

## Chemical Hazards in Food\*

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This review gives a broad overview of the types of chemical hazards that can occur in foodstuffs, indicating how they arise and how they are measured and controlled. The examples given are representative of the many types of issues that the food industry has to face on a daily basis.

Further information on individual chemicals can be found in [Campden BRI Review 52 - Understanding chemical hazards in support of risk assessment](#). Also, advice on the risk assessment of raw materials will be published in March 2011 in Campden BRI Guideline 65 - *Risk assessment and management of raw materials*. Meanwhile, analysis is covered in the Campden BRI [Key Topic No. 10 Chemical analysis of foods: an introduction](#). A summary of [chemistry](#) and [quality management](#) services available to the food industry can be found on the Campden BRI website ([www.campden.co.uk](http://www.campden.co.uk)).

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## CHEMICAL HAZARDS IN FOOD

### INTRODUCTION

The purpose of this review is to give a broad overview of the types of chemical hazards that can occur in foodstuffs, to indicate how they arise and how they are measured and controlled. The examples given are representative of the many types of issues that the food industry has to face on a daily basis. The risks posed by allergens have been omitted, as this is covered by a separate IUFOST Scientific Information Bulletin (<http://iufost.org/iufost-scientific-information-bulletins-sib>)

### ORIGIN OF CHEMICAL HAZARDS IN FOODS

It has been said that 99% of all toxins are naturally occurring, and also that all things are toxic at a high enough concentration. Certainly, many food raw materials contain chemicals, which, if consumed in excess, might lead to health problems. Cooking and processing in general can remove or inactivate many chemicals (e.g. protease inhibitors, lectins) that are either directly toxic or inhibit digestion or absorption of nutrients. However, some chemicals have arisen as problems associated with food processing techniques developed in the last 100 years or so, e.g. trans fatty acids resulting from chemical hydrogenation of unsaturated fats, or 3-monochloropropanediol from the chemical hydrolysis of proteins. One recently publicised example of a process-derived chemical hazard in food is the formation of acrylamide in baked products. Although this has been occurring for centuries (e.g. in home baking of bread, potatoes and other starch-based foods), it was not discovered until 2002. A further area of concern is the migration of chemicals from packaging materials into foods, which has recently become a large problem for food manufacturers.

Other hazards are contaminants introduced by accident during the production of the food raw materials - sometimes these are unavoidable and sometimes they are to a greater or lesser extent caused by poor growing, post-harvest or processing conditions. Mycotoxins produced by moulds on grain or nut products are one example; nitrate accumulation in leafy vegetables, and heavy metal accumulation in seafoods are others.

Most difficult to predict or control are the chemical hazards introduced deliberately, generally as a consequence of fraudulent trading (e.g. addition of melamine to milk to boost the apparent protein content).

Chemical hazards can thus be divided into five broad categories:

- Inherent ('Natural') toxins
- Natural and environmental contaminants
- Process and storage-derived contaminants
- Deliberately added contaminants
- Pesticides and veterinary residues

## EXAMPLES OF CHEMICAL HAZARDS

The examples below give a flavour of the many different chemicals that can be hazardous in food. They are chosen to give a broad picture of their nature, rather than to be exhaustive. Some, such as trans fatty acids, have not been included, as these were discussed in a previous IUFOST Scientific Information Bulletin in 2010 (<http://iufost.org/iufost-scientific-information-bulletins-sib>).

### *i) Natural toxins*

These chemicals occur as regular constituents of the food in question (e.g. lectins in kidney beans), or at increased levels as a response of the foodstuff to some sort of stress (e.g. glycoalkaloids in potatoes, an increased production of which can be stimulated when the tuber is exposed to light), and are inherent to the food raw material. There are also some instances of a processing regime potentially releasing a toxin from a non-toxic starting material (as occurs with cyanogenic glycosides in some canned stone fruits). The latter could be considered under the process-derived hazards category, but is essentially 'natural'. There are many types of natural toxins produced from many species of plants and the following examples serve to illustrate the importance of controlling the risk from these chemicals.

#### *Lectins*

Lectins occur in a wide variety of plants including beans of the *Phaseolus* genus (e.g. kidney beans and lima beans), broad beans, castor beans, soya beans, lentils, peas, field beans, peanuts, potatoes and cereals, as well as a range of non-food plants. In many cases the lectins have no or minimal toxic effect. Others are toxic to a greater or lesser extent, but in most cases normal cooking procedures eliminate this toxicity entirely, and consumption of moderate levels of most types of uncooked beans or peas will have no adverse effect. However, there are some specific exceptions, the most well known and significant of which (because of the way we consume them) is red kidney beans.

Raw kidney beans are significantly toxic due to the presence of lectins and must be cooked adequately before consumption. One form of wording suggested for the labelling of beans for sale is:

"After soaking overnight and throwing away the water, these beans should be boiled briskly for at least 10 minutes and then cooked until soft, otherwise they may cause stomach upsets. Never cook in a small casserole unless the beans have first been soaked and boiled in this way. Do not eat raw beans"

#### *Glycoalkaloids*

Potato glycoalkaloids are a good example of naturally occurring toxins that can and have caused problems when consumed in large quantities, but which we have learned to avoid without too much difficulty.

Potatoes contain two main glycoalkaloids, solanine and chaconine, with chaconine being the more toxic. Symptoms of acute poisoning can range from abdominal pain, vomiting and diarrhoea (similar to bacterial food poisoning) to confusion, fever, hallucination, paralysis, convulsions and occasionally death. There is an unofficial, but widely accepted safety limit of 200 mg glycoalkaloid/kg fresh potato. Levels of glycoalkaloids in

modern varieties are usually well below this value, but they can exceed the limit under certain circumstances. The associated bitterness that accompanies these increases means that the chances of ingesting a toxic dose are small unless the bitterness has been masked with other highly flavoured ingredients.

Thus, although the chances of someone eating potatoes with high levels of glycoalkaloids are small, the possibility does exist, and hence the food industry must take precautions to eliminate the risk as far as is possible. Glycoalkaloids can only be made by the living potato tissue, and therefore will be halted by cooking and any other process that kills the tissue. However, they are heat stable and therefore preformed toxin will remain after processing. Glycoalkaloid levels in potatoes are highest in the flowers and in the sprouts on the tubers. Within the mass of the tuber itself, they are concentrated in the outer 2mm, so that unpeeled potato products are a higher risk than flesh-only products. Levels vary from one variety to another, and are generally higher in early varieties than in main crop varieties. Smaller potatoes tend to have higher levels than large potatoes, largely as a consequence of the increased surface area/volume ratio.

Increased levels arise through various stress factors, such as pest and disease damage, drought, waterlogging, and extremes of temperature. During post-harvest handling, bruising, abrasion and other types of mechanical damage can all cause increases in levels, as can peeling (although the act of peeling will remove much of the glycoalkaloid content unless the peel is added back into the product). Light can also induce glycoalkaloid formation. Light also induces chlorophyll formation, causing the potatoes to turn green on the surface.

### *Oxalates*

Oxalic acid and oxalates are widely distributed in plant foods, highest levels being found in spinach (0.3-1.2%), rhubarb (0.2-1.3%), tea (0.3-2.0%) and cocoa (0.5-0.9%). Although there is no question that the ingestion of sufficient oxalic acid as crystals or in solution can be fatal, there is considerable debate as to whether serious food poisoning from oxalate is usually due to food.

The eating of rhubarb leaves has been a well-known cause of illness for centuries. Rhubarb leaves contain high amounts of oxalate. However, the levels of oxalate in rhubarb stalks are sufficiently high that consumption of normal levels of rhubarb stalks will result in at least as much oxalate intake as from small to moderate amounts of leaves.

There is some debate as to whether it is the oxalate in rhubarb leaves that is responsible for toxicity. Whatever the toxic principle, consumer perception is that rhubarb leaves are toxic, and hence consumer complaints about small fragments of leaves in canned rhubarb are well-known.

### *Cyanogenic glycosides*

Many fruits and other plant foods contain compounds that have the potential to release cyanide. These compounds are usually glycosides - i.e. they consist of a sugar molecule linked to a cyanide group, usually indirectly through another component. The release of cyanide from these compounds occurs by enzymic hydrolysis, usually when the plant tissue is crushed or otherwise disrupted (allowing the active enzyme to reach the substrate), but it can also occur in the digestive system after the food has been eaten. Some plants are toxic because of their high levels of these compounds. Other foods are considered safe to consume, despite their having moderate levels of cyanogenic glycosides. The most well-known of these compounds is amygdalin, a cyanogenic glycoside first identified in bitter almonds, which on hydrolysis by an enzyme complex known as emulsin yields glucose, benzaldehyde and hydrogen cyanide.

Cassava or manioc is a staple food for large numbers of the world's population. It is the world's seventh largest food crop in terms of production area. The toxic potential of cassava has been known for hundreds of years, and traditional methods of food preparation from cassava have been developed to reduce cyanide content. These include leaching out the linamarin precursor, washing in running water before cooking (bruising of the cassava

root during harvesting often results in considerable cyanide release), and boiling in uncovered pots so that the cyanide can evaporate. Fermentation also significantly reduces cyanogenic potential.

### *Trypsin inhibitors*

There are substances which have the ability to inhibit the proteolytic activity of certain enzymes which are found throughout the plant kingdom. The most common of these are chemicals which inhibit the activity of the enzyme trypsin which is important for digestion of proteins in the stomach. Examples of plants containing trypsin inhibitors are Lima beans and soya beans. Most of the inhibitor molecules are proteins which are inactivated by heating, hence cooking is a key step to increasing the nutritional value of foods containing these inhibitors.

### **ii) Natural and Environmental Contaminants**

All plants and animals during their lifetime will accumulate various chemicals from their environment. Some of these chemicals, if they are accumulated at high enough levels, might be of toxicological significance to us when we eat the food. Specific examples that are of concern are nitrates in leafy vegetables, heavy metals in various foods, and specific toxins in shellfish. In many cases, the best way to control levels of these unwanted substances is to control the environment in which the food is produced. However, this is generally a long-term control measure and more immediate steps have to be taken to protect human health. As many of the toxins can not be 'processed out', the short term controls are usually based around the setting of maximum permitted levels, and the removal from the supply chain of food that does not meet the required standard. These contaminants are divided below into 'natural' (of biological origin) and 'environmental', but they are linked in that the food plant or animal acquires them from its surroundings during its growth.

### **'Natural' Contaminants**

#### *Mycotoxins*

Mycotoxins are a group of chemically diverse naturally occurring substances produced by a range of filamentous fungi or moulds. They have toxic effects on both humans and animals ranging from acute toxicity and death, through reduced egg and milk production, lack of weight gain, impairment or suppression of immune function to tumour formation, cancers and other chronic diseases. The mycotoxins of greatest concern are produced by mould species from three main genera - *Aspergillus*, *Penicillium* and *Fusarium*. These are mainly storage moulds affecting commodities such as nuts, dried fruits and cereals. The moulds grow and produce toxins when commodities are stored incorrectly - usually at too high moisture levels. Specific mycotoxins of greatest concern are detailed below:

#### *- Aflatoxins*

Aflatoxins are produced mainly by some strains of *Aspergillus flavus* and most, if not all, strains of *A. parasiticus*. There are four main aflatoxins, B1, B2, G1 and G2, plus two additional ones that are significant, M1 and M2. The aflatoxins are potent liver toxins in most animals and carcinogens in some, with aflatoxin B1 being the most toxic and carcinogenic. Mould growth and aflatoxin production are greatest in warm temperatures and high humidity, particularly in tropical and sub-tropical regions, mainly on corn (maize), peanuts, cottonseed and tree nuts.

#### *- Ochratoxins*

Ochratoxins are a group of related compounds produced by *Aspergillus ochraceus* and related species, as well as *Penicillium verrucosum*. The main toxin in the group is Ochratoxin A, which causes liver damage in rats, dogs and pigs. Ochratoxins are also teratogenic to mice, rats and chicken embryos, and are now thought to be carcinogenic in humans.

#### - Patulin

Patulin is produced by numerous *Penicillium* and *Aspergillus* species and by *Byssoschlamys nivea*. However, the most common producer of patulin is *Penicillium expansum*, which occurs commonly in rotting apples, as a result of which patulin has frequently been found in commercial apple juice. Patulin is toxic to many biological systems, including bacteria, mammalian cell cultures, higher plants and animals. Its role in causing animal and human disease is unclear, but it is believed to be carcinogenic.

#### - Cyclopiazonic acid (CPA)

Cyclopiazonic acid (CPA) is produced by several moulds which occur on agricultural products or are used in some food fermentations. It also occurs naturally in infected corn (maize) and peanuts. It affects rats, dogs, pigs and chickens, where it may cause anorexia, weight loss, diarrhoea, pyrexia, dehydration and other symptoms. Organs affected include liver, spleen, kidneys, and pancreas. It has the ability to chelate metal ions such as calcium, magnesium and iron, which may be an important mechanism of toxicity.

#### - Zearalenone

Zearalenone (also known as F-2 toxin) is produced by several *Fusarium* species. It occurs naturally in high moisture corn (maize) in late autumn and winter, mainly from the growth of *F. culmorum* in Northern Europe and *F. graminearum* in North America. Production of this and other *Fusarium* toxins is favoured by high humidity and low temperatures, conditions which often occur in temperate regions during autumn harvest. It has been found in mouldy hay, high-moisture corn (maize), corn infected before harvest and pelleted feed rations, so it is an important contaminant of animal feed. The involvement of zearalenone in human disease is unconfirmed, but it is regarded as an endocrine disruptor and hence a potential hazard.

#### - Tricothecenes

The tricothecenes are a group of over 20 chemically related toxins produced by several *Fusarium* species. These include deoxynivalenol (DON), T-2 toxin, diacetoxyscirpenal, neosolaniol, nivalenol, diacetylnivalenol, HT-2 toxin and fusarenon X. The most commonly occurring of these is deoxynivalenol or DON, which causes vomiting in animals, hence its other name of vomitoxin. It may also be a teratogen and has been found in commodities such as corn (maize) and wheat as well as some processed food products.

#### - Fumonisin

The fumonisins are a group of compounds mainly produced by *Fusarium moniliforme* and *F. proliferatum*. They have been linked to several diseases, including liver cancer and oesophageal cancer in humans.

#### - Moniliformin

Moniliformin is so called because it was first thought to be produced by *F. moniliforme* isolated from corn (maize). However, it has since been shown to be produced mainly by other species of *Fusarium*. It has been shown to be highly toxic in experimental animals, causing rapid death without severe cellular damage.

#### - Other mycotoxins

Other mycotoxins include sterigmatocystin, reported in green coffee, mouldy wheat and the rind of some hard cheese, citrinin, penicillic acid, mycophenolic acid,  $\beta$ -nitropropionic acid, tremorgens (penitrem) and rubratoxin.

The Institute of Food Science and Technology website contains information statements on mycotoxins

- see [www.ifst.org/about\\_ifst/hotspot/29514/updated\\_Mycotoxins\\_Information\\_Statement](http://www.ifst.org/about_ifst/hotspot/29514/updated_Mycotoxins_Information_Statement)

#### Shellfish toxins

There are several types of shellfish poisoning including neurotoxic (NSP), diarrhoeic (DSP), paralytic (PSP), amnesic (ASP), and ciguatera fish poisoning (CFP). Shellfish toxins are not produced by the shellfish themselves, but are accumulated through the ingestion of planktonic dinoflagellates in the diet of the shellfish. The term shellfish generally refers to both marine crustaceans (lobsters, crab, shrimp etc), and molluscs.

However, it is the bivalve molluscs – oysters, mussels, clams and scallops - which accumulate these algae by filter feeding, that are the major areas of concern. See Lawley et al. (2008) for a review of these different types.

Paralytic shellfish poisoning is a global problem which has increased dramatically since the 1970s. The most significant toxins in PSP are saxitoxin and its derivatives, though the exact composition differs amongst algal species and amongst regions of occurrence. Generally the population density of such algae is not high enough to cause problems, but on occasion when environmental conditions (nutrients, temperature, sunlight etc) are favourable, population explosions called 'algal blooms' occur. Problems can arise if the algal bloom is of a species which produces toxins, such as the *Alexandrium* genus. Such toxins can then accumulate within the flesh of the filter-feeding bivalve at levels which cause disorder in humans after consumption. The toxins can persist within shellfish at dangerous levels for weeks or months after the algae are no longer present in the waters. Seafood containing saxitoxin looks and tastes normal, and cooking or steaming only partially destroys toxins. Therefore one of the most effective methods in preventing outbreaks of PSP is the detection of the toxins before the shellfish are harvested.

Amnesic shellfish poisoning is also caused by algae in the diet of shellfish; domoic acid is the principal toxin and is produced by various species, but the diatom *Pseudo-nitzschia* is the primary source. It can work its way up through the food chain, so illness can result from consumption of other contaminated seafood. As with PSP, decontamination of foodstuffs is not effective and detection of areas where the contamination exists is the best method of preventing problems.

Ciguatera fish poisoning is an intoxication caused by the consumption of coral reef fish which feed on certain marine plankton which contain specific toxins. It is one of the commonest marine food poisonings worldwide and a significant health problem with as many as 50,000 cases occurring each year. Toxins accumulate as they move up the food chain so that the larger carnivorous fish are more toxic. Symptoms are extremely varied and include gastrointestinal and cardiovascular problems, though most patients recover. The toxins are not easy to detect so the only effective control option is to avoid consumption of susceptible fish species.

## **'Environmental' contaminants**

### *Dioxins/Polychlorinated biphenyls (PCBs)*

PCBs and dioxins are persistent contaminants with a wide range of chemical structures. They have been found in soil, water, sediment, plants and animal tissue in all parts of the world. Dioxins and PCBs are heterocyclic organic molecules, with PCBs being chlorinated. They have long half-lives in the environment and many have been reported to have toxicological effects in humans. PCBs and dioxins are man-made chemicals used by industry and their release to the environment is generally through by-products of fires and by some manufacturing processes. Their widespread environmental occurrence means that PCBs and dioxins are present in virtually all foods, which is the main route to human exposure. The highest concentrations are in fatty foods such as oily fish and the main sources of dioxins in the diet are meat and milk. Levels accumulate as they move through the food chain.

Control options are based on prohibiting the use of dioxins and PCBs by industry and hence their release into the environment and the EU put into force a ban on the use of most PCBs from 1978. Legislative limits have been imposed within the EU for many foods (Regulation EC 1881/2006) as have methods for sampling (EC 1883/2006). No limits exist in the US although the FDA considers all detectable levels to be of concern.

### *Polycyclic aromatic hydrocarbons*

Polycyclic aromatic hydrocarbons (PAHs) are a group of compounds comprising two or more fused aromatic rings. Many individual PAHs exist, the most simple of which is naphthalene. A variety of toxic properties have

been related to PAH exposure, including the capacity to produce genotoxic and carcinogenic effects in mammals.

PAHs are found in petroleum and coal, and can also be formed by the incomplete combustion of these and other organic materials. These compounds have been detected in air, water, soil and foods. Foods may become contaminated through direct environmental exposure, migration from packaging material or during thermal processing of food, e.g., baking, grilling, frying and smoking.

The occurrence of PAHs in fruit, vegetables and cereals is primarily due to soil and air exposure. Although levels detected in foods of animal origin tend to be low, high levels have been recorded in smoked meats and animals farmed on contaminated land. Shellfish can accumulate PAHs from oils spilt by grounded tankers or from waste oils which have been incorrectly disposed of. PAHs can also be formed during the heating and drying processes which allow combustion products to come into contact with the food substance. Direct fire-drying and heating processes used during the production of food oils can result in high levels of PAHs.

The complexity and number of individual PAH compounds means that it is not easy to produce specific limits for regulation of levels. Benzo (a)pyrene has been used as a marker for PAH levels and limits for this have been set by the European Commission (EC 1881/2006) for a range of foods, although there is currently discussion about widening this to include other marker compounds.

Specific foods of concern are fish which are farmed in oil contaminated waters, fats and oils including cocoa butter, and smoked foods. Refining processes are generally ineffective in eliminating PAHs from foods so the main control measure is to limit their production during processing and to screen out foods known to contain high levels.

### *Heavy metals*

Heavy metals are those with a high atomic mass, including, for example, mercury, cadmium, arsenic and lead, although other metals (e.g. tin) may also be included within this category of contaminant. They are natural components which originate from the earth's crust and are found all over the world. They are toxic in low amounts and have been recognised as a health hazard for many years. There are other routes for metal contamination of products such as migration from packaging (e.g. antimony from plastic bottles, and tin in canned food).

Metals can occur in a variety of foodstuffs of plant and animal origin. Mostly, they arise indirectly in foodstuffs from the environment – e.g. they are in soil that the crop is grown in, or on the grass that a cow is eating or in the water in which a fish is living. As such, once they become incorporated into the food they cannot be removed. There is a risk to crops and animals themselves from metals in the environment (e.g. they can kill plants and reduce yields) and to humans from eating crop and livestock products. Metals which can be particularly harmful to animals and man include lead, cadmium, arsenic, mercury, copper, selenium and molybdenum. These elements can accumulate in primary products that are otherwise growing satisfactorily, but still affect animals and man.

Of particular relevance to crop products as food raw materials are lead and cadmium. Lead is a widespread environmental pollutant, deriving from such human activities as lead mining, smelting and processing, and burning of fossil fuels. The main route of crop contamination is via uptake from the soil. Soil contamination with both lead and cadmium is primarily from aerial deposition.

Maximum levels for heavy metal contaminants have been established in many countries so it is important to be aware of the legislative limits which apply if exporting. Each metal has a specific limit which is food-type-dependent and is a reflection on both the occurrence of the metal in that food and its toxicological effect. Control of raw materials is the only mechanism for ensuring that levels do not become unsafe. A particular problem has been lead and cadmium in cereals and close monitoring of levels in flour mills and maltings has been necessary

to ensure that limits are not exceeded. The legislation in this area is constantly changing so food manufacturers need to keep abreast of proposed new limits and use horizon scanning methods to maintain vigilance for problems.

### *Nitrates*

In general nitrates in agriculture are considered more of a hazard to the environment and water than in foods. However, nitrate intake from water and food has received considerable publicity because of its role in methaemoglobinaemia in infants and its reported implication in various types of cancer. Methaemoglobinaemia is caused by nitrate being reduced, under the conditions found in the infant stomach, to nitrite, which then combines with haemoglobin in the bloodstream. Methaemoglobinaemia, sometimes known as the "blue baby syndrome", can be fatal.

The possible involvement of nitrate in cancer is via its role in the generation of nitrosamines. Nitrosamines are known to be very potent carcinogens and are produced by the reaction of nitrate, when reduced to nitrite, with certain nitrogenous compounds found in proteinaceous substrates. Whilst nitrosamines can be formed in the body, the link between high nitrate exposure and the incidence of cancer is often not clear.

Nitrates in food might, therefore, have some adverse health effect, but the levels in most crops are not generally considered a food safety hazard. However, green leafy vegetables usually contain higher levels of nitrate than most other foods, and maximum levels have been set in the EU and by Codex for nitrates in spinach and fresh lettuce. There are a number of factors which affect the levels of nitrates in these crops, including nitrate availability in the soil, seasonal variations, applications of nitrate fertilisers shortly before harvest and environmental influences.

### *Fluoride*

Fluoride can be found dissolved in waters at high levels in certain parts of the world, and in some cases is above the WHO maximum limit. There is some controversy as to whether the presence of fluoride is a benefit or a threat to human health. The benefits for protection of dental health are well known and in fact fluoride is routinely added to water and/or toothpaste in many countries. There are also reports that fluoride can be a hazard to human health with links to cancer, bone health and endocrine disruption having been cited. There is no doubt that the debate regarding fluoridation of public water supplies will continue given the emotion regarding mass medication. Information regarding the hazardous effects of long term ingestion of fluorides is required to determine whether this policy is acceptable.

### **iii) Process-derived contaminants**

The production of toxic chemicals in foodstuffs through processing is a recently discovered phenomenon, although historically these chemicals will have always been present. The first three examples below serve to show how unexpected contaminants may arise. In addition, the contamination of food with chemicals from packaging, pesticide and veterinary medicine applications could also loosely be described as process-derived.

### *Acrylamide*

In 2002, Swedish scientists unexpectedly discovered acrylamide in food when they were carrying out a study into occupational acrylamide exposure. As part of the study, people who were not believed to have been exposed to acrylamide were included as controls and were also found to have significant acrylamide in their blood; further research determined that this unknown source was food. Subsequent research has now revealed that acrylamide is formed in food by traditional cooking methods such as baking, frying and roasting (i.e. high temperatures). It is formed at highest levels in starch containing foods and varies widely among different products and between production batches of the same foods. Examples of foods most at risk are potato products such as crisps and chips, coffee, savoury snacks such as cracker type biscuits, and bread and other

cereal products. For a more extensive review of acrylamide please see <http://iufost.org/iufost-scientific-information-bulletins-sib>

Considerable research has been carried out to understand the mechanism of acrylamide formation and a major international project took place bringing together scientists from all over the world (HEATOX). The output from this project formed the basis of industry guidelines aimed at minimising the formation of acrylamide during food processing (the CIAA 'Toolbox': [http://www.ciaa.be/asp/documents/brochures\\_form.asp?doc\\_id=65](http://www.ciaa.be/asp/documents/brochures_form.asp?doc_id=65)). This guide recommends measures such as avoiding sources of asparagine (such as certain potato varieties), avoiding long cooking times and high cooking temperatures, and replacing ammonium bicarbonate as a processing aid in bread.

Although acrylamide is a known carcinogen, it is still unclear whether it has any major effect on health when consumed in food. It is certainly known that at high levels it has neurotoxic and genotoxic effects though these are unlikely at levels found in most foods. The Joint FAO/WHO Expert Committee on Food Additives (JECFA) has recommended that the food industry should use the guidelines in the CIAA 'Toolbox' to reduce acrylamide levels to as low as possible in critical food groups. Although there is not yet specific legislation, or maximum levels for acrylamide, there is a comprehensive programme on monitoring in many countries. It is possible that in future limits could be set for food manufacturers so the food industry is keen to demonstrate that it is doing all it can to reduce levels where possible through good manufacturing practice.

There is more information at [www.food.gov.uk/safereating/chemsafe/acrylamide\\_branch](http://www.food.gov.uk/safereating/chemsafe/acrylamide_branch)

### *Chloropropanols*

Chloropropanols are a group of chemical contaminants, the most notable of which is 3-monochloropropane-1,2-diol (3-MCPD). 3-MCPD can occur in foods and food ingredients at low levels as a result of processing, migration from packaging materials during storage, or domestic cooking. It has been found in a variety of foods, such as cooked/cured meats and fish, cheese, bread and toast, malt extracts and baked products, as well as in teabag paper, tissue and sausage casings. A major area of concern is its occurrence in food following the reaction between hydrochloric acid and lipids, particularly in foods processed at high temperatures such as soy sauce. In laboratory animal studies it has been shown that 3-MCPD is a carcinogen; it was originally classified as a genotoxic carcinogen, but more recent studies suggest that there is a lack of evidence of in vivo genotoxicity. However, the issue with 3-MCPD has meant that industry has looked to use enzymic methods of producing HVP rather than acid hydrolysis. Control of processing conditions and selection of ingredients is the main strategy being used by industry to control levels of chloropropanols. The level of 3-MCPD in the EU is prescribed by EC 1881/2006 though for other chloropropanols there are no limits and manufacturers are requested to reduce levels as far as is technically possible.

### *Furans*

Furan is a colourless, volatile liquid used in some chemical manufacturing industries, which was occasionally found in foods. Recently, it has been discovered that furan is formed in some foods more commonly than previously thought. This discovery is probably a result of our ability to detect compounds at exceedingly low levels rather than a change in the presence of furan. It is believed that furan forms in food during traditional heat treatment techniques, such as cooking, bottling, and canning. Furan has been found in such canned or bottled foods as soups, sauces, beans, pasta meals, and baby foods.

### *Packaging migrants*

There is a risk with any packaging material that its components may be transferred in some way to the food that it is surrounding. In most cases, the level of transfer is extremely slight and the components transferred are

innocuous. However, there are instances where a realistic hazard exists and must be controlled. There are no official internationally agreed guidelines, but in the EU there is a general requirement that food packaging components must not be transferred into food during its normal shelf-life to the detriment of the food (i.e. to pose a health risk, or to adversely affect the quality of the food - its flavour, texture or appearance).

Transfer of monomers and additives such as plasticizers in plastic packaging materials are the major area of concern. In the EU, there is a list of approved monomers and of additives that can be used in food contact plastic materials (this covers all contact with food, not just packaging materials) and also limits for the migration of these constituents into food. The general limit for containers and sealing devices is 60mg per kg of food. For other contact materials it is 10mg/dm<sup>2</sup>. To determine whether a particular plastic formulation meets these criteria, there are four model simulants that are used in laboratory trials to assess the plastic's properties. These are: distilled water; a 3% aqueous solution of acetic acid; 10% ethanol in water solution (or greater, if the alcoholic beverage in question has a higher alcohol content); and rectified olive oil. The regulations specify which simulants should be used for each category of food. In general, there are no simulants listed for dried foods, which can be considered to not take up plastics constituents from contact materials.

#### *Tin*

Tin can be considered to be a specific type of packaging-derived contaminant. Although there is no evidence that excess tin intake has any long-term health effects, some studies have shown that intake of high concentrations (above about 250ppm) may cause short-term gastrointestinal problems. For most foods, this is of no significance, but for foods packed in cans with some unlacquered tinfoil, high levels can sometimes occur. Tin dissolution in unlacquered tinfoil cans is essential in that it confers electrochemical protection to the iron, which makes up the structural component of the can and so maintains the can's integrity. Without it, the can would quickly become corroded by the contents of the can; this could cause serious discoloration and off-flavours in the product and swelling of the can. Tin is also involved in maintaining product quality (it helps prevent undesirable colour changes amongst other things, by mopping up any residual oxygen left in the headspace), so there is an advantage in some products of having some exposed (i.e. unlacquered) tinfoil. As tin dissolution tends to be accelerated by oxygen, for products where exposed tin is considered to be beneficial, the base, lid and ends of the can may be lacquered, with the rest being unlacquered.

Tin pick-up is normally relatively slow and does not give rise to excessive levels in the product within its shelf-life. However, certain natural variations within the product can cause problems.

#### ***iv) Deliberately added contaminants***

There is no limit to what chemical contaminants might be deliberately added to foods during manufacture in order to cause harm to the consumer. In most cases, however, the aim is not to cause harm, but to defraud for financial gain. However, potential harm can still result, as evidenced from two of the examples given below.

#### *Illegal or unauthorised dyes*

The Sudan I-IV group of chemicals are synthetic azo dyes which have been historically used in industry to colour products such as shoe polish, automotive paints and petroleum derivatives. They are not permitted food colours.

During the summer of 2003 it became apparent that chilli powder and related products in the European market, and originating from India, were contaminated with Sudan I-IV at levels between 2.8 and 3500 mg/kg. Although the Sudan dyes were deemed to be toxic, the levels at which they were found were probably not a major health concern because of the very low concentrations in which they were detected in final products. However, such dyes are not permitted for food use and were being added to the chilli powder in order to make it appear to be of better quality than it actually was. The chilli powder was incorporated into various sauces, which were themselves used as ingredients in a range of ready meals. With the significant dilution effect of this, analysing the final food for Sudan dyes became a problem, as the levels involved were now very small. A major

traceability program had to be launched to identify and remove all affected products. This involved the withdrawal of over 1000 products, at a very significant cost to the food industry. However, with laboratories now routinely testing for the presence of the Sudan dyes, the contamination spread progressively to a wide range of other dyes in order to avoid detection, including Para Red, Rhodamine B, Orange II, Red G, Butter Yellow and Metanil Yellow. As well as many dyes which were not permitted for any food use, these new colours included some, such as Bixin, which were permitted in some foods, but not in the spices to which they were being added.

### *Melamine*

Melamine is an industrial chemical found in plastics. It can be combined with formaldehyde to produce melamine resin, a very durable thermosetting plastic used in Formica, and melamine foam, a polymeric cleaning product. The end products include countertops, dry erase boards, fabrics, glues, house wares, guitar saddles, guitar nuts, and flame retardants. Melamine is one of the major components in Pigment Yellow 150, a colorant in inks and plastics. It is also used in the manufacture of plasticizers for concrete.

In 2007 it was discovered in the US that melamine had been fraudulently added to wheat gluten and rice protein from China, which was subsequently used in pet foods. This was a widespread problem and resulted in a petfood recall initiated by manufacturers who had found that their products had been contaminated. Further vegetable protein imported from China was later implicated. It was claimed that some of the animals that had eaten the contaminated food had become ill, although melamine was not previously believed to have been significantly toxic at low doses.

Melamine has no nutritional value but because it is high in nitrogen (66% by mass), its addition to food makes it appear to have more protein than it actually does and so meet required contractual obligations. Standard tests such as the Kjeldahl and Dumas tests estimate protein levels by measuring the nitrogen content, so values obtained can be increased by adding nitrogen-rich compounds such as melamine.

By early 2006, melamine production in mainland China was reported to be in "serious surplus". In September 2008, it was discovered that melamine was present in infant milk powder produced in China. Six infants are believed to have died as a result, and over 300,000 were reported to have been made ill. Traces of melamine were subsequently found in other dairy-based products in the region. Melamine has also been detected in other products, including eggs, originating in China. Actions taken in 2008 by the Government of China have reduced the practice of adulteration, with the goal of eliminating it. Court trials began in December 2008 for six people linked to the scandal and ended in January 2009 with those convicted being sentenced to death and executed.

Melamine is described as being "Harmful if swallowed, inhaled or absorbed through the skin. Chronic exposure may cause cancer or reproductive damage. Eye, skin and respiratory irritant." However, the short-term lethal dose is on a par with common table salt with an LD<sub>50</sub> of more than 3 grams per kilogram of bodyweight. However, it is thought that when melamine and cyanuric acid are absorbed together into the bloodstream, they concentrate and interact in the urine-filled microtubules in the kidneys, then crystallize and form large numbers of round, yellow crystals, which block and damage the renal cells that line the tubes, causing the kidneys to malfunction. Toxicology studies conducted after recalls of contaminated pet food concluded that the combination of melamine and cyanuric acid in the diet does lead to acute renal failure in cats and rats.

The European Union set a standard for acceptable human consumption of melamine at 0.5 milligrams per kg of body mass (reduced to 0.2 mg per kg in April 2010). Member States of the European Union are required under Commission Decision 2008/757/EC to ensure that all composite products containing at least 15% of milk product, originating from China, are systematically tested before import into the Community and that all such products which are shown to contain melamine in excess of 2.5 mg/kg are immediately destroyed.

*More recently there has been concern about the migration of melamine from food contact materials. In addition, there have been reports of melamine residues as a result of the use of cyromazine, an insecticide derived from melamine.*

### *Spanish Toxic Oil Syndrome*

This incident started as a deliberate act of fraudulent adulteration. A large volume of rapeseed oil had been treated with aniline to downgrade it for industrial use. Some unscrupulous traders decided to refine, decolourise and deodorise this oil, mix it with other oils, package and label it as olive oil, and then illegally introduced it on to the Spanish market. Unfortunately, the oil contained a highly toxic substance formed in a reaction between the aniline and fatty acids in the oil, resulting in the deaths of up to 600 people and over 20,000 people affected by health problems. A full account of the incident can be found in Wood et al. (1994).

## **v) Pesticides and veterinary residues**

### *Pesticides*

Pesticides include chemical and biological products specifically designed to control pests, weeds and diseases, particularly in the production of food. These include insecticides, fungicides, herbicides, rodenticides and molluscicides.

Pesticides are licensed for use against specific target organisms, and their use and application are strictly regulated to control the risks to the operator involved in applying them, and the surrounding environment, and to prevent significant residues being left in or on the food. Regulations include restrictions on the target organisms the chemical may be used against, the crops on which it may be used, the concentrations that may be applied and the number of applications permitted. There are strict limits on the levels of pesticide residues allowed in food and this is closely monitored by regulatory authorities worldwide.

Pesticides can be classified by target organism, chemical structure, and physical state. They can be classed as inorganic, synthetic, or biological (biopesticides). Biopesticides include microbial pesticides and biochemical pesticides. Plant-derived pesticides include the pyrethroids, rotenoids, and nicotinoids.

Many pesticides can be grouped into chemical families. The main insecticide families include organochlorines, organophosphates and carbamates. These operate by disrupting the sodium/potassium balance of the nerve fibre, forcing the nerve to transmit continuously. Toxicities of these chemicals vary greatly, but they have been largely phased out because of their persistence and potential to bioaccumulate. The organochlorines have been largely replaced by the organophosphates and carbamates. Both of these operate through inhibiting the enzyme acetylcholinesterase, allowing acetylcholine to transfer nerve impulses indefinitely and causing a variety of symptoms such as weakness or paralysis. However, organophosphates are quite toxic to vertebrates, and they have in some cases been replaced by the less toxic carbamates. Prominent families of herbicides include phenoxy and benzoic acid herbicides (e.g. 2,4-D), triazines (e.g. atrazine), ureas (e.g. diuron), and chloroacetanilides (e.g. alachlor). Phenoxy compounds are designed as selective weedkillers to kill broadleaved weeds rather than grasses. The phenoxy and benzoic acid herbicides function in a similar way to plant growth hormones, and cause cells to grow without normal cell division, affecting the plant's nutrient transport system. Triazines interfere with photosynthesis. Many commonly used pesticides such as glyphosate are not included in these families.

In the UK, there is a national monitoring programme overseen by the Pesticides Residues Committee, which measures the levels of pesticide residues in a wide range of foods, to check that they are within legal and safe limits. The limits apply both to food produced both in the UK and that imported from elsewhere. A number of different statutory bodies are involved in regulating which pesticides may be used and how. There are particularly strict limits on the levels of pesticides allowed in infant formulae and manufactured baby foods.

## *Veterinary residues*

The use of medicines used to treat animals raised for food is regulated in a similar manner to that for pesticides used on food crops. There are a wide variety of chemicals for different uses, including:

- Antimicrobials such as sulphadiazine, enrofloxacin, ciprofloxacin, chlortetracycline, amoxicillin and oxytetracycline used to control bacterial diseases
- Pain-killers and anti-inflammatory medicines such as NSAIDs, including ibuprofen and phenylbutazone
- Dips to control external parasites, including organochlorine or organophosphorus insecticides (see above)
- Wormers to control internal parasites, such as ivermectin
- Coccidiostats to control protozoal diseases, particularly in poultry, such as nicarbazin
- Steroids such as boldenone

## **HOW ARE MAXIMUM LIMITS SET?**

As can be seen from the above examples, there are a variety of chemical hazards that could enter food. Some of these are unpredictable (e.g. those that are deliberately added), but most can be, and are, controlled. The main route for this is Good Manufacturing Practice and monitoring of environmental conditions and the quality of incoming ingredients and raw materials. However, part of the control at a national or international level may be in the form of the setting of maximum legal limits. What these limits are and how they are determined may vary from one part of the world to another, depending on specific circumstances, but in general three main areas are taken into consideration.

- Toxicity evidence: How toxic is the contaminant believed to be and how sound is the evidence for this belief?
- Good Manufacturing Practice: What is technologically achievable and how costly is it?
- Analytical capability: What are the limits of detection or quantification?

In all instances, safety is the primary concern, and maximum limits are usually set at about 100 times below the level at which a toxic effect is noted. However, maximum limits to control contaminant levels are only meaningful if they can be monitored by analysis (see below). In addition, even if a contaminant is only mildly toxic, it may be possible to reduce levels to well below the toxicity/100 threshold by Good Manufacturing Practice. This approach is taken with many pesticides, where good agricultural practice (including correct application regimes and suitable intervals between application and harvesting) will result in no remaining residues. Maximum levels are therefore set at the 'limit of detection' or 'limit of quantification'.

The maximum limit for a chemical will often be different for different food types - and there may well be a limited number of foods for which a maximum limit is set. It may be unnecessary to specify a maximum limit in cases where the chemical would not be expected to be found in the food. In contrast, it may very difficult to limit a chemical in some food types, and so higher limits are set, based on what is realistically achievable (bearing in mind that safety is still the over-riding factor). Nitrates provide a good example of this. In Europe, high nitrate levels are only a significant issue in leafy vegetables (spinach and lettuce), and it is these products for which limits have been set. However, levels will vary depending on growing conditions and season, and so different maxima have been set for different situations. These are typically in the range 2000-3000 ppm.

The Codex Alimentarius Commission (Codex, 2009) ([www.codexalimentarius.net/download/standards/17/CXS\\_193e.pdf](http://www.codexalimentarius.net/download/standards/17/CXS_193e.pdf)) has set maximum and guideline levels for the following chemical hazards that are an inherent risk in certain foods. The figures given are typical but may vary in some cases depending on product type and whether consumed raw or further processed. In particular the levels set for foods for infants and young children are often much lower than those for the general population.

The figures given are merely for illustration; for any individual contaminant in a particular foodstuff, the original text should be consulted.

#### Mycotoxins

*Aflatoxins* (15µg/kg in peanuts; 0.5µg/kg M1 in milk)

*Patulin* (50µg/kg in apple juice)

#### Heavy metals

*Arsenic* (typically 0.1mg/kg)

*Cadmium* (typically 0.05-0.2mg/kg)

*Lead* (typically 0.1-1mg/kg)

*Mercury* (0.001mg/kg in natural mineral water; 0.1mg/kg in food grade salt)

*Methylmercury* (0.5mg/kg in fish - 1mg/kg in predatory fish)

*Tin* (150mg/kg in canned beverages; 250mg/kg in canned fruit and vegetables)

Radionuclides (1-10,000 Bq/kg, depending on individual radionuclide - generally 10-fold lower in infant foods)

#### Others

*Plastic monomers* (typically 60mg/kg of food or 10mg/dm<sup>2</sup> of package surface)

*Acrylonitrile* (0.02mg/kg)

*Vinylchloride monomer* (0.01mg/kg)

As a comparison, in the EU, the following have been set (see <http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=CONSLEG:2006R1881:20090701:EN:PDF>)

Nitrates (typically 2000-4500 mg/kg)

#### Mycotoxins

*Aflatoxins* (typically 4-15µg/kg in total; 0.05µg/kg M1 in milk)

*Ochratoxin A* (typically 2-10 µg/kg)

*Patulin* (50 µg/kg in apple juice; 25 µg/kg in solid apple products)

*Deoxynivalenol* (typically 500-1750µg/kg)

*Zearalenone* (typically 50-200 µg/kg)

*Fumonisin* (200-2000 µg/kg)

## Metals

*Lead* (0.02-1.5mg/kg)

*Cadmium* (0.05-1mg/kg)

*Mercury* (0.5-1mg/kg)

*Tin* (100mg/kg in canned beverages; 200mg/kg in other canned foods)

3-MCPD (20 µg/kg in HVP and soy sauce)

Dioxins and PCBs (usually picogram levels per gram of fat)

## Polycyclic aromatic hydrocarbons

*Benzo[a]pyrene* (1-10 µg/kg)

In addition, there are limits for many components of plastic packaging materials.

## ANALYTICAL APPROACHES

As mentioned above, robust analytical methods are essential if the occurrence of chemical hazards in food is to be monitored and controlled. The type of method used will depend primarily on the chemical concerned, as well as the levels likely to be present and the food matrix.

Analysts have at their disposal a wider range of analytical techniques than ever before, and the sophistication of many of these would have been almost unimaginable just a few decades ago. This means that the analyst can now measure lower levels of a wide range of compounds in many different sample types. But it also means that the analyst has to be careful about the approach taken. Getting the right result requires the correct approach – and this includes using the right method of analysis.

A method of analysis typically involves several stages, and can involve a combination of techniques. Following sample receipt and the associated administrative requirements, the sample may need to be pre-treated (e.g. ground or blended), before the analyte is extracted (e.g. by solvent extraction). This latter stage may involve an initial crude extraction, followed by a purification stage (e.g. on an affinity chromatography column). Only then can the analyte be measured. Following analysis, the results have to be correctly interpreted and reported. In many cases, the extraction and/or purification stages are combined with the actual analytical stages, as happens with liquid or gas chromatography techniques linked with mass spectrometry.

Given the breadth of chemical hazards that might be present in food, the variability in their nature, and the many different types of food matrices, it is impossible to describe in any detail the types of analytical techniques that could be used. Some of the many generic techniques available are described in Jones (2005).

In some cases it is possible to analyse many related chemicals in one sweep - screening. This is possible for a wide range of pesticides, for example, and for some of the illegal dyes. In many other cases targeted analysis is required, i.e. a specific procedure for an individual chemical.

When looking to analyse any chemical hazard in food (or indeed any chemical), there are a few basic points to note:

- Purpose of the analysis - it is important for those commissioning the analysis to be clear about the reasons for the analysis and how the result is to be used

- Sampling - samples should be representative of the product being analysed. Once taken, the samples should be handled, stored and prepared properly, so that they are not altered in any way that would affect the analysis
- Method suitability - the analytical method has to be fit for purpose - even if a method has been devised for the specific hazard in question, it may have to be adapted or modified for a particular foodstuff or to take into account other chemicals present that may interfere with the analysis
- Validation - following on from the above, the method, if it is new or modified, will have to be validated - i.e. tested to show that it works
- Quality control and standardisation - although the method itself has been shown to be fit for purpose, there needs to be evidence that it can produce consistent results over a period of time and in the hands of different analysts.
- Measurement uncertainty - no method will ever give exactly the right result all the time - in fact, in any analysis the result obtained will only ever be an approximation (adequately close, if the method is suitable) to the 'true' answer. It is important to understand where the potential sources of error might arise, and which are the most significant, when interpreting the results.

## PREVENTING CHEMICAL SAFETY BREAKDOWNS IN THE FOOD CHAIN

### HACCP

The most effective and efficient way of minimising the chances of chemical (and any other) safety issues arising in the food chain is through the use of HACCP (Hazard Analysis and Critical Control Points) systems. In the EU, it is a requirement throughout the industry to use HACCP-based systems to ensure food safety. In essence this means identifying which chemicals may be a problem in a particular food, and the measures to limit (or eliminate) their occurrence or remove them. It is then a case of monitoring and documenting what is being done and sampling the final product from time to time to ensure that the protocol is working.

The HACCP approach is based on seven internationally recognized simple principles:

1. Conduct a hazard analysis: prepare a flow diagram of the steps in the process; identify and list the hazards associated with the process and specify how they are going to be controlled.
2. Determine the critical control points (CCPs), i.e. those stages at which hazard control is essential for the production of a safe end-product.
3. Establish critical limits for each hazard at each CCP, i.e. the levels for each individual hazard that must not be exceeded if a safe product is going to be achieved. This may, for example, be a requirement to boil red kidney beans vigorously for 10 minutes in order to eliminate haemagglutinin (lectin) activity.
4. Set up a system to monitor control of each CCP by scheduled testing and observations, to ensure that the hazard remains within critical limits.
5. Establish what corrective action needs to be taken if monitoring indicates that a particular CCP is not under control *or is moving out of control*, i.e. is going beyond critical limits – this means stopping something going wrong before it happens, if at all possible.
6. Set up procedures to make sure that the overall HACCP plan is working as desired; this may include some end-product testing and a regular review of the system.
7. Establish thorough documentation of the system, process and procedures, and of all measurements taken relating to the monitoring of the process.

## *Surveillance*

General monitoring of levels of specific chemicals in foods is part of the HACCP process, but in addition to this there are general government-initiated surveillance programmes for specific chemicals. These may be long-term studies to determine trends in levels of well-known hazards in the environment, such as dioxins or nitrates, or may be as a result of a specific problems that arise. As an example, both the UK's Food Standards Agency (FSA) and the EU's European Food Safety Authority (EFSA) publish reports of surveillance exercises. In addition, there are also systems in place to inform the industry of specific incidents as they arise.

The latest EFSA Annual Report on the Rapid Alert system is available at [http://ec.europa.eu/food/food//rapidalert/report2008\\_en.pdf](http://ec.europa.eu/food/food//rapidalert/report2008_en.pdf)

The FSA's Annual Report of Incidents for 2009 is available at <http://www.food.gov.uk/multimedia/pdfs/incidents09.pdf>

## *Traceability*

Maintaining adequate traceability in the food supply chain is a prerequisite to controlling the hazards which may be present in many food ingredients. This is a mandatory requirement in many countries and should include robust supplier assurance programmes as well as full records of all transactions as food is traded, processed and placed on sale. In the event of a recall due to the identification of a food hazard it will be necessary to identify through the records all possible products implicated and to remove them from sale and/or consumption.

## **FURTHER READING**

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