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Food production and food safety

T A B Sanders

Most food is now produced by large farms, processed industrially, and sold in supermarkets and multi-national food outlets. Modern food production has reduced the cost and increased the variety of food available, but this centralisation of the food supply presents an opportunity for foodborne pathogens and toxins to infect and poison large numbers of consumers.¹ Furthermore, the globalisation of food trade means that food can become contaminated in one country and cause outbreaks of foodborne illness in another.²⁻⁴ Modern food production is so complex that a systematic approach is needed to identify the hazards at each point in the food chain.

Methods

I made an electronic search of the Medline database between January 1990 and May 1999, using the search terms food poisoning and epidemiology, food additives and adverse effects, pesticides and poisoning, and food contamination. Statistical information on the incidence of food poisoning and adverse reactions was obtained from the Public Health Laboratory Services; Centers for Disease Control and Prevention, Atlanta; and the UK Department of Health. Data on food surveillance was obtained from the Ministry of Agriculture, Fisheries, and Food. Information on risk assessment was derived from working papers of the WHO/FAO

Summary points

The centralisation and globalisation of foods increase the likelihood of pandemics of foodborne disease

People in developing countries are at greater risk from naturally occurring toxicants, foodborne disease, and contaminants in the food chain

The hazard critical control point concept is essential for assessing and managing risk

Special consideration is needed with regard to fish and shellfish

Concerted action needs to be taken to prohibit the use of antibiotics as growth promoters in animal production

Internationally agreed food standards are essential to facilitate trade in food between areas with food surplus and those with food deficit

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Codex Alimentarius Commission and the European Commission Scientific Committee for Food.

Seven steps of HACCP

- Analyse hazards: potential hazards associated with a food and measures to control those hazards are identified
- Identify critical control points: these are points in the food chain at which the potential hazard can be controlled or eliminated
- Establish preventive measures with critical limits for each control point: for a cooked food, for example, this might include setting the minimum cooking temperature and time required to ensure the elimination of any microbes
- Establish procedures to monitor the critical control points
- Establish corrective actions to be taken when monitoring shows that a critical limit has not been met
- Establish procedures to verify that the system is working properly
- Establish effective record keeping to document the HACCP system: this would include records of hazards and their control methods, the monitoring of safety requirements, and action taken to correct potential problems

Adapted from the USA National Food Safety Initiative⁶

Hazards from food

An important development in improving food safety has been the application of the hazard critical control point concept (HACCP), which is a systematic approach to identifying, assessing, and controlling hazards, borrowed from the aerospace industry.⁵ It can be applied to all sectors of the food chain from primary production through food processing, manufacture, distribution, and retailing, to the point of consumption. Its strength is that it focuses on identifying the main avenues of risk and tackling them (box).

A food hazard is defined as “a biological, chemical or physical agent in, or condition of, food with the potential to cause an adverse health effect.”⁷ The acute hazards resulting from the consumption of food, such as allergy and food poisoning, are much easier to document than are the chronic harmful effects. The hazards associated with nutritional deficiency or nutritional imbalance (table 1) are recognised to be of great public health importance but are beyond the scope of this paper.

Risk is defined as “a function of the probability of an adverse health effect and the severity of that effect, consequential to a hazard(s) in food.”⁷ Risk analysis consists of three components: risk assessment, risk management, and risk communication. Risk assessment is the science of understanding hazards, how likely they are to occur, and the consequences if they do occur. The proc-

ess involves identifying and characterising the hazards, assessing exposure, and characterising the risk (box). Risk management is the process of weighing policy alternatives in the light of the results of risk assessment and, if required, selecting and implementing appropriate control options, including regulatory measures. It is essential that the risk assessment and risk management processes are transparent and separated, as one is scientific and the other is political. Risk communication is defined as “the interactive exchange of information and opinions concerning risk among risk assessors, risk managers, consumers and other interested parties.”⁷ It is perhaps this last stage that is hardest to deal with, as the distinction between risk assessment and risk management becomes blurred. A major barrier to risk communication is a general lack of understanding by the public of relative risk as opposed to absolute risk.⁸ Furthermore, public perception of risk is distorted by media reporting.⁹

Naturally occurring toxicants in food

Naturally occurring toxicants are ubiquitous in plants.¹⁰ People in developing countries are at much greater risk from naturally occurring toxicants because they have a limited dietary repertoire, they may out of necessity eat food which would otherwise be regarded as unfit for human consumption, and they may lack the resources to process it effectively into a safe form.^{11 12} Naturally occurring toxicants pose a relatively low risk to health in developed countries because effective food processing and a varied diet decreases exposure. Two exceptions to this rule are the toxicants present in wild mushrooms and herbal products.¹⁰

Microbiological hazards

The effects of foodborne infection are not restricted to the gastrointestinal tract, as illustrated by viral hepatitis, tuberculosis, and haemolytic-uraemic syndrome caused by *Escherichia coli* O157. Microbiological contamination of food and water is the main cause of diarrhoea, which contributes to about 3 million deaths among children aged under 5 (mainly in developing countries).¹³ Foodborne parasitic diseases are also a major public health problem in developing countries but not in developed countries. In addition, mycotoxins such as aflatoxin are known to present acute and chronic health hazards, particularly in tropical countries. In developed countries routine surveillance of mycotoxins,¹⁴ controls on the imports of potentially contaminated materials, use of fungicides, and good

Table 1 Risks associated with food hazards

Food hazards	Risk level	
	Developing countries	Developed countries
Nutritional deficiency	High	Low
Nutritional imbalance (for example, obesity, excess intakes of salt, saturated and <i>trans</i> fats)	Moderate	High
Natural occurring toxicants in food (for example, alkaloids, legume toxins, cyanogenic glycosides)	High	Low
Microbiological contamination (bacteria, viruses, parasites, mould, and algal toxins)	Very High	Moderate
Contaminants in food (heavy metals, organic chemicals)	Moderate	Low

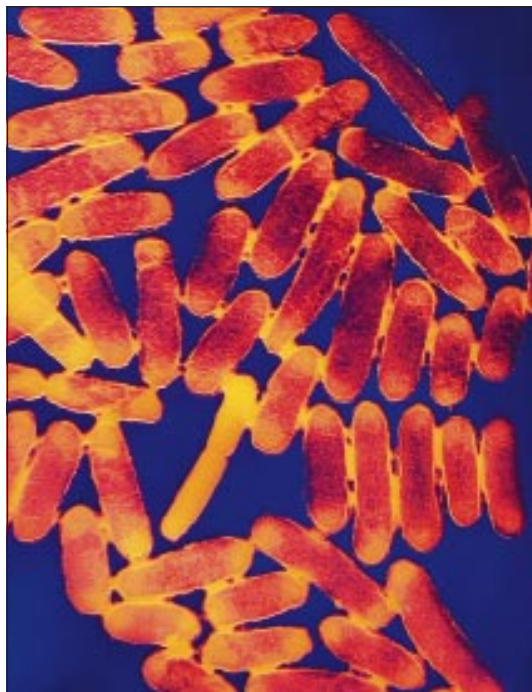
Risk analysis framework

- Risk assessment
 - Hazard identification
 - Hazard characterisation
 - Exposure assessment
 - Risk characterisation
- Risk management
 - Assess policy alternatives
 - Select and implement appropriate options
 - Interactive exchange of information and opinions
- Risk communication

storage conditions minimise exposure to mycotoxins. Algal toxins that accumulate in the marine food chain are a considerable hazard for some fish eating populations. Ciguatera is a sporadic form of human poisoning caused by the consumption of contaminated subtropical and tropical marine finfish (barracuda, grouper, snappers, mackerel) that causes neuropathy and can be fatal.¹⁵ Red tides of toxic algae known to cause paralytic and diarrhoeic shellfish poisoning affect waters around the British Isles between the months of May and August.¹⁶ Monitoring programmes minimise exposure of the population to these algal toxins.

The US National Food Safety Initiative attributes 9000 deaths and between 6.5 million and 33 million episodes of illness annually to foodborne microbial illness.⁶ In England and Wales, 300 deaths and 35 000 hospital admissions are attributed annually to infectious gastrointestinal diseases.¹⁷ Laboratory reports indicate that campylobacter, salmonella, rotavirus A, and small round structured viruses are the most commonly detected pathogens. Surveillance reports underestimate the true incidence of infectious gastrointestinal disease by two orders of magnitude; it is estimated that there are 9.4 million cases of infectious gastrointestinal disease in England each year.¹⁷ Table 2 shows the number of outbreaks reported by cause in the United Kingdom.

The emergence of new foodborne pathogens such as *E coli* O157, which has been detected in the faeces of up to 15% of British cattle, is of particular concern as beef is often consumed undercooked or rare. Intensive poultry production is linked to the epidemic of *Salmonella enteritidis* phage type 4 that has emerged in Europe and the United States: *S enteritidis* can be detected in 1% of eggs and in about a fifth of all poultry.¹⁹ There is, however, some evidence that application of the hazard critical control point concept in poultry production is



E coli O157 has been detected in the faeces of up to 15% of British cattle

Table 2 Infectious intestinal disease general outbreaks in England and Wales¹⁸

Organism	No of outbreaks						
	1992 (n=372)	1993 (n=456)	1994 (n=486)	1995 (n=833)	1996 (n=734)	1997 (n=561)	1998 (n=570)*
<i>Clostridium perfringens</i>	32	36	22	25	22	28	11
<i>Salmonella enteritidis</i> PT 4	94	108	60	73	61	79	44
<i>Salmonella enteritidis</i>	18	15	14	12	22	44	24
<i>Salmonella typhimurium</i>	22	19	20	19	18	20	7
<i>Salmonella virchow</i>	5	2	6	6	1	2	0
Other salmonellas	18	4	7	9	11	9	6
Scambrotoxin	1	2	8	9	6	7	4
<i>Campylobacter</i>	5	6	9	4	8	10	15
<i>Clostridium difficile</i>	4	8	36	32	30	9	23
<i>Cryptosporidium</i>	9	8	6	6	5	9	3
<i>Escherichia coli</i> O157	5	8	5	10	10	16	11
Rotavirus	5	8	17	23	31	12	16
<i>Shigella sonnei</i>	28	14	4	0	4	1	1
Small round structured virus	55	133	154	367	314	128	197
Other	11	11	24	22	23	6	2
Unknown	53	73	92	215	163	181	206

*Data for 1998 are provisional.

leading to a decline in *S enteritidis* infections in the United States and Europe.²⁰ Providing that poultry and eggs are cooked properly, the risk of food poisoning is low. Poor food hygiene and inadequate processing, particularly within the home, contribute to causing infectious intestinal disease but cannot be blamed for food poisoning outbreaks associated with shellfish, especially molluscs, which are particularly linked to viral infections.

Hazards from food production

Technological inputs (selective breeding, fertilisers, herbicides, pesticides, fungicides, etc) into farming have increased the efficiency of food production. However, inappropriate animal feeding practices and the use of agrochemicals may pose hazards to human health. Foodborne exposure to agricultural and environmental chemicals results in much public concern in the United Kingdom. Owing to exquisitely sensitive methods of detection, trace amounts of potentially harmful chemicals can be detected in many foods. However, the levels of human exposure to these chemicals are generally well below the tolerable daily intakes in the United Kingdom.^{14 21 22} In most developed countries the use and application of agrochemicals is carefully regulated, monitored, and reviewed. The appropriate use of agrochemicals in food production is a not a great hazard to human health.

The safety assessment of genetically modified foods poses a new challenge. The classical toxicological approach used for chemicals, which involves feeding animals intakes 100 times the amounts likely to be consumed by humans to demonstrate toxic effects, is not appropriate when applied to foods that may contribute up to 20% of the dietary intake. The UK Advisory Committee on Novel Foods and Processes considers each genetically modified food to ensure that there are no hazards associated with the method used to transfer the gene, that the genetic modification is stable, that the processing of the food denatures the DNA, that there are no new allergens, and that the food

Genetically modified foods approved for use in Britain²³

- Genetically modified foods:
 - Improved baker's yeast (1990)
 - Improved brewer's yeast (1991)
 - Processed products from glyphosate tolerant soybeans (1995)
 - Oil from glufosinate tolerant rapeseed (1995)
 - Oil from glyphosate tolerant rapeseed (1996)
 - Flavr Savr tomato paste (1995) and fresh Flavr Savr tomato (1996)
 - Processed products from glufosinate tolerant and BT toxin containing maize (1997)
 - Oil from bromoxynil tolerant cotton seed (1997)
- Food ingredients made from genetically modified micro-organisms:
 - Chymosin (rennet)—currently used to make most cheese in the United Kingdom (1991, 1992)
 - Amylase—used to clear haze in fruit juice (1994)
 - Riboflavin (1997)

is substantially equivalent in terms of chemical composition to the unmodified parent organism. Few genetically modified foods have been approved for food use in the United Kingdom (box), but many more have been approved and been in the food chain for a few years in Canada and United States.

The potential environmental and health risks from genetically modified foods have been discussed elsewhere.²⁴ No adverse reactions in humans to approved genetically modified foods have yet been reported. The continued use of antibiotics as growth promoters for poultry and pigs is of concern²⁵ because it has resulted in the emergence of multidrug resistant strains of pathogenic bacteria such as quinolone resistant *Campylobacter jejuni*²⁶ and *Salmonella enterica* serotype typhimurium DT104.²⁷ The sewage sludge generated from intensive poultry and pig meat production might be an important origin for the spread of antibiotic resistant genes and pathogenic bacteria into the food chain. The process of feeding infected bovine and ovine offal to cattle was responsible for the epidemic of bovine spongiform encephalopathy in the United Kingdom and is almost certainly responsible for new variant Creutzfeldt-Jakob disease, although the exact mode of transmission remains uncertain.²⁸ The *S enteritidis* and bovine spongiform encephalopathy epidemics underscore the importance of applying the hazard critical control point concept to the production of food animals.

Hazards from the industrial processing of food

The benefits of modern food processing are often taken for granted: increased availability of food, decreased cost, and convenience. Food processing is essential to feed a large urban population: it destroys naturally occurring toxicants and inhibits the growth and spread of pathogenic and spoilage organisms. Raw or unpasteurised milk and eggs are an important cause of food poisoning in both the United Kingdom and the United States.^{29 30}

In processed foods, food additives act as preservatives and processing aids and replace the colour and

nutrients lost during processing. The safety and use of food additives is strictly controlled by legislation. Allegations widely reported in the media a decade ago that food additives are a major cause of food allergy have not been substantiated.⁹ The hazards of allergic reactions to naturally occurring foodstuffs such as nuts, shellfish, and soy are much greater. Peanut allergy may affect 1-2% of the UK population³¹; it tends to be lifelong and can result in life threatening anaphylaxis.³² In the United Kingdom, products containing even traces of peanuts need to be clearly labelled. It is a paradox that peanut allergy is virtually unknown in tropical countries, where peanuts are eaten almost daily. One explanation for this could be that frequent infections in childhood dampen the response to allergens.

The mass packaging of food is an important barrier against microbiological contamination. However, the hazards resulting from the leaching of potentially accumulatively toxic compounds (such as vinyl chloride, phthalates, dioxins) from packaging material into foods, especially those with a high fat content, are currently being assessed.²²

Challenge of the future

Advances in technology have enabled world food supply to keep pace with population growth. However, each technology has its own risks. The world population is forecast to double over the next 50 years, and food production must increase to meet demands. The availability of water is a major constraint on food production in many parts of the world, and efforts will be needed to conserve water for food production. Biotechnology could help achieve the goal of sustainable development, which recognises the need for technology without environmental damage. An efficient food industry and distribution system can also decrease waste.

To achieve a safe food supply it is necessary to apply the hazard critical control point concept and risk analysis to the food chain and to enact legislation, where appropriate, to ensure that training is undertaken and practices are followed and that monitoring and surveillance occur. Education is also needed to alert consumers to risks from food and how to minimise them. Special consideration is needed for fish and shellfish as they are particularly prone to both environmental and microbiological contamination.^{22 33} Global warming could dramatically change the geographical distribution of algal toxins. Finally, internationally agreed food standards are essential to facilitate trade between countries. All of these challenges require a sophisticated infrastructure, which in some parts of the world, particularly Africa, is being destroyed by war.

Competing interests: TABS has been paid for participating in workshops on food safety by ILSI Europe and acts as a consultant to Seven Seas Ltd and the Nutrasweet Information Service.

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A lesson learnt

The virus and the hookworm

Hookworm infestation with the nematode *Necator americanus* is endemic in the highlands of Sri Lanka. Patients present profoundly anaemic with a characteristic facial appearance that often lends itself to a "spot diagnosis." Indeed, it was common for an intern to tell a colleague in passing, "I see you've got another hookworm coming in."

Hepatitis A, or infectious hepatitis as it was known a few decades ago (to distinguish it from hepatitis B or serum hepatitis), was also a common infection. At any time, the medicine ward would include three or four patients so afflicted. As inspection of the urine was a better index of jaundice than examination of the eyes, clear glass jars containing a morning specimen of urine could be seen by each patient's bedside. Then, as now, the treatment was largely supportive. As managed care was a phrase yet to come, patients remained in bed for about three weeks, and when it was deemed that the patient had convalesced enough, plans for discharge were initiated.

When I was an intern, it was such a patient who taught me a lesson that I shall never forget. Examination of the stools for parasitic ova and cysts was routine for all inpatients, regardless of the reason for admission. Helminthiasis was so prevalent that eradication of asymptomatic infestation was the usual practice. The patient's stool had yielded hookworm ova, and on the day before discharge I ordered the standard dose of trichlorethylene (TCE). This was the treatment of the day, and, although not as effective as the drugs now available, it had a high success rate in eliminating the parasite.

Of course, a recurrence of illness was the rule rather than the exception, and there was no way you could tell whether the recurrence was because of incomplete eradication or reinfection. Nor did it matter. On the morning of discharge the patient was drowsy, and I rather naively attributed his somnolence to a poor night's sleep. The consultant was more impressed by the patient's appearance than by my explanation. He reached for the chart and studied it. "I see that you have prescribed TCE for this patient," he said, and misinterpreting this as a compliment I

responded, "Yes, because his stools contained hookworm ova, and I thought it best to treat him before he left hospital." "Do you know the formula for trichlorethylene?" he asked, and with increasing pride I replied, "Yes Sir: C₂HCl₃." "And what," he asked, "are the agents used for the experimental induction of hepatic necrosis?"

I still suspected nothing. Remembering an old mnemonic from pathology, P for phosphorus that causes peripheral necrosis, and C for carbon tetrachloride that results in centrilobular necrosis, I answered with some satisfaction, "Phosphorus and carbon tetrachloride." "And what," continued the consultant, "is the formula of carbon tetrachloride?" And that was when the penny dropped, as did my heart. As I responded, "CCl₄," I knew what was coming next. "I hope you realise," he said, "that you have administered a highly hepatotoxic drug to a patient whose liver is recovering from hepatitis; a drug that is different by but two atoms from a powerful toxin." I said nothing; what could I say? And then he used the same phrase that has been used before, "Never again."

Results of the thymol flocculation and zinc turbidity—liver function tests used at the time—confirmed what we already knew; there was a marked deterioration. Fortunately, a few more days of tender loving care resulted in complete recovery. As I saw the patient walk out of the ward, I said to myself, "Never again."

Sundaram V Ramanan, *associate professor of clinical medicine, University of Connecticut School of Medicine*

We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake*, or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk. Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.