetables	0.1	ML	Adopted	2005	FAC	Excluding potato and celeriac	
VS 0078	Stalk and stem vegetables	0.1	ML	Adopted	2005	FAC		
GC 0081	Cereal grains, except buckwheat, canihua and quinoa	0.1	ML	Adopted	2001	FAC	Excluding wheat and rice; and bran and germ	
CM 1205	Rice, polished	0.4	ML	Adopted	2006	FAC		
GC 0654	Wheat	0.2	ML	Adopted	2005	FAC		
IM 0152	Cephalopods	2	ML	Adopted	2006	FAC	Without viscera	
IM 0151	Marine bivalve molluscs	2	ML	Adopted	2006	FAC	Excluding oysters and scallops	
	Natural mineral waters	0.003	ML	Adopted	CS 108-1981	NMW	Expressed in mg/l	
	Salt, food grade	0.5	ML	Adopted	CS 150-1985	NFSDU		

At the 61th JECFA (2003) it was estimated that the total intake of cadmium ranged from 2.8 to 4.2 µg/kg bw per week. This was calculated from available data on concentrations and food consumption taken from the GEMS/Food regional diets and corresponds to approximately 40-60% of the current PTWI of 7 µg/kg bw/week. Regarding major dietary sources of cadmium, the following foods contributed 10% or more to PTWI in at least one of the GEMS/Food regions: rice, wheat, starchy roots/tubers, and molluscs. Vegetable (excluding leafy vegetables) contribute >5% to the PTWI in two regions.

The 36th CCFAC (2004) decided to discontinue the work on developing MLs for cadmium in fruits, meat of cattle, pigs, sheep and poultry; horse meat; herbs, fresh; fungi (edible); celeriac; soya beans (dry); and peanuts as no levels were necessary because these foods were no major contributors to cadmium intake (ALINORM 04/27/12, para. 176).

The 64th JECFA (2005) conducted intake and impact assessment requested by the 36th session of CCFAC for the seven commodity groups; rice, wheat, potatoes, stem and root vegetables, leafy vegetables, other vegetables and molluscs taking into account different MLs. The JECFA concluded that the effect of different MLs on the overall intake of cadmium

Cadmium

would be very small.

Cadmium is a relatively rare element, released to the air, land, and water by human activities. In general, the two major sources of contamination are the production and utilization of cadmium and the disposal of wastes containing cadmium. Increases in soil cadmium content will result in an increase in the uptake of cadmium by plants; the pathway of human exposure from agricultural crops is thus susceptible to increases in soil cadmium. The cadmium uptake by plants from soil is greater at low soil pH. Edible free-living food organisms such as shellfish, crustaceans, and fungi are natural accumulators of cadmium. Similar to humans, there are increased levels of cadmium in the liver and kidney of horses and some feral terrestrial animals. Regular consumption of these items can result in increased exposure. Tobacco is an important source of cadmium uptake in smokers. (Environmental health criteria for cadmium; International Programme on Chemical Safety (IPCS); 1992)

Data from experimental animals and humans show that pulmonary absorption is higher than gastrointestinal absorption. The gastrointestinal absorption of cadmium is influenced by the type of diet and nutritional status. Cadmium absorbed from the lungs or the gastrointestinal tract mainly accumulates in the liver and kidneys. Although cadmium accumulates in the placenta, transfer to the fetus is low. Excretion is normally slow, and the biological half-time is very long (decades). The binding of intracellular cadmium to metallothionein in tissues protects against the toxicity of cadmium. Excretion occurs mainly via urine. (Environmental health criteria for cadmium; International Programme on Chemical Safety (IPCS); 1992)

The kidney is considered the critical target organ for the general population as well as for occupationally exposed populations. The accumulation of cadmium in the kidney leads to renal dysfunction. Chronic obstructive airway disease is associated with long-term high-level occupational exposure by inhalation. (Environmental health criteria for cadmium; International Programme on Chemical Safety (IPCS); 1992).

The IARC classified cadmium and cadmium compounds in group 1, carcinogenic to humans (1993).

Metals

Lead

Reference to JECFA: 10 (1966), 16 (1972), 22 (1978), 30 (1986), 41 (1993), 53 (1999)
Toxicological guidance value: PTWI 0.025 mg/kg bw (1986, maintained in 1993 & 1999)
Residue definition: Lead, total
Synonyms: Pb
Related code of practice: Code of Practice for the Prevention and Reduction of Lead Contamination in Foods (CAC/RCP 56-2004)
Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
FT 0026	Assorted (sub)tropical fruits, edible peel	0.1	ML	Adopted	2001	FAC		
FI 0030	Assorted (sub)tropical fruits, inedible peel	0.1	ML	Adopted	2001	FAC		
FB 0018	Berries and other small fruits	0.2	ML	Adopted	2001	FAC		
FC 0001	Citrus fruits	0.1	ML	Adopted	2001	FAC		
FP 0009	Pome fruits	0.1	ML	Adopted	2001	FAC		
FS 0012	Stone fruits	0.1	ML	Adopted	2001	FAC		
VB 0040	Brassica vegetables	0.3	ML	Adopted	2001	FAC	Excluding kale	
VA 0035	Bulb vegetables	0.1	ML	Adopted	2001	FAC		
VC 0045	Fruiting vegetables, Cucurbits	0.1	ML	Adopted	2001	FAC		
VO 0050	Fruiting vegetables, other than Cucurbits	0.1	ML	Adopted	2001	FAC	Excluding mushrooms	
VL 0053	Leafy vegetables	0.3	ML	Adopted	2001	FAC	Including Brassica leafy vegetables but excluding spinach.	
VP 0060	Legume vegetables	0.2	ML	Adopted	2001	FAC		
VD 0070	Pulses	0.2	ML	Adopted	2001	FAC		
VR 0075	Root and tuber vegetables	0.1	ML	Adopted	2001	FAC	Including peeled potatoes	
	Canned fruit cocktail	1	ML	Adopted	CS 78-1981	PFV		
	Canned mangoes	1	ML	Adopted	CS 159-1987	PFV		
	Canned pineapple	1	ML	Adopted	CS 42-1981	PFV		
	Canned raspberries	1	ML	Adopted	CS 60-1981	PFV		
	Canned strawberries	1	ML	Adopted	CS 62-1981	PFV		
	Canned tropical fruit salad	1	ML	Adopted	CS 99-1981	PFV		
	Jams (fruit preserves) and jellies	1	ML	Adopted	CS 79-1981	PFV		
	Mango chutney	1	ML	Adopted	CS 160-1987	PFV		
	Table olives	1	ML	Adopted	CS 66-1981	PFV		
	Canned asparagus	1	ML	Adopted	CS 56-1981	PFV		

Metals

Lead

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Canned carrots	1	ML	Adopted	CS 116-1981	PFV		
	Canned green beans and canned wax beans	1	ML	Adopted	CS 16-1981	PFV		
	Canned green peas	1	ML	Adopted	CS 58-1981	PFV		
	Canned mature processed peas	1	ML	Adopted	CS 81-1981	PFV		
	Canned mushrooms	1	ML	Adopted	CS 55-1981	PFV		
	Canned palmito	1	ML	Adopted	CS 144-1985	PFV		
	Canned sweet corn	1	ML	Adopted	CS 18-1981	PFV		
	Pickled cucumbers (cucumber pickles)	1	ML	Adopted	CS 115-1981	PFV		
JF 0175	Fruit juices	0.05	ML	Adopted		FAC	Including nectars; Ready to drink	
GC 0081	Cereal grains, except buckwheat, canihua and quinoa	0.2	ML	Adopted	2001	FAC		
	Canned chestnuts and canned chestnuts puree	1	ML	Adopted	CS 145-1985	PFV		
MM 0097	Meat of cattle, pigs and sheep	0.1	ML	Adopted	2001	FAC	Also applies to the fat from meat	
PM 0110	Poultry meat	0.1	ML	Adopted	2001	FAC		
MO 0812	Cattle, Edible offal of	0.5	ML	Adopted	2001	FAC		
MO 0818	Pig, Edible offal of	0.5	ML	Adopted	2001	FAC		
PO 0111	Poultry, Edible offal of	0.5	ML	Adopted	2001	FAC		
	Fish	0.3	ML	Adopted	2006	FAC		
	Edible fats and oils	0.1	ML	Adopted	CS 19-1981,	FO	Edible fats and oils not covered by individual standards	1)
	Fat spreads and blended spreads	0.1	ML	Adopted	CS 256-2007	FO		1)
	Named animal fats	0.1	ML	Adopted	CS 211-1999	FO	Lard, rendered pork fat, premier jus and edible tallow.	
OR 0305	Olive oil, refined	0.1	ML	Adopted	CS 33-1981	FO		
OC 0305	Olive oil, virgin	0.1	ML	Adopted	CS 33-1981	FO		
OR 5330	Olive, residue oil	0.1	ML	Adopted	CS 33-1981	FO	Olive pomace oil	
PF 0111	Poultry fats	0.1	ML	Adopted	2001	FAC		
OC 0172	Vegetable oils, Crude	0.1	ML	Adopted	CS 210-1999, 2001	FO, FAC	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize,	

Metals

Lead

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
							mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein.	
OR 0172	Vegetable oils, Edible	0.1	ML	Adopted	CS 210-1999, 2001	FO, FAC	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein.	
ML 0106	Milks	0.02	ML	Adopted	2001	FAC	A concentration factor applies to partially or wholly dehydrated milks.	The previous footnote “For dairy products, an appropriate concentration factor should apply” was changed by the 35th CCFAC.2)
LS	Secondary milk products	0.02	ML	Adopted	2001	FAC	As consumed	
	Natural mineral waters	0.01	ML	Adopted	CS 108-1981	NMW	Expressed in mg/l	
	Infant formula	0.02	ML	Adopted	2001	FAC	Ready to use	CCNFSDU is revising at Step 6 the Codex Standard for Infant formula which includes an ML for lead at the same level.
	Salt, food grade	2	ML	Adopted	CS 150-1985	NFSDU		
FF	Wine	0.2	ML	Adopted	2001	FAC		The OIV requested special consideration to be given to levels of lead in wines that had been stored for long periods of time (ALINORM 01/41, para.123).

1) The revised Standards for oils and fats contain the following wording for the mentioned contaminant MLs: "The products covered by the provisions of this Standard shall comply with MLs being established by the CAC but in the meantime the following limits will apply." The Codex Standard for Edible Fats and Oils Not Covered by Individual Standards contains the same contaminant provisions as the other recent Standards for oils and fats (only applying to Pb and As).

2) The 32nd CAC (2001) requested reevaluation of the lead MLs in milk and milk fat (ALINORM 01/41, para. 121); see also ALINORM 03/12 para. 135-137. The 35th CCFAC (2004) discussed the issue of the necessity of an ML for milk, as milk was not a major contributor to the intake of lead. However, in view of opinions that milk is a major contributor to the exposure of infants and young children, the ML for milk was maintained. The Committee decided to inform the CAC that the current level for lead in milk fat (0.1 mg/kg) should be revoked (no documentation of such a decision is found in the CAC 2003 report however).

Exposure to lead can occur from many sources but usually arises from industrial use. Lead and its compounds can enter the environment during mining, smelting, processing, use, recycling, or disposal. The main uses of lead are in batteries, cables, pigments, plumbing, gasoline, solder and steel products, food packaging, glassware, ceramic products, and pesticides. The main exposure of the general non-smoking adult population is from food and water. Airborne lead may contribute significantly to exposure, depending on such factors

Lead

as use of tobacco, occupation, and proximity to sources such as motorways and lead smelters. Food, air, water, and dust or soil are the main potential sources of exposure of infants and young children (WHO Food Additives Series 44, 2000 with reference to Environmental health criteria for inorganic lead, International Programme on Chemical Safety (IPCS), 1995).

The rate of absorption of lead after ingestion can range from 3% to 80%. It is heavily influenced by food intake, much higher rates of absorption occurring after fasting than when lead is ingested with a meal. Absorption is also affected by age, the typical absorption rates in adults and infants being 10% and 50%, respectively. Up to 50% of the inhaled lead compound may be absorbed. After its absorption and distribution in blood, lead is initially distributed to soft tissues throughout the body. Eventually, bone accumulates lead over much of the human life span and may serve as an endogenous source of lead. The half-life for lead in blood and other soft tissues is about 28-36 days, but it is much longer in the various bone compartments. The percentage retention of lead in body stores is higher in children than adults. Inorganic lead is not metabolized. Lead that is not distributed is mainly excreted through the kidney. (WHO Food Additives Series 44, 2000 with reference to Environmental health criteria for inorganic lead, International Programme on Chemical Safety (IPCS), 1995)

Lead is a classical chronic or cumulative poison. In humans, lead can result in a wide range of biological effects depending upon the level and duration of exposure. Health effects are generally not observed after a single exposure. Many of the effects that have been observed in laboratory animals have also been observed in humans, including hematological effects, neurological and behavioral effects, renal effects, cardiovascular effects, and effects on the reproductive system. In addition, lead has been shown to have effects on bone and on the immune system in laboratory animals. Children are more vulnerable to the effects of lead than adults. Lead has been shown to be associated with impaired neurobehavioral functioning in children. Impaired neurobehavioral development was considered to be the most critical effect. (Food Additives Series 44, 2000 with reference to Environmental health criteria for inorganic lead, International Programme on Chemical Safety (IPCS), 1995).

Inorganic lead compounds are classified by the IARC as probably carcinogenic to humans (Group 2A; Vol. 87, 2006)

Metals

Mercury

Reference to JECFA: 10 (1966), 14 (1970) , 16 (1972), 22 (1978)
Toxicological guidance value: PTWI 0.005 mg/kg bw (1978)
Residue definition: Mercury, Total
Synonyms: Hg
Related code of practice: Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Natural mineral waters	0.001	ML	Adopted	CS 108-1981	NMW	Expressed in mg/l	
	Salt, food grade	0.1	ML	Adopted	CS 150-1985	NFSDU		

No CCFAC position document was available about mercury.

Mercury is a naturally occurring metallic element which can be present in foodstuffs by natural causes; elevated levels can also occur due to e.g. environmental contamination by industrial or other uses of mercury. Methylmercury and also total mercury levels in terrestrial animals and plants are usually very low; the use of fish meal as animal feed can however also lead to higher methyl mercury levels in other animal products.

Metals

Methylmercury

Reference to JECFA: 22 (1978), 33 (1988), 53 (1999), 61 (2003), 67 (2006)
Toxicological guidance value: PTWI 0.0016 mg/kg bw (2003; confirmed in 2006,)
Residue definition: Methylmercury
Related code of practice: Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Fish	0.5	GL	Adopted	1991	FAC, FFP	Except predatory fish Intended for methylmercury in fresh or processed fish and fish products moving in international trade. a)	
	Predatory fish	1	GL	Adopted	1991	FAC, FFP	Predatory fish such as shark (WS 0131), swordfish, tuna (WS 0132), pike (WF 0865) and others. Intended for methylmercury in fresh or processed fish and fish products moving in international trade. a)	1)

1) The GLs for methylmercury in fish were adopted by the CAC-19 in 1991 (ALINORM 91/40, para. 202), on the understanding that the levels would be kept under review by the CCFAC as well as the CCFFP, especially as to the identification of predatory species of fish to which the higher GL applies.

The 53rd JECFA（1999）calculated the human exposure to methylmercury in regional diets to range from 0.3-1.5 ug/kg bw/week. Nationally reported dietary exposures are in the range 0.1 –2.0 mcg/kg bw/week. The 53rd JECFA maintained the PTWI of 3.3 ug bw for methylmercury set in the previous meetings of the JECFA and recommended that methylmercury be re-evaluated in 2002 when new information on the cohort in one of the studies could be assessed and possibly other new relevant data could be available. The JECFA also recommended that the nutritional benefits of fish consumption are weighted against the possibility of harm when limits on methylmercury concentrations in fish or on fish consumption are being considered.

The 67th JECFA confirmed the PTWI of 1.6 µg/kg bw, set in 2003, based on the most sensitive toxicological end-point (developmental neurotoxicity) in the most susceptible species (humans). However, the Committee noted that life-stages other than the embryo and fetus may be less sensitive to the adverse effects of methylmercury. The Committee considered that intakes of up to about two times higher than the existing PTWI would not pose any risk of neurotoxicity in adults, except for women of childbearing age in order to protect the embryo and fetus. Concerning infant and children up to about 17 years no firm conclusions could be drawn; it is clear that they are not more sensitive than the embryo or fetus, but may be more sensitive than adults because significant development of the brain continues in infancy and childhood. Therefore, no level of intake higher than the existing PTWI could be identified of infants and children.

The 67th JECFA recommended that:
- Known benefits of fish consumption need to be taken in consideration in any advice aimed at different populations, since fish make an important contribution to nutrition, especially in certain regional and ethnic diets. Risk managers may wish to consider whether specific advice should be given concerning children and adults after weighing the potential risks and benefits.
- Setting of guideline levels for methyl mercury in fish may not be an effective way of reducing exposure for the general population, however advice to population subgroups that may be at risk may provide an effective method for lowering the number of individuals with exposures greater than the PTWI.

Metals

Methylmercury

The 24th CCFAC (1992) informed the CAC and the CCFFP that the recommended GLs for mercury in fish referred to total mercury rather than methylmercury. The 20th CAC (1993) decided to maintain the GLs for methylmercury in fish as previously adopted, while recommending that the establishment of corresponding GLs for total mercury in fish be considered by the CCFAC at its next meeting. The 26th CCFAC (1994) noted that analysis of total mercury was generally adequate to ensure that GLs for methylmercury were not exceeded and decided that the establishment of GLs for total mercury in fish was not necessary. The 29th CCFAC (1997) noted that the 43rd CXEXEC (1996) had recommended that the CCFAC initiate a new risk analysis on methylmercury. It was decided to defer any decision on the question of GLs based on methylmercury or total mercury until JECFA had performed the risk assessment. The 32nd CCFAC (2000) took note of these recommendations made by the 53rd JECFA.

The 37th CCFAC agreed that the revision of the GLs requires more comprehensive consideration by CCFAC in order to take into account all factors related to the consumption of fish, in particular, risks and benefits. In the meantime, the existing GLs can be retained with the understanding that enforcement can be performed by determination of total mercury as a screening method (for facilitating control/monitoring). Methylmercury needs only be determined for verification purposes (ALINORM 05/28/12, para. 202).

The 38th CCFAC agreed: to forward a request to the CAC for an FAO/WHO expert consultation on health risks associated with methylmercury and dioxins and dioxin-like PCBs in fish and health benefits of fish consumption; to postpone consideration on the need to revise the guideline levels for methylmercury in fish pending the outcome of the requested FAO/WHO consultation and to retain the current Codex guideline levels; not to start compiling data on the ratio of methylmercury to total mercury in different fish species; and to postpone
discussion on the risk communication aspects of methylmercury in fish (ALINORM 06/29/12, paras 191-194).

The First CCCF was informed by the WHO Representative that JECFA's conclusion with respect to guideline levels must be considered in relation to the fact that guidelines already in place in some national jurisdictions had already influenced the range of observed mercury concentrations by eliminating fish containing high concentrations of mercury from the market. The First CCCF reaffirmed the decision made by the 38th Session of the CCFAC to postpone consideration of the need to revise the guideline levels for methylmercury in fish pending the outcomes of a joint FAO/WHO expert consultation on health risks associated with methylmercury and dioxins and dioxin-like PCBs in fish and the health benefits of fish consumption and to retain the current Codex guideline levels for the time being. (ALINORM 07/30/41, paras 34-35)

The 30th Commission recalled that its 29th Session had requested FAO and WHO for scientific advice on the health risks associated with methylmercury and dioxins and dioxin-like PCBs in fish and the health benefits of fish consumption. The Representative of FAO, speaking on behalf of FAO and WHO, informed the Commission that a step-wise preparatory process was being taken, given the complex nature of the issue and the need for innovative principles and methodology. The Representative indicated that, possibly at a first stage, FAO and WHO would consider conducting qualitative risk-benefit assessment of fish consumption, specifically addressing issues related to the impact of methylmercury exposure on women of child-bearing age and at a later stage, conducting quantitative assessment including the intake of dioxin and dioxin-like PCBs, taking into account consumption of fatty fish, considered as a significant source of beneficial fatty acids. (ALINORM 07/30/REP, para. 192)

Methylmercury is the most toxic form of mercury and is formed in aquatic environments. Methylmercury therefore is found mainly in aquatic organisms. It can accumulate in the food chain; the levels in large predatory fish species are therefore higher than in other species and fish is the predominant source of human exposure to methylmercury. Methylmercury and also total mercury levels in terrestrial animals and plants are usually very low; the use of fish meal as animal feed can however also lead to higher methyl mercury levels in other animal products.

In all experimental animal species evaluated, methylmercury was readily absorbed (up to 95%) after oral exposure. Methylmercury crossed both the blood–brain barrier and the placenta effectively, resulting in higher concentrations of mercury in the brain of the fetus than of the mother. Methylmercury is eliminated mainly via the bile and faeces, neonatal animals having a lower excretory capacity than adults. Methylmercury is toxic to the nervous system, kidney, liver and reproductive organs, neurotoxicity being the most sensitive end-point (WHO Food additives Series 52; 2004).

Metals

Methylmercury

a) Lots should be considered as being in compliance with the guideline levels if the level of methylmercury in the analytical sample, derived from the composite bulk sample, does not exceed the above levels. Where these Guideline levels are exceeded, governments should decide whether and under what circumstances, the food should be distributed within their territory or jurisdiction and what recommendations, if any, should be given as regards restrictions on consumption, especially by vulnerable groups such as pregnant women.

Metals

Tin

Reference to JECFA: 10 (1966), 14 (1970), 15 (1971), 19 (1975), 22 (1978), 26(1982), 33(1988), 55 (2000), 64 (2005)
Toxicological guidance value: PTWI 14 mg/kg bw (1988, Expressed as Sn; includes tin from food additive uses; maintained in 2000)
Residue definition: Tin, total (Sn-tot) when not otherwise mentioned; inorganic tin (Sn-in); or other specification
Synonyms: Sn
Related code of practice: Code of Practice for the Prevention and Reduction of Inorganic Tin Contamination in Canned Foods (CAC/RCP 60-2005)
Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)		Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Canned beverages	150	C	ML	Adopted	2007	FAC 99-07		Changed from 200 mg/kg in 2005.
	Cooked cured chopped meat	50		ML	Adopted	CS 98-1981	PMPP	For products in other containers than tinplate container	
	Cooked cured ham	50		ML	Adopted	CS 96-1981	PMPP	For products in other containers than tinplate container	
	Cooked cured pork shoulder	50		ML	Adopted	CS 97-1981	PMPP	For products in other containers than tinplate container	
	Corned beef	50		ML	Adopted	CS 88-1981	PMPP	For products in other containers than tinplate container	
	Luncheon meat	50		ML	Adopted	CS 89-1981	PMPP	For products in other containers than tinplate container	
	Canned foods (other than beverages)	250	C	ML	Adopted	2007	FAC 99-07		

In previous JECFA meetings it was noted that inorganic tin compounds generally have low systemic toxicity in animals, because of limited absorption from the gastrointestinal tract, low accumulation in tissues, and rapid passage through the gastrointestinal tract. Insoluble tin compounds are less toxic than soluble tin salts.

The 33rd JECFA (1988) established a PTWI for inorganic tin of 14 mg/kg bw.

At the 55th JECFA (2000), it was concluded that the acute toxicity of inorganic tin in animals and humans results from irritation of the mucosa of the gastrointestinal tract, which may lead to vomiting, diarrhea, anorexia, depression, ataxia, and muscular weakness. There was insufficient data available to establish an ARfD for inorganic tin. The committee did not consider studies on organic tin compounds, since it had concluded at the 22nd JECFA (1978), that these compounds differ considerably from inorganic tin compounds with respect to toxicity and should be considered separately.

The 55th JECFA (2000) maintained the existing PTWI and reiterated that limited human data available indicated that concentrations of 150mg/kg tin in canned beverages and 250 mg/kg in other canned foods may produce acute manifestations of gastric irritation in certain individuals. This is considered to be a reversible effect however, which may occur in a limited number of sensitive subject only.

Metals

Tin

Following the discussions in the 34th CCFAC (2002) and in the 35th CCFAC (2003) (ALINORM 03/12, para.146 and ALINORM 03/12A, para.160), the proposed MLs were repeatedly returned to step 3. The 35th CCFAC changed the terminology of the commodities to which the proposed draft MLs apply, which previously was “liquid canned foods resp. solid foods”, to “canned beverages” and “canned foods other than beverages”. The Committee decided to ask JECFA to evaluate current tin level in canned foods and to determine an acute reference dose; it was noted that new data would become available. The 36th CCFAC (2004) decided to hold the proposed MLs and reconsider these MLs in the light of the 64th JECFA re-evaluation (ALINORM 04/27/12, para.171).

The 64th JECFA (2005) concluded that the data available indicated that it is inappropriate to establish and ARfD for inorganic tin since whether or not irritation of gastrointestinal tract occur after ingestion of a food containing tin depends on the concentration and nature of in the product, rather than on the dose ingested on a body-weight basis.

The First CCCF (2007) agreed to forward the draft MLs to the 30th Commission for adoption at Step 8 and noted that the adoption of the ML for tin in canned foods (other than beverages) would result in consequential changes to MLs for tin in certain canned products (i.e. products in tin-layered cans), currently included in Schedule 1. (ALINORM 07/30/41, para. 81).

The 30th Commission adopted these MLs at Step 8 with the understanding that the existing MLs for tin in certain canned foods included in Schedule I of the GSCTF would be replaced by the adopted MLs. (ALINORM 07/30/REP)

Tin is mainly used in tinplated containers, but it is also extensively used in solders, in alloys including dental amalgams. Inorganic tin compounds, in which the element may be present in the oxidation states of +2 or +4, are used in a variety of industrial processes for the strengthening of glass, as a base for colours, as catalysts, as stabilizers in perfumes and soaps, and as dental anticariogenic agents. On the whole, contamination of the environment by tin is only slight. Food is the main source of tin for man. Small amounts are found in fresh meat, cereals, and vegetables. Larger amounts of tin may be found in foods stored in plain cans and, occasionally, in foods stored in lacquered cans. Some foods such as asparagus, tomatoes, fruits, and their juices tend to contain high concentrations of tin if stored in unlaquered cans (Environmental health criteria for tin; International Programme on Chemical Safety (IPCS); 1980). Inorganic tin is found in food in the +2 and +4 oxidation states; it may occur in a cationic form (stannous and stannic compounds) or as inorganic anions (stannites or stannates).

Mycotoxins

Aflatoxins, Total

Reference to JECFA:	31 (1987), 46 (1996), 49 (1997) , 68 (2007)
Toxicological guidance value:	Carcinogenic potency estimates for aflatoxins B, G, M (1997, Intake should be reduced to levels as low as reasonably possible.)
Residue definition:	Aflatoxins total (B1 +B2 + G1 + G2)
Synonyms:	Abbreviations, AFB, AFG, with numbers, to designate specific compounds
Related code of practice:	Code of Practice for the Prevention and Reduction of Aflatoxin Contamination in Peanuts (CAC/RCP 55-2004) Code of Practice for the Prevention and Reduction of Aflatoxin Contamination in Tree Nuts (CAC/RCP 59-2005) Code of Practice for the Reduction of Aflatoxin B1 in Raw Materials and Supplemental Feedingstuffs for Milk Producing Animals (CAC/RCP 45-1997) Proposed Draft Code of Practice for the Prevention and Reduction of Aflatoxin Contamination in Dried Figs (Step 3)

Code	Food/product	Level (ug/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
TN 0660	Almonds	15	ML	7		FAC 03-07	For further processing	
TN 0660	Almonds	8	ML	7		FAC 03-07	Ready-to-eat	
TN 0666	Hazelnuts	15	ML	7		FAC 03-07	For further processing	
TN 0666	Hazelnuts	8	ML	7		FAC 03-07	Ready-to-eat	
SO 0697	Peanut	15	ML	Adopted	1999	FAC	Applies to peanuts intended for further processing. For sampling plan, see Schedule I.	1)
TN 0675	Pistachio nut	15	ML	7		FAC 03-07	For further processing	
TN 0675	Pistachio nut	8	ML	7		FAC 03-07	Ready-to-eat	

1) The 1994 CCCPL decided not to proceed with the proposed GL for processed peanuts and to advance the proposed GL for raw peanuts (intended for further processing), associated with a specific sampling plan because the contamination is usually very inhomogeneous in a lot. It is assumed that raw peanuts are the major commodity in international trade.

The 23rd CCFAC (1991) decided to discontinue the development of a ML for aflatoxins in foods in general, and to discuss the problems on a commodity basis.

It is acknowledged that for primary plant products the aflatoxin contamination is often not homogeneous and a sampling plan is necessary to assure reasonable application of MLs. A general position paper on aflatoxins in food and feeds (CX/FAC 97/16) was presented to the 29th CCFAC (1997).
- Corn was included in a Technical Consultation on sampling plans for aflatoxins in commodities. See FAO Food and nutrition Paper 55 (Rome, 1993).
- The 26th CCFAC (1994) decided to discontinue the establishment of GLs for AFB1 in supplementary feedingstuffs for milk-producing animals (previously proposed at the level of 5 ug/kg), based on the assumption that the relationship between aflatoxins in milk and feeds is not (completely) clear and that there is not much international trade in (composite) supplementary feedingstuffs. International trade mostly is in the form of individual commodities which can be used as feed components in various quantities, directed to other feed uses than milk producing animals, or to other uses in general, or be decontaminated etc. Therefore, a Code of Practice for the reduction of aflatoxin B1 in raw materials and supplemental feedingstuffs for milk-producing animals was developed and adopted as RCP 045-1997.

The 35th CCFAC (2003) agreed to the elaboration of MLs for aflatoxins in almonds, hazelnuts and pistachios, based on the ALARA principle and with the understanding that related sampling plans need to be established (ALINORM 03/12A para.129). The 37th CCFAC (2005) advanced the ML for unprocessed almonds, hazelnuts and pistachios while the Committee decided to circulate for comments at step 3 the ML for processed almonds, hazelnuts and pistachios(ALINORM 05/28/12 para.141). The 38th CCFAC (2006) agreed to rename “processed” and “unprocessed” tree nuts to as “ready-to-eat” and tree nuts “for further processing” respectively and to hold at step 7 the ML in tree nuts for further processing and to advance to step 5 the ML in ready-to-eat tree nuts(ALINORM 06/29/12 para.132). The 29th CAC (2006) adopted ML for ready-to-eat tree nuts and advanced to step 6.

Mycotoxins

Aflatoxins, Total

The 38th CCFAC agreed to request JECFA to conduct a dietary exposure assessment on ready-to-eat tree nuts and impact on exposure taking into account hypothetical levels of 4, 8, 10 and 15 µg/kg, putting in the context of exposure from other sources and previous exposure assessments on maize and groundnuts. The 38th CCFAC decided to expand the discussion paper on the aflatoxin level in ready-to-eat tree nuts, considering I) the detailed data on distribution on aflatoxins between lots, ii) consumer health risk assessment of different levels of aflatoxin in ready-to-eat tree nuts, iii) sampling plan for tree nuts, iv) effect of code of practice and v) terminology of "ready-to-eat" and "for further processing" for consideration at the 1st session of CCCF (ALINORM 06/29/12 paras 129-130).

The First CCCF agreed to hold both the draft ML of 15 µg/kg for total aflatoxins in almonds, hazelnuts and pistachios "for further processing" and the draft ML of 8 µg/kg for total aflatoxins in almonds, hazelnuts and pistachios "ready-to-eat" at Step 7 and to resume discussion on these draft MLs at its next session, after the results of the forthcoming 68th JECFA evaluation are available. The CCCF also agreed to establish an electronic working group lead by the European Community to update the discussion paper which would provide useful information for further discussion on the MLs at its next session. (ALINORM 07/30/41, paras 57-58)

The 68th JECFA concluded that consumption of almonds, Brazil nuts, hazelnuts, pistachios and dried figs contributes to more than 5% of the total aflatoxin dietary exposure in only five of the 13 GEMS/Food cluster diets (clusters B, C, D, E and M). Setting an ML of 20 µg/kg for these products would only have an impact on the relative contribution to aflatoxin dietary exposure in these clusters (including the high-level consumers of tree nuts). This can solely be attributed to the elevated aflatoxin level in pistachios. For the tree nuts other than pistachios, as well as dried figs, setting an ML has no effect on aflatoxin dietary exposure. Also, enforcing an ML of 4, 8, 10 or 15 µg/kg has little further impact on the overall dietary exposure to aflatoxin compared to an ML of 20 µg/kg.

(Sampling Plan)

The 38th CCFAC agreed to further elaborate the proposed draft sampling plan once a ML had been established by Committee and to include considerations on the draft sampling plan for tree nuts in the discussion paper on total aflatoxin levels in processed tree-nuts (ALINORM 06/29/12 para.125).

The First CCCF agreed that the proposed draft Sampling Plan for Aflatoxin Contamination in Almonds, Brazil Nuts, Hazelnuts and Pistachios be returned to Step 2 for redrafting by an electronic working group, lead by the USA, with a view to circulation at Step 3 and consideration at Step 4 at the next session of the CCCF. It was also agreed that the working document to be considered at the next session of the CCCF incorporate a revised proposed draft Sampling Plan as well as an explanatory text in support of the consideration of the Sampling Plan. (ALINORM 07/30/41, para. 62)

<u>Code of Practice for the Prevention and Reduction of Aflatoxin Contamination in Tree Nuts</u>

The 29th CAC adopted the Appendix to Code of Practice for the Prevention and Reduction on Aflatoxin Contamination in Tree Nuts to address additional measures for Brazil Nuts.

<u>Discussion paper on aflatoxins in Brazil nuts</u>

The First CCCF agreed that the discussion paper on aflatoxin contamination in Brazil nuts would be updated by the Delegation of Brazil, incorporating additional data that would become available on the contribution of the shell to aflatoxin contamination of Brazil nuts, for consideration at the next session of the Committee. (ALINORM 07/30/41, para. 66)

<u>Discussion paper on aflatoxins in dried figs</u>

Mycotoxins

Aflatoxins, Total

The First CCCF agreed to forward the project document proposing new work to the 59th Executive Committee for critical review and for approval by the 30th Commission. It also agreed to establish an electronic working group led by Turkey to prepare a draft proposed code of practice on the prevention and reduction of aflatoxin contamination in dried figs at Step 2, with a view to its circulation for comments at Step 3 and its consideration at Step 4 at the second session, pending the formal approval of new work by the Commission. (ALINORM 07/30/41, paras 120-121)

The 30th Commission approved the above new work. (ALINORM 07/30/REP, Appendix VII)

Aflatoxins are a group of highly toxic mycotoxins produced by fungi of the genus *Aspergillus*. The four main aflatoxins found in contaminated plant products are B1, B2, G1 and G2 and are a group of structurally related difuranocoumarin derivatives that usually occur together in varying ratios, AFB1 usually being the most important one. These compounds pose a substantial hazard to human and animal health. IARC (1992) classified aflatoxin B1 in Group 1 (human carcinogen) and aflatoxin M in Group 2B (probable human carcinogen). The liver is the primary target organ.

A wide range of foods may be contaminated with aflatoxins; they are most commonly found in groundnuts (peanuts), dried fruit, tree nuts (such as almonds, pecans, walnuts, pistachio and brazil nuts), spices, figs, crude vegetable oils, cocoa beans, maize, rice, cottonseed and copra. Aflatoxin B1 present in animal feed can partly be transferred to milk in the form of the metabolite aflatoxin M1 (mostly 1-2%, but higher percentages are found at low contamination levels in high producing animals.) Aflatoxin contamination is responsible for considerable economic losses and efforts are being made to reduce contamination of food and feedingstuff.

Mycotoxins

Aflatoxin M1

Reference to JECFA: 56 (2001)

Toxicological guidance value: Cancer potency estimates at specified residue levels (2001, Using worst-case assumptions, the additional risks for liver cancer predicted with use of proposed maximum levels of aflatoxin M1 of 0.05 and 0.5 µg/kg are very small. The potency of aflatoxin M1 appears to be so low in HBsAg- individuals that a carcinogenic effect of M1 intake in those who consume large quantities of milk and milk products in comparison with non-consumers of these products would be impossible to demonstrate. Hepatitis B virus carriers might benefit from a reduction in the aflatoxin concentration in their diet, and the reduction might also offer some protection in hepatitis C virus carriers.)

Residue definition: Aflatoxin M1

Synonyms: AFM1

Code	Food/product	Level (ug/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
ML 0106	Milks	0.5	ML	Adopted	2001	FAC		

The 24th CCFAC (1993) decided to stop the development of a specific standard for aflatoxin M1 in milk destined for use in baby foods. The CCFAC has discussed 2 options for a standard for aflatoxin M1 in milk: 0.05 ug/kg and 0.5 ug/kg. At the request of the 32nd CCFAC (2000), the 56th JECFA (2001) examined exposure to aflatoxin M1 and conducted a quantitative risk assessment to compare the consequences of setting the maximum level in milk at 0.05 ug/kg and 0.5 ug/kg. The estimates of the potency of aflatoxin M1 were combined with estimates of intake from the GEMS/Food European regional diet. JECFA noted that the calculation showed that, with worst case assumptions, the projected risks for liver cancer at the proposed maximum levels of aflatoxin M1 of 0.05 and 0.5 ug/kg are very small. As a result, 0.5 ug/kg was forwarded to the 33rd CCFAC (2001) which adopted this draft ML, noting that data supporting the lower level, if and when available, could be examined by the CCFAC at a future meeting when necessary.

It is acknowledged that the aflatoxin M1 level in milk is related to the aflatoxin B1 level in the animal feed. See notes under Aflatoxins, total.

Mycotoxins

Deoxynivalenol

Reference to JECFA:	56 (2001)
Toxicological guidance value:	PMTDI 0.001 mg/kg bw (2001)
Synonyms:	Vomitoxin; Abbreviation, DON
Related code of practice:	Code of Practice for the Prevention and Reduction of Mycotoxin Contamination in Cereals, Including Annexes on Ochratoxin A, Zearalenone, Fumonisins and Tricothecenes (CAC/RCP 51-2003)

Code	Food/product	Level (ug/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

The available data did not permit JECFA to set an acute reference dose however. The PMTDI is based on a chronic dietary study with mice, applying a safety factor of 100. An intake at the level of the PMTDI is not expected to result in effects of DON on the immune system, growth or reproduction, which are the most critical effects. JECFA recommended that toxic equivalency factors relative to DON be developed for the other trichothecenes commonly occurring in cereal grains, if sufficient data become available.

The JECFA estimated that the PMTDI for DON could be exceeded in 4 out of 5 GEMS/Food regional diets.

The situation regarding deoxynivalenol has been reviewed in a discussion paper (last version CX/FAC 03/35); the 35th CCFAC (2003) discontinued the consideration of this discussion paper and agreed to commence work on the elaboration of MLs for DON (ALINORM 03/12A, paras 180-182).

The 26th CAC (2003) approved the development of maximum levels for DON as new work (ALINORM 03/41, Appendix VIII).

The 36th CCFAC (2004) agreed to discontinue the consideration of maximum levels for deoxynivalenol for the time being. Instead, it agreed to request information on: the occurrence of deoxynivalenol in cereals; the influence of processing, decontamination, sorting, etc. to lower the level of DON in a lot; national levels or guideline levels for DON; sampling procedures and methods of analysis; etc. for consideration by the 37th Session of the Committee (ALINORM 04/27/12, paras 156-158).

The 37th CCFAC (2005) decided to establish an electronic Working Group to develop a discussion paper to provide comprehensive relevant data, including the occurrence of deoxynivalenol and the effects of processing on the levels of DON, for consideration at the 38th session (ALINORM 05/28/12, paras 148-150).

The 38th CCFAC (2006) agreed to endorse the recommendation of the ad hoc Working Group on Contaminants and Toxins in Foods to update the Discussion Paper on DON with: more data from regions where data on DON levels are missing or inadequate; additional data, especially on DON levels in maize; information on the effect on levels of seasonal variation; and information on the effect of processing on DON levels in foods (ALINORM 06/29/12, paras 137-138).

In view of the need for more occurrence data, including regional data on incidence and levels of DON in cereals over a period of several years, and for adequate information on consumption patters for various countries as a prerequisite to developing international standards, the First CCCF agreed to discontinue consideration of this item for the time being and to encourage countries to submit data on DON contamination to GEMS/Food Databases electronically and in the prescribed format. (ALINORM 07/30/41, para. 108)

The First CCCF noted that sufficient data on DON occurrence in food and fate at processing would not be available before the end of 2008 and that no information was provided on the availability of toxicological data. It agreed that DON remain on the priority list. (ALINORM 07/30/41, para. 126)

Mycotoxins

Deoxynivalenol

Deoxynivalenol (DON) is the major compound of a group of chemically related mycotoxins called type B trichothecenes (which are epoxy-sesquiterpenoid compounds) and is produced by certain Fusarium species, which are pathogens of several cereal grains. Closely related compounds are e.g. nivalenol and several acetyl-DON derivatives. DON is water-soluble and chemically very stable under most normal food processing conditions. DON contamination is commonly found in various cereals and cereal products. It undergoes rapid metabolism and elimination in livestock species and the transfer from feed to animal products is probably negligible. Maximum levels in feed are not needed to product public health, but are useful for the protection of animal health and productivity. Especially pigs are vulnerable.

In animals, decreased feed consumption, diarrhea and vomiting have been observed as acute effects. JECFA recognized that DON can lead to outbreaks of acute illness in humans.

Mycotoxins

Fumonisins

Reference to JECFA:	56 (2001)
Toxicological guidance value:	PMTDI 0.002 mg/kg bw (2001)
Synonyms:	(Several related compounds have been described, notably fumonisin B1, B2 and B3 (abbreviation: FB1 etc.))
Related code of practice:	Code of Practice for the Prevention and Reduction of Mycotoxin Contamination in Cereals, Including Annexes on Ochratoxin A, Zearalenone, Fumonisins and Tricothecenes (CAC/RCP 51-2003)

Code	Food/product	Level (ug/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

A position paper has been prepared for fumonisins (last version CX/FAC 00/22). The 32nd CCFAC (2000) asked the USA to finalize the position paper as a potential basis for future work (ALINORM 01/12 paras 106-109). No MLs have been proposed.

The Representative of WHO, speaking on behalf of the JECFA Secretariats, clarified at the First CCCF that there was no plan for JECFA to update the risk assessment conducted by the 56th JECFA meeting ant that an updated risk assessment could be conducted only when new data became available. (ALINORM 07/30/41, para. 135)

Fumonisins are a class of recently identified mycotoxins that are produced mainly by certain Fusarium species, especially F. moniliforme which is a pathogen of corn (Zea mays). Fumonisins are a structurally related group of diesters of propane-1,2,3-tricarboxylic acid and various 2-amino-12,16-dimethylpolyhydroxyeicosanes. There are at least 12 fumonisin analogues identified, classified into series A, B, F and P. The B-series, consisting mainly of fumonisin B1 and fumonisin B2, is believed to be the most abundant and most toxic group. A typical ratio between these analogues is B1:B2:B3 as 10:3:1. The worldwide occurrence of fumonisins in corn and corn-based products is well documented: sporadic natural occurrence in sorghum, rice and navy beans has been reported. Fumonisins are heat-stable, so cooking and other heat processes do not substantially reduce their levels in foods. Processing involving treatment of wet milling fractions may, however, lead to elimination of most fumonisin. The human exposure via food can vary to a large extent because of the large range of fumonisin contents found in practice. Fumonisins undergo rapid metabolism and elimination in livestock species and the transfer from feed to animal products is probably negligible. Maximum levels in feed are not needed to protect public health but are useful for the protection of animal health and productivity. In animals, various adverse effects have been observed. The horse appears to be the most sensitive species, and equine leukoencephalomalacia (ELEM) is the most frequently encountered disease. Fumonisins are also associated with liver damage, often also kidney lesions and changes in certain lipid classes, especially sphingolipids, in all animals studied. Carcinogenic effects have been observed in animals exposed to high dietary levels.

Nephrotoxicity, observed in several strains of rat, was considered by JECFA to be the most sensitive toxic effect. On the basis of the NOEL for renal toxicity and a safety factor of 100, the PMTDI was established. National estimates for the mean or median intake were generally much lower than the PMTDI (the highest being 0.2 ug/kg bw).

Mycotoxins

Ochratoxin A

Reference to JECFA:	37 (1990), 44 (1995), 56 (2001) , 68 (2007)
Toxicological guidance value:	PTWI 0.0001mg/kg bw (2001)
Residue definition:	Ochratoxin A
Synonyms:	(The term “ochratoxins” includes a number of related mycotoxins (A, B, C and their esters and metabolites), the most important one being ochratoxin A)
Related code of practice:	Code of Practice for the Prevention and Reduction of Mycotoxin Contamination in Cereals, Including Annexes on Ochratoxin A, Zearalenone, Fumonisins and Tricothecenes (CAC/RCP 51-2003)
	Code of Practice for the Prevention and Reduction of Ochratoxin A Contamination in Wine (CAC/RCP 63-2007)

Code	Food/product	Level (ug/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
GC 0640	Barley	5	ML	7		FAC 91-04,07		
GC 0650	Rye	5	ML	7		FAC 91-04,07		
GC 0654	Wheat	5	ML	7		FAC 91-04,07		

The situation regarding ochratoxins has been reviewed in a position paper (last version CX/FAC 99/14).

The draft ML of 5 ug/kg for ochratoxin A in raw wheat, barley and rye and derived products was forwarded for adoption at step 8 by the 34th CCFAC (2002) (ALINORM 03/12, paras 111-114), on the basis of the assumption that this level was ALARA. The 26th CAC (2003) discussed this proposal (ALINORM 03/41, paras 45-47). Many delegations were of the opinion that this proposed ML was too low and, taking account of the evaluation of the 56th JECFA (2001), noted that a ML of 20 ug/kg could be adequate in terms of public health and safety. The CAC concluded that there was a lack of consensus both regarding the appropriate ML and regarding the reference to derived products and returned the standard to step 6 for further work by the CCFAC. The 36th CCFAC (2004) noted that given the wide range of derived products and that many of them were of little or no importance in international trade, the maximum level should be limited to raw wheat, barley, and rye. The Committee agreed to hold the maximum level of 5 ug/kg for Ochratoxin A in raw wheat, barley, and rye at Step 7. The Committee also agreed, depending upon the available data, that JECFA should perform a comprehensive risk assessment by 2006, so that the Committee might reconsider this issue in the light of the outcome of the JECFA evaluation at its Session in 2007 (ALINORM 04/27/12, paras 132-137).

The ad hoc working group of the 38th CCFAC (2006) agreed to forward to the CAC for approval of new work, the project document “Code of practices for the prevention and reduction of Ochratoxin A contamination in wine”, and agreed that MLs for ochratoxin A in wine might be considered in future, pending collection of data on levels in wine and the outcomes of the elaboration of the Code. The Committee agreed to endorse the recommendation of the ad hoc Working Group on Contaminants and Toxins in Foods to start new work on the elaboration of the Code and clarified that the scope of this work should be limited to wine only. The Committee also agreed that the proposed draft Code would be circulated for comments at Step 3 and considered at the next session of the Committee. The 29th CAC (2006) approved the development of the Code as a new work (ALINORM 06/29/12, paras 139-142).

The 38th CCFAC agreed with the recommendations of the ad hoc Working Group to establish two electronic Working Groups to prepare separate discussion papers on ochratoxin A in coffee and ochratoxin A in cocoa, respectively, for circulation, comments and consideration at its next Session that might allow the Committee to decide if the development of Codes of Practice was appropriate (ALINORM 06/29/12, paras 143-145).

The First CCCF agreed to retain the draft MLs at Step 7 and to inform the Executive Committee that work on this item would be completed by 2009 (ALINORM 07/30/41, para. 50).

Mycotoxins

Ochratoxin A

The 68th JECFA (2007) retained the PTWI of 100 ng/kg bw. The estimated overall dietary exposure to Ochratoxin A from cereals (mainly European data) was adjusted to 8-17 ng/kg bw/week (processed cereals), compared with the 25 ng/kg bw/week (raw cereals) in the previous assessment. This is well below the PTWI. Moreover, contamination levels in the majority of raw cereal samples were below 5 µg/kg and only a few samples were above the highest proposed limit of 20 µg/kg. The 68th JECFA concluded that it would be unlikely that an ML of 5 or 20 µg/kg has an impact on dietary exposure to Ochratoxin A. The committee was unable to reach a conclusion regarding developing countries due to the lack of adequate data to consider.

Discussion paper on OTA in coffee

The First CCCF decided to establish an electronic working group, to be chaired by Brazil, to prepare a revised discussion paper for consideration at the second session. The revised discussion paper should incorporate new data and other relevant information including those submitted to the first session, and be accompanied by a project document proposing new work and possibly an outline of the proposed draft code of practice. (ALINORM 07/30/41, para. 113)

Discussion paper on OTA in cocoa

The First CCCF decided to establish and electronic working group, to be chaired by Ghana, to update the discussion paper with new data and other relevant information, and taking into account the comments made at the first session, for consideration at the second session. (ALINORM 07/30/41, para. 117)

Ochratoxin A is the major compound of a group of chemically related mycotoxins produced by species of the genera Aspergillus and Penicillium. Ochratoxin A contamination is commonly found in various cereals, some pulses, coffee, cocoa, figs, grapes, wine, nuts and coconut products. It can also be transferred through the feed to animal products and concentrates especially in the kidney, but may also be found in meat and milk. Most ochratoxin A is, however, converted to the less harmful ochratoxin-alpha in the rumen of ruminants. Ochratoxin A is a nephrotoxic mycotoxin, which is carcinogenic to rodents and has also teratogenic, immunotoxic and possibly neurotoxic properties. It has been associated with Balkan Endemic Nephropathy.

Mycotoxins

Patulin

Reference to JECFA: 35 (1989), 44 (1995)
Toxicological guidance value: PMTDI 0.0004 mg/kg bw (1995)
Residue definition: Patulin
Related code of practice: Code of Practice for the Prevention and Reduction of Patulin Contamination in Apple Juice and Apple Juice Ingredients in Other Beverages (CAC/RCP 50-2003)

Code	Food/product	Level (ug/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
JF 0226	Apple juice	50	ML	Adopted	2003	FAC	The ML also covers apple juice as ingredient in other beverages.	

The situation regarding patulin was reviewed in a position paper (last version CX/FAC 99/16).

The 26th CAC in 2003 adopted the ML. The possible reduction of the ML from 50 to 25 ug/kg will be reconsidered by the CCFAC once the Code of Practice has been implemented (i.e., after 4 years). More data are requested on the level of patulin in apple juice and apple juice ingredients for other beverages.

The First CCCF agreed to take patulin out of the priority list, noting that there was an existing maximum level and this topic was no longer considered a high priority. (ALINORM 07/30/41, para. 127)

Patulin is a low molecular weight hemiacetal lactone mycotoxin produced by species of the genera Aspergillus, Penicillium and Byssochlamys. The major sources of patulin contamination are apples with brown rot and blue mould. Because patulin does not spread much from spoilt tissue, the main human exposure can be expected from processed products, like apple juice and apple sauce, in which the contamination is not visible. Because fermentation destroys patulin, it is not normally present in cider and perry, unless unfermented apple juice has been added after fermentation. Patulin may also be a contaminant of soft fruits, some vegetables, barley, wheat and corn.

Potential health problems related to patulin are connected to cytotoxic, immunotoxic, neurotoxic, gastrointestinal and other effects observed in animals. Patulin is mostly eliminated within a few days after ingestion.

The PMTDI was set by applying a safety factor of 100 from the lowest NOAEL of 43 ug/kg bw/day in rats.

Mycotoxins

T2 and HT-2 Toxin

Reference to JECFA: 56 (2001)
Toxicological guidance value: PMTDI 0.00006 mg/kg bw (2001, Group PMTDI for T-2 and HT-2 toxins, alone or in combination)
Related code of practice: Code of Practice for the Prevention and Reduction of Mycotoxin Contamination in Cereals, Including Annexes on Ochratoxin A, Zearalenone, Fumonisins and Tricothecenes (CAC/RCP 51-2003)

Code	Food/product	Level (ug/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

No further action on T-2 and HT-2 toxin has been recommended by the 33rd CCFAC (2001), probably based on the understanding that the (limited) information available suggested that intakes would not exceed the PMTDI (ALINORM 01/12A, para. 16).

T-2 and HT-2 toxin are closely related compounds belonging to a group of chemically related mycotoxins called type A trichothecenes (which are epoxy-sesquiterpenoid compounds) and are produced by certain Fusarium species, which are pathogens of several cereal grains. The most important producer is F. sporotrichioides, a saprophyte which only will grow at high water activities. As a consequence, T-2 and HT-2 toxins are not normally found in grain at harvest, but result from water damage when it remains wet for longer periods in the field or after harvest. T-2 and HT-2 toxin undergo rapid metabolism and elimination in livestock species and the transfer from feed to animal products is probably negligible. Maximum levels in feed are not needed to protect public health, but are useful for the protection of animal health and productivity. Especially pigs are vulnerable. In animals, decreased feed consumption, diarrhea and vomiting have been observed as acute effects.

T-2 toxin is a potent inhibitor of protein synthesis, both in vivo and in vitro. T-2 toxin is linked to outbreaks of acute poisoning of humans, in which the adverse effects reported include nausea, vomiting, pharyngeal irritation, abdominal pain, diarrhea, bloody stool, dizziness and chills. Co-occurrence of T-2 toxin with other trichothecenes in these cases is likely. T-2 toxin is also associated with food-related poisoning incidents in 1931- 1947 referred to as alimentary toxic aleukia, in the former Soviet Union.

The PMTDI is based on a 3-week dietary study with pigs, applying a safety factor of 500 to a LOEL for changes in white and red cell counts. The average intake of T-2 and HT-2 toxin via the human diet was estimated by JECFA as 8 resp. 9 ng/kg bw, which is lower than the group PMTDI. An intake at the level of the PMTDI is not expected to result in effects of T-2 and HT-2 toxin on the immune system and to haematotoxicity, which are considered critical effects after short-term intake. JECFA recommended that toxic equivalency factors relative to DON be developed for the other trichothecenes commonly occurring in cereal grains, if sufficient data become available.

Mycotoxins

Zearalenone

Reference to JECFA:	53 (1999)
Toxicological guidance value:	PMTDI 0.0005 mg/kg bw (1999, The total intake of zearalenone and its metabolites (including alpha-zearalenol (zeranol)) should not exceed the PMTDI.)
Synonyms:	(Zearalenone is the most important of a group of related mycotoxins and relevant metabolites. Abbreviation, ZEN. Its metabolite, alpha-zearalenol (zeranol) is used as veterinary drug.)
Related code of practice:	Code of Practice for the Prevention and Reduction of Mycotoxin Contamination in Cereals, Including Annexes on Ochratoxin A, Zearalenone, Fumonisins and Tricothecenes (CAC/RCP 51-2003)

Code	Food/product	Level (ug/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

The situation regarding ZEN has been reviewed in a position paper (last version CX/FAC 00/19).

Preliminary intake calculations indicate values well below the PMTDI. It is mentioned however that further action seems required to reduce the levels of ZEN in risk products (especially maize containing products) for especially children with a high intake of these products.

The 31st CCFAC (1999) agreed that, recognizing that there were no identified trade problems with ZEN, Codex MLs were not necessary for the time being. The MRLs for ZAL in cattle liver and muscle have been established by Codex (CCRVDF) because of recognized use of zeranol in cattle; they are relevant for the CCFAC in so far that feed contamination with ZEN can lead to residues of both ZEN and ZAL (and other metabolites) in cattle liver and muscle.

Zearalenone (ZEN) is the most important of a group of resorcyclic acid lactone mycotoxins, produced by several species of Fusarium moulds. It is found worldwide in a number of cereal crops and also in derived products like beer. It has been implicated in numerous incidents of mycotoxicosis in farm animals, especially pigs. ZEN is rapidly metabolized in and excreted from animals; residues of this mycotoxin in animal products are probably not significant from a health point of view. A metabolite of ZEN, alpha-zearalenol (zeranol, abbreviated here as ZAL) is, however, relevant relating to its potential use as a veterinary drug. Also beta-zearalenol (taleranol) has hormonal activity. Besides these substances which can be used as anabolic growth promoters, also alpha- and beta-zearalenol (ZEL) and zearalenone (ZAN) are mentioned as possibly occurring metabolites of or co-occurring substances with ZEN.

The PMTDI for ZEN was set by applying a safety factor of 100 from the lowest NOAEL, related to the estrogenic effect in pigs. ZAL has an ADI of 0.5 ug/kg bw (ref. JECFA 26, 27 and 32).

Residues of ZEN and ZAL together in an animal product may be regarded as evidence that the animal feed was contaminated with ZEN. In order to distinguish between contamination of the feed with mycotoxins of the ZEN group or use of ZAL as veterinary drug, it may be necessary to determine the relative proportions of the different residues, e.g. as ZEN + alpha- and beta-ZEL against ZAL. A ratio of 5 or more probably indicates only contamination by mycotoxins.

Other Chemical Contaminants (except radionuclides)

Acrylamide

Reference to JECFA: 64 (2005)
Toxicological guidance value: (Intake estimates: mean 0.001 mg/kg bw/day; high 0.004 mg/kg bw/day
Margin of exposure (MOE): morphological changes in nerves (NOEL 0.2 mg/kg bw/day), mean intake 200, high intake 50; reproductive, developmental and other non-neoplastic effects (NOEL 2 mg/kg bw/day), mean intake 2000, high intake 500; cancer (BMDL 0.3 mg/kg bw/day), mean intake 300, high intake 75.)

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

JECFA was asked by the 36th Session of CCFAC (2004) to evaluate acrylamide.

The 64th JECFA (2005) concluded that a dietary intake of 1 µg/kg/day of acrylamide represents the average for the general population and an intake of 4 µg/kg/day represents the high consumers; this includes children. Comparison of these intakes with the NOEL of 0.2 mg/kg bw/day for morphological changes in nerves would provide MOEs of 200 and 50, respectively. Comparison with the NOEL of 2 mg/kg bw/day for reproductive, developmental and other non-neoplastic effects would provide MOEs of 2000 and 500, respectively. For the induction of tumors, the MOE is calculated by comparing those intakes with the BMDL of 0.3 mg/kg bw/day for mammary tumours in rats to be 300 and 75, respectively.

The 64th JECFA (2005) concluded that adverse effects on morphological changes in nerves and on reproductive, developmental and other non-neoplastic effects are unlikely at the estimated average intakes, but that morphological changes in nerves cannot be excluded for some individuals with very high intakes. It considered the MOEs (induction of tumors - mean and high intakes) to be low for a compound that is genotoxic and carcinogenic and that they may indicate a human health concern. Therefore, appropriate efforts to reduce acrylamide concentrations in food stuffs should continue.

Recommendations by the 64th JECFA:
- Acrylamide be re-evaluated when results of ongoing carcinogenicity and long-term neurotoxicity studies become available.
- Work should be continued on using PBPK modeling to better link human biomarker data with exposure assessments and toxicological effects in experimental animals.
- Appropriate efforts to reduce acrylamide concentrations in food should continue.
- In addition, the Committee noted that it would be useful to have occurrence data on acrylamide in foods as consumed in developing countries. This information will be useful in conducting intake assessments as well as considering mitigation approaches to reduce human exposure.(Sixty-fourth Report of the Joint FAO/WHO Expert Committee on Food Additives, pages 8-26).

The 37th CCFAC (2005) agreed to revise the discussion paper which would include an outline of a code of practice and a project document for starting new work on the elaboration of the code of practice, taking into account the 64th JECFA evaluation of acrylamide; national mitigation strategies; and the role of food processors, catering services, and consumers. (ALINORM 05/28/12, paras 193-196)

The 38th CCFAC (2006) agreed to forward to the CAC for approval as new work the project document on the elaboration of a Code of Practice for Reduction of Acrylamide in Food, and agreed that, subject to the approval of the CAC, an electronic working group would elaborate an initial draft Code of Practice for comment at Step 3. (ALINORM 06/29/12, paras 184 & 185)

The First CCCF (2007) decided to maintain paras 52 and 53 describing recommendations to national authorities on consumer practices since consumer practices were considered to add significantly to acrylamide exposure and similar recommendations had already been incorporated in other codes of practice. The Committee, noting the opinion of the ad hoc

Acrylamide

physical working group that the document was not yet ready for advancement in the Codex Procedure, agreed that a revised proposed draft should be prepared, taking account of additional data and information which would become available in the coming year from ongoing studies. The Committee agreed to return the proposed draft Code of Practice to Step 2 for redrafting by an electronic working group chaired by the USA and the UK on the basis of the written comments received and the discussion in the ad hoc Working Group and the in the First Session of the Committee, with a view to circulation for comments at Step 3 and consideration at Step 4 at the next session of the Committee. (ALINORM 07/30/41, paras 95, 96, 97)

Acrylamide is an important industrial chemical used since the mid 1950s as a chemical intermediate in the production of polyacrylamides, which are used as flocculants for clarifying drinking water and other industrial applications. Recently, attention was drawn to the formation of acrylamide at high temperatures during frying, baking or other thermal processing of a variety of foods, typically plant commodities high in carbohydrates and low in protein. In this Maillard reaction, the most important precursor amino acid asparagine reacts with reducing sugars. After its formation acrylamide seems to be stable in a large majority of the affected foods. Acrylamide levels in commodities are highly variable because its formation is dependent on the exact conditions of time and temperature used to heat process the food and the composition of the food. Research on acrylamide formation is ongoing; mitigation could be accomplished by adjustments in existing production procedures.

In experimental animals, acrylamide is rapidly and extensively absorbed following oral administration and widely distributed to the tissues, as well as the fetus. It has also been found in breast milk. The major metabolite is glycidamide, formed by a CYP2E1-mediated oxidation, which is much more reactive with DNA than acrylamide itself. Acrylamide and metabolites are rapidly eliminated via urine.

The neurotoxicity of acrylamide in humans is well-known from occupational and accidental exposures. In addition, experimental studies in animals have shown reproductive, genotoxic and carcinogenic properties. The nervous system is the principal site of toxic actions of acrylamide, which is expressed by morphological changes. Degenerative changes in nerves (NOEL 0.2 mg/kg/day, based on a study in rats). Reproduction studies showed reduced fertility, adverse effects on sperm-count and -morphology in male rodents, however, no adverse effects have been observed in female rodents (NOEL 2 mg/kg/day). Furthermore, acrylamide was not teratogenic in mice or rats. Acrylamide is genotoxic, however, metabolism to glycidamide appears to be a prerequisite.

Acrylamide was evaluated by IARC in 1994 and classified as probably carcinogenic to humans on the basis of a positive cancer bioassay and evidence that acrylamide is efficiently biotransformed to the genotoxic metabolite glycidamide. BMDL for 10% extra risk of tumors was established by the JECFA to be 0.3 mg/kg/day.

A wide range of commodities may be contaminated with acrylamide, such as cereals and cereals-based products, fish and seafood, meat and offals, milk and milk products, nuts and oilseeds, pulses, potato and potato products, coffee, sugars and honey, vegetables.

Studies conducted in Sweden in 2002 showed the formation of high levels of acrylamide during frying or baking of a variety of food.

Other Chemical Contaminants (except radionuclides)

Acrylonitrile

Reference to JECFA: 28 (1984)

Toxicological guidance value: Provisional Acceptance (1984, the use of food-contact materials from which acrylonitrile may migrate is provisionally accepted on condition that the amount of the substance migrating into food is reduced to the lowest level technologically attainable.)

Residue definition: acrylonitrile (monomer)

Synonyms: 2-Propenenitrile; vinyl cyanide (VCN); cyanoethylene; abbreviations, AN, CAN.

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Food	0.02	GL	Adopted	1991	FAC		

Guideline Levels for Acrylonitrile in Food and Vinyl Chloride Monomer in Food and Food Packaging Materials were adopted by the CAC at its 19th session (1991) with the understanding that the AOAC and the ISO would be requested to elaborate appropriate sampling plans and methods of analysis. (ALINORM 91/40, paras 203-204)

Acrylonitrile monomer is the starting substance for the manufacture of polymers which are used as fibres, resins, rubbers and also as packaging material for foods. Acrylonitrile is not known to occur as a natural product. Acrylonitrile is classified by IARC as possibly carcinogenic to humans (Group 2B). Polymers derived from acrylonitrile may still contain small amounts of free monomer. Migration of possibly harmful substances from food contact materials has been discussed in the CCFA/CCFAC in the period 1986-1991. (IARC Vol. 71, 43-108)

Other Chemical Contaminants (except radionuclides)

Chloropropanols

Reference to JECFA:	41 (1993; for 1,3-dichloro-2-propanol only), 57 (2001), 67 (2006)
Toxicological guidance value:	PMTDI 0.002 mg/kg bw (2001, for 3-chloro-1,2-propanediol; maintained in 2006. Establishment of tolerable intake was considered to be inappropriate for 1,3-dichloro-2-propanol because of the nature of the toxicity (tumorogenic in various organs in rats and the contaminant can interact with chromosomes and/or DNA.) BMDL 10 cancer, 3.3 mg/kg bw/day (for 1,3-dichloro-2-propanol); MOE, 65000 (general population), 2400 (high level intake, including young children))
Residue definition:	3-MCPD
Synonyms:	Two substances are the most important members of this group: 3-monochloropropane-1,2-diol (3-MCPD, also referred to as 3-monochloro-1,2-propanediol) and 1,3-dichloro-2-propanol (1,3-DCP)
Related code of practice:	Draft Code of Practice for the Reduction of 3-Monochloropropane-1,2-diol (3-MCPD) during the Production of Acid-Hydrolyzed Vegetable Protein (Acid-HVPs) and Products that Contain Acid-HVPs (Step 6)

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Liquid condiments containing acid-hydrolyzed vegetable protein (excluding naturally fermented soy sauce)	0.4	ML	6		FAC 05-07		

The 57th JECFA (2001) noted that the dose that caused tumours in rats (19 mg/kg bw/day) was about 20000 times the highest estimated intake of 1,3-DCP by consumers of soya sauce (1mg/kg bw/day). The available evidence suggests that 1,3-DCP is associated with high concentrations of 3-MCPD in food. Regulatory control of the latter would therefore obviate the need for specific controls on 1,3-DCP. (57th Report of the Joint FAO/WHO Expert Committee on Food Additives, pages 118-121）

High levels of chloropropanols (up to 100 mg/kg and more) have especially been found in products like non-traditionally fermented soy sauces and hydrolyzed vegetable proteins (HVP). There is an obvious connection with the conditions of the production method; levels of chloropropanols in these products are shown to be declining in the last decade since the problem was noticed and measures have been taken to reduce the formation of chloropropanols. These compounds have also been found, however, in many other foods, including baked goods, bread, cooked/cured meat/fish and malt ingredients. There are (inconclusive) indications that cooking/grilling (high temperature treatment) could result in some formation of 3-MCPD. Also the resins in packaging materials and paper used for processing of food may contain 3-MCPD and could contribute to exposure via food, this has led to the development of resins with significantly lower levels of 3-MCPD. The available evidence suggests that 1,3-DCP occurs at lower levels than 3-MCPD in soy sauce (and related products) and in acid-HVP food ingredients. However, in meat products the concentrations of 1,3-DCP are generally higher than the levels of 3-MCPD as concluded at the 65th JECFA. Further information is required on the levels of chloropropanols in foods and food ingredients, on the dietary exposure to these compounds, on the origin and formation and on production methods which can be utilized to avoid chloropropanol contamination of foodstuffs.

The 37th CCFAC (2005) agreed to request JECFA to conduct an exposure assessment for chloropropanols from all sources (ALINORM 05/28/12, para. 189).

The 67th JECFA (2006) estimated the average exposure to 3-MCPD (at the national level in a wide range of foods including soya-sauce and soya-sauce related products) to be 1% to 35% of the PMTDI in the general population. For consumers at the 95th percentile the estimated intakes ranged from 3% to 85%, and for young children up to 115% of the PMTDI. The Committee noted that a reduction in the concentration of 3-MCPD in soya sauce and related products made with acid-HVP could substantially reduce the intake of this contaminant by certain consumers.

Other Chemical Contaminants (except radionuclides)

Chloropropanols

The 67th JECFA concluded that the critical effect of 1,3-DCP is carcinogenicity. Negative results were found in two new studies on genotoxicity in vivo. However, limitations in these studies, positive findings in in vitro test for genotoxicity as well as lack of knowledge on the modes of action operative at the various tumor locations led the Committee to the conclusion that a genotoxic mode of action could not be excluded.

The estimated intake of 1,3-DCP was calculated at 0.051µg/kg bw/day and 0.136 µg/kg bw/day, respectively for the general population and the high-level intake (including young children). Comparison of these intakes with the lowest BMDL10 of 3.3 mg/kg bw/day (incidence data on tumour-bearing animals for all treatment-affected locations) resulted in a margin of exposure (MOE) of approximately 65000 and 24000, respectively. Based on these MOEs the Committee concluded that the estimated intakes of 1,3-DCP were of low concern for human health.

The 67th JECFA recommended that studies should be undertaken to evaluate the intake or toxicological significance of fatty acid esters of 3-MCPD, which have been reported to be present in foods.

MLs:

A position paper was written; the 35th CCFAC (2003) agreed that the paper should be revised on the basis of the discussions and of submitted comments and data (ALINORM 03/12A, para. 179).

The setting of MLs for 3-MCPD in foodstuffs was asked to be considered at the 35th session of the CCFAC. The CCFAC could not reach a consensus on a ML of 1 mg/kg for acid-HVP soy sauce as proposed, and deferred the elaboration of MLs in different foodstuffs until its next session; the revised position paper should include proposals for the elaboration of MLs for chloropropanols in relevant foods (ALINORM 03/12A, paras 173-179).

The 36th CCFAC (2004) agreed to commence work on the establishment of a maximum level for 3-MCPD in acid-HVPs and acid-HVP containing products subject to approval as new work. In addition, the CCFAC agreed that a working group would prepare an updated discussion paper (ALINORM 04/27/12, paras 193-194).

The 37th CCFAC (2005) agreed to use as a starting point a maximum level of 0.4 mg/kg for 3-MCPD in liquid condiments containing acid-HVP (excluding naturally fermented soya sauce). Due to the need to better define the products for which maximum levels should be set, the Committee agreed to prepare a discussion paper that will define the different acid HVP containing products and collect information on other products that contain 3-MCPD. (ALINORM 05/28/12, paras 188 and 189).

The 38th CCFAC (2006) agreed to update the discussion paper in view of the results of the JECFA evaluation and other information relevant for discussions on the Maximum Levels and to maintain the proposed draft Maximum Level at Step 4 (ALINORM 06/29/12, paras 176 and 177).

The First CCCF agreed to forward the proposed draft ML of 0.4 mg/kg to the 30th Commission for adoption at Step 5. It was agreed that the draft ML should be further considered in light of finalization and implementation of the Codex of Practice for the Reduction of 3-MCPD during the Production of Acid-Hydrolyzed Vegetable Proteins (acid-HVPs) and Products that Contain Acid-HVPs. (ALINORM 07/30/41)

The 30th Commission adopted the proposed draft ML and advanced it to Step 6. (ALINORM 07/30/REP, paras 80 and 94)

Other Chemical Contaminants (except radionuclides)

Chloropropanols

COP:

The 37th CCFAC (2005) agreed to forward to the Commission for approval as new work the project document on the elaboration of a Code of Practice for Reduction of the reduction of chloropropanols during the production o acid HVPs and products that contain acid HVPs and pending the approval of the Commission, to elaborate the proposed draft Code of Practice. (ALINORM 05/28/12, para. 183)

The 38th CCFAC (2006) agreed to urge professional organisations and governments to provide additional data on measures to reduce the presence of chloropropanols in acid HVP produced under industrial conditions, thereby considering, in particular, that which was feasible from an organoleptic point-of-view and the Committee also agreed to revise the proposed draft. In revising the Code of Practice, the electronic Working Group should consider revision of the title to specifically refer to 3-MCPD, on account of the co-occurrence of 3-MCPD and other chloropropanols. (ALINORM 06/29/12, paras 172 and 173)

The First CCCF (2007) agreed to most of the amendments proposed by the ad hoc physical working group and two additional changes and forwarded the proposed draft Code of Practice, as amended at the session, to the 30th Session of the Commission for adoption at Step 5. (ALINORM 07/30/41, paras 92-93)

The 30th Commission adopted the proposed draft COP and advanced it to Step 6 (ALINORM 07/30/REP, para. 80)

Chloropropanols can be formed in foods as a result of specific processing and storage conditions. The main source is acid hydrolyzation of vegetable proteins for the production of savoury food ingredients (e.g. soy sauce). In this process the use of hydrochloric acid at high temperatures can result in chlorination of lipids present in the protein starting materials. 3-MCPD has been shown to be a precursor for 1,3-DCP-formation and control of the levels of 3-MCPD is expected to obviate the need for specific control on 1,3-DCP.

Toxicity of 3-MCPD:

3-MCPD crosses the blood-testis barrier and the blood-brain barrier and is widely distributed in the body fluids. The parent compound is partly detoxified by conjugation with glutathione, resulting in excretion of the corresponding mercapturic acid, and is partly oxidized further to oxalic acid. Intermediate formation of an epoxide has been postulated but not proven. The incidence of tubule hyperplasia in the kidneys of treated rats was the most sensitive end-point for deriving a tolerable intake. This effect was seen in the long-term study of toxicity and carcinogenicity in rats in a dose-related manner. 3-MCPD is neither genotoxic in vitro at concentrations at which other toxic effects are observed, nor genotoxic in vivo. (Fifty-seventh Report of the Joint FAO/WHO Expert Committee on Food Additives, pages 114-118)

Toxicity of 1,3-DCP:

Although only a few studies of kinetics, metabolism, short- and long-term toxicity and reproductive toxicity were available for evaluation, the results clearly indicated that 1,3-dichloro-2-propanol was genotoxic in vitro, was hepatotoxic and induced a variety of tumours in various organs in rats. The JECFA concluded that it would be inappropriate to estimate a tolerable intake because of the nature of the toxicity observed:
- The results of the long-term study of toxicity and carcinogenicity showed significant increases in the incidences of both benign and malignant neoplasms in at least three different tissues.
- It has been shown unequivocally that this contaminant can interact with chromosomes and/or DNA; however, the tests were confined to bacterial and mammalian test systems in vitro, and there were no data on intact mammalian organisms or humans. (57th Report of the Joint FAO/WHO Expert Committee on Food Additives, pages 118-121)

Other Chemical Contaminants (except radionuclides)

Dioxins

Reference to JECFA: 57 (2001)
Toxicological guidance value: PTMI 70 pg TEQ/kg bw (2001, Including coplanar PCBs)
Synonyms: Polychlorinated dibenzo-dioxins and -furans
Related code of practice: Code of Practice for the Prevention and Reduction of Dioxin and Dioxin-like PCB Contamination in Food and Feeds (CAC/RCP 62-2006)

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

The situation regarding dioxins has been reviewed in a discussion paper (last version CX/FAC 00/26). The 32nd CCFAC (2000) requested an additional position paper in which recent intake assessments and national regulations regarding dioxins are assembled. This was presented to the 33rd CCFAC (2001). A revision of this document was requested, with also data on dioxin levels in food and feedingstuffs and breast-milk; the latest version is CX/FAC 03/32. The 34th CCFAC (2002) agreed that it should not draft MLs for dioxins at the time. The 35th CCFAC (2003) requested a revision of the position paper, including the insertion of a new section to cover ranges of data on background levels of dioxins and dioxin-like PCBs in food and feed. The 36th CCFAC (2004) encouraged Codex members to submit data on dioxins and dioxin-like PCBs in foods, and it agreed to request WHO to report in a detailed way to the Committee on the data submitted within three years time. In view of this, the CCFAC agreed to discontinue the consideration of the position paper (ALINORM 04/27/12, paras 188-189).

The 29th CAC (2006) agreed to invite the CCMAS to review the sections on sampling and analytical methods and assess the need for future revisions of the Code, taking into account the comments made at the 29th CAC (ALINORM 06/29/41, paras 60-62)

The term dioxins refers to a group of polychlorinated planar aromatic compounds. The group 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-tetra-CDD (TCDD) is the most toxic and most studied congener. Dioxins are ubiquitously present as contaminants in the environment and in food, be it in minute amounts. Dioxins are lipophilic compounds which bind to sediment and organic matter in the environment and tend to be absorbed in animal and human fatty tissue. They are extremely resistant towards chemical and biological transformation processes and are consequently persistent in the environment and accumulate in the food chain. Dioxins are formed as unwanted by-products in combustion processes or industrial processes. Most of the dioxins enter the environment by emission to air. The Ah receptor is an important factor in the toxicological effects of dioxins. Activation of this receptor can result in endocrine and paracrine disturbances and alterations in cell functions including growth and differentiation.

Developmental neurobehavioral (cognitive) and reproductive effects and immunotoxic effects belong to the most sensitive endpoints of dioxin toxicology. TCDD is classified by IARC as Group 1 human carcinogen. It has been shown to be carcinogenic in several animal species at multiple sites, but TCDD is not an initiator of carcinogenesis and the tumour promotion in animal studies indicated a non-genotoxic mechanism.

The toxic equivalency concept has been developed for application to dioxins in order to assess the toxicity of a mixture of congeners as it exists in practice. Toxic Equivalency Factors (TEFs) have been established in relation to TCDD and the total toxicity of a mixture can thus be calculated as total toxic equivalents (TEQs). It has been shown that also some PCB-congeners (those with a planar dioxin-like structure) have effects on the Ah receptor and thus they are given TEFs and can be combined with the dioxins for the calculation of total TEQ of a sample.

Ethyl carbamate

Reference to JECFA: 64 (2005)
Toxicological guidance value: (Intake estimates: from food (=mean) 15 ng/kg bw/day; from food and alcoholic beverages (=high) 80 ng/kg bw/day Margin of Exposure (MOE): cancer (BMDL 0.3 mg/kg bw/day), mean intake 20 000, high intake 3 800.)
Synonyms: Urethane; abbreviation, EC

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

When ethyl carbamate was discussed in the CCFAC in 1991, a Danish national TDI of 0.2 ug/kg bw was reported. The intake of a person consuming some of the higher contaminated food products was estimated to be more than 50% of this TDI. Therefore measures aimed at reducing the EC formation were seen as necessary. No specific health effects by ethyl carbamate in humans related to dietary exposure are reported however.

Some countries mentioned national GLs for EC. No trade problems are reported however. The 27th CCFAC (1995) decided that no further action was needed at present.

The 64th JECFA evaluated the national estimates of intake submitted to the committee by Denmark, Switzerland, USA (assessments conducted in the early 1990s) and South Korea, Australia, New Zealand (assessments conducted more recently). The committee noted that mitigation measures have been effective in reducing residual concentrations of ethyl carbamate, and that, consequently the older data published in the early 1990s and used to make the initial estimates of intake of ethyl carbamate no longer accurately reflect current intake from alcoholic beverages. The committee estimated the mean intake of ethyl carbamate from food to be approximately 15 ng/kg bw/day, this was based on the relevant foods, including bread, fermented milk products and soy sauce; alcoholic beverages were not included. With the inclusion of alcohol beverages the estimated intake is 80 ng/kg bw/day. High consumption of stone-fruit brandies could lead to higher intakes of ethyl carbamate.

The 64th JECFA concluded that intake of ethyl carbamate from foods excluding alcoholic beverages would be of low concern (MOE: 20 000). However, the MOE from all intakes, food and alcoholic beverages combined (MOE: 3800), is of concern and therefore mitigation measures to reduce concentrations of ethyl carbamate in some alcoholic beverages should be continued.

The 37th CCFAC (2005) observed the matter of ethyl carbamate was relevant but not of a high priority and that, due to the limited resources, it should be taken up at a later stage. (ALINORM 05/28/12, para. 41)

The First CCCF noted that the 64th JECFA had concluded that health risks for the general population were low and that only sub-populations consuming a high quantity of specific alcoholic beverages might be exposed to certain health risks. (ALINORM 07/30/41, para. 137)

Ethyl carbamate can be formed from various substances derived from food and beverages, including hydrogen cyanide, urea, citrulline and other N-carbamyl compounds. Cyanate is probably the ultimate precursor, reacting with ethanol to form the carbamate ester. Over the past years, major reductions in concentrations of EC have been achieved using two approaches: first, by reducing the concentration of the main precursor substances in the food and beverages; second, by reducing the tendency for these precursor substances to react to form cyanate, e.g. by the exclusion of light from bottled spirits. Also, diethylpyrocarbonate, an inhibitor of fermentation, and azodicarbonamide, a blowing agent for sealing gaskets, can form ethyl carbamate. Diethylpyrocarbonate is revoked by the JECFA at its 17th meeting, azodicarbonamide is not recommended for bottling alcoholic beverages.

Ethyl carbamate is well absorbed from the gastrointestinal tract and is rapidly distributed throughout the body. Elimination is also rapid, with most being excreted as carbon dioxide as studied in mice. CYP2E1 activity is responsible for most of the metabolism of EC to carbon dioxide. EC may also undergo metabolic activation to vinyl carbamate epoxide, which binds

Ethyl carbamate

covalently to nucleic acids and proteins. Moreover, hydrolysis to ethanol and ammonia may occur.

The acute oral toxicity of EC is low; however, high doses caused anesthesia in rodents. Effects on lung, liver, kidney, heart, spleen, lymph nodes, thymus, bone marrow and ovaries were seen during chronic exposure to EC, as studied in mice and rats. Reproduction studies showed high rates of embryonic/fetal mortality and malformations. Ethyl carbamate is genotoxic and carcinogenic. Single doses, short-term and long-term oral dosing of ethyl carbamate have been shown to induce tumors in all species tested (BMDL 0.3 mg/kg bw/day). IARC classified ethyl carbamate in Group 2B, possibly carcinogenic to humans (1974). No quality data for humans are available.

Other Chemical Contaminants (except radionuclides)

Polybrominated diphenyl ethers

Reference to JECFA: 64 (2005)
Toxicological guidance value: (Intake estimates: mean approximately 4 ng/kg bw/day
Based on limited toxicity data, the 64th JECFA concluded that there appeared to be a large MOE for a non-genotoxic compound which, despite the inadequacy of the data on toxicity and intake, gave reassurance that intakes of PBDEs are not likely to be a significant health concern.)
Synonyms: PBDEs

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

In 1994, WHO published an Environmental Health Criteria document on PBDEs. Recent analysis of samples from environment and from human collected over the last 3-4 decades demonstrated significant increases in concentrations of PBDEs. At its 35th session the CCFAC requested to evaluate the potential risks associated with the presence of PBDEs in food.

The 64th JECFA noted that the available data on PBDEs were not adequate to allocate a PTWI or PMTDI, because:
- PBDEs represent a complex group of related chemicals and the pattern of PDBE congeners in food is not clearly defined by a single commercial mixture;
- Data are inadequate to establish a common mechanism of action that would allow a single congener to be used as a surrogate for total exposure or, alternatively, as the basis for establishing toxic equivalence factors;
- There is no systematic database on toxicity including long-term studies on the main congeners present in diet, using standardized testing protocols that could be used to define a NOEL for individual PBDEs of importance;
- Several of the reported effects are biological outcomes for which the toxicological significance remains unclear;
- Studies with purified PBDE congeners in vitro have shown a lack of Ah receptor activation, however, many of the adverse effects reported are similar to those found with dioxin-like contaminants, suggesting that some toxicity data may be confounded by the presence of traces of impurities that are potent Ah receptor agonists.

The 64th JECFA recognized the preliminary nature of the data on concentrations of PBDEs in food and human milk and estimated the dietary intake for the sum of all measured PBDE congeners to be approximately 4 ng/kg bw/day, while intake by breastfeeding infants could be up to 100 ng/kg bw/day. Adverse effects for PBDE congeners would be unlikely to occur at doses of less than approximately 100 ug/kg bw/day.

Based on limited toxicity data, The 64th JECFA concluded that there appeared to be a large MOE for a non-genotoxic compound which, despite the inadequacy of the data on toxicity and intake, gave reassurance that intakes of PBDEs are not likely to be a significant health concern. The committee considered that continuing studies of PBDEs in samples from humans, including human milk, would be useful in assessing the overall exposures to PBDEs in foods and other possible sources.

Polybrominated diphenyl ethers (PBDEs) are anthropogenic chemicals that are added to a wide variety of consumer/commercial products (e.g. plastics, polyurethane foam, textiles) in order to improve their fire resistance. Theoretically, 209 distinct PBDE isomers are possible, however, each commercial mixture usually only contains a limited number of congeners from each homologue group. PDBEs have been produced primarily as three main commercial products (mixtures): pentabromodiphenyl oxide or ether (PentaBDE), octabromodiphenyl oxide or ether (OctaBDE) and decabromodiphenyl oxide or ether (DecaBDE). Some variability in composition is known to exist between products from different manufacturers. The worldwide demand for PBDEs in 2001 was estimated to be almost 70 000 tonnes, with DecaBDE accounting for almost 80% of the total market.

Polybrominated diphenyl ethers

Absorption of PBDEs is directly related to the extent of bromination of the parent diphenyl ether; as a general rule, greater substitution of bromine leads to a decrease in bioavailability. The metabolism of PBDEs consists of hydroxylation and methoxylation reactions and, in the case of congeners with a higher degree of bromination, oxidative debromination. Faecal excretion appears to be the dominant route of elimination, however, species differences exist. Limited data are available regarding the half-lives, however, preliminary values ranged from 30 to 90 days for the tetra- to hexa-substituted congeners. Moreover, limited pharmacokinetic data are available for humans, however, based on the observed increase in concentrations of PBDEs in tissue in time, PDBEs are absorbed and bioaccumulate.

The acute toxicity of mixtures of PBDEs is low in rodents, however, increased mortality, neurobehavioral effects, changes in gross pathology, induction of enzymes, changes in levels of hormones have been observed. In short-term studies the main effects of mixtures of PBDEs were seen in the liver (enlargement, 'round bodies', vacuolization, necrosis), kidney (hyaline degenerative cytoplasmic changes) and thyroid (hyperplasia). Embryo and fetus may be more sensitive to PBDEs than maternal animals; exposure to OctaBDE mixtures caused an increase in the incidence of developmental abnormalities. The results of the majority of tests for genotoxicity indicated that PBDE mixtures and single congeners are not genotoxic. The only long-term study was conducted with the DecaBDE mixture in mice and rat, however, evidence for the carcinogenicity of DecaBDE is limited. No information is available on the carcinogenic potential of other PBDE mixtures. Available studies in humans are not adequate to evaluate whether exposure to PBDEs is associated with adverse health effects. Some toxicity data may be confounded by the presence of traces of impurities that are Ah-receptor agonists (e.g. dioxin).

Other Chemical Contaminants (except radionuclides)

Polychlorinated biphenyls

Reference to JECFA: 35 (1989)
Toxicological guidance value: Not established (, For coplanar PCBs (dioxin-like PCBs), see the toxicological guidance value of Dioxins)
Synonyms: Abbreviations, PCBs
Related code of practice: Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

PCBs were discussed by the 35th JECFA (1989); it was difficult to come to clear conclusions about the toxicity of PCBs as such because impurities such as dioxins and related compounds (e.g., PCDFs) probably were present in the PCB-mixtures used for the animal studies. The Committee concluded that 0.04 mg/kg bw was the NOEL in monkey studies. However, because of the limitations of the data and the ill-defined nature of the materials used in the study, no tolerable intake for humans could be established. One of the complications is that humans are exposed to biologically filtered mixtures of congeners, which are rather different from the industrial PCB-mixtures that were used for the studies. No toxicological monograph was prepared (see however EHC 140).

The major foods in which contamination with PCBs can be significant are fish, milk and dairy products, meat and eggs. Because PCBs bioaccumulate, the levels will usually be higher in animals which are higher in the food chain, but local pollution and feed composition may have major influence on the levels in animal products. Humans with a considerable intake of animal fats also may accumulate high levels of PCBs and as a consequence also PCB-levels in breast milk and in human adipose fat may be high. The JECFA, however, considered that the advantages to the infant of breast-feeding outweigh any potential hazards due to the PCB-content of breast milk. The JECFA recommended that PCB-levels in foods are monitored , preferably by quantifying the most important individual congeners. Safety studies should be carried out on the toxicological potential of the PCB-congeners which are predominantly present in foods. It is evident that in relation to the persistent nature of PCBs and ongoing environmental contamination, it is still valid to pay due attention to PCBs. JECFA pointed out that a long-term goal should be the reduction of PCBs in the diet to a minimum.

PCBs are related to other chlorinated hydrocarbons, such as polybrominated biphenyls (PBBs), polychlorinated terphenyls (PCTs), tetrachlorobenzyltoluenes, and polychlorinated dibenzodioxins and dibenzofurans. Coplanar PCBs were integrated included in the toxicological evaluation of dioxins (see the PTMI of 3.08 Dioxins), but it has to be borne in mind that the toxicological effects of PCBs are broader than the dioxin-related effects. The CCFAC discussed PCBs from 1990 to 1994 on the basis of CX/FAC 90/20-Add.1 and further related documents. It was noted that several countries have established MLs for PCBs in food, so that trade issues might arise. Some of these countries have introduced MLs for the sum of some specific PCB-congeners, which is probably the best defined way of analyzing and reporting PCBs. The most important congeners for analysis of the general content of PCBs in foods are usually considered to be IUPAC numbers 28, 52, 101, 118, 138, 153 and 180.

The CCFAC also acknowledged that source-directed measures were most important to reduce contamination with PCBs. The Committee agreed in 1992 that it was premature to set (maximum) levels for these contaminants at this stage. The discussions later were focused on dioxins and the dioxin-related PCBs.

PCBs are a class of stable chlorinated aromatic hydrocarbons which (mostly prior to the 1970s) have been produced since 1930 and used extensively in a wide range of industrial applications. One of the main uses which still persists is as dielectric and heat exchange fluids. Despite increasing withdrawal of the use and restrictions on the production, large amounts of PCBs continue to be present in the environment, either in use in existing industrial systems, or in waste materials, or dispersed as persistent pollutants. PCBs are mixtures of related chemicals which are formed by the chlorination of biphenyl. Theoretically, 209 congeners are possible; in practice about 130 are likely to occur in commercial products. Also related by-products are formed, such as polychlorinated dibenzofurans (PCDFs), and may be found in technical PCB-mixtures. Some of the trade names for technical PCB-mixtures as

Polychlorinated biphenyls

they were produced are Aroclor, Clophen, Kanechlor. The different congeners in PCB-mixtures can be designated by their IUPAC number, and different industrial PCB-mixtures can be characterized by their composition in terms of the relative percentages of the congeners.

Degradation of PCBs in the environment depends on the degree of chlorination (higher chlorinated compounds are generally more persistent against photolytic, microbial and animal metabolic degradation) and on the position of the chlorine atoms in the molecule. All congeners are lipophilic and accumulate in the food chain.

PCBs were evaluated by IARC in 1978 and 1987. The conclusion was that PCBs are carcinogenic for laboratory animals and are probably carcinogenic for humans (IARC, 1987). Extensive documentation about PCBs is gathered in EHC 140 (WHO, 1993).

The PCB-congeners that most easily adopt a co-planar configuration (the non-ortho substituted PCBs, numbers 77, 126 and 169) are potent Ah receptor agonists. Mono-ortho substituted PCBs are less potent but are included with a TEQ-factor for dioxin-like activity (nos 105, 114, 118, 123, 156, 157, 167, 189). Sometimes also PCB 81 and two di-ortho substituted PCBs (170 and 180) were included in the discussion about the TEF-approach for dioxins because of their ability to induce P4501A1 enzymes and their occurrence and persistence in the environment; they however were not incorporated in the WHO-recommendation about the TEF-approach for dioxin-related compounds (1998). The PCBs with a TEF form usually only a few percent of the total PCBs, but are relevant because of this specific toxicity, which can form an important contribution to the total TEQ for dioxins in a sample of food and in the human diet.

Other Chemical Contaminants (except radionuclides)

Polycyclic aromatic hydrocarbons

Reference to JECFA: 64 (2005)
Toxicological guidance value: (Intake estimates for benzo[a]pyrene as marker for PAHs: mean 4 ng/kg bw/day; high 10 ng/kg b/day
Margin of exposure (MOE): Cancer (BMDL for benzo[a]pyrene as marker for mixtures of PAHs 100 000 ng/kg bw/day), mean intake 25 000; high intake 10 000.)
Synonyms: PAHs

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
No ML								

At the 37th JECFA the committee evaluated benzo[a]pyrene and recognized that it was one member of a family of PAHs that should be considered as a class. The most significant toxicological effect was carcinogenicity and it was noted that the estimated average daily intake of benzo[a]pyrene by humans was about four orders of magnitude lower than that reported to be without effect on the incidence of tumors in rats. However, the committee was unable to establish a tolerable intake for benzo[a]pyrene, based on the available data.

The 64th JECFA evaluated 33 compounds. Some were found to be clearly genotoxic and carcinogenic (benz[a]anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, chrysene, dibenz[a,h]anthracene, dibenzo[a,e]pyrene, dibenzo[a,h]pyrene, dibenzo[a,l]pyrene, dibenzo[a,l]pyrene, indeno[1,2,3-cd]pyrene, 5-methylchrysene), whereas others were not. There is limited or no evidence on the reproductive toxicity of individual PAHs, other than benzo[a]pyrene, which showed impaired fertility in the offspring of female mice. Developmental toxicity after oral administration has been reported for benz[a]anthracene, benzo[a]pyrene, dibenz[a,h]anthracene and naphthalene. A NOEL for reproductive toxicity has not been established. Using parenteral administration, it was shown that PAHs exert immunosuppressive effects, probably via the Ah receptor. The NOEL for immunosuppressive effects of benzo[a]pyrene was 3 mg/kg bw/day. No quality data for humans are available.

To evaluate the combined toxicity of PAHs, the 64th JECFA decided to use a surrogate approach, with benzo[a]pyrene being used as a marker of exposure to, and effect of the 13 genotoxic and carcinogenic PAHs. A BMDL equivalent to 0.1 mg benzo[a]pyrene kg bw/day was derived for mixtures of PAHs in food. The committee concluded that a representative mean intake of benzo[a]pyrene of 0.004 µg/kg bw/day and high-level intake of 0.01 µg/kg bw/day could be used in the evaluation. Comparison of these mean and high-level intakes with the BMDL indicates MOEs of 25 000 and 10 000, respectively. Based on these MOEs, the committee concluded that the estimated intakes of PAHs were of low concern for human health. Measures to reduce intake of PAHs could include avoiding contact of foods with flames, and cooking with the heat source above rather than below the food. Efforts should be made to reduce contamination with PAHs during drying and smoking processes by replacing direct smoking (with smoke developed in the smoking chamber, traditionally in smokehouses) with indirect smoking. Washing or peeling fruit and vegetable before consumption would help to remove surface contaminants.

Recommendations by 64th JECFA:
- Future monitoring should include, but not be restricted to, analysis of the 13 PAHs identified as being genotoxic and carcinogenic.

The 37th CCFAC (2005) agreed to revise the discussion paper with particular attention to the 64th JECFA evaluation (ALINORM 05/28/12 para.199). The 38th CCFAC (2006) agreed to the elaboration of a Code of Practice for the reduction of PAH contamination in food and to limit its scope to smoking and direct drying process (ALINORM 06/29/12 para.187). An initial draft Code of Practice is to be considered at the 1st session of CCCF.

The First CCCF (2007) agreed to address smoke flavours in the introductory part only in the Code. The CCCF agreed to return the proposed draft Code of Practice to Step 2 for redrafting by an electronic working group led by Denmark with a view to circulation for comments at Step 3 and consideration at Step 4 at its next session. (ALINORM 07/30/41, para. 102)

Polycyclic aromatic hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) constitute a large class of organic compounds containing two or more fused aromatic rings. Foods can be contaminated by two major routes: firstly, by environmental PAHs present in air, soil and water; secondly, PAHs can be formed during processing (drying, smoking) or cooking (grilling, roasting, frying) of foods.

Absorption of dietary PAH is determined by size and lipophilicity of the molecule and the lipid content of the food. PAHs are metabolized by oxidation of the aromatic rings, followed by formation of glutathione, glucuronide and sulfate conjugates. Oxidation can generate electrophilic metabolites that bind covalently to nucleic acids and proteins. Some PAH and PAH metabolites bind to the aryl hydrocarbon (Ah) receptor, resulting in upregulation of enzymes involved in PAH metabolism.

The major foods containing higher concentrations of PAHs are meat and fish products, particularly grilled and barbecued products, oils and fats, cereals and dry foods.

Vinyl chloride monomer

Reference to JECFA: 28 (1984)
Toxicological guidance value: Provisional Acceptance (1984, the use of food-contact materials from which vinyl chloride may migrate is provisionally accepted, on condition that the amount of the substance migrating into food is reduced to the lowest level technologically
Residue definition: Vinyl chloride monomer
Synonyms: Monochloroethene, chloroethylene; abbreviation VC or VCM

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Food	0.01	GL	Adopted	1991	FAC	The GL in food packaging material is 1.0 mg/kg.	

Migration of possibly harmful substances from food contact materials has been discussed in the CCFA/CCFAC in the period 1986-1991.

Guideline levels for vinyl chloride monomer and acrylonitrile in food and packaging material were adopted by the CAC at its 19th session (1991) on the understanding that the AOAC International and the ISO would develop appropriate sampling plans and methods of analysis.

Vinyl chloride monomer is the main starting substance for the manufacture of polymers which are used as resins, as packaging material for foods. Vinyl chloride is not known to occur as a natural product. Residues of vinyl chloride monomer may be still present in the polymer. Vinyl chloride is considered by IARC to be a human carcinogen (as has been shown in occupational exposure situations). IARC Vol. 19, 377-438 (1979)

Radionuclides

^{238}Pu, ^{239}Pu, ^{240}Pu, ^{241}Am

Residue definition: ^{238}Pu, ^{239}Pu, ^{240}Pu, ^{241}Am

Code	Food/product	Level (Bq/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Foods other than infant foods	10	GL	Adopted	2006	FAC		
	Infant foods	1	GL	Adopted	2006	FAC	When intended for use as such.	

See Schedule I

Radionuclides

^{90}Sr, ^{106}Ru, ^{129}I, ^{131}I, ^{235}U

Residue definition: ^{90}Sr, ^{106}Ru, ^{129}I, ^{131}I, ^{235}U

Code	Food/product	Level (Bq/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Foods other than infant foods	100	GL	Adopted	2006	FAC		
	Infant foods	100	GL	Adopted	2006	FAC	When intended for use as such.	

See Schedule I

Radionuclides

^{35}S, ^{60}Co, ^{89}Sr, ^{103}Ru, ^{134}Cs, ^{137}Cs, ^{144}Ce, ^{192}Ir

Residue definition: ^{35}S, ^{60}Co, ^{89}Sr, ^{103}Ru, ^{134}Cs, ^{137}Cs, ^{144}Ce, ^{192}Ir; ^{35}S represents the value for organically bound sulphur.

Code	Food/product	Level (Bq/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Foods other than infant foods	1000	GL	Adopted	2006	FAC		
	Infant foods	1000	GL	Adopted	2006	FAC	When intended for use as such.	

See Schedule I

Radionuclides

^{3}H, ^{14}C, ^{99}Tc

Residue definition: ^{3}H, ^{14}C, ^{99}Tc; ^{3}H represents the value for organically bound tritium.

Code	Food/product	Level (Bq/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Foods other than infant foods	10000	GL	Adopted	2006	FAC		
	Infant foods	1000	GL	Adopted	2006	FAC	When intended for use as such.	

See Schedule I

Quality factors

Copper

Reference to JECFA: 10 (1966), 14 (1970), 26 (1982)
Toxicological guidance: PMTDI 0.05-0.5 mg/kg bw (1982)
Residue definition: Copper, total
Synonyms: Cu

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Edible fats and oils, refined (not covered by other standards)	0.1	ML	Adopted	CS 19-1981	FO	Edible fats and oils not covered by individual standards. This ML is mentioned to be a quality characteristic, for voluntary application by commercial partners and not for application by governments.	
	Edible fats and oils, virgin and cold pressed (not covered by other standards)	0.4	ML	Adopted	CS 19-1981	FO	This ML is mentioned to be a quality characteristic, for voluntary application by commercial partners and not for application by governments.	
	Named animal fats	0.4	ML	Adopted	CS 211-1999	FO	Lard, rendered pork fat, premier jus and edible tallow. This ML is mentioned to be a quality characteristic, for voluntary application by commercial partners and not for application by governments.	1)
OC 0172	Vegetable oils, Crude	0.4	ML	Adopted	CS 210-1999,	FO	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein.	
OR 0172	Vegetable oils, Edible	0.1	ML	Adopted	CS 210-1999	FO	Named vegetable oils from arachis, babasu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein. This ML is mentioned to be a quality characteristic, for voluntary application by commercial partners and not for application by governments.	1)
	Natural mineral waters	1 mg/l	ML	Adopted	CS 108-1981	NMW		

1) The revised Standards for oils and fats contain the following wording for the mentioned contaminant MLs: "The products covered by the provisions of this Standard shall comply with MLs being established by the CAC but in the meantime the following limits will apply."

Quality factors

Iron

Reference to JECFA: 27 (1983)
Toxicological guidance: PMTDI 0.8 mg/kg bw (1983, Group PMTDI, applies to iron from all sources except for iron oxides used as colouring agent, supplemental iron taken during pregnancy and lactation, and supplemental iron for specific clinical requirements)
Residue definition: Iron, total
Synonyms: Fe

Code	Food/product	Level (mg/kg)	Type	Step	Reference or Adoption year	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC
	Edible fats and oils, refined (not covered by other standards)	2.5	ML	Adopted	CS 19-1981	FO	This ML is mentioned to be a quality characteristic, for voluntary application by commercial partners and not for application by governments.	
	Edible fats and oils, virgin and cold pressed	5	ML	Adopted	CS 19-1981	FO	This ML is mentioned to be a quality characteristic, for voluntary application by commercial partners and not for application by governments.	
OC 0172	Vegetable oils, Crude	5	ML	Adopted	CS 210-1999	FO	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein.	
OR 0172	Vegetable oils, Edible	2.5	ML	Adopted	CS 210-1999	FO	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein.	

Quality factors

Zinc

Reference to JECFA: 10 (1966), 26 (1982)
Toxicological guidance PMTDI 0.3-1 mg/kg bw (1982)
Residue definition: Zinc, total
Synonyms: Zn

Commodity/Product Code	Name	Level mg/kg	Suffix	Type	Step	Reference	Ref to CC	Notes/Remarks for Codex Alimentarius	Notes for CCFAC

No ML

Zinc is a naturally occurring element, which naturally is never found in its metallic form, but which occurs usually in the form of insoluble or soluble salts. In the soil and in plants and animal tissues it is normally always present in small quantities. Zinc is an essential element, but toxic concentrations could be reached by environmental contamination or by specific conditions in connection with uses of zinc compounds.

The MLs for zinc should probably not be considered as contaminant MLs in the context of the activities of the CCFAC. The MLs should accordingly be characterized as quality characteristics. This notion however has not yet been expressed in the commodity standards in which MLs for zinc are established.

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SCHEDULE I - MAXIMUM AND GUIDELINE LEVELS FOR CONTAMINANTS AND TOXINS IN FOODS

INDEX OF CONTAMINANTS IN ALPHABETICAL ORDER

EXPLANATORY NOTES

Reference to JECFA:	References to JECFA meeting in which the contaminant was evaluated and the year of that meeting
Toxicological guidance value:	Toxicological advice about the tolerable intake level of the contaminant for humans, expressed in milligrammes (mg) per kg body weight (bw). The year of recommendations and additional explanation are included.
Residue definition:	Definition of the contaminant in the form of which the ML applies or which may or should be analyzed in commodities.
Synonyms:	Symbols, synonyms abbreviations, scientific descriptions and identification codes used to define the contaminant.
Related code of practice:	Name of any code(s) of practice related to the contaminant and its (their) reference number(s).
Commodity code:	The code for food commodities is according to the food and feed categorization system as contained in Annex V of the GSCTF. The food/feed categorization. The food/feed categorization system also specifies the part of Commodity which should be analyzed and to which the ML applies, unless a specific commodity definition is provided as an annex to the ML. For those maximum levels contained in Codex commodity standards, the relevant standard numbers are referred, if the code numbers are not readily available for these commodities.
Type:	Indicates whether the value is Codex maximum level (ML) or Codex guideline level (GL). See also the definitions of these terms in the preamble of the GSCTF.

Reference or adoption year:	Reference number of the commodity standard in which the maximum level is established or the year of adoption of the maximum level following the recommendation of the Codex Committee on Food Additives and Contaminants (up to 2006).

Qualification of MLs

C:	In canned products only

Definitions of some toxicological terms

PMTDI:	*(Provisional Maximum Tolerable Daily Intake)* The endpoint used for contaminants with no cumulative properties. Its value represents permissible human exposure as a result of the natural occurrence of the substance in food and in drinking-water. In the case of trace elements that are both essential nutrients and unavoidable constituents of food, a range is expressed, the lower value representing the level of essentiality and the upper value the PMTDI.
PTWI:	*(Provisional Tolerable Weekly Intake)* An endpoint used for food contaminants such as heavy metals with cumulative properties. Its value represents permissible human weekly exposure to those contaminants unavoidably associated with the consumption of otherwise wholesome and nutritious foods.
PTMI:	*(Provisional Tolerable Monthly Intake)* An endpoint used for a food contaminant with cumulative properties that has a very long half-life in the human body. Its value represents permissible human monthly exposure to a contaminant unavoidably associated with otherwise wholesome and nutritious foods

Arsenic

Reference to JECFA: 5 (1960), 10 (1967), 27 (1983), 33 (1988)
Toxicological guidance value: PTWI 0.015 mg/kg bw (1988, for inorganic arsenic)
Residue definition: Arsenic: total when not otherwise mentioned or inorganic arsenic; or other specification
Synonyms: As
Related code of practice: Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Edible fats and oils	0.1	ML	CS 19-1981	Edible fats and oils not covered by individual standards
	Fat spreads and blended spreads	0.1	ML	CS 256-2007	
	Named animal fats	0.1	ML	CS 211-1999	Lard, rendered pork fat, premier jus and edible tallow.
OR 0305	Olive oil, refined	0.1	ML	CS 33-1981	
OC 0305	Olive oil, virgin	0.1	ML	CS 33-1981	
OR 5330	Olive, residue oil	0.1	ML	CS 33-1981	Olive pomace oil
OC 0172	Vegetable oils, Crude	0.1	ML	CS 210-1999	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein.
OR 0172	Vegetable oils, Edible	0.1	ML	CS 210-1999	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein.
	Natural mineral water	0.01	ML	CS 108-1981	Expressed in total As mg/l
	Salt, food grade	0.5	ML	CS 150-1985	

Arsenic is a metalloid element which is normally occurring in mineral bound form in the earth's crust and which can become more easily available by natural sources such as volcanic activity and weathering of minerals, and by anthropogenic activity causing emissions in the environment, such as ore smelting, burning of coal and specific uses, such as arsenic-based wood preservatives, pesticides or veterinary or human medicinal drugs. As a result of naturally occurring metabolic processes in the biosphere arsenic occurs as a large number of organic or inorganic chemical forms in food (species). Especially in the marine environment arsenic is often found in high concentrations of organic forms, up to 50 mg/kg of arsenic on a wet weight basis in some seafood including seaweed, fish, shellfish and crustaceans. In fresh water and in the terrestrial environments arsenic is normally found in much lower levels (typically 0-20 ug/kg) in crop plants and in livestock. Higher levels may be found in rice, mushrooms and sometimes in poultry which is fed fish meal containing arsenic. Levels of arsenic in drinking water are of concern in many countries; levels exceeding 200 mg/l have been reported, which can adversely affect the health of consumers. The most toxic forms of arsenic are the inorganic arsenic (III) and (V) compounds; the inorganic arsenic trioxide is well known as a rat poison, which was also sometimes used for homicide. Methylated forms of arsenic have a low acute toxicity; arsenobetaine which is the principal arsenic form in fish and crustaceans is considered non-toxic. In shellfish, mollusks and seaweed dimethylarsinylriboside derivatives occur ("arsenosugars"), the possible toxicity of which is not known in detail. Only a few percent of the total arsenic in fish is present in inorganic form, which is the only form about which a PTWI has been developed by JECFA. The human epidemiological data used for this risk assessment is based on exposure to inorganic arsenic in drinking water. IARC has classified inorganic arsenic as a human carcinogen, and the estimated lifetime risk for arsenic-induced skin cancer which may be caused by drinking water at or in excess of the WHO guideline for arsenic in drinking water is estimated at 6x 10-4.

Arsenic

The analysis of total arsenic in food has up to date suffered from difficulties with respect to accuracy and precision. Furthermore, specified data for arsenic are strongly needed because of the large differences in toxicity to humans of the various forms of arsenic.

The intake of total arsenic in the human diet is usually dominated by organic arsenic derived from seafood. The available data about the possible human exposure to inorganic arsenic (often using the assumption that non-seafood commodities contain only inorganic arsenic) suggest that the PTWI will normally not be exceeded, unless there is a large contribution from drinking water. Further research is needed about the fate of organic arsenicals and the possibility that they might be converted to more toxic inorganic forms of arsenic, whether by processing or by metabolism in animals or humans.

Cadmium

Reference to JECFA:	16 (1972), 33 (1988), 41 (1993), 55 (2000), 61 (2003), 64 (2005)
Toxicological guidance value:	PTWI 0.007 mg/kg bw (1988, maintained in 2000 & 2003)
Residue definition:	Cadmium, total
Synonyms:	Cd
Related code of practice:	Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
VB 0040	Brassica vegetables	0.05	ML	2005	
VA 0035	Bulb vegetables	0.05	ML	2005	
VC 0045	Fruiting vegetables, cucurbits	0.05	ML	2005	
VO 0050	Fruiting vegetables, other than cucurbits	0.05	ML	2005	Excluding tomatoes and edible fungi.
VL 0053	Leafy vegetables	0.2	ML	2005	
VP 0060	Legume vegetables	0.1	ML	2001	
VR 0589	Potato	0.1	ML	2005	Peeled
VD 0070	Pulses	0.1	ML	2001	Excluding soya bean (dry)
VR 0075	Root and tuber vegetables	0.1	ML	2005	Excluding potato and celeriac
VS 0078	Stalk and stem vegetables	0.1	ML	2005	
GC 0081	Cereal grains, except buckwheat, canihua and quinoa	0.1	ML	2001	Excluding wheat and rice; and bran and germ
CM 1205	Rice, polished	0.4	ML	2006	
GC 0654	Wheat	0.2	ML	2005	
IM 0152	Cephalopods	2	ML	2006	Without viscera
IM 0151	Marine bivalve molluscs	2	ML	2006	Excluding oysters and scallops
	Natural mineral waters	0.003	ML	CS 108-1981	Expressed in mg/l
	Salt, food grade	0.5	ML	CS 150-1985	

Cadmium is a relatively rare element, released to the air, land, and water by human activities. In general, the two major sources of contamination are the production and utilization of cadmium and the disposal of wastes containing cadmium. Increases in soil cadmium content will result in an increase in the uptake of cadmium by plants; the pathway of human exposure from agricultural crops is thus susceptible to increases in soil cadmium. The cadmium uptake by plants from soil is greater at low soil pH. Edible free-living food organisms such as shellfish, crustaceans, and fungi are natural accumulators of cadmium. Similar to humans, there are increased levels of cadmium in the liver and kidney of horses and some feral terrestrial animals. Regular consumption of these items can result in increased exposure. Tobacco is an important source of cadmium uptake in smokers. (Environmental health criteria for cadmium; International Programme on Chemical Safety (IPCS); 1992)

Data from experimental animals and humans show that pulmonary absorption is higher than gastrointestinal absorption. The gastrointestinal absorption of cadmium is influenced by the type of diet and nutritional status. Cadmium absorbed from the lungs or the gastrointestinal tract mainly accumulates in the liver and kidneys. Although cadmium accumulates in the placenta, transfer to the fetus is low. Excretion is normally slow, and the biological half-time is very long (decades). The binding of intracellular cadmium to metallothionein in

Cadmium

tissues protects against the toxicity of cadmium. Excretion occurs mainly via urine. (Environmental health criteria for cadmium; International Programme on Chemical Safety (IPCS); 1992)

The kidney is considered the critical target organ for the general population as well as for occupationally exposed populations. The accumulation of cadmium in the kidney leads to renal dysfunction. Chronic obstructive airway disease is associated with long-term high-level occupational exposure by inhalation. (Environmental health criteria for cadmium; International Programme on Chemical Safety (IPCS); 1992).

The IARC classified cadmium and cadmium compounds in group 1, carcinogenic to humans (1993).

Lead

Reference to JECFA: 10 (1966), 16 (1972), 22 (1978), 30 (1986), 41 (1993), 53 (1999)
Toxicological guidance value: PTWI 0.025 mg/kg bw (1986, maintained in 1993 & 1999)
Residue definition: Lead, total
Synonyms: Pb
Related code of practice: Code of Practice for the Prevention and Reduction of Lead Contamination in Foods (CAC/RCP 56-2004)
Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
FT 0026	Assorted (sub)tropical fruits, edible peel	0.1	ML	2001	
FI 0030	Assorted (sub)tropical fruits, inedible peel	0.1	ML	2001	
FB 0018	Berries and other small fruits	0.2	ML	2001	
FC 0001	Citrus fruits	0.1	ML	2001	
FP 0009	Pome fruits	0.1	ML	2001	
FS 0012	Stone fruits	0.1	ML	2001	
VB 0040	Brassica vegetables	0.3	ML	2001	Excluding kale
VA 0035	Bulb vegetables	0.1	ML	2001	
VC 0045	Fruiting vegetables, Cucurbits	0.1	ML	2001	
VO 0050	Fruiting vegetables, other than Cucurbits	0.1	ML	2001	Excluding mushrooms
VL 0053	Leafy vegetables	0.3	ML	2001	Including Brassica leafy vegetables but excluding spinach.
VP 0060	Legume vegetables	0.2	ML	2001	
VD 0070	Pulses	0.2	ML	2001	
VR 0075	Root and tuber vegetables	0.1	ML	2001	Including peeled potatoes
	Canned fruit cocktail	1	ML	CS 78-1981	
	Canned mangoes	1	ML	CS 159-1987	
	Canned pineapple	1	ML	CS 42-1981	
	Canned raspberries	1	ML	CS 60-1981	
	Canned strawberries	1	ML	CS 62-1981	
	Canned tropical fruit salad	1	ML	CS 99-1981	
	Jams (fruit preserves) and jellies	1	ML	CS 79-1981	
	Mango chutney	1	ML	CS 160-1987	
	Table olives	1	ML	CS 66-1981	
	Canned asparagus	1	ML	CS 56-1981	
	Canned carrots	1	ML	CS 116-1981	

Lead

Code	Food/product	Level (mg/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Canned green beans and canned wax beans	1	ML	CS 16-1981	
	Canned green peas	1	ML	CS 58-1981	
	Canned mature processed peas	1	ML	CS 81-1981	
	Canned mushrooms	1	ML	CS 55-1981	
	Canned palmito	1	ML	CS 144-1985	
	Canned sweet corn	1	ML	CS 18-1981	
	Pickled cucumbers (cucumber pickles)	1	ML	CS 115-1981	
JF 0175	Fruit juices	0.05	ML		Including nectars; Ready to drink
GC 0081	Cereal grains, except buckwheat, canihua and quinoa	0.2	ML	2001	
	Canned chestnuts and canned chestnuts puree	1	ML	CS 145-1985	
MM 0097	Meat of cattle, pigs and sheep	0.1	ML	2001	Also applies to the fat from meat
PM 0110	Poultry meat	0.1	ML	2001	
MO 0812	Cattle, Edible offal of	0.5	ML	2001	
MO 0818	Pig, Edible offal of	0.5	ML	2001	
PO 0111	Poultry, Edible offal of	0.5	ML	2001	
	Fish	0.3	ML	2006	
	Edible fats and oils	0.1	ML	CS 19-1981,	Edible fats and oils not covered by individual standards
	Fat spreads and blended spreads	0.1	ML	CS 256-2007	
	Named animal fats	0.1	ML	CS 211-1999	Lard, rendered pork fat, premier jus and edible tallow.
OR 0305	Olive oil, refined	0.1	ML	CS 33-1981	
OC 0305	Olive oil, virgin	0.1	ML	CS 33-1981	
OR 5330	Olive, residue oil	0.1	ML	CS 33-1981	Olive pomace oil
PF 0111	Poultry fats	0.1	ML	2001	
OC 0172	Vegetable oils, Crude	0.1	ML	CS 210-1999, 2001	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm olein, stearin and superolein.
OR 0172	Vegetable oils, Edible	0.1	ML	CS 210-1999, 2001	Named vegetable oils from arachis, babassu, coconut, cottonseed, grapeseed, maize, mustardseed, palm kernel, palm, rapeseed, safflowerseed, sesameseed, soya bean, and sunflowerseed, and palm

Lead

Code	Food/product	Level (mg/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
					olein, stearin and superolein.
ML 0106	Milks	0.02	ML	2001	A concentration factor applies to partially or wholly dehydrated milks.
LS	Secondary milk products	0.02	ML	2001	As consumed
	Natural mineral waters	0.01	ML	CS 108-1981	Expressed in mg/l
	Infant formula	0.02	ML	2001	Ready to use
	Salt, food grade	2	ML	CS 150-1985	
FF	Wine	0.2	ML	2001	

Exposure to lead can occur from many sources but usually arises from industrial use. Lead and its compounds can enter the environment during mining, smelting, processing, use, recycling, or disposal. The main uses of lead are in batteries, cables, pigments, plumbing, gasoline, solder and steel products, food packaging, glassware, ceramic products, and pesticides. The main exposure of the general non-smoking adult population is from food and water. Airborne lead may contribute significantly to exposure, depending on such factors as use of tobacco, occupation, and proximity to sources such as motorways and lead smelters. Food, air, water, and dust or soil are the main potential sources of exposure of infants and young children (WHO Food Additives Series 44, 2000 with reference to Environmental health criteria for inorganic lead, International Programme on Chemical Safety (IPCS), 1995).

The rate of absorption of lead after ingestion can range from 3% to 80%. It is heavily influenced by food intake, much higher rates of absorption occurring after fasting than when lead is ingested with a meal. Absorption is also affected by age, the typical absorption rates in adults and infants being 10% and 50%, respectively. Up to 50% of the inhaled lead compound may be absorbed. After its absorption and distribution in blood, lead is initially distributed to soft tissues throughout the body. Eventually, bone accumulates lead over much of the human life span and may serve as an endogenous source of lead. The half-life for lead in blood and other soft tissues is about 28-36 days, but it is much longer in the various bone compartments. The percentage retention of lead in body stores is higher in children than adults. Inorganic lead is not metabolized. Lead that is not distributed is mainly excreted through the kidney. (WHO Food Additives Series 44, 2000 with reference to Environmental health criteria for inorganic lead, International Programme on Chemical Safety (IPCS), 1995)

Lead is a classical chronic or cumulative poison. In humans, lead can result in a wide range of biological effects depending upon the level and duration of exposure. Health effects are generally not observed after a single exposure. Many of the effects that have been observed in laboratory animals have also been observed in humans, including hematological effects, neurological and behavioral effects, renal effects, cardiovascular effects, and effects on the reproductive system. In addition, lead has been shown to have effects on bone and on the immune system in laboratory animals. Children are more vulnerable to the effects of lead than adults. Lead has been shown to be associated with impaired neurobehavioral functioning in children. Impaired neurobehavioral development was considered to be the most critical effect. (Food Additives Series 44, 2000 with reference to Environmental health criteria for inorganic lead, International Programme on Chemical Safety (IPCS), 1995).

Inorganic lead compounds are classified by the IARC as probably carcinogenic to humans (Group 2A; Vol. 87, 2006)

Mercury

Reference to JECFA:	10 (1966), 14 (1970) , 16 (1972), 22 (1978)
Toxicological guidance value:	PTWI 0.005 mg/kg bw (1978)
Residue definition:	Mercury, Total
Synonyms:	Hg
Related code of practice:	Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Natural mineral waters	0.001	ML	CS 108-1981	Expressed in mg/l
	Salt, food grade	0.1	ML	CS 150-1985	

Mercury is a naturally occurring metallic element which can be present in foodstuffs by natural causes; elevated levels can also occur due to e.g. environmental contamination by industrial or other uses of mercury. Methylmercury and also total mercury levels in terrestrial animals and plants are usually very low; the use of fish meal as animal feed can however also lead to higher methyl mercury levels in other animal products.

Methylmercury

Reference to JECFA: 22 (1978), 33 (1988), 53 (1999), 61 (2003), 67 (2006)
Toxicological guidance value: PTWI 0.0016 mg/kg bw (2003; confirmed in 2006,)
Residue definition: Methylmercury
Related code of practice: Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Fish	0.5	GL	1991	Except predatory fish Intended for methylmercury in fresh or processed fish and fish products moving in international trade. a)
	Predatory fish	1	GL	1991	Predatory fish such as shark (WS 0131), swordfish, tuna (WS 0132), pike (WF 0865) and others. Intended for methylmercury in fresh or processed fish and fish products moving in international trade. a)

Methylmercury is the most toxic form of mercury and is formed in aquatic environments. Methylmercury therefore is found mainly in aquatic organisms. It can accumulate in the food chain; the levels in large predatory fish species are therefore higher than in other species and fish is the predominant source of human exposure to methylmercury. Methylmercury and also total mercury levels in terrestrial animals and plants are usually very low; the use of fish meal as animal feed can however also lead to higher methyl mercury levels in other animal products.

In all experimental animal species evaluated, methylmercury was readily absorbed (up to 95%) after oral exposure. Methylmercury crossed both the blood–brain barrier and the placenta effectively, resulting in higher concentrations of mercury in the brain of the fetus than of the mother. Methylmercury is eliminated mainly via the bile and faeces, neonatal animals having a lower excretory capacity than adults. Methylmercury is toxic to the nervous system, kidney, liver and reproductive organs, neurotoxicity being the most sensitive end-point (WHO Food additives Series 52; 2004).

a) Lots should be considered as being in compliance with the guideline levels if the level of methylmercury in the analytical sample, derived from the composite bulk sample, does not exceed the above levels. Where these Guideline levels are exceeded, governments should decide whether and under what circumstances, the food should be distributed within their territory or jurisdiction and what recommendations, if any, should be given as regards restrictions on consumption, especially by vulnerable groups such as pregnant women.

Tin

Reference to JECFA:	10 (1966), 14 (1970), 15 (1971), 19 (1975), 22 (1978), 26(1982), 33(1988), 55 (2000), 64 (2005)
Toxicological guidance value:	PTWI 14 mg/kg bw (1988, Expressed as Sn; includes tin from food additive uses; maintained in 2000)
Residue definition:	Tin, total (Sn-tot) when not otherwise mentioned; inorganic tin (Sn-in); or other specification
Synonyms:	Sn
Related code of practice:	Code of Practice for the Prevention and Reduction of Inorganic Tin Contamination in Canned Foods (CAC/RCP 60-2005) Code of Practice for Source Directed Measures to Reduce Contamination of Food with Chemicals (CAC/RCP 49-2001)

Code	Food/product	Level (mg/kg)		Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Canned beverages	150	C	ML	2007	
	Cooked cured chopped meat	50		ML	CS 98-1981	For products in other containers than tinplate container
	Cooked cured ham	50		ML	CS 96-1981	For products in other containers than tinplate container
	Cooked cured pork shoulder	50		ML	CS 97-1981	For products in other containers than tinplate container
	Corned beef	50		ML	CS 88-1981	For products in other containers than tinplate container
	Luncheon meat	50		ML	CS 89-1981	For products in other containers than tinplate container
	Canned foods (other than beverages)	250	C	ML	2007	

Tin is mainly used in tinplated containers, but it is also extensively used in solders, in alloys including dental amalgams. Inorganic tin compounds, in which the element may be present in the oxidation states of +2 or +4, are used in a variety of industrial processes for the strengthening of glass, as a base for colours, as catalysts, as stabilizers in perfumes and soaps, and as dental anticariogenic agents. On the whole, contamination of the environment by tin is only slight. Food is the main source of tin for man. Small amounts are found in fresh meat, cereals, and vegetables. Larger amounts of tin may be found in foods stored in plain cans and, occasionally, in foods stored in lacquered cans. Some foods such as asparagus, tomatoes, fruits, and their juices tend to contain high concentrations of tin if stored in unlaquered cans (Environmental health criteria for tin; International Programme on Chemical Safety (IPCS); 1980). Inorganic tin is found in food in the +2 and +4 oxidation states; it may occur in a cationic form (stannous and stannic compounds) or as inorganic anions (stannites or stannates).

Aflatoxins, Total

Reference to JECFA:	31 (1987), 46 (1996), 49 (1997)
Toxicological guidance value:	Carcinogenic potency estimates for aflatoxins B, G, M (1997, Intake should be reduced to levels as low as reasonably possible.)
Residue definition:	Aflatoxins total (B1 +B2 + G1 + G2)
Synonyms:	Abbreviations, AFB, AFG, with numbers, to designate specific compounds
Related code of practice:	Code of Practice for the Prevention and Reduction of Aflatoxin Contamination in Peanuts (CAC/RCP 55-2004) Code of Practice for the Prevention and Reduction of Aflatoxin Contamination in Tree Nuts (CAC/RCP 59-2005) Code of Practice for the Reduction of Aflatoxin B1 in Raw Materials and Supplemental Feedingstuffs for Milk Producing Animals (CAC/RCP 45-1997)

Code	Food/product	Level (ug/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
SO 0697	Peanut	15	ML	1999	Applies to peanuts intended for further processing. For sampling plan, see below.

Aflatoxins are a group of highly toxic mycotoxins produced by fungi of the genus *Aspergillus*. The four main aflatoxins found in contaminated plant products are B1, B2, G1 and G2 and are a group of structurally related difuranocoumarin derivatives that usually occur together in varying ratios, AFB1 usually being the most important one. These compounds pose a substantial hazard to human and animal health. IARC (1992) classified aflatoxin B1 in Group 1 (human carcinogen) and aflatoxin M in Group 2B (probable human carcinogen). The liver is the primary target organ.

A wide range of foods may be contaminated with aflatoxins; they are most commonly found in groundnuts (peanuts), dried fruit, tree nuts (such as almonds, pecans, walnuts, pistachio and brazil nuts), spices, figs, crude vegetable oils, cocoa beans, maize, rice, cottonseed and copra. Aflatoxin B1 present in animal feed can partly be transferred to milk in the form of the metabolite aflatoxin M1 (mostly 1-2%, but higher percentages are found at low contamination levels in high producing animals.) Aflatoxin contamination is responsible for considerable economic losses and efforts are being made to reduce contamination of food and feedingstuff.

SAMPLING PLAN FOR TOTAL AFLATOXINS IN PEANUTS INTENDED FOR FURTHER PROCESSING

INTRODUCTION

1. The sampling plan calls for a single 20 kg laboratory sample of shelled peanuts (27 kg of unshelled peanuts) to be taken from a peanut lot (sub-lot) and tested against a maximum level of 15 micrograms per kilogram (μg/kg) total aflatoxins.

2. This sampling plan has been designed for enforcement and controls concerning total aflatoxins in bulk consignments of peanuts traded in the export market. To assist member countries in implementing the Codex sampling plan, sample selection methods, sample preparation methods and analytical methods required to quantify aflatoxin in bulk peanut lots are described in this document.

Aflatoxins, Total

A. Definitions

Lot: an identifiable quantity of a food commodity delivered at one time and determined by the official to have common characteristics, such as origin, variety, type of packing, packer, consignor or markings.

Sublot: designated part of a large lot in order to apply the sampling method on that designated part. Each sublot must be physically separate and identifiable.

Sampling plan: is defined by an aflatoxin test procedure and an accept/reject limit. An aflatoxin test procedure consists of three steps: sample selection, sample preparation and aflatoxin quantification. The accept/reject limit is a tolerance usually equal to the Codex maximum limit.

Incremental sample: a quantity of material taken from a single random place in the lot or sublot.

Aggregate sample: the combined total of all the incremental samples taken from the lot or sublot. The aggregate sample has to be at least as large as the 20 kg laboratory sample.

Laboratory sample: smallest quantity of peanuts comminuted in a mill. The laboratory sample may be a portion of or the entire aggregate sample. If the aggregate sample is larger than 20 kg, a 20 kg laboratory sample should be removed in a random manner from the aggregate sample. The sample should be finely ground and mixed thoroughly using a process that approaches as complete a homogenization as possible.

Test portion: portion of the comminuted laboratory sample. The entire 20 kg laboratory sample should be comminuted in a mill. A portion of the comminuted 20 kg sample is randomly removed for the extraction of the aflatoxin for chemical analysis. Based upon grinder capacity, the 20 kg aggregate sample can be divided into several equal sized samples, if all results are averaged.

B. Sampling

Material to be Sampled

3. Each lot which is to be examined must be sampled separately. Large lots should be subdivided into sublots to be sampled separately. The subdivision can be done following provisions laid down in Table 1 below.

4. Taking into account that the weight of the lot is not always an exact multiple of the weight of the sublots, the weight of the sublot may exceed the mentioned weight by a maximum of 20 %.

Aflatoxins, Total

Table 1: Subdivision of Large Lots into Sublots for Sampling

Commodity	Lot weight – tonne (T)	Weight or number of sublots	Number of incremental samples	Laboratory Sample Weight (kg)
Peanuts	≥ 500	100 tonnes	100	20
	>100 and <500	5 sublots	100	20
	≥ 25 and ≤ 100	25 tonnes	100	20
	>15 and <= 25	--1 sublot	100	20

Number of Incremental Samples for Lots of Less than 15 Tonnes

5. The number of incremental samples to be taken depends on the weight of the lot, with a minimum of 10 and a maximum of 100. The figures in the following Table 2 may be used to determine the number of incremental samples to be taken. It is necessary that the total sample weight of 20 kg is achieved.

Table 2: Number of Incremental Samples to be Taken Depending on the Weight of the Lot

Lot weight tonnes – (T)	N° of incremental samples
T≤ 1	10
1 <T ≤ 5	40
5< T ≤ 10	60
10<T < 15	80

Incremental Sample Selection

6. Procedures used to take incremental samples from a peanut lot are extremely important. Every individual peanut in the lot should have an equal chance of being chosen. Biases will be introduced by the sample selection methods if equipment and procedures used to select the incremental samples prohibit or reduce the chances of any item in the lot from being chosen.

7. Since there is no way to know if the contaminated peanut kernels are uniformly dispersed through out the lot, it is essential that the aggregate sample be the accumulation of many small portions or increments of the product selected from different locations throughout the lot. If the aggregate sample is larger than desired, it should be blended and subdivided until the desired laboratory sample size is achieved.

Static Lots

Aflatoxins, Total

8. A static lot can be defined as a large mass of peanuts contained either in a single large container such as a wagon, truck, or railcar or in many small containers such as sacks or boxes and the peanuts are stationary at the time a sample is selected. Selecting a truly random sample from a static lot can be difficult because the container may not allow access to all peanuts.

9. Taking a aggregate sample from a static lot usually requires the use of probing devices to select product from the lot. The probing devices used should be specially designed for the type of container. The probe should (1) be long enough to reach all product, (2) not restrict any item in the lot from being selected, and (3) not alter the items in the lot. As mentioned above, the aggregate sample should be a composite from many small increments of product taken from many different locations throughout the lot.

10. For lots traded in individual packages, the sampling frequency (SF), or number of packages that incremental samples are taken from, is a function of the lot weight (LT), incremental sample weight (IS), aggregate sample weight (AS) and the individual packing weight (IP), as follows :

Equation 1 : SF = (LT x IS)/(AS x IP). The sampling frequency (SF) is the number of packages sampled. All weights should be in the same mass units such as kg.

Dynamic Lots

11. True random sampling can be more nearly achieved when selecting an aggregate sample from a moving stream of peanuts as the lot is transferred, for example, by a conveyor belt from one location to another. When sampling from a moving stream, take small increments of product from the entire length of the moving stream; composite the peanuts to obtain an aggregate sample; if the aggregate sample is larger than the required laboratory sample, then blend and subdivide the aggregate sample to obtain the desired size laboratory sample.

12. Automatic sampling equipment such as cross-cut samplers are commercially available with timers that automatically pass a diverter cup through the moving stream at predetermined and uniform intervals. When automatic equipment is not available, a person can be assigned to manually pass a cup though the stream at periodic intervals to collect incremental samples. Whether using automatic or manual methods, small increments of peanuts should be collected and composited at frequent and uniform intervals throughout the entire time peanuts flow past the sampling point.

13. Cross-cut samplers should be installed in the following manner: (1) the plane of the opening of the diverter cup should be perpendicular to the direction of flow; (2) the diverter cup should pass through the entire cross sectional area of the stream; and (3) the opening of the diverter cup should be wide enough to accept all items of interest in the lot. As a general rule, the width of the diverter cup opening should be about three times the largest dimensions of the items in the lot.

14. The size of the aggregate sample (S) in kg, taken from a lot by a cross cut sampler is :

Equation 2 : S = (D x LT) / (T x V). D is the width of the diverter cup opening (in cm), LT is the lot size (in kg), T is interval or time between cup movement through the stream (in seconds), and V is cup velocity (in cm/sec).

15. If the mass flow rate of the moving stream, MR (kg/sec), is known, then the sampling frequency (SF), or number of cuts made by the automatic sampler cup is :

Equation 3 : SF = (S x V) / (D x MR).

16. Equation 2 can also be used to compute other terms of interest such as the time between cuts (T). For example, the required time (T) between cuts of the diverter cup to obtain a 20 kg aggregate sample from a 30,000 kg lot where the diverter cup width is 5.08 cm (2 inches), and the cup velocity through the stream 30 cm/sec. Solving for T in Equation 2,

T = (5.08 cm x 30,000 kg)/(20 kg x 30 cm/sec) = 254 sec

17. If the lot is moving at 500 kg per minute, the entire lot will pass through the sampler in 60 minutes and only 14 cuts (14 incremental samples) will be made by the cup through the lot. This may be considered too infrequent, in that too much product passes through the sampler between the time the cup cuts through the stream.

Weight of the Incremental Sample

18. The weight of the incremental sample should be approximately 200 grams or greater, depending on the total number of increments, to obtain an aggregate sample of 20kg.

Packaging and transmission of samples

19. Each laboratory sample shall be placed in a clean, inert container offering adequate protection from contamination and against damage in transit. All necessary precautions shall be taken to avoid any change in composition of the laboratory sample which might arise during transportation or storage.

Sealing and labelling of samples

20. Each laboratory sample taken for official use shall be sealed at the place of sampling and identified. A record must be kept of each sampling, permitting each lot to be identified unambiguously and giving the date and place of sampling together with any additional information likely to be of assistance to the analyst.

C. Sample Preparation

Precautions

21. Daylight should be excluded as much as possible during the procedure, since aflatoxin gradually breaks down under the influence of ultra-violet light.

Homogenization – Grinding

22. As the distribution of aflatoxin is extremely non-homogeneous, samples should be prepared - and especially homogenized - with extreme care. All laboratory sample obtained from aggregate sample is to be used for the homogenization/grinding of the sample.

Aflatoxins, Total

23. The sample should be finely ground and mixed thoroughly using a process that approaches as complete a homogenization as possible.

24. The use of a hammer mill with a #14 screen (3.1 mm diameter hole in the screen) has been proven to represent a compromise in terms of cost and precision. A better homogenization (finer grind – slurry) can be obtained by more sophisticated equipment, resulting in a lower sample preparation variance.

Test portion

25. A minimum test portion size of 100 g taken from the laboratory sample.

D. Analytical Methods

Background

26. A criteria-based approach, whereby a set of performance criteria is established with which the analytical method used should comply, is appropriate. The criteria-based approach has the advantage that, by avoiding setting down specific details of the method used, developments in methodology can be exploited without having to reconsider or modify the specified method. The performance criteria established for methods should include all the parameters that need to be addressed by each laboratory such as the detection limit, repeatability coefficient of variation, reproducibility coefficient of variation, and the percent recovery necessary for various statutory limits. Utilizing this approach, laboratories would be free to use the analytical method most appropriate for their facilities. Analytical methods that are accepted by chemists internationally (such as AOAC) may be used. These methods are regularly monitored and improved depending upon technology.

Performance Criteria for Methods of Analysis

Table 3: Specific Requirements with which Methods of Analysis Should Comply

Criterion	Concentration Range	Recommended Value	Maximum Permitted Value
Blanks	All	Negligible	-
Recovery-Aflatoxins Total	1 - 15 µg/kg	70 to 110 %	
	> 15 µg/kg	80 to 110 %	
Precision RSD_R	All	As derived from Horwitz Equation	2 x value derived from Horwitz Equation
Precision RSD_r may be calculated as 0.66 times Precision RSD_R at the concentration of interest			

- The detection limits of the methods used are not stated as the precision values are given at the concentrations of interest;

Aflatoxins, Total

- The precision values are calculated from the Horwitz equation, i.e.:

$$RSD_R = 2^{(1-0.5\log C)}$$

where:

* RSD_R is the relative standard deviation calculated from results generated under reproducibility conditions [$(s_R / \bar{x}) \times 100$]
* C is the concentration ratio (i.e. 1 = 100g/100g, 0.001 = 1,000 mg/kg)

27. This is a generalized precision equation which has been found to be independent of analyte and matrix but solely dependent on concentration for most routine methods of analysis.

Aflatoxins M1

Reference to JECFA: 56 (2001)

Toxicological guidance value: Cancer potency estimates at specified residue levels (2001, Using worst-case assumptions, the additional risks for liver cancer predicted with use of proposed maximum levels of aflatoxin M1 of 0.05 and 0.5 µg/kg are very small. The potency of aflatoxin M1 appears to be so low in HBsAg- individuals that a carcinogenic effect of M1 intake in those who consume large quantities of milk and milk products in comparison with non-consumers of these products would be impossible to demonstrate. Hepatitis B virus carriers might benefit from a reduction in the aflatoxin concentration in their diet, and the reduction might also offer some protection in hepatitis C virus carriers.)

Residue definition: Aflatoxin M1

Synonyms: AFM1

Code	Food/product	Level (ug/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
ML 0106	Milks	0.5	ML	2001	

Deoxynivalenol

Reference to JECFA:	56 (2001)
Toxicological guidance value:	PMTDI 0.001 mg/kg bw (2001)
Synonyms:	Vomitoxin; Abbreviation, DON
Related code of practice:	Code of Practice for the Prevention and Reduction of Mycotoxin Contamination in Cereals, Including Annexes on Ochratoxin A, Zearalenone, Fumonisins and Tricothecenes (CAC/RCP 51-2003)

Deoxynivalenol (DON) is the major compound of a group of chemically related mycotoxins called type B trichothecenes (which are epoxy-sesquiterpenoid compounds) and is produced by certain Fusarium species, which are pathogens of several cereal grains. Closely related compounds are e.g. nivalenol and several acetyl-DON derivatives. DON is water-soluble and chemically very stable under most normal food processing conditions. DON contamination is commonly found in various cereals and cereal products. It undergoes rapid metabolism and elimination in livestock species and the transfer from feed to animal products is probably negligible. Maximum levels in feed are not needed to product public health, but are useful for the protection of animal health and productivity. Especially pigs are vulnerable.

In animals, decreased feed consumption, diarrhea and vomiting have been observed as acute effects. JECFA recognized that DON can lead to outbreaks of acute illness in humans.

Fumonisins

Reference to JECFA:	56 (2001)
Toxicological guidance value:	PMTDI 0.002 mg/kg bw (2001)
Synonyms:	(Several related compounds have been described, notably fumonisin B1, B2 and B3 (abbreviation: FB1 etc.))
Related code of practice:	Code of Practice for the Prevention and Reduction of Mycotoxin Contamination in Cereals, Including Annexes on Ochratoxin A, Zearalenone, Fumonisins and Tricothecenes (CAC/RCP 51-2003)

Fumonisins are a class of recently identified mycotoxins that are produced mainly by certain Fusarium species, especially F. moniliforme which is a pathogen of corn (Zea mays). Fumonisins are a structurally related group of diesters of propane-1,2,3-tricarboxylic acid and various 2-amino-12,16-dimethylpolyhydroxyeicosanes. There are at least 12 fumonisin analogues identified, classified into series A, B, F and P. The B-series, consisting mainly of fumonisin B1 and fumonisin B2, is believed to be the most abundant and most toxic group. A typical ratio between these analogues is B1:B2:B3 as 10:3:1. The worldwide occurrence of fumonisins in corn and corn-based products is well documented: sporadic natural occurrence in sorghum, rice and navy beans has been reported. Fumonisins are heat-stable, so cooking and other heat processes do not substantially reduce their levels in foods. Processing involving treatment of wet milling fractions may, however, lead to elimination of most fumonisin. The human exposure via food can vary to a large extent because of the large range of fumonisin contents found in practice. Fumonisins undergo rapid metabolism and elimination in livestock species and the transfer from feed to animal products is probably negligible. Maximum levels in feed are not needed to protect public health but are useful for the protection of animal health and productivity. In animals, various adverse effects have been observed. The horse appears to be the most sensitive species, and equine leukoencephalomalacia (ELEM) is the most frequently encountered disease. Fumonisins are also associated with liver damage, often also kidney lesions and changes in certain lipid classes, especially sphingolipids, in all animals studied. Carcinogenic effects have been observed in animals exposed to high dietary levels.

Nephrotoxicity, observed in several strains of rat, was considered by JECFA to be the most sensitive toxic effect. On the basis of the NOEL for renal toxicity and a safety factor of 100, the PMTDI was established. National estimates for the mean or median intake were generally much lower than the PMTDI (the highest being 0.2 ug/kg bw).

Ochratoxin A

Reference to JECFA:	37 (1990), 44 (1995), 56 (2001) , 68 (2007)
Toxicological guidance value:	PTWI 0.0001mg/kg bw (2001)
Residue definition:	Ochratoxin A
Synonyms:	(The term “ochratoxins” includes a number of related mycotoxins (A, B, C and their esters and metabolites), the most important one being ochratoxin A)
Related code of practice:	Code of Practice for the Prevention and Reduction of Mycotoxin Contamination in Cereals, Including Annexes on Ochratoxin A, Zearalenone, Fumonisins and Tricothecenes (CAC/RCP 51-2003) Code of Practice for the Prevention and Reduction of Ochratoxin A Contamination in Wine (CAC/RCP 63-2007)

Ochratoxin A is the major compound of a group of chemically related mycotoxins produced by species of the genera Aspergillus and Penicillium. Ochratoxin A contamination is commonly found in various cereals, some pulses, coffee, cocoa, figs, grapes, wine, nuts and coconut products. It can also be transferred through the feed to animal products and concentrates especially in the kidney, but may also be found in meat and milk. Most ochratoxin A is, however, converted to the less harmful ochratoxin-alpha in the rumen of ruminants. Ochratoxin A is a nephrotoxic mycotoxin, which is carcinogenic to rodents and has also teratogenic, immunotoxic and possibly neurotoxic properties. It has been associated with Balkan Endemic Nephropathy

Patulin

Reference to JECFA: 35 (1989), 44 (1995)
Toxicological guidance value: PMTDI 0.0004 mg/kg bw (1995)
Residue definition: Patulin
Related code of practice: Code of Practice for the Prevention and Reduction of Patulin Contamination in Apple Juice and Apple Juice Ingredients in Other Beverages (CAC/RCP 50-2003)

Code	Food/product	Level (ug/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
JF 0226	Apple juice	50	ML	2003	The ML also covers apple juice as ingredient in other beverages.

Patulin is a low molecular weight hemiacetal lactone mycotoxin produced by species of the genera Aspergillus, Penicillium and Byssochlamys. The major sources of patulin contamination are apples with brown rot and blue mould. Because patulin does not spread much from spoilt tissue, the main human exposure can be expected from processed products, like apple juice and apple sauce, in which the contamination is not visible. Because fermentation destroys patulin, it is not normally present in cider and perry, unless unfermented apple juice has been added after fermentation. Patulin may also be a contaminant of soft fruits, some vegetables, barley, wheat and corn.

Potential health problems related to patulin are connected to cytotoxic, immunotoxic, neurotoxic, gastrointestinal and other effects observed in animals. Patulin is mostly eliminated within a few days after ingestion.

The PMTDI was set by applying a safety factor of 100 from the lowest NOAEL of 43 ug/kg bw/day in rats.

T2 and HT-2 Toxin

Reference to JECFA:	56 (2001)
Toxicological guidance value:	PMTDI 0.00006 mg/kg bw (2001, Group PMTDI for T-2 and HT-2 toxins, alone or in combination)
Related code of practice:	Code of Practice for the Prevention and Reduction of Mycotoxin Contamination in Cereals, Including Annexes on Ochratoxin A, Zearalenone, Fumonisins and Tricothecenes (CAC/RCP 51-2003)

T-2 and HT-2 toxin are closely related compounds belonging to a group of chemically related mycotoxins called type A trichothecenes (which are epoxy-sesquiterpenoid compounds) and are produced by certain Fusarium species, which are pathogens of several cereal grains. The most important producer is F. sporotrichioides, a saprophyte which only will grow at high water activities. As a consequence, T-2 and HT-2 toxins are not normally found in grain at harvest, but result from water damage when it remains wet for longer periods in the field or after harvest. T-2 and HT-2 toxin undergo rapid metabolism and elimination in livestock species and the transfer from feed to animal products is probably negligible. Maximum levels in feed are not needed to protect public health, but are useful for the protection of animal health and productivity. Especially pigs are vulnerable. In animals, decreased feed consumption, diarrhea and vomiting have been observed as acute effects.

T-2 toxin is a potent inhibitor of protein synthesis, both in vivo and in vitro. T-2 toxin is linked to outbreaks of acute poisoning of humans, in which the adverse effects reported include nausea, vomiting, pharyngeal irritation, abdominal pain, diarrhea, bloody stool, dizziness and chills. Co-occurrence of T-2 toxin with other trichothecenes in these cases is likely. T-2 toxin is also associated with food-related poisoning incidents in 1931- 1947 referred to as alimentary toxic aleukia, in the former Soviet Union.

The PMTDI is based on a 3-week dietary study with pigs, applying a safety factor of 500 to a LOEL for changes in white and red cell counts. The average intake of T-2 and HT-2 toxin via the human diet was estimated by JECFA as 8 resp. 9 ng/kg bw, which is lower than the group PMTDI. An intake at the level of the PMTDI is not expected to result in effects of T-2 and HT-2 toxin on the immune system and to haematotoxicity, which are considered critical effects after short-term intake. JECFA recommended that toxic equivalency factors relative to DON be developed for the other trichothecenes commonly occurring in cereal grains, if sufficient data become available.

Zearalenone

Reference to JECFA:	53 (1999)
Toxicological guidance value:	PMTDI 0.0005 mg/kg bw (1999, The total intake of zearalenone and its metabolites (including alpha-zearalenol (zeranol)) should not exceed the PMTDI.)
Synonyms:	(Zearalenone is the most important of a group of related mycotoxins and relevant metabolites. Abbreviation, ZEN. Its metabolite, alpha-zearalenol (zeranol) is used as veterinary drug.)
Related code of practice:	Code of Practice for the Prevention and Reduction of Mycotoxin Contamination in Cereals, Including Annexes on Ochratoxin A, Zearalenone, Fumonisins and Tricothecenes (CAC/RCP 51-2003)

Zearalenone (ZEN) is the most important of a group of resorcyclic acid lactone mycotoxins, produced by several species of Fusarium moulds. It is found worldwide in a number of cereal crops and also in derived products like beer. It has been implicated in numerous incidents of mycotoxicosis in farm animals, especially pigs. ZEN is rapidly metabolized in and excreted from animals; residues of this mycotoxin in animal products are probably not significant from a health point of view. A metabolite of ZEN, alpha-zearalenol (zeranol, abbreviated here as ZAL) is, however, relevant relating to its potential use as a veterinary drug. Also beta-zearalenol (taleranol) has hormonal activity. Besides these substances which can be used as anabolic growth promoters, also alpha- and beta-zearalenol (ZEL) and zearalenone (ZAN) are mentioned as possibly occurring metabolites of or co-occurring substances with ZEN.

The PMTDI for ZEN was set by applying a safety factor of 100 from the lowest NOAEL, related to the estrogenic effect in pigs. ZAL has an ADI of 0.5 ug/kg bw (ref. JECFA 26, 27 and 32).

Residues of ZEN and ZAL together in an animal product may be regarded as evidence that the animal feed was contaminated with ZEN. In order to distinguish between contamination of the feed with mycotoxins of the ZEN group or use of ZAL as veterinary drug, it may be necessary to determine the relative proportions of the different residues, e.g. as ZEN + alpha- and beta-ZEL against ZAL. A ratio of 5 or more probably indicates only contamination by mycotoxins.

Acrylonitrile

Reference to JECFA: 28 (1984)
Toxicological guidance value: Provisional Acceptance (1984, the use of food-contact materials from which acrylonitrile may migrate is provisionally accepted on condition that the amount of the substance migrating into food is reduced to the lowest level technologically attainable.)
Residue definition: acrylonitrile (monomer)
Synonyms: 2-Propenenitrile; vinyl cyanide (VCN); cyanoethylene; abbreviations, AN, CAN.

Code	Food/product	Level (mg/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Food	0.02	GL	1991	

Acrylonitrile monomer is the starting substance for the manufacture of polymers which are used as fibres, resins, rubbers and also as packaging material for foods. Acrylonitrile is not known to occur as a natural product. Acrylonitrile is classified by IARC as possibly carcinogenic to humans (Group 2B). Polymers derived from acrylonitrile may still contain small amounts of free monomer. Migration of possibly harmful substances from food contact materials has been discussed in the CCFA/CCFAC in the period 1986-1991. (IARC Vol. 71, 43-108)

Dioxins

Reference to JECFA:	57 (2001)
Toxicological guidance value:	PTMI 70 pg TEQ/kg bw (2001, Including coplanar PCBs)
Synonyms:	Polychlorinated dibenzo-dioxins and -furans
Related code of practice:	Code of Practice for the Prevention and Reduction of Dioxin and Dioxin-like PCB Contamination in Food and Feeds (CAC/RCP 62-2006)

The term dioxins refers to a group of polychlorinated planar aromatic compounds. The group 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-tetra-CDD (TCDD) is the most toxic and most studied congener. Dioxins are ubiquitously present as contaminants in the environment and in food, be it in minute amounts. Dioxins are lipophilic compounds which bind to sediment and organic matter in the environment and tend to be absorbed in animal and human fatty tissue. They are extremely resistant towards chemical and biological transformation processes and are consequently persistent in the environment and accumulate in the food chain. Dioxins are formed as unwanted by-products in combustion processes or industrial processes. Most of the dioxins enter the environment by emission to air. The Ah receptor is an important factor in the toxicological effects of dioxins. Activation of this receptor can result in endocrine and paracrine disturbances and alterations in cell functions including growth and differentiation.

Developmental neurobehavioral (cognitive) and reproductive effects and immunotoxic effects belong to the most sensitive endpoints of dioxin toxicology. TCDD is classified by IARC as Group 1 human carcinogen. It has been shown to be carcinogenic in several animal species at multiple sites, but TCDD is not an initiator of carcinogenesis and the tumour promotion in animal studies indicated a non-genotoxic mechanism.

The toxic equivalency concept has been developed for application to dioxins in order to assess the toxicity of a mixture of congeners as it exists in practice. Toxic Equivalency Factors (TEFs) have been established in relation to TCDD and the total toxicity of a mixture can thus be calculated as total toxic equivalents (TEQs). It has been shown that also some PCB-congeners (those with a planar dioxin-like structure) have effects on the Ah receptor and thus they are given TEFs and can be combined with the dioxins for the calculation of total TEQ of a sample.

Vinyl chloride monomer

Reference to JECFA:	28 (1984)
Toxicological guidance value:	Provisional Acceptance (1984, the use of food-contact materials from which vinyl chloride may migrate is provisionally accepted, on condition that the amount of the substance migrating into food is reduced to the lowest level technologically
Residue definition:	Vinyl chloride monomer
Synonyms:	Monochloroethene, chloroethylene; abbreviation VC or VCM

Code	Food/product	Level (mg/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Food	0.01	GL	1991	The GL in food packaging material is 1.0 mg/kg.

Vinyl chloride monomer is the main starting substance for the manufacture of polymers which are used as resins, as packaging material for foods. Vinyl chloride is not known to occur as a natural product. Residues of vinyl chloride monomer may be still present in the polymer. Vinyl chloride is considered by IARC to be a human carcinogen (as has been shown in occupational exposure situations). IARC Vol. 19, 377-438 (1979)

^{238}Pu, ^{239}Pu, ^{240}Pu, ^{241}Am

Residue definition: ^{238}Pu, ^{239}Pu, ^{240}Pu, ^{241}Am

Code	Food/product	Level (Bq/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Foods other than infant foods	10	GL	2006	
	Infant foods	1	GL	2006	When intended for use as such.

See Appendix 1.

^{90}Sr, ^{106}Ru, ^{129}I, ^{131}I, ^{235}U

Residue definition: ^{90}Sr, ^{106}Ru, ^{129}I, ^{131}I, ^{235}U

Code	Food/product	Level (Bq/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Foods other than infant foods	100	GL	2006	
	Infant foods	100	GL	2006	When intended for use as such.

See Appendix 1.

^{35}S, ^{60}Co, ^{89}Sr, ^{103}Ru, ^{134}Cs, ^{137}Cs, ^{144}Ce, ^{192}Ir

Residue definition: ^{35}S, ^{60}Co, ^{89}Sr, ^{103}Ru, ^{134}Cs, ^{137}Cs, ^{144}Ce, ^{192}Ir; ^{35}S represents the value for organically bound sulphur.

Code	Food/product	Level (Bq/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Foods other than infant foods	1000	GL	2006	
	Infant foods	1000	GL	2006	When intended for use as such.

See Appendix 1.

^{3}H, ^{14}C, ^{99}Tc

Residue definition: ^{3}H, ^{14}C, ^{99}Tc; ^{3}H represents the value for organically bound tritium.

Code	Food/product	Level (Bq/kg)	Type	Reference or Adoption year	Notes/Remarks for Codex Alimentarius
	Foods other than infant foods	10000	GL	2006	
	Infant foods	1000	GL	2006	When intended for use as such.

See Appendix 1.

APPENDIX I

Scope: The Guideline Levels apply to radionuclides contained in foods destined for human consumption and traded internationally, which have been contaminated following a nuclear or radiological emergency[3]. These guideline levels apply to food after reconstitution or as prepared for consumption, i.e., not to dried or concentrated foods, and are based on an intervention exemption level of 1 mSv in a year.

Application: As far as generic radiological protection of food consumers is concerned, when radionuclide levels in food do not exceed the corresponding Guideline Levels, the food should be considered as safe for human consumption. When the Guideline Levels are exceeded, national governments shall decide whether and under what circumstances the food should be distributed within their territory or jurisdiction. National governments may wish to adopt different values for internal use within their own territories where the assumptions concerning food distribution that have been made to derive the Guideline Levels may not apply, e.g., in the case of wide-spread radioactive contamination. For foods that are consumed in small quantities, such as spices, that represent a small percentage of total diet and hence a small addition to the total dose, the Guideline Levels may be increased by a factor of 10.

Radionuclides: The Guideline Levels do not include all radionuclides. Radionuclides included are those important for uptake into the food chain; are usually contained in nuclear installations or used as a radiation source in large enough quantities to be significant potential contributors to levels in foods, and; could be accidentally released into the environment from typical installations or might be employed in malevolent actions. Radionuclides of natural origin are generally excluded from consideration in this document.

In the Table, the radionuclides are grouped according to the guideline levels rounded logarithmically by orders of magnitude. Guideline levels are defined for two separate categories "infant foods" and "other foods". This is because, for a number of radionuclides, the sensitivity of infants could pose a problem. The guideline levels have been checked against age-dependent ingestion dose coefficients defined as committed effective doses per unit intake for each radionuclide, which are taken from the "International Basic Safety Standards" (IAEA, 1996)[4].

Multiple radionuclides in foods: The guideline levels have been developed with the understanding that there is no need to add contributions from radionuclides in different groups. Each group should be treated independently. However, the activity concentrations of each radionuclide within the same group should be added together[5].

ANNEX 1

SCIENTIFIC JUSTIFICATION FOR PROPOSED DRAFT REVISED GUIDELINE LEVELS FOR RADIONUCLIDES IN FOODS CONTAMINATED FOLLOWING A NUCLEAR OR RADIOLOGICAL EMERGENCY

The proposed draft revised Guideline Levels for Radionuclides in Foods and specifically the values presented in Table 1 above are based on the following general radiological considerations and experience of application of the existing international and national standards for control of radionuclides in food.

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3 For the purposes of this document, the term "emergency" includes both accidents and malevolent actions.

4 Food and Agriculture Organization of the United Nations, International Atomic Energy Agency, International Labour Office, OECD Nuclear Energy Agency, Pan American Health Organization, World Health Organization (1996) International Basic Safety Standards for Protection against Ionizing Radiation and for the Safety of Radiation Sources, IAEA, Vienna.

5 For example, if ^{134}Cs and ^{137}Cs are contaminants in food, the guideline level of 1000 Bq/kg refers to the summed activity of both these radionuclides.

Significant improvements in the assessment of radiation doses resulting from the human intake of radioactive substances have become available since the Guideline Levels were issued by the Codex Alimentarius Commission in 1989[6] (CAC/GL 5-1989).

Infants and adults: The levels of human exposure resulting from consumption of foods containing radionuclides listed in Table 1 at the suggested guideline levels have been assessed both for infants and adults and checked for compliance with the appropriate dose criterion.

In order to assess public exposure and the associated health risks from intake of radionuclides in food, estimates of food consumption rates and ingestion dose coefficients are needed. According to Ref. (WHO, 1988) it is assumed that 550 kg of food is consumed by an adult in a year. The value of infant food and milk consumption during first year of life used for infant dose calculation equal to 200 kg is based on contemporary human habit assessments (F. Luykx, 1990[7]; US DoH, 1998[8]; NRPB, 2003[9]). The most conservative values of the radionuclide-specific and age-specific ingestion dose coefficients, i.e. relevant to the chemical forms of radionuclides which are most absorbed from the gastro-intestinal tract and retained in body tissues, are taken from the (IAEA, 1996).

Radiological criterion: The appropriate radiological criterion, which has been used for comparison with the dose assessment data below, is a generic intervention exemption level of around 1 mSv for individual annual dose from radionuclides in major commodities, e.g. food, recommended by the International Commission on Radiological Protection as safe for members of the public (ICRP, 1999)[10].

Naturally occurring radionuclides: Radionuclides of natural origin are ubiquitous and as a consequence are present in all foodstuffs to varying degrees. Radiation doses from the consumption of foodstuffs typically range from a few tens to a few hundreds of microsieverts in a year. In essence, the doses from these radionuclides when naturally present in the diet are unamenable to control; the resources that would be required to affect exposures would be out of proportion to the benefits achieved for health. These radionuclides are excluded from consideration in this document as they are not associated with emergencies.

One-year exposure assessment: It is conservatively assumed that during the first year after major environmental radioactive contamination caused by a nuclear or radiological emergency it might be difficult to readily replace foods imported from contaminated regions with foods imported from unaffected areas. According to FAO statistical data the mean fraction of major foodstuff quantities imported by all the countries worldwide is 0.1. The values in Table 1 as regards foods consumed by infants and the general population have been derived to ensure that if a country continues to import major foods from areas contaminated with radionuclides, the mean annual internal dose of its inhabitants will not exceed around 1 mSv (see Annex 2). This conclusion might not apply for some radionuclides if the fraction of contaminated food is found to be higher than 0.1, as might be the case for infants who have a diet essentially based on milk with little variety.

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6 The Codex Alimentarius Commission at its 18th Session (Geneva 1989) adopted Guideline Levels for Radionuclides in Foods Following Accidental Nuclear Contamination for Use in International Trade (CAC/GL 5-1989) applicable for six radionuclides (^{90}Sr, ^{131}I, ^{137}Cs, ^{134}Cs, ^{239}Pu and ^{241}Am) during one year after the nuclear accident.

7 F. Luykx (1990) Response of the European Communities to environmental contamination following the Chernobyl accident. In: Environmental Contamination Following a Major Nuclear Accident, IAEA, Vienna, v.2, 269-287.

8 US DoHHS (1998) Accidental Radioactive Contamination of Human Food and Animal Feeds: Recommendations for State and Local Agencies. Food and Drug Administration, Rockville.

9 K. Smith and A. Jones (2003) Generalized Habit Data for Radiological Assessments. NRPB Report W41.

10 International Commission on Radiological Protection (1999). Principles for the Protection of the Public in Situations of Prolonged Exposure. ICRP Publication 82, Annals of the ICRP.

Long-term exposure assessment: Beyond one year after the emergency the fraction of contaminated food placed on the market will generally decrease as a result of national restrictions (withdrawal from the market), changes to other produce, agricultural countermeasures and decay.

Experience has shown that in the long term the fraction of imported contaminated food will decrease by a factor of a hundred or more. Specific food categories, e.g. wild forest products, may show persistent or even increasing levels of contamination. Other categories of food may gradually be exempted from controls. Nevertheless, it must be anticipated that it may take many years before levels of individual exposure as a result of contaminated food could be qualified as negligible.

ANNEX 2

ASSESSMENT OF HUMAN INTERNAL EXPOSURE WHEN THE GUIDELINE LEVELS ARE APPLIED

For the purpose of assessment of the mean public exposure level in a country caused by the import of food products from foreign areas with residual radioactivity, in implementing the present guideline levels the following data should be used: annual food consumption rates for infants and adults, radionuclide- and age-dependent ingestion dose coefficients and the import/production factors. When assessing the mean internal dose in infants and adults it is suggested that due to monitoring and inspection the radionuclide concentration in imported foods does not exceed the present guideline levels. Using cautious assessment approach it is considered that all the foodstuffs imported from foreign areas with residual radioactivity are contaminated with radionuclides at the present guideline levels.

Then, the mean internal dose of the public, E (mSv), due to annual consumption of imported foods containing radionuclides can be estimated using the following formula:

$$E = GL(A) \cdot M(A) \cdot e_{ing}(A) \cdot IPF$$

where:

GL(A) is the Guideline Level (Bq/kg)

M(A) is the age-dependent mass of food consumed per year (kg)

$e_{ing}(A)$ is the age-dependent ingestion dose coefficient (mSv/Bq)

IPF is the import/production factor[11] (dimensionless).

Assessment results presented in Table 2 both for infants and adults demonstrate that for all the twenty radionuclides doses from consumption of imported foods during the 1st year after major radioactive contamination do not exceed 1 mSv. It should be noted that the doses were calculated on the basis of a value for the IPF equal to 0.1 and that this assumption may not always apply, in particular to infants who have a diet essentially based on milk with little variety.

[11] The import/production factor (***IPF***) is defined as the ratio of the amount of foodstuffs imported per year from areas contaminated with radionuclides to the total amount produced and imported annually in the region or country under consideration.

It should be noted that for ^{239}Pu as well as for a number of other radionuclides the dose estimate is conservative. This is because elevated gastro-intestinal tract absorption factors and associated ingestion dose coefficients are applied for the whole first year of life whereas this is valid mainly during suckling period recently estimated by ICRP to be as average first six months of life (ICRP, 2005[12]). For the subsequent six months of the first year of life the gut absorption factors are much lower. This is not the case for ^{3}H, ^{14}C, ^{35}S, iodine and cesium isotopes.

As an example, dose assessment for ^{137}Cs in foods is presented below for the first year after the area contamination with this nuclide.

For adults: E = 1000 Bq/kg ·550 kg ·$1.3\cdot10^{-5}$ mSv/Bq ·0.1 = 0.7 mSv;
For infants: E = 1000 Bq/kg ·200 kg ·$2.1\cdot10^{-5}$ mSv/Bq ·0.1 = 0.4 mSv

TABLE. ASSESSMENT OF EFFECTIVE DOSE FOR INFANTS AND ADULTS FROM INGESTION OF IMPORTED FOODS IN A YEAR

Radionuclide	Guideline Level (Bq/kg)		Effective dose (mSv)	
	Infant foods	Other foods	1st year after major contamination	
			Infants	Adults
^{238}Pu	1	10	0.08	0.1
^{239}Pu			0.08	0.1
^{240}Pu			0.08	0.1
^{241}Am			0.07	0.1
^{90}Sr	100	100	0.5	0.2
^{106}Ru			0.2	0.04
^{129}I			0.4	0.6
^{131}I			0.4	0.1
^{235}U			0.7	0.3

Radionuclide	Guideline Level (Bq/kg)		Effective dose (mSv)	
	Infant foods	Other foods	1st year after major contamination	
			Infants	Adults
^{35}S*	1000	1000	0.2	0.04
^{60}Co			1	0.2
^{89}Sr			0.7	0.1
^{103}Ru			0.1	0.04
^{134}Cs			0.5	1
^{137}Cs			0.4	0.7
^{144}Ce			1	0.3
^{192}Ir			0.3	0.08
^{3}H**	1000	10000	0.002	0.02
^{14}C			0.03	0.3
^{99}Tc			0.2	0.4

* This represents the value for organically bound sulphur.
** This represents the value for organically bound tritium.

[12] International Commission on Radiological Protection (2005) Doses to Infants from Radionuclides Ingested in Mothers Milk. To be published.