chapter four

Contaminants

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4.1 Introduction

Food contaminants are substances that are included unintentionally in foods. Some are harmless and others are hazardous because of the toxicological risks from their intake to the consumer. Harmless contaminants may still have the disadvantage of interfering with food processing and causing interactions during storage. Examples are metal ions and plant pigments. This chapter deals with contaminants that are hazardous.

Contamination can occur at every step on the way from raw material to consumer. Raw material of plant origin can be contaminated with environmental pollutants, such as heavy metals, pesticide residues, industrial chemicals, and products from fossil fuels (in the exhaust gases of combustion engines). The sources of contaminants in raw materials of animal origin — mainly fish and milk — are to a large extent comparable to those of raw materials originating from plants. In animal products also, residues of veterinary drugs and growth promoting substances may be present.

During processing, food can be contaminated with processing aids, such as filtering and cleaning agents, and with metals coming from the equipment.

Finally, contaminants can be included in foods during packaging and storage. These can originate from plastics, coatings, and tins.

A number of important examples of hazardous contaminants originating from the above sources are dealt with in this chapter. Toxic contaminants of natural origin have already been discussed in Chapter 2. The formation and inclusion of the well-known environmental pollutants polycyclic aromatic hydrocarbons are dealt with in Chapter 6.

4.2 *Contamination with heavy metals*

4.2.1 *Mercury*

The widespread use of mercury and its derivatives in industry and agriculture has resulted in serious environmental pollution. This has led to increased levels of mercury in foods. Fish products in particular can be contaminated with mercury, as methylmercury accumulates extensively in fish. Data on mercury residues in food are shown in Table 4.1.

The toxicity of mercury depends on the chemical form involved: elemental, inorganic, or organic. Exposure to organic mercury compounds, especially methylmercury, is more dangerous than exposure to elemental or inorganic mercury. Organic mercury compounds easily pass across biomembranes and are lipophilic.

The primary target for mercury is the central nervous system. Human response data are available from epidemics of methylmercury poisoning in Japan and Iraq. The first epidemic was caused by consumption of fish from water that was heavily contaminated by industrial waste water. In Iraq, the poisoning appeared to result from the ingestion of wheat treated with a fungicidal mercurial. The total daily intake of mercury per individual in the US and in Western Europe is estimated at 1 to 20 µg. The tolerable weekly intake (TWI) is 300 µg, of which not more than 200 µg should be in the form of methylmercury. The TWI is an estimate of the amount of a contaminant in food or drinking water, which can be ingested weekly over a lifetime by humans without appreciable health risk. Compare to Part 3, Chapter 16, Section 16.3.2.1. The US has set limit values for seafood only.

4.2.2 *Lead*

In Roman times, extensive lead poisoning occurred as a result of drinking wine treated with lead salts for neutralizing the sour taste. Over a long period of time contamination of

Table 4.1 Levels of mercury residues in food in several countries

Foods	Hg in µg/kg (ppb)		
	United States	United Kingdom	Japan
Cereal (grains)	2–25	5	12–48
Bread and flour		20	
Meats[a]	1–150	10–40	310–360
Fish[b]	0–60	70–80	35–540
Dairy products			
Milk	8	10	3–7
Cheese	80	170	–
Butter	140	10	–
Fruits	4–30	10–40	18
Vegetables (fresh)	0–20	10–25	30–60
Canned	2–7	20[c]	0
Eggs			
White	10	ND[d]	80–125
Yolk	62		330–670
Beer	4		

[a] Includes beef, pork, beef liver, canned meats, and sausages.

[b] Includes canned salmon, shellfish, and whitefish.

[c] Canned peas.

[d] Not detectable.

Table 4.2 Ranges and means of lead content of food

Food	Pb, µg/100 g Range	Mean	Food	Pb, µg/100 g Range	Mean
Cereal grains	0–62	22	Cider, apple		90 µg/l
Cereal grain products	0–749	10.5	Vinegar, cider		100 µg/l
Seafood, raw	17–250	62	Cola (2 samples)	18–65 µg/l	
Canned	6–30	16	Ginger ale		10 µg/l
Meats	7–37	19	Beer, canned		40 µg/l
Gelatin		57	Wine, red		50 µg/l
Eggs, whole	0–15	7	Sugar, white granulated	0–7	
Vegetables, leafy	0–126	37			
Legumes, raw, dried or frozen	0–16	7	Molasses		53
			Backing powder		150
Canned	3–11	7	Yeast, dry		117
Apple, raw		38	Black pepper		40
Pear, raw		3	Cinnamon		11
Milk, whole, fresh		0	Nutmeg		41
Skim, dried and packaged		2	Allspice		64
Skim, bulk package		2	Chili powder		18
evaporated	4–5	4.5	Bay leaves		55
Tea, leaves		1.37			
Cocoa, dry		0.10			

food and water with lead occurred in improperly lead-glazed earthenware containers, tins with lead solders, or lead water pipes.

At the moment, the main causes of environmental contamination with lead are industrialization and the use of leaded gasoline. The lead content of food, however, has not significantly increased. The soil retains lead effectively. Nevertheless, the diet, including drinking water, is believed to be the principal source of the total body burden of lead (Table 4.2).

Chronic lead intoxication has been reported to lead to central and peripheral nervous system effects, anemia, and disturbance of renal function and weight loss. Lead intoxications following the intake of contaminated food and water rarely happen. In the majority of cases, lead poisoning of adults is occupational.

Lead compounds are hardly soluble in water. As a result, their absorption is low. Approximately 10% of the ingested lead is absorbed. The levels of lead in bones, hair, and teeth increase with age, suggesting a gradual accumulation of lead in the body. Therefore, contamination of food with lead and the possibility of chronic lead intoxication through the diet needs constant monitoring.

Annual FDA Total Diet Studies have shown that the average lead intake (by adults) in the US has decreased from 90 µg/day in 1974 to 8.1 µg/day in 1989. The TWI through food is 3 mg for adults, and 25 µg per kg body weight for children. The US has set the safety limit for lead in calcium supplements at 5.0 µg/g. Further, the lead content of drinking water and bottled water should not exceed 5 ppb. Decontamination of plant foods by trimming or dehulling is sometimes possible.

4.2.3 *Cadmium*

Cadmium is widely distributed in the environment, due to extensive industrial use. Sewage sludge, which is used as fertilizer and soil conditioner, is an important source of soil pollution with cadmium. In food, only inorganic cadmium salts are present. Organic

Table 4.3 Cadmium content of foods in the US in µg/100 g

Class of foodstuffs	1972–1973		1973–1974		1974–1975		1975	1976
	Range	Average[a]	Range	Average[a]	Range	Average[a]	Range	Average[a]
Dairy products	1–6	trace (5/30)	1–14	1 (4/30)	trace	trace (4/20)	1–2	0.2 (3/20)
Meat, fish, and poultry	1–6	1 (12/30)	1–6	2 (21/30)	trace	trace (11/20)	1–3	1.0 (17/20)
Grains and cereals	2–5	1 (30/30)	2–5	3 (29/30)	5–8	trace (19/20)	2–5	3.0 (20/20)
Potatoes	2–12	5 (30/30)	2–13	5 (29/30)	5–12	4 (20/20)	2–9	5.0 (20/20)
Leafy vegetables	1–28	5 (30/30)	1–14	4 (28/30)	5–14	5 (20/20)	2–10	4.0 (19/20)
Legumes	1–3	trace (10/30)	1–10	1 (8/30)	trace	trace (3/30)	1–7	1.0 (14/20)
Root vegetables	1–6	2 (24/24)	1–31	3 (24/30)	trace	trace (16/20)	1–8	2.7 (19/20)
Garden fruits	1–6	2 (25/25)	1–10	2 (23/30)	trace	trace (17/10)	1–4	2.0 (18/20)
Other fruits	1–2	trace (4/30)	1–6	trace (3/30)	trace	trace (5/20)	1–2	0.3 (5/20)
Oils, fats, shortening	1–6	3 (29/30)	1–7	2 (24/30)	trace	trace (17/20)	1–3	1.6 (18/20)
Sugars and adjuncts	1–6	1 (13/30)	1–9	1 (12/30)	trace	trace (8/20)	1–3	1.1 (14/20)
Beverages	1–8	trace (5/30)	1–3	trace (6/30)	trace	trace (1/20)	0–1	0.2 (3/20)

[a] For numbers in parentheses, numerators represent positive composites; the denominators, the total number of composites analyzed.

cadmium compounds are very unstable. In contrast to lead and mercury ions, cadmium ions are readily absorbed by plants. They are equally distributed over the plant.

Foods of animal origin that can be contaminated with cadmium, include liver, kidney and milk. Table 4.3 shows the cadmium content of foods in the US.

Cadmium accumulates in the human body, especially in the liver and kidney. In experimental animals it can cause anemia, hypertension, and testicular damage. Chronic cadmium intoxication in humans occurred in Japan after the consumption of rice heavily contaminated as a result of environmental pollution. 0.1 to 1 mg/day were ingested for a period of possibly more than 12 years. The painful disease that developed was characterized by skeletal deformation, reduced body height and multiple fractures. Vitamin D deficiency appeared to be a predisposing factor in this case.

The intake of cadmium in the US from 1982 to 1991 ranged from 3.7 to 14.4 µg/day (also determined on the basis of data obtained in FDA Total Diet Studies). The absorption from food varies, depending on genetic factors, age, and nutritional factors. Infants absorb and accumulate more cadmium than adults. Calcium or iron deficiency can increase the absorption of cadmium. Pyridoxine deficiency appears to decrease its absorption. The TWI is 0.4 to 0.5 mg. The US has set a safety limit for drinking water and bottled water: 0.005 mg/l. For foods, no limit values have been set.

4.3 Nitrate

Contamination of the biosphere with nitrogen compounds can result in a nitrate concentration increase in groundwater. This can ultimately lead to increases in the nitrate concentration of drinking water as well as in the nitrate level of food (of plant origin).

Public waterworks use both groundwater and surface water as sources of drinking water. At the moment, it is not common practice to remove nitrate during drinking water production. Where there is no connection with the water system, groundwater is also used as a source of drinking water through private wells.

There are a number of nitrate sources in the soil. For example, nitrate can originate from microbial fixation of nitrogen in symbiotic relationships with leguminous plants, i.e., from the only biological way of binding nitrogen. Other sources are soil pollution caused by the use of fertilizers in agriculture and manure production in cattle breeding and dairy farming. Microbial nitrification is responsible for the conversion of ammonia and urea to nitrate in the soil. The toxicological risks due to intake of nitrate are attributed to its reduction product nitrite.

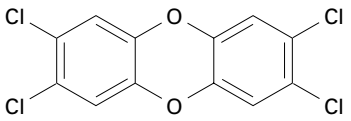
Nitrite can oxidize hemoglobin to methemoglobin. In acidic environments, it may react with secondary amines under the formation of nitrosamines. Numerous alkyl- or alkylarylnitrosamines are carcinogenic in experimental animals. Nitrite and nitrosamines can be formed *in situ* in food, but also in the body (after ingestion of nitrate). Oral and intestinal bacteria can convert nitrate to nitrite.

Certain vegetables tend to accumulate nitrate: beets, celery, lettuce and spinach. The nitrate levels in some foods are listed in Table 4.4.

The intake of nitrate via food consumption is estimated at 1.4 to 2.5 mg/kg/day, and from water at 0.3 mg/kg/day. The acceptable daily intake (A.D.I.) of nitrate is 3.64 mg/kg/day. (See Part 3, Chapter 17, Sections 17.3.2 and 17.3.3)

4.4 2,3,7,8-Tetrachlorodibenzo-p-dioxin

2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) is a well-known environmental pollutant, formed from chlorinated hydrocarbons at the high temperatures reached in incinerators. Further, it is also a contaminant of the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T).



Tetrachlorodibenzo–*p*–dioxin (TCDD)

TCDD is highly toxic on acute exposure. The oral LD_{50} in guinea pigs of either sex is 1 µg/kg. It induces a variety of adverse effects in experimental animals: liver damage, porphyria, teratogenic effects, immune suppression and increased tumor incidence. It also causes enzyme induction. In man, the following effects have been reported (based on occupational exposures and industrial accidents): chloracne, porphyria, liver damage and polyneuropathies.

The main route of exposure to TCDD is dietary intake. TCDD can reach food via:

(a) spraying of crops with 2,4,5-T;
(b) ingestion of contaminated feed by livestock;
(c) magnification via food chains;
(d) contamination of fruits and vegetables in the proximity of incinerators.

Table 4.4 Average nitrate contents of common foods in the US and per capita daily intake

Food	Nitrate, mg/100 g	
	Content	Ingestion
Total vegetables	1.3–27.6	8609.1
Asparagus	2.1	2.8
Beet	276	546.0
Beans, dry	1.3	10.0
Beans, lima	5.4	6.6
Beans, snap	25.3	258.0
Broccoli	78.3	127.0
Cabbage	63.5	548.0
Carrot	11.9	104.0
Celery	234.0	1600.0
Corn	4.5	77.0
Cucumber	2.4	7.8
Eggplant	30.2	14.8
Lettuce	85.0	1890.0
Melon	43.3	935.0
Onion	13.4	159.0
Peas	2.8	19.8
Pepper, sweet	12.5	33.5
Pickles	5.9	56.0
Potato	11.9	1420.0
Potato, sweet	5.3	26.4
Pumpkin/squash	41.3	38.0
Spinach	186.0	420.0
Sauerkraut	19.1	33.2
Tomato and tomato products	6.2	198.0
Breads	2.2	198.0
All fruits	1.0	130.0
Juices	0.2	10.7
Cured meats	20.8	1554.0
Milk and milk products	0.05	25.0
Water	0.071	71.0

Data on the dietary intake of TCDD are scarce. An indication for the body burden may be the 2 ppt, measured in mother's milk.

The ADI is set at 10 pg/kg/day (by a World Health Organization Expert Committee).

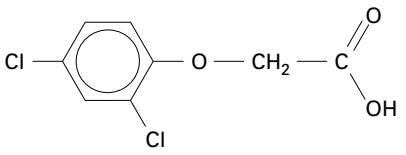
4.5 Pesticide residues

Pesticides are chemicals developed and produced for use in the control of agricultural and public health pests. The main groups of pesticides are insecticides, herbicides, and fungicides. Pesticides are of vital importance in the fight against diseases, e.g., malaria, and for the production and storage of food. In spite of their extensive use, an average of 35% of the produce is lost worldwide.

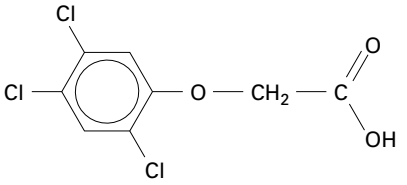
Common classes of pesticides include organochlorine compounds, organophosphates, and carbamates.

Many members of the various classes are highly toxic. A common misconception is that pesticides have the same mode of action. The ways in which they act are as diverse as their chemistry. Chlorinated cyclodiene insecticides (e.g., aldrin) are neurotoxicants that

interfere with γ-aminobutyric acid transmitters in the brain. In humans and experimental animals, seizures have been reported, in addition to symptoms such as nausea, vomiting, and headache. The toxicity mechanism of the chlorophenoxy herbicides 2,4-dichlorophenoxyacetic acid and 2,4,5- trichlorophenoxyacetic acid is poorly understood. They induce their herbicidal effects by acting as growth hormones in plants. However, they do not act as hormones in experimental animals. In animals, effects such as stiffness of the extremities, inability to coordinate muscular movements, paralysis, and eventually coma have been observed.



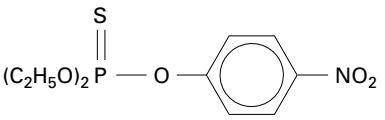
2, 4 – Dichlorophenoxyacetic acid



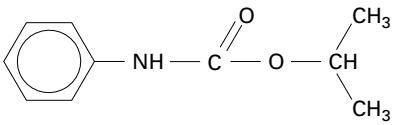
2, 4, 5 – Trichlorophenoxyacetic acid

The organophosphorous insecticides (e.g., parathion) inhibit acetylcholinesterase, resulting in symptoms (that mimic the action of acetylcholine) such as lachrymation, pupillary constriction, convulsions, respiratory failure, and coma.

Carbamate herbicides such as propham (isopropyl-N-carbanilate) have relatively low acute toxicities. The oral LD_{50} of propham in rats is 5 g per kg. Herbicidal carbamates are not inhibitors of cholinesterase.



Parathion



Propham (isopropyl carbanilate)

The toxicological risks from residues of s ynthetic pesticides in foods are minimal because of careful food safety legislation and regulation. Contamination of vegetables may result from treatment as well as from conditions such as improper use of pesticides, residues from preceding treatments in the soil and cross-contamination (particularly during harvesting). Sources of residues in products of animal origin include contaminated water or feed, pesticide-treated housing, and contaminated milk (during weaning).

Table 4.5 lists the pesticide residue levels in food in the US.

Organochlorine insecticides deserve particular attention, as they are very stable and can accumulate in food chains. Products of animal origin as well as mother's milk almost always contain residues of organochlorine compounds. The residue content of mother's milk is 10 to 30 times higher than that of cow's milk.

From May 1990 through July 1991, 806 milk samples from 63 metropolitan areas in the US were collected and analyzed for pesticide residues by the FDA. In the samples from eight of the metropolitan areas, no residues could be detected. Pesticide residues appeared to contaminate 398 milk samples though. The most frequently occurring residues were *p,p′*-DDE (4,4′-dichlorodiphenyltrichloroethane) (in 212 samples) and dieldrin (in 172 samples). The highest residue level measured was 0.02 ppm *p,p′*-DDE (whole milk basis). These chlorinated pesticides have not been registered for agricultural use for about 20 years.

Table 4.5 Pesticide residues in food in the US in 1991

Food	Origin	Number of samples	Samples with residues below permissible level in %	Samples with residues above permissible level in %
Grains/grain products	Domestic	495	40.8	0.8
	Import	396	25.5	2.3
Milk/dairy products/eggs	Domestic	809	12.5	0
Milk/dairy products	Import	216	10.2	0
Fish/shellfish/other meats	Domestic	536	41.6	0.2
Fish/shellfish	Import	611	23.2	0.2
Fruits	Domestic	2168	50.9	0.5
	Import	3481	34.1	1.3
Vegetables	Domestic	3811	30.6	1.3
	Import	4311	28.3	3.3
Other	Domestic	462	19.5	0
	Import	918	17.9	3.5

Source: Food and Drug Administration Pesticide Program, Residue Monitoring 1991 (5th annual report).

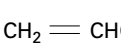
The use of organochlorine compounds is decreasing in favor of that of organophosphates and carbamates. Both latter classes of pesticidal chemicals are much more readily degraded, in the environment as well as during processing.

Many of the techniques presently used in food processing give a considerable reduction of pesticide residue levels. Many types of residues are degraded to harmless products during processing due to heat, steam, light, and acid or alkaline conditions. In addition, major reductions of residue levels result from their physical removal by peeling, cleaning or trimming of foods such as vegetables, fruits, meat, fish and poultry.

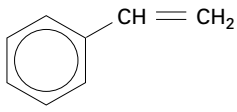
Table 4.6 lists the results of the Total Diet Study 1991 on the occurrence of pesticides in food. In general, residues present at or above 1 ppb could be measured. Malathion continues to be the residue most frequently found; it is used on a wide variety of crops, including many post-harvest uses on grains. From 1987 to 1991, the occurrence of malathion has decreased from 23 to 18% (see Table 4.6), and that of DDT from 22 to 10%.

4.6 *Food contaminants from packaging material*

Contact of packaging material with food may result in the transfer of trace quantities of particular chemicals, such as monomers and plasticizers. Well-known chemicals used in the production of polymers are vinyl chloride and styrene. Vinyl chloride is the monomer of polyvinyl chloride, and styrene is used in the manufacturing of a number of plastics. Important plasticizers in polyvinyl chloride plastics are the phthalic acid esters di-(2-ethylhexyl) phthalate (DEHP) and di-*n*-butyl phthalate (DBP).



Vinyl chloride



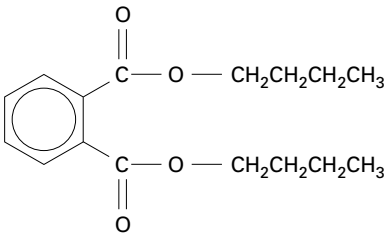
Styrene

Table 4.6 Occurrence of pesticides in total diet study in 1991

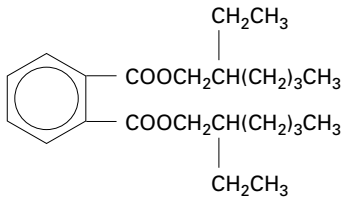
Pesticide[a]	Number of food items contaminated with	Occurrence %[b]
Malathion	167	18
Chlorpyrifos-methyl	97	10
DDT	93	10
Dieldrin	73	8
Endosulfan	67	7
Methamidophos	58	6
Chlorpyrifos	51	5
Dicloran	44	5
Acephate	42	4
Diazinon	42	4
Dimethoate	34	4
Chlorpropham	28	3
Heptachlor	24	3
Lindane	22	2
Omethoate	22	2
Ethion	21	2
Hexachlorobenzene	20	2
Permethrin	16	2
BHC, alpha	13	1
Chlordane	12	1
Parathion	12	1
Quintozene	12	1
Dicofol	10	1

[a] Including parent compounds, isomers, metabolites and related compounds.

[b] On the basis of 936 items. NB: a food item can contain several pesticides.



Dibutyl phthalate



Di(2 – ethylhexyl) phthalate

Vinyl chloride has been identified as a liver carcinogen in animal models as well as in humans. Acute intoxication causes depression of the central nervous system and hepatic damage. Vinyl chloride leaches out of packaging materials into water as well as into fatty material. Mineral water (stored in polyvinyl chloride bottles) has been shown to take up vinyl chloride. After 6 months, a concentration of 170 mg per l was measured. This may lead to a daily intake of 120 ng per person in countries where polyvinyl chloride bottled drinking water is used. In cooking oils, higher concentrations have been found, viz. 14.8 mg/kg.

Styrene-induced toxic effects include renal and hepatic damage, pulmonary edema, and cardiac arrhythmia. The oral LD_{50} in rats is relatively low: 5 g/kg. Styrene appears to leach out of polystyrene packaging material, preferably into the fatty components of food.

Average concentrations of 27 ppb have been measured in high-fat yogurt, 71 ppb in fruit yogurt, 20 to 70 ppb in other desserts, 18 to 180 ppb in meat products and 5 ppb in packed fruit and vegetable salads. For styrene, a provisional ADI of 40 ng per kg has been calculated.

The phthalic acid esters DEHP and DBP have low acute toxicities. The intraperitoneal LD_{50} in mice are 14.2 and 4.0 g/kg, respectively. However, liver or lung damage by the leached plasticizers has been suggested. DEHP and BBP appear to be non-genotoxic carcinogens.

Since they are widely distributed in materials involved in transportation, construction, clothing, medicine, and packaging, the concern about their health effects has increased.

4.7 Summary

Food contaminants are substances unintentionally included in foods. Some are harmless but others may be hazardous. Contamination can occur at every step on the way from raw material to consumer. Raw materials of plant origin can be contaminated with environmental pollutants, such as heavy metals, pesticide residues, industrial chemicals, and products from fossil fuels. The sources of contaminants from raw materials of animal origin are to a large extent comparable with those from raw materials of plant origin. During processing, food can be contaminated with processing aids (filter and cleaning agents) and equipment materials (e.g., metals), and during packaging and storage with components of the packaging material. A number of important examples of hazardous food contaminants originating from the above sources were dealt with, namely heavy metals (mercury, lead, and cadmium), nitrate, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, pesticides, vinyl chloride, styrene, and plasticizers di-(2-ethylhexyl) phthalate and di-*n*-butyl phthalate.

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