ISSN 1671-4083 Acta Pharmacol Sin 中国药理学报 2002 Mar; 23 (3): 193-202
http://www.chinaphar.com

Review

Potential food contaminants and associated health risks

Sharda Shah PESHIN, Shyam Bala LALL, Suresh Kumar GUPTA[1] (*National Poisons Information Centre, Department of Pharmacology, All India Institute of Medical Sciences, Ansari Nagar, New Delhi-110029, India*)

KEY WORDS food contamination; organophosphorus compounds; oils; aromatic hydrocarbons; heavy ions; microbiology; fungi

ABSTRACT

The potential toxicants in food are derived from natural or industrial sources. Compounds like lectins and glycoalkaloids that are toxic to man are naturally present in some vegetables like potatoes or legumes. A wide variety of marine toxins mostly produced by dinoflagellates occuring secondarily in molluscs and mussels are usually ingested by human beings causing poisoning. On the other hand, toxic compounds find their way into food during manufacture, storage, or transportation. These include largely the industrial contaminants, persistent organic pollutants (POP), pesticides, heavy metals, and toxins of fungal and bacterial origin. Further, toxic compounds like higher alcohols may be produced as byproducts during processing. Migration of compounds from packaging materials into packaged food like contamination with lead from solder in certain metal cans is well known. Additives (emulsifiers, preservatives, and antioxidants) could also influence the quality of foods. Solvent residues may find their way into food as a result of their use in extraction processes like the use of trichloroethylene and methylene chloride in decaffeination of coffee. In addition, poor hygiene, storage, and preparation may also lead to food contamination by various microbes and ova or cysts of nematodes. The problem of food contamination can be overcome to a great extent by regular surveillance and monitoring programmes and strict implementation of food and adulteration act. In the present review some of these aspects of food contamination have been discussed in detail.

[1] Correspondence to Dr Suresh Kumar GUPTA.
Phn 91-11-685-9391. E-mail skgup@hotmail.com
Received 2001-08-01 Accepted 2001-12-29

INTRODUCTION

There is a growing awareness that chemical and biological contaminants in foodstuffs may play an etiological role in various human diseases. The potential toxicants of concern to consumers are intentional additives (adulterants) or unintentional contaminants. Adulteration involves deliberate addition of undesirable, cheap agent for disguising inferior commodities and earning undue profits while contamination refers to accidental incorporation of toxicants during production, processing, packaging, or storage. A number of food contamination episodes resulting in great morbidity and mortality have occurred in the past. Epidemics of ergotism have occurred due to ingestion of bread contaminated with the fungus *Claviceps purpurea* over the centuries[1]. Inadvertent use of wheat seeds treated with hexachlorobenzene resulted in porphyria cutanea tarda in Turkey in 1956[2]. Epping Jaundice, an outbreak of toxic hepatitis in England was due to accidental contamination of a sac of flour with methylene dianiline during transport to a bakery[3]. In Japan, in 1968 contamination of rice oil with polychlorinated biphenyls (PCB) and polychlorinated dibenzofurans (PCDF) led to an illness called Yusho disease. A similar epidemic referred to as Yu-Cheng followed in Taiwan in 1979[4]. Another toxic oil syndrome characterised by multisystem disorder affected a large number of people in Spain in 1981 after consuming rape seed oil contaminated with 2 % aniline[5]. Heavy metals like lead, arsenic, mercury, and cadmium have also been involved in various episodes of food contamination in the past. An episode known as Devonshire colic is reported due to ingestion of lead contaminated cider[6]. Aquatic food contamination with methyl mercury resulted in infamous Minimata disease in Japan during 1950's. A similar episode took place in Iraq in 1971 due to consumption of homemade bread baked from wheat contaminated with methylmercury[7-9]. Itai-itai disease with typical symptoms of extreme bone pain and

ISSN 1671-4083 Acta Pharmacol Sin 中国药理学报 2002 Mar; 23 (3)
E-mail aps@mail.shcnc.ac.cn Phn/Fax 86-21-6474-2629

ostomalacia in Japan resulted from contamination of local water supply with waste water run off from zinc-lead-cadmium mine[10]. Ingestion of contaminated bengal gram pulse/flour with *Lathyrus sativus* crippled thousands of people in the past[11,12]. In Calcutta in 1988, an epidemic of polyneuritis especially of hands and feet was caused due to contamination of rape seed oil with tricresyl phosphate. Several episodes of epidemic dropsy have occurred since 1937 in the country due to adulteration and or contamination of mustard oil with argemone oil[13-15].

Thus surveillance and monitoring of food contaminants becomes mandatory for each country so that timely preventive and control measures can be initiated. One of the important federal agencies in USA which protects the public from hazards of unrecognized toxins is the Food and Drug Administration (FDA), the other three being Consumer Product Safety and Health Commission (CPSC), Environmental Protection Agency (EPA), and Occupational Safety and Health Administration (OSHA). Though in India, the Prevention of Food Adulteration Act (1954) lays down regulations to be followed for sampling procedures and methods of analysis of food, however, the routine sampling of food is mostly done by export laboratories only. Chemical analysis is usually carried out for the disputed foods by state laboratories and hence there is no routine sampling and testing of raw materials or cooked food to maintain prescribed limits of contaminants. A similar situation exists in other developing countries as well.

In this review an attempt is made to discuss some common contaminants and their health effects. Toxicovigilance of such toxicants may help in prevention and implementation of legal measures.

POLYCHLORINATED BIPHENYLS AND DIOXINS

Industrial toxicants, polychlorinated biphenyls (PCB), polybrominated biphenyls (PBB) and dibenzofurans may be present together as contaminants. Due to their accumulation in the environment, animal and human tissues, the potential for chronic or delayed toxicity is significant. More than twenty years back an incident of mass poisoning in central Taiwan referred to as Yucheng (oil disease) took place due to consumption of rice bran oil contaminated with PCB and their degraded byproducts[4]. Similarly in some parts of Japan Yusho disease was caused due to consumption of contaminated rice oil with polychlorodibenzo-*p*-dioxins (PCDD) and its related compounds like polychlorodibenzofurans (PCDF). Characteristic toxic manifestations in the affected population included chloracne, hyperpigmentation, subcutaneous facial edema, cheese like discharge from eyes, swelling of eye lids, transient visual disturbances, hyperemia of conjunctiva, and hearing difficulty. Increased incidence of liver cancer and adverse reproductive effects have been reported in the exposed populations. Serum levels of congener specific PCB/PCDF were found to be good indicators of previous exposure in women[16]. Blood PCB levels were high in patients with typical eye and skin symptoms[17]. Children born to mothers who had consumed the contaminated oil are reported to have higher prevalence of middle ear diseases than their matched controls[18]. Food animals have been found to contain PCB, PBB, and dioxins[19]. Consumption of fatty fish like salmon and herring from Baltic sea has been found to be an important source of human exposure to PCBs, PCDDs, and PCDFs[20]. Aquatic food products from industrialized countries have been found to contain markedly high levels of PCB and chlordane[21]. Dietary intake of fish contaminated with persistent organochlorine compounds has been associated with low birth weight babies and endocrine abnormalities[22,23]. Great increase of blood levels of PCDD/PCDF in the population consuming fish and shell fish from contaminated areas has been observed[24].

Dioxins are among the most toxic anthropogenic chemicals in the environment, contaminating the soil thus entering the food chain. Pathways of entry into foods are atmospheric transport of emissions and subsequent deposition on plants, soil, and water. These products are not generally taken up and translocated by the plants, resulting in negligible residues in foods and feeds derived from seeds. However, the main sources are animal diet contaminated with soil that has accumulated dioxin residues from the environment[25]. It is estimated that 98 % of human exposure is through foods particularly meat, fish, and dairy products. A survey has shown that about 25 % or more of marine products are contaminated by trace amounts of dioxins and related compounds[26]. Dioxins are practically insoluble in water and get concentrated in lipids of biological systems and groups at risk for highest exposure are nursing infants and people on high fish/meat diet. An association between exposure via mother's milk to dioxins and developmental dental defects has been found[27].

Dioxins are reported to be plausible myelomagens[28].

PESTICIDES

Contamination of food with pesticides and residues is of special concern because of their inherently high toxicity. Organochlorine pesticides because of their persistence in environment, susceptibility to biomagnification, toxicity to higher animals and ecological catastrophy, have been banned in advanced nations. However, DDT and several others are still used for vector control in many developing countries in Asia and South Pacific region, with the result human populations are still exposed to these compounds mainly through bovine milk and milk produce. After a ban for ten years in Hong Kong, a survey of milk in local markets in 1993 – 1995 showed residues of both DDT and HCH[29]. Data from India indicate DDT and HCH to be the major residues in food. Though their concentration has declined in farm products like food grains and vegetables, milk and milk products are still the major sources. Dietary intake of these residues in Indians is reported to be 100 fold greater than that seen in the population of developed countries. In addition concentration of more than 1 $\mu g/g$ of aldrin, dieldrin, and heptachlor have also been found in vegetables and in untreated water. In Malaysia and Thailand meat was found to be contaminated with aldrin, dieldrin, and DDT, however, in Australia and New Zealand chlordane and PCB were most prevalent organochlorines in food stuffs. The greatest concern has been for infants and children who are exposed through human and dairy milk. Malnutrition also increases the risk of toxicity. The estimated daily intake of DDT by infants in India has been found to be at least 100 fold greater than the acceptable daily intake of FAO/WHO[21]. DDT has been listed as a possible carcinogen in humans. Toxaphene has been found to be a major organochlorine in fish[30]. Chronic exposure to these pesticides may be associated with birth defects, endocrine disorders (estrogenic effects), and neurological deficits[31,32].

Organophosphates (OP) are widely used for home and garden pest control. Though they degrade relatively rapidly in the environment, a few like demeton and demeton methyl are translocated from soil to plant tissues which could ultimately lead to contamination of the products. Exposure to residues of OP in lettuce and cauliflower plants has led to anticholinesterase poisoning in farm workers[33]. Prothiofos and cyanophos residues are reported to migrate from insecticides impregnated paper bags to fruits[34]. Pregnant women believed to have consumed trichlorfon contaminated fish during pregnancy are reported to have developed a cluster of congenital anomalies in Hungary[35]. Myeloid leukemia has been reported after chronic exposure to isofenfos contaminated drinking well water[36]. Consumption of food contaminated with OP is reported to cause mass food borne poisoning outbreaks especially in developing countries and rural areas[37,38].

NITRITES AND HETEROCYCLIC AMINES

Nitrites are ubiquitous and potentially hazardous inorganic ions. They are used for meat curing, coloring and preservation. In certain green vegetables nitrates and nitrites may be formed by microbial reduction after preparation and storage. Acute effect of nitrates and nitrites is toxic methemoglobinemia[39,40]. Outbreaks of methemoglobinemia have been reported due to nitrite contamination of drinking water from corrosion inhibitor[41]. Nitrites may further react with a number of nitrogenous compounds like amines and indoles to form nitroso compounds. Nitrosoamines may also be formed by nitrosation of secondary amines[42]. Volatile *N*-nitrosoamines like *N*-nitrosodimethylamine (NDMA) has been detected in 52 % of Spanish beers[43]. Relatively high content of various *N*-nitrosoamines in different diets is suspected as one of the etiological factors for oesophageal cancer. Heterocyclic amines which are formed during cooking of fast food meat products can also contribute to mutagenic and carcinogenic effects[44]. 3β-Chlorosteroids formed during the production of flavour enhancers though not acutely toxic in animals could however be harmful chronically, as small amounts are absorbed by intestinal tract and accumulated in adipose tissue[45].

POLYCYCLIC AROMATIC HYDROCARBONS AND PHTHALATES

Polycyclic aromatic hydrocarbons (PAH) and other related chemicals are reported to be mutagenic and carcinogenic[46]. Direct human exposure is from diverse sources like tobacco smoke, automobile engine exhausts, high boiling petroleum distillates, carbon black, coal, pitch rubber, *etc*. However, food contamination occurs by polluted air, water, and during cooking. Common foods contaminated with PAH are edible oils (soyabean,

ISSN 1671-4083 Acta Pharmacol Sin 中国药理学报 2002 Mar; 23 (3)
E-mail aps@mail.shcnc.ac.cn Phn/Fax 86-21-6474-2629

coconut, cotton seed, and sunflower), fats, margarine, mayonnaise, and marine products. Smoked and grilled foods and over heated oil fried products also have PAH. Wax paper packaging has led to contamination of bread, biscuits, and breakfast cereals[47].

Packaging in polymer based materials can lead to changes in product flavour due to aroma sorption and transfer of undesirable flavours from packing to food. Certain packaging materials have the potential to contribute plasticizers like phthalate and di-2-ethyl hexyl adipate (DEHA) into the food items especially wrapped meat, poultry, fish, cheese, and ready to eat foods. Low levels of diethyl hexyl phthalate esters have been found with the use of phthalate ester cap or lid seals in a variety of glass packaged foods. The di-2-ethyl hexyl, dibutyl, and butyl benzyl phthalate esters have been found to migrate from aluminium foil paper laminates into butter and margarine, *etc*. In most cases plasticizers found in food have been detected in associated packages also[48]. Certain foods or liquids acquire estrogenic activity after leaching of bisphenol-A from lacquer coating in food cans[49].

MONOSODIUM GLUTAMATE

Monosodium glutamate (MSG) marketed as a flavouring agent causes burning, facial pressure, headache, flushing, chest pain, nausea, and vomiting. Life threatening bronchospasm and angioedema is rarely seen. There is a significant variation in individual responses, however, the intensity and duration of symptoms are also dose-related. In young children sudden attacks or a seizure-like syndrome is reported. There is rapid absorption in the fasting state with typical burning symptoms rapidly spreading over the back, neck, shoulders, abdomen, and occasionally thighs[50].

OIL CONTAMINATION

Contamination of oil has caused poisoning episodes in the past. High concentrations of benzo-(a)-pyrene was reported in Brazilian corn oils[51]. Toxic oil syndrome in Spain caused an epidemic due to contamination of rape seed oil with aniline resulting in multisystem disorder[5,52]. The syndrome was characterized by pneumonitis, eosinophilia, pulmonary hypertension, scleroderma like features, and neuromuscular weakness. Contamination of mustard oil with argemone seeds has caused several outbreaks of epidemic dropsy in India and other parts of the world. The manifestations of the poisoning include vomiting, diarrhea, nausea, abdominal pain, edema of feet and legs, erythema, breathlessness, lethargy, and low grade fever. The cardiovascular manifestations include tachycardia, precardial pain, ECG changes and congestive heart failure. Among the various ocular manifestations are itching and buring of eyes with superficial retinal hemorrhages, disc edema, and glaucoma[13-15]. Several outbreaks have been reported after consumption of tricresyl phosphate (TOCP) contaminated foodstuffs and oil[53]. Major toxicity of TOCP is a delayed neuropathy of the dying back or peripheral axonopathy variety. It is known to induce delayed neurotoxicity[54].

HEAVY METALS

Heavy metals are widespread in nature and their presence has been found from traces to macro levels. Increased environmental load with respect to metallic pollutants is a serious concern. Significant contamination of seeds, plants, and plant products with heavy metals through soil, fertilizers, and water may cause chronic toxicity. Industrial effluents are released into sea, rivers, or even to irrigation channels and fields which facilitates the entry of metallic constituents of effluents into the foodchain. The Minimata disease was caused by contamination of water due to discharge of effluents containing mercury into Minimata bay. The disease was characterised by chronic brain damage, tunnel vision, deafness, and severe congenital defects[7]. Children exposed in-utero developed cerebral palsy, chorea, ataxia, tremors, seizures, and profound mental retardation[55]. Similarly in Iraq an outbreak of organomercurial poisoning took place due to consumption of home-made bread baked from wheat treated with methylmercury used as seed dressing to prevent wheat bunt and other crop diseases. The symptoms included speech disturbances, abnormal behaviour, loss of visual and auditory acuity, and ataxia. Severe disabilities and deaths were reported in this episode[56]. Elemental mercury originating from natural and anthropogenic sources is in the inorganic form predominantly as metallic vapour, which is microbiologically transformed into lipophilic organic compound methylmercury in aquatic environments. The lipophilic states make mercury more prone to biomagnification in aquatic foodchains. Consequently, populations with a traditionally high

dietary intake of food originating from either fresh-water or marine environments have highest exposure to methylmercury. Ingestion of contaminated pork with organic mercury is reported to result in severe toxicity[57]. Populations like polar Eskimos in North West Greenland have a high dietary access to methylmercury due to intake of foods originating from either fresh water or marine environments[58,59]. Studies in humans chronically exposed to food contaminated with methylmercury showed increased sister chromatid exchange rates, chromosomal aberration, and aneuploidy in the lymphocytes[60,61]. Contamination of rice with cadmium led to Itai-itai disease in Japan manifested by extreme bone pain and osteomalacia[10,62]. Cadmium has also been documented to cause hypertension and nephrotoxicity[63,64]. It is reported to induce immunosuppression in experimental studies[65-67]. Markedly high blood mercury levels have been reported after consumption of sea bass containing remarkable mercury levels[68]. In certain fish eaters high mercury levels are reported in urine and hair[69]. Wild mushrooms growing in viscinity of mercury and copper smelter areas have shown extremely high concentrations of mercury, copper, lead, and cadmium[70,71]. Children living in viscinity of mercury contaminated industrial sites are the most sensitive group mainly exposed by soil ingestion which makes up nearly 80 % ADI[72]. Lead and cadmium are reported to get transfered from contaminated soil to crop plants or food of plant origin[73]. Maternal exposure to mercury can lead to impaired intellectual and motor development in breast fed infants.

With rapid increase in the consumption of canned food products throughout the world, the possibility of food contamination with different metals is expected. The metals involved could be arsenic, aluminium, copper, iron, lead, tin, and zinc. Metals used in food packaging material or food processing equipment may contribute to contamination[74]. Leaching of metals like iron, chromium, and nickle from stainless steel utensils into food is reported[75]. Acute poisoning is less common however food contaminated with arsenic oxide is reported[76]. Arsenic salts are also used as rodent baits, insecticidal fruit sprays, and herbicides, so food or fruits may get accidentally contaminated. Severe arsenic poisoning has been reported due to contamination of wells in Minnesota[77]. In West Bengal in India and Bangladesh drinking water is also contaminated with arsenic[78]. A large number of population is suffering from peripheral neuropathy, hyperkeratoses, and skin cancer. Remarkable arsenic concentrations have been found in sea food especially shell fish. Remarkable elevations of urinary monomethyl and dimethyl arsenic acid are reported after mussel ingestion[79,80]. Excessive intake of manganese due to food contamination coupled with low intake of magnesium from drinking water has led to motor neuron disease in Japan[81]. Aluminium salts are sometimes used as food additives and aluminium may also leach from aluminium utensils and cans[82]. Processed cheese and wheat flour may contain aluminium containing additives[83]. The metal leaching tends to increase with increasing acid concentration of soft drinks in the cans. However, possible intake through ingestion is negligible[84]. Leaching of aluminium from aluminium-lead alloy utensils is reported to be enhanced by water containing high concentration of fluoride ions at a low pH than from high quality utensils of aluminium-manganese alloy[85]. Inadvertent addition of a massive amount of aluminium sulfate during water treatment resulted in very high aluminium concentrations as compared to maximum admissible limits and led to symptoms like nausea, vomiting, diarrhea, mouth ulcers, skin rashes, and arthritic pains[86]. A number of commercial teas have been found to contain aluminium. However its absorption from tea may be quite low[87,88]. Some parenteral and dialysing solutions containing aluminium salts and in uremic patients on dialysis metabolic bone disease can occur[89].

Lead contamination could occur from ceramic glazed pottery. A correlation between the use of lead glazed ceramicware for cooking or food preparations and elevated lead levels has been found. More leaching (> 0.5 mmol/L) occurs into acidic foods cooked or stored in lead ceramicware than neutral foods. Sequential extractions of acidic foods yield variable but declining levels of lead[90]. Leaching from old ceramic dinnerware with 4 % acetic acid or 0.5 % citric acid has been reported after microwave heating. Use of such dishes for microwaving of common foods could result in ingestion of dangerously large amounts of lead. Further, dishes with uranium and copper containing glazes could also lead to unsafe concentration of lead in microwave leachates[91]. Automobile radiaters used as condensors for illegally distilled whisky add lead to the final products. Baking processes of bread may contribute to contamination due to lead and benzo-(a)-pyrene[92]. Lead is also present in lead arsenate, a pesticide used for spraying apples. Anemia and lead colic have been reported after prolonged use. The children show weight loss, weakness, and

ISSN 1671-4083 Acta Pharmacol Sin 中国药理学报 2002 Mar; 23 (3)
E-mail aps@mail.shcnc.ac.cn Phn/Fax 86-21-6474-2629

neurobehavioural deficits. Vague gastrointestinal and CNS effects are manifested in adults[93]. Barium salts have been investigated as one of the alternatives to lead in frit formulations for glazes. The potential hazard associated with the leaching of barium from glazed ceramicware is reported to vary with the level of use[94]. Food contamination with lead and its salts could occur in occupationally exposed people working with unrecognized sectors through transfer of metallic particles on clothes and hands. Drinking water may be contaminated by lead pipes. Experimental evidence suggests that lead induces immunotoxicity[95]. Metal contamination may also occur from the food processing equipments and corrosion of packing materials. Nickel is used in hydrogenation of vegetable oils, however the health effects are not known.

FUNGAL TOXINS

Fungal toxins are one of the major food contaminants. Three groups of fungi that easily grow on food stuffs include *Aspergillus*, *Fusarium*, and *Penicillium*. These fungi elaborate several hundred toxic metabolites. A variety of consumable articles like cereals, pulses, oil seeds, vegetables, dry fruits, spices, dried fish, milk and its products, and meat get contaminated by fungi. The common mycotoxins include aflatoxins, ochratoxins, citrinin, zearalenone, deoxyvalenol, fumonisins, trichothecenes, patulin, tenuazoic acid, and pencillic acid, *etc*. However, aflatoxins being toxic are potent carcinogenic, mutagenic, and teratogenic compounds[96,97]. In India, the problem is of greater magnitude due to congenial climatic conditions, socio-economic backwardness, and traditional and unscientific agronomic and storage practices. The genetic make up of the aflatoxin strains and the environmental factors determine the degree of aflatoxic poisoning to a great extent. The main environmental factors include moisture, temperature, length of storage, level of carbon dioxide, and oxygen. Besides these abiotic factors, the biotic factors like insect vectors, birds, rodents, crop varieties, and fungal interactions affect the contamination. Reports indicate insect and bird damaged samples of crops have high levels of aflatoxins as compared to the healthy samples. Of the various aflatoxins, aflatoxin B1 is the most toxic with proven carcinogenicity. It constitutes an important risk factor for hepatocellular carcinoma in exposed populations[98]. It exerts its effects after conversion to the reactive compound aflatoxin B1-epoxide by the action of cytochrome P-450-dependent enzymes in the body[99]. Aflatoxin B1 and G1 have been found in sorghum seeds during storage under controlled conditions[100]. In Philippines, toxic levels have been found in corn and peanuts[101]. Black pepper may be heavily contaminated with aflatoxin B1[102]. An outbreak of acute hepatic encephalopathy due to severe aflatoxicosis has been reported from Malaysia due to ingestion of contaminated noodles[103]. On the basis of reports of natural occurrence and level of aflatoxins from different parts of India maize, groundnut, cotton seed, rice, and millet can be categorized as high risk crops. Further, rice bran, a valuable product utilized for various purposes in food and feed industry, is also reported to be a good substrate for natural occurrence of *Aspergills flavus*. Through infected animal feed milk may get contaminated. Oil seeds also provide good substrate for growth of toxicogenic fungi and toxin elaboration. A ten year Indian study showed remarkable contamination of groundnut and deoiled groundnut cake, maize, and mixed feeds[104]. Patulin, a metabolite of *Aspergillus* and *Penicillium* having carcinogenic, teratogenic and mutagenic properties is detected in some fruits. Kojic acid, a common metabolite of *Aspergills flavus* is reported to be a convulsant.

Ergot alkaloids of various species of *Claviceps* infect millet and produce peripheral, vascular, neurohumoral, and CNS effects (ergotism). Streigmatocystin, a potent toxin from heavily fungal infected wheat can contaminate processed foods. Ochratoxin A (OA) is the most toxic of ochratoxins produced by a number of *Aspergillus* and *Penicillium* species and is carcinogenic, teratogenic, mutagenic, and immunosuppressive. It grows in cereals, legumes and green coffee beans. Though porcine nephrotoxicity is established, acute human nephrotoxicity seems to be rare[105]. It has been found in coffee beans besides being present predominantly in cereals. OA has also been found in brew[106]. Levels of 0.1 – 12 μg/kg of OA have been found in the breast milk of donor mothers in Italy[107].

Fusarium mycotoxins (fumosinins, nivalenol, deoxynivalenol, and zearalenone) may contaminate cereals and animal feeds and cause health risks in domestic animals and humans[108,109]. Contamination of commercially available corn based food stuffs for human consumption is reported from Taiwan[110]. Zearalenone and its derivatives are reported to show estrogenic effects in animals. Nivalenol has been reported in high

concentrations (4700 μg/kg) in Swedish cereals[111]. Certain toxins survive the brewing process in the manufacture of beers. These mycotoxins could originate from malted grain or from adjuncts. Although increased incidence and concentrations of aflatoxins and zearalenone have been found in local beers brewed in Africa, no aflatoxins have been found in European beers. In naturally contaminated corn used for ethyl alcohol fermentation and wet milling operations fumosinins B1 and B2 are stable to conditions for fermentations and tend to concentrate in distillers dried grain generally used for animal feeds[112]. Trichothecenes may cause severe hemorrhage in intestines, nose and throat, leukopenia, dermatitis, cough, and rhinitis[113].

Ingestion of moldy sugarcane has resulted in acute encephalopathy, delayed dystonia, and bilateral lentricular lucencies. Such cases have been reported from China and the most likely etiologic agent is 3-nitropropionic acid produced by *Arthrinium* species of fungus[114].

MICROORGANISMS

Common microorganisms that contaminate food include *Salmonella*, *Staphylococcus aureus*, *Bacillus cereus*, *Clostridium perfringes*, and *Campylobacter jejuni*. Botulism caused by Clostridium botulinium is manifested as acute decending paralysis due to blocking of neuromuscular transmission[115]. The growth and toxin production of *Clostridium botulinium* in food is enhanced by high moisture, low salt, low acid, reduced oxygen, and increased temperature conditions[116]. Milk products, potato salads, custards, ham, creamy pastry, and cheese provide good medium for growth of *Staphylococi* and enterotoxins released can cause vomiting, diarrhea, and dehydration. *Bacillus cereus*, which grows on inadequately refrigerated starchy foods and cereals causes both diarrhea and emesis[117]. *Clostridium perfringes* present in previously cooked, inadequately refrigerated poultry and meat products causes diarrhea and clostridial myonecrosis[118-120]. Milk and cream are the source of *Campylobacter* associated with food borne disease. Human infection due to *Salmonella* is frequent and widespread and the types of foods most often associated with the poisoning are meat, milk, and eggs. Usual symptoms include headache, chills, abdominal pain, nausea, vomiting, diarrhea, and fever[121]. Systemic anaphylaxis has been reported after ingestion of storage mite contaminated food. Mite contaminated wheat flour can lead to anaphylaxis and an increased frequency of sensitivity to aspirin and other nonsteroidal anti-inflammatory drugs (NSAID)[122,123].

The problem of food contamination can be overcome to a great extent by the use of good quality raw materials and maintenance of absolute hygienic conditions, undertaking regular surveillance, and monitoring programmes. There is an absolute need for formulating maximum permissible standards for each country taking into account food habits, social, cultural, and economic status. Further, widespread contamination of cereals, pulses, poultry, dairy products with pesticide residues warrants caution and efforts to ensure any further rise. This can be achieved by ensuring absolute ban or restricted use and disposal of organochlorine pesticides. In addition proper disposal of effluents from industries dealing with persistent organochlorines and heavy metals should be ensured and lastly there should be a strict implementation of food and adulteration act.

REFERENCES

1 Merchoff GC, Porter JM. Ergot intoxication: historical review and description of unusual clinical manifestations. Ann Surg 1974; 180: 773-9.
2 Schmid R. Cutaneous porphyria in Turkey. N Engl J Med 1960; 263: 397-8.
3 Koppelman H, Robertson MH, Saunders PG. The Epping jaundice. Br Med J 1976; 1: 514-6.
4 Jones GRN. Polychlorinated biphenyls: where do we stand now. Lancet 1989; 2: 791-4.
5 Kilbourne EM, Rigau-Perez JG, Heath CW Jr, Zack MM, Falk H, Martin-Marcos M, *et al*. Clinical epidemiology of toxic-oil syndrome. Manifestations of a new illness. N Engl J Med 1983; 309: 1408-14.
6 Waldron HA. The Devonshire colic. J Hist Med Allied Sci 1970; 25: 383-413.
7 Powell PP. Minimata disease: a story of mercury's malevolence. South Med J 1991; 84: 1352-8.
8 Tsuchiya K. The discovery of the causal agent of Minimata disease. Am J Ind Med 1992; 21: 275-80.
9 Bakir F, Damluji SF, Amin-Zaki L, Murtadha M, Khalidi A, al-Rawi NY, *et al*. Methyl mercury poisoning in Iraq. Science 1973; 181: 230-41.
10 Cadmium pollution and Itai-itai disease. Lancet 1971; 1: 382-3.
11 Shourie KL. An outbreak of lathyrism in central India. Indian J Med Res 1945; 33: 239-47.
12 Haque A, Hossain M, Wouters G, Lambein F. Epidemiological study of lathyrism in districts of Bangladesh. Neuroepidemiology 1996; 15: 83-91.
13 Sharma BD, Malhotra S, Bhatia V, Rathee M. Epidemic dropsy in India. Postgrad Med J 1999; 75: 657-61.
14 Singh NP, Anuradha S, Dhadwal DK, Singh K, Prakash A, Madan K, *et al*. Epidemic dropsy — a clinical study of the Delhi outbreak. J Assoc Physicians India 2000; 48: 877-

ISSN 1671-4083 Acta Pharmacol Sin 中国药理学报 2002 Mar; 23 (3)
E-mail aps@mail.shcnc.ac.cn Phn/Fax 86-21-6474-2629

80.
15 Das M, Khanna SK. Clinicoepidemiological toxicological and safety evaluation studies on argemone oil. Crit Rev Toxicol 1997; 27: 273 – 97.
16 Guo YL, Ryan JJ, Lau BP, Yu ML, Hsu CC. Blood serum levels of PCBs and PCDFs in Yucheng women 14 years after exposure to a toxic rice oil. Arch Environ Contam Toxicol 1997; 33: 104 – 8.
17 Hirota Y, Tokunaqaa S, Kalaoka K, Shinohara S. Symptoms and blood PCB level among chronic Yusho patients, twenty five years after outbreak. Fukuoka Igaku Zasshi 1997; 88: 220 – 5.
18 Chao WY, Hsu CC, Guo YI. Middle ear disease in children exposed prenatally to polychlorinated biphenyls and polychlorinated dibenzofurans. Arch Environ Health 1997; 52: 257 – 62.
19 Headrick ML, Hollinger K, Lovell RA, Matheson JC. PBBs, PCBs and dioxins in food animals, their public health implications. Vet Clin North Am Food Anim Pract 1999; 15: 109 – 31.
20 Svensson BG, Hallberg T, Nilsson A, Schutz A, Hagmar L. Parameters of immunological competence in subjects with high consumption of fish contaminated with persistent organochlorine compounds. Int Arch Occup Environ Health 1994; 65: 351 – 8.
21 Kannan K, Tanabe S, Giesy JP, Tatsukawa R. Organochlorine pesticides and polychlorinated biphenyls in food stuffs from Asian and oceanic countries. Rev Environ Contam Toxicol 1997; 152: 1 – 55.
22 Rylander L, Stromberg U, Hagmar L. Dietary intake of fish contaminated with persistent organochlorine compounds in relation to low birth weight. Scand J Work Environ Health 1996; 22: 260 – 6.
23 Colborn T, vom Saal FS, Soto AM. Developmental effects of endocrine-disrupting chemicals in wildlife and humans. Environ Health Perspect 1993; 101: 378 – 84.
24 Johansen HR, Alexander J, Rossland OJ, Planting S, Lovik M, Gaarder PI, *et al*. PCDDs, PCDFs, and PCBs in human blood in relation to consumption of crabs from a contaminated Fjord area in Norway. Environ Health Perspect 1996; 104: 756 – 64.
25 Fries GF. A review of the significance of animal food products as potential pathways of human exposures to dioxins. J Anim Sci 1995; 73: 1639 – 50.
26 Saito Y. The interim report on survey of polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans in marine products and estimation of exposure through fishes and shellfishes. Yakaguku Zasshi 1997; 117: 850 – 63.
27 Alaluusua S, Lukinmaa PL, Koskimies M, Pirinen S, Holtta P, Kallio M, *et al*. Developmental dental defects associated with long breast feeding. Eur J Oral Sci 1996; 104: 493 – 7.
28 Schwartz GG. Multiple myeloma: clusters, clues and dioxins. Cancer Epidemiol Biomakers Prev 1997; 6 : 49 – 56.
29 Wong SK, Lee WO. Survey of organochlorine pesticide residues in milk in Hong Kong (1993 – 1995). J AOAC Int 1997; 80: 1332 – 5.
30 Braune B, Muir D, DeMarch B, Gamberg M, Poole K, Currie R, *et al*. Spatial and temporal trends of contaminants in Canadian Arctic freshwater and terrestrial ecosystems: a review. Sci Total Environ 1999; 230: 145 – 207.
31 Hall RH. A new threat to public health, organochlorine and food. Health J 1992; 8: 33 – 43.
32 Mattison DR, Plowchalk DR, Meadows MJ, Al-Juburi AZ, Gandy J, Malek A. Reproductive toxicity: male and female reproductive systems as targets for chemical injury. Med Clin North Am 1990; 74: 391 – 411.
33 Midtling JE, Barnett PG, Coye MJ, Velasco AR, Romero P, Clements CL, *et al*. Clinical management of field worker organophosphate poisoning. West J Med 1985; 142: 514 – 8.
34 Katami T, Nakamura M, Yasuhara A, Shibamoto T. Migration of organophosphorous insecticides cyanophos and prothiofos residues from impregnated paper bags to Japanese apple-pears. J Agric Food Chem 2000; 48: 2499 – 501.
35 Czeizel AE, Elek C, Gundy S, Metneki J, Nemes E, Reis A, *et al*. Environmental trichlorfon and cluster of congenital abnormalities. Lancet 1993; 341: 539 – 42.
36 Boros LG, Williams RD. Chronic isofenos poisoning: case report of agnogenic myeloid metaplasia with a rapid progression into acute myeloid leukemia. Leuk Res 1998; 22: 849 – 51.
37 Greenway C, Orr P. A foodborne outbreak causing cholinergic syndrome. J Emergency Med 1996; 14: 339 – 44.
38 Chaudhury R, Lall SB, Mishra B, Dhawan B A foodborne outbreak of organophosphate poisoning. Br Med J 1998; 317: 268 – 9.
39 Askew CL, Finelli L, Cenese CA, Sorhage FE, Sosin DM, Spitalny KC. Boiler baisse: an outbreak of methemoglobinemia in New Jersey in 1992. Pediatrics 1994; 94: 381 – 4.
40 Chan TY. Food-borne nitrates and nitrites as cause of methemoglobinemia. Southeast Asian J Trop Med Public Health 1996; 27: 189 – 92.
41 Bradberry SM, Gazzard B, Vale JA. Methemoglobinemia caused by the accidental contamination of drinking water with sodium nitrite. Clin Toxicol 1994; 32: 173 – 8.
42 Silla Santos MH. Biogenic amines: their importance in foods. Int J Food Microbiol 1996; 29: 213 – 31.
43 Izquierdo Pulido M, Barbour JF, Scanlan RA. *N*-nitrosodimethylamine in Spanish beers. Food Chem Toxicol 1996; 34: 297 – 9.
44 Knize MG, Sinha R, Rothman N, Brown ED, Salmon CP, Levander OA, *et al*. Heterocyclic amine content in fast-food meat products. Food Chem Toxicol 1995; 33: 545 – 51.
45 Weber N, Richter KD. 3-Beta-chlorosteroids: artefacts and possible contaminants in food and feed toxicological effects in mice. Food Chem Toxicol 1994; 32: 297 – 303.
46 Guillen MD, Sopelana P, Partearroyo MA. Food as a source of polycyclic aromatic carcinogens. Rev Environ Health 1997; 12: 133 – 46.
47 Castle L, Nichol J, Gilbert J. Migration of mineral hydrocarbons into foods. 4. Waxed paper for pakaging dry goods including bread, confectionery and for domestic use including microwave cooking. Food Addit Contam 1994; 11: 79 – 89.
48 Page BD, Lacroix GM. The occurrence of phathalate ester and di-2-ethylhexyl adipate plasticizers in Canadian packaging and food sampled in 1985 – 1989: a survey. Food Addit Contam 1995; 12: 129 – 51.
49 Brokons JA, Olea-Serrano MF, Villalobos M, Pedraza V, Olen N. Xenoestrogens released from lacquer coatings in foods cans. Environ Health Perspect 1995; 103: 608 – 12.
50 Goldfrank LR, Tunik MG. Food poisoning. In: Goldfrank LR, Flomenbaum NE, Lewin NA, Weisman RS, Howland

MA, Hoffman RS, editors. Goldfrank's toxicologic emergencies. Connecticut: Appleton Lange; 1994. p 921 – 48.

51 Pupin AM, Toledo MC. Benzo (a) pyrene in Brazilian vegetable oils. Food Addit Contam 1996; 13: 639 – 45.

52 Posada de la Paz M, Philen RM, Borda IA, Silica Socias JM, Gomez de la Camara A, Kilbourne EM. Toxic oil syndrome: traceback of the toxic oil and evidence for a point source epidemic. Food Chem Toxicol 1996; 34: 251 – 7

53 Hayes WJ Jr, Laws EK Jr, editors. Handbook of pesticide toxicology; v 2. San Diego (CA): Academic Press; 1991. p 957 – 72.

54 Craig PH, Barth ML. Evaluation of hazards of industrial exposure to tricresyl phosphate: a review and interpretation of the literature. J Toxicol Environ Health B Crit Rev 1999; 2: 281 – 300.

55 Winship KA. Toxicity of mercury and its inorganic salts. Adv Drug React 1985; 3: 129 – 60.

56 Bakir F, Rustam H, Tikriti S, Al-Damluji SF, Shihristani H. *Clinical and epidemiological aspects of methyl mercury* poisoning. Postgrad Med J 1980; 56: 1 – 10.

57 Davis LE, Kornfeld M, Mooney HS, Fielder KJ, Haaland KY, Orrison WW, *et al*. Methyl mercury poisoning: long term clinical, radiological, toxicological and pathological studies of an affected family. Ann Neurol 1994; 35: 680 – 8.

58 Ilansen JC, Danscher G. Organic mercury: an environmental threat to the health of dietary exposed societies? Rev Environ Health 1997; 12: 107 – 16.

59 Hansen JC. Environmental contaminants and human health in the Arctic. Toxicol Lett 2000; 112 – 3: 119 – 25.

60 IARC monographs on the evaluation of carcinogenic risks to humans: beryllium, cadmium, mercury and exposures in the glass manufacturing industry. IARC Monogr Eval Carcinog *Risks Hum* 1993; 58: 239 – 88.

61 Franchi E, Loprieno G, Ballardin M, Petrozzi L, Migliore L. Cytogenetic monitoring of fishermen with environmental mercury exposure. Mutat Res 1994; 320: 23 – 9.

62 Dunnick JK, Fowler BA. Cadmium. In: Seiler HG, Sigel H, editors. Handbook on toxicity of inorganic compounds; New York: Marcel Dekker; 1988. p 55 – 174.

63 Lall SB, Peshin SS, Gulati K, Khattar S, Das N, Seth SD. Involvement of RAS in hypertensive effect of cadmium. Indian J Exp Biol 1997; 35: 338 – 41.

64 Lall SB, Das N, Rama R, Peshin SS, Khattar S, Gulati K, Seth SD. Cadmium induced nephrotoxicity in rats. Indian J Exp Biol 1997; 35: 151 – 41.

65 Lall SB, Dan G. Sympathetic regulation of immune system in Cd exposed rats. Neuro Endocrinol Lett 1998; 19: 21 – 5.

66 Dan G, Lall SB. Neuroendocrine modulation of immune system. Indian J Pharmacol 1998; 30: 129 – 40.

67 Lall SB, Dan G. Role of corticosteroids in Cd induced immunotoxicity. Drug Chem Toxicol 1999; 22: 401 – 9.

68 Kobeloch LM, Ziarnik M, Anderson HA, Dodson VN. Imported seabass as a source of Hg exposure a Wisconsin case study. Environ Health Perspect 1995; 103: 604 – 6.

69 Abe T, Ohtsuka R, Hongo T, Suzuki T, Tohyama C, Nakuno A, *et al*. High hair and urinary mercury levels of fish eaters in non polluted environment of Papua, New Guinea. Arch Environ Health 1995; 50: 367 – 73.

70 Kalae P, Niznanska M, Bevilaqua D, Sbaskova L. Concentrations of mercury, copper, cadmium and lead in fruiting bodies of edible mushrooms in the viscinity of a mercury smelter and a copper smelter. Sci Total Environ 1996; 177: 251 – 8.

71 Svoboda L, Zimmermannova K, Kalac P. Concentrations of mercury, cadmium, lead and copper in fruiting bodies of edible mushrooms in an emission area of copper smelter and mercury smelter. Sci Total Environ 2000; 246: 61 – 7.

72 Nusslein F, Feicht EA, Schulte Hostede S, Seltmann U, Kettrup A. Exposure analysis of the inhabitants living in the neighbourhood of a mercury contaminated industrial site. Chemosphere 1995; 30: 2241 – 8.

73 Dudka S, Miller WP. Accumulation of potentially toxic elements in plants and their transfer to human food chain. J Environ Sci Health B 1999; 34: 681 – 708.

74 Morgan JN. Effects of processing of heavy metal content of foods. Adv Exp Med Biol 1999; 459: 195 – 211.

75 Kumar R, Srivastava PR, Srivastava SP. Leaching of heavy metals (Cr, Fe, Ni) from stainless steel utensils in food simulants and food materials. Bull Environ Contam Toxicol 1994; 53: 259 – 66.

76 Dong HQ, Wang KL, Ma YJ. A clinical analysis of 117 cases of acute arsenic poisoning. Chin J Intern Med 1993; 32: 813 – 5.

77 Feinglass EJ. Arsenic intoxication from well water in the United States. N Engl J Med 1973; 288: 828 – 30.

78 GuhaMazumdar DN, Das Gupta J, Santra A, Pal A, Ghose A, Sarkar S. Chronic arsenic toxicity in West Bengal — the worst calamity in the world. J Indian Med Assoc 1998; 96: 4 – 7.

79 Buchet JP, Pauwels J, Lauwerys R. Assessment of exposure to inorganic arsenic, a human carcinogen due to consumption of sea food. Arch Toxicol 1996; 70: 773 – 8.

80 Buchet JP, Pauwels J, Lauwerys R. Assessment of exposure to inorganic arsenic following ingestion of marine organisms by volunteers. *Environ Res* 1994; 66: 44 – 51.

81 Iwami O, Watanabe T, Moon CS, Nakatsuka I, Ikeda M. Motor neuron disease on the Kii Peninsula of Japan: excess manganese intake from food coupled with low magnesium in drinking water as a risk factor. Sci Total Environ 1994; 149: 121 – 35.

82 Wang L, Su DZ, Wang YF. Studies on the aluminium content in Chinese foods and the maximum permitted levels of aluminium in wheat flour products. Biomed Environ Sci 1994; 7: 91 – 9.

83 Pennigton JA, Schoen SA. Estimates of dietary exposure to aluminium. Food Addit Contam 1995; 12: 119 – 28.

84 Seruga M, Grgic J, Mandic M. Aluminium content of soft drinks from aluminium cans. Z Lebensm Unters Forsch 1994; 198: 313 – 6.

85 Rao KS, Rao GV. Aluminium leaching from utensils — a kinetic study. Int J Food Sci Nutr 1995; 46: 31 – 8.

86 Eastwood JP, Levin GE, Pazianes M, Taylor AP, Denton J, Freemont AJ. Aluminium deposition in bone after contamination of drinking water supply. Lancet 1990; 336: 462 – 4.

87 Koch KR, Pougnet MAB, de Villiers S, Monteagudo F. Increased urinary excretion of aluminium after drinking tea. Nature 1988; 333: 122.

88 Powell JJ, Greenfield SM, Parkes HG, Nicholson JK, Thompson RP. Gastrointestinal availability of aluminium from tea. Food Chem Toxicol 1993; 31: 449 – 54.

89 Klein GL. Aluminium in parenteral solutions revisited again.

ISSN 1671-4083 Acta Pharmacol Sin 中国药理学报 2002 Mar; 23 (3)
E-mail aps@mail.shcnc.ac.cn Phn/Fax 86-21-6474-2629

Am J Clin Nutr 1995; 61: 449 - 56.
90 Gonzalez de Mejia E, Craigmill AL. Transfer of lead from lead glazed ceramics to food. Arch Environ Contam Toxicol 1996; 31: 581 - 4.
91 Sheets RW, Turpen SL, Hill P. Effect of microwave heating on leaching of lead from old ceramic dinnerware. Sci Total Environ 1996; 182: 187 - 91.
92 Ahmed MT, Abdel Hadi el S, el-Samahy S, Youssof K. The influence of baking fuel on residues of polycyclic aromatic hydrocarbons and heavy metals in bread. J Hazard Mater 2000; 80: 1 - 8.
93 Needleman HL, Gatsonis CA. Low level lead exposure and IQ of children: a metaanalysis of modern studies. J Am Med Assoc 1990; 263: 673 - 8.
94 Assimon SA, Adams MA, Jacobs RM, Boiger PM. Preliminary assessment of potential health hazards associated with barium leached from glazed ceramicware. Food Addit Contam 1997; 14: 483 - 90.
95 Lall SB, Das N. Role of free radicals in lead induced immunotoxicity. Indian J Pharmacol 2000; 32: 167.
96 Park DL. Surveillance programmes for managing risks from naturally occuring toxicants. Food Addit Contam 1995; 12: 361 - 71.
97 Wang JS, Groopman JD. DNA damage by mycotoxins. Mutat Res 1999; 424: 167 - 81.
98 Jackson PE, Groopman JD. Aflatoxin and liver cancer. Baillieres Best Pract Res Clin Gastroenterol 1999; 13: 545 - 55.
99 De-Oliveira CA, Germano PM. Aflatoxins: current concepts on mechanisms of toxicity and their involvement in *the etiology of hepatocellular carcinoma*. Rev Saude Publica 1997; 31: 417 - 24.
100 Mukherjee K, Lakshminarasimhan AV. Aflatoxin contamination of sorghum seeds during storage under controlled conditions. Int J Med Microbiol Virol Parasitol Infect Dis 1995; 282: 237 - 43.
101 Arim RH. Present status of the aflatoxin situation in the Phillippines. Food Addit Contam 1995; 12: 291 - 6.
102 Delcourt A, Rousset A, Lemaitre JP. Microbial and mycotoxic contamination of peppers and food safety. Boll Chim Farm 1994; 133: 235 - 6.
103 Lye MS, Gazali AA, Mohan J, Alwin N, Nair RC. An acute outbreak of acute hepatic encephalopathy due to severe aflatoxicosis in Malaysia. Am J Trop Med Hyg 1995; 53: 68 - 72.
104 Dhavan AS, Chowdary MR. Incidence of aflatoxins in animal feedstuffs: a decades scenario in India. J AOAC Int 1995; 78: 693 - 8.
105 Fillastre JP. Experimental and human nephrotoxicity induced by ochratoxins. Bull Acad Natl Med 1997; 181: 1447 - 63.
106 Studer-Rohr I, Dietrich DR, Schlatter J, Schlatter C. The occurrence of ochratoxin A in coffee. Food Chem Toxicol 1995; 33: 341 - 55.
107 Micco C, Miraglia M, Brera C, Cornell S, Ambruzzi A. Evaluation of ochratoxin A level in human milk in Italy. Food Addit Contam 1995; 12: 351 - 4.
108 Yuwai KE, Rao KS, Singh K, Tanaka T, Ueno Y. Occurrence of nivalenol, deoxynivalenol and zearalenone in imported cereals in papua New Guinea. Nat Toxins 1994; 2: 19 - 21.
109 Lu G, Li L, Xue Y. Studies on deoxynivalenol contamination in grain and its products in Anhui province. Chin J Prev Med 1994; 28: 27 - 30.
110 Tseng TC, Liu CY. Occurrence of fumonisin Bland BZ in corn based food stuffs in Taiwan market. Mycopathologia 1997; 137: 57 - 61.
111 Pettersson H, Hedman R, Engstrom B, Elwinger K, Fossum O. Nivalenol in Swedish cereals occurrence, production and toxicity towards chickens. Food Addit Contam 1995; 12: 373 - 6.
112 Bennet GA, Richard JL, Eckhott SR. Distribution of fumonisins in food and feed products prepared from contaminated corn. Adv Exp Med Biol 1996; 392: 317 - 22.
113 Watson SA, Mirocha CJ, Hayes AW. Analysis for trichothecenes in samples for southest Asia associated with yellow rain. Fundam Appl Toxicol 1984; 4: 700.
114 Ming L. Moldy sugarcane poisoning — a case report with a brief review. J Toxicol Clin Toxicol 1995; 33: 363 - 7.
115 Arnon SS. Infant botulism. In : Anaerobic infections in humans. San Diego: Academic Press; 1989. p 601 - 9.
116 Pierson MD, Reddy NR. Clostridium botulinium. Food Technol 1988; 42: 196 - 8.
117 Kramer JM, Gilbert RJ. Bacillus cerus and other bacillus species. In: Doyle MP, editor. Food borne bacterial pathogens. New York: Marcel Dekker; 1989. p 21 - 70.
118 Bariello S, Larsonl H, Welch A, Barclayl F, Stringer M, Bartholomew B. Clostridium perfringens: A possible cause of antibiotic associated diarrhoea. Lancet 1984; 1: 305 - 7.
119 Luzzi I, Caprioli A, Falbo V, Guarino A, Capano G, Alessio M, *et al*. Detection of clostridial toxins in stools from children with diarrhoea. J Med Microbiol 1986; 22: 29 - 31.
120 Murrel TG, Ingham B, Moss JR, Taylor W. A hypothesis concerning *Clostridium perfringens* type A enterotoxin and sudden infant death syndrome (SIDS). Med Hypotheses 1987; 22: 401 - 31.
121 Gast RK, Beard CW. Isolation of *Salmonella enteritidis* from internal organs of experimentally infected hens. Avian Dis 1990; 34: 991 - 3.
122 Matsumoto T, Hisano T, Hamaguchi M, Miike T. Systemic anaphylaxis after eating storage mite contaminated food. Int Arch Allergy Immunol 1996; 109: 197 - 200.
123 Sanchez-Borges M, Capriles-Hulett A, Capriles-Behrens E, Fernandez-Caldas E. A new triad: sensitivity to aspirin, allergic rhinitis, and severe allergic reaction to ingested aeroallergens. Cutis 1997; 59: 311 - 4.

潜在的食品污染剂和与之相关的健康危险

关键词 食品污染；有机磷化合物；油类；芳香烃类；重离子；微生物学；真菌

（责任编辑 朱倩蓉）