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Food safety in the human food chain

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2 The magnitude of the problems

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INTRODUCTION

Absolute food safety is impossible. There is virtually no component of our food supply that is without some risk for some part of the population. An *objective* ranking of the hazards associated with food consumption can be produced by considering the severity, incidence and onset of their physiological consequences (Wodicka, 1971a). *Severity* indicates the type of effect involved, ranging from mild and temporary discomfort through more serious but reversible effects, to irreversible effects, incuding death. *Incidence* refers to the number of cases or rate of occurrence of a given effect. *Onset* denotes the time of occurrence of the effect after exposure to a hazard and can range from immediate to long term. The risk from microbial contamination is a thousandfold greater than that due to environmental contamination, while the risk due to pesticide residues and food additives is about a hundred times smaller again. Naturally occurring compounds in food are far more likely to cause toxicity than intentional food additives (Roberts, 1981).

The *perception* of the risk from natural toxicants is very different. Hall (1971) ranked these same hazards according to the perceptions of various sections of society – the media, the food industry, the regulatory authorities and that section of consumers who were particularly vocal in calling for safer food. In all cases natural toxicants were regarded as the least significant.

MICROBIOLOGICAL HAZARDS

Despite the care and efforts of those involved in the whole of the food chain it is apparent that the number of reported cases of food-borne illness is increasing in developed countries. At this point it is helpful to consider which are the micro-organisms of particular concern (see Table 1). Listing recent major food-borne outbreaks of illness and their reported costs gives an indication of the extent of the microbiological problems. Thus in the 1960s and '70s there were outbreaks of intoxication associated with *Staphylococcus aureus* in cheddar cheese. More recently, *Salmonella* has caused illness after consumption of a variety of foods including chocolate, cheddar cheese, pasteurised milk, infant dried milk and fermented meat, while *Listeria monocytogenes* in cheese and *Clostridium botulinum* type B in hazelnut yoghurt have also produced outbreaks of food poisoning in the last four years. It is important to note that some of these outbreaks have not only been very large in terms of numbers reported ill and the fatalities produced but have also been extremely expensive to the companies concerned. Thus there are reported to be law suits amounting to over £400 million associated with *L. monocytogenes* in cheese in the USA, while *Salmonella* in infant dried milk cost the company concerned over £22 million.

The total annual cost of food-borne diarrhoeal disease in the USA has been estimated as between $5 and $17 billion (1985 figures). These costs include only those for medical care and lost productivity of acute episodes and exclude the effects of chronic disease, death, loss of leisure time, cost of litigation and cost to industry (including lost business, product recalls, seizure and destruction) (Archer, 1986). Even fear of food-related disease can be expensive and earlier this year the UK Government was prepared to spend £20 million in compensation for egg producers when concern about *Salmonella enteritidis* in eggs was at its height.

The major outbreaks indicate some of the main food-poisoning micro-organisms but the number of individual cases reported perhaps provides a better guide to the more important micro-organisms concerned with food-borne illness.

In England and Wales the Central Public Health Laboratory Service at Colindale regularly publishes statistics on food-borne disease. Their studies show that reported cases of food poisoning caused by *Salmonella* and by *Campylobacter jejuni* have increased rapidly over the last four years.

Salmonella

In particular, within the last two years *Salmonella enteritidis* has caused special problems and now represents something like 56% of the reported cases of salmonellosis (with phage type 4 causing 72% of this 56%). Despite the unusually high temperatures and the increased publicity, reported cases of *Salmonella* are only about 7% more than those reported last year (Anon,

Table 1
Cost of outbreaks of food-borne illness

Year & Country	Food	Number reported ill	Cause	Cost £
1965-USA	Cheddar cheese	42	*Staphyloccoccus aureus*	250 000
1977-Canada	Cheddar cheese	15	*Staphyloccoccus aureus*	300 000
1982-UK (Italy)	Chocolate bar	245	*Salmonella*	505 000
1983-USA	Milk	49 (14 deaths)	*Listeria monocytogenes*	294 000
1984-Canada	Cheddar cheese	2700	*Salmonella*	5 880 000
1985-USA	Pasteurised milk	18 000 (2 deaths)	*Salmonella*	Dairy closed
1985-USA	'Mexican Style' cheese	142 (47 deaths)	*Listeria monocytogenes*	Reported lawsuit of 411 764 000
1985-UK	Infant dried milk	76 (48 infants) (1 death)	*Salmonella*	22 058 000
1986-UK	Pasteurised milk	54	*Salmonella*	167 650
1987-Switzerland	Vacherin Mont D'or cheese	30 deaths	*Listeria monocytogenes*	882 352
1988-UK (Germany)	Pepperami	81	*Salmonella*	>1 000 000
1989-UK	Hazelnut yoghurt	27 (1 death)	*Clostridium botulinum* Type B toxin in hazelnut purée	?

Source: Author's own data.

1989). A similar problem has also been reported from other countries, including the United States where a different strain of *S. enteritidis* was reported as causing salmonellosis some five years before the problem became widespread in the United Kingdom.

Such figures, however, reflect only the number of *reported* cases and the *total* number of cases may well be much higher. An American study has suggested multiplying the number of reported cases by a hundred-fold to

arrive at the total, although more usual multipliers are thirty- or ten-fold. Certainly many cases of food-borne illness are never reported because the illness is slight. However, serious cases of illness or those involving large numbers of individuals are always reported and, although the reported cases represent the tip of the iceberg, they are the most important part. In addition, it is necessary to get the whole problem of food-borne disease into perspective. Given that there are about 50 000 food-borne illnesses reported each year in England and Wales, taking the worst-case scenario and assuming that this represents only 1% of the total number of cases, this would represent 5 million meals a year which produced illness. However, such a figure would be only 0.01% of the total number of meals eaten.

Listeria

With *Listeria monocytogenes* the number of reported cases of listeriosis have increased from 25 in 1967 to 291 in 1988, with a near doubling between 1986 and 1987, although the increase in 1988 was much less. Unfortunately, listeriosis has a high fatality rate with about 30% of reported cases resulting in death if abortions are included. Nevertheless, up to the end of 1988 in England and Wales only four documented cases of listeriosis were proven to be food related. In these cases *L. monocytogenes* bacteria of the same serogroup and phage type were isolated from both the patient and the incriminated food (see Table 2). There are a few other reported cases in which a suggestive link to a particular food has been detected but in none of these cases is the evidence supporting the link convincing.

L. monocytogenes is common in the environment and its level in agriculture and hence in food may be increasing because of changes in agricultural practice (Fenlon, 1985). The organism is not especially heat resistant and is destroyed by cooking or by pasteurisation. However, surveys by the PHLS have found that some mould-ripened cheeses may carry more

Table 2
Confirmed cases of food-borne listeriosis in England and Wales to December 1988

Year	Cases	Food Vehicle	Serotype
1986	1	Imported soft cheese	4b
1988	1	UK goats' milk cheese	4b
1988	1	Cook-chilled chicken	4*
1988	1	Vegetable rennet	4*

* Serotyping not yet completed.
Source: From R J Gilbert (1989).

than 10 000 organisms/g and there is an obvious need for research on the ability of *L. monocytogenes* to grow in such cheeses during ripening and storage, as well as on the sources of the organism within both natural and man-made environments. Information on the minimum infective dose is also required but is very difficult to obtain, although the possibility that there are strain differences in virulence requires further research.

Sources of food contamination

In terms of preventing food-borne disease and intoxication, there is much evidence from England and Wales, as well as from the USA and Canada, that the majority of incidents of food poisoning result from mistakes during preparation of food, rather than errors produced during food manufacture (Table 3). Given the widespread occurrence of such organisms as *Salmonella* and *Listeria monocytogenes* in the environment, it will never be possible to produce food free of risk, unless it is given a commercial sterilization, as in canning. Even canned food is not risk free, as the presence of *C. botulinum* in canned salmon showed in Birmingham in 1976. Given this background it would seem that the known microbiological problems must be the major ones associated with food.

Other micro-organisms

Bovine spongiform encephalopathy

In addition, there are other microbiological problems. For example, bovine spongiform encephalopathy (BSE) has recently caused concern amongst consumers, given that it may be derived from scrapie by ingestion of contaminated sheep meat. The apparent ability of this infectious agent to jump across normal species barriers is unexpected and it has been suggested, therefore, that BSE could also pose a risk to man. At present there is no scientific evidence for this, although because of the long time taken for symptoms to appear with such slow agents of infection, it would be unlikely for anyone to develop symptoms for perhaps ten years after infection. Work is currently underway to determine if there is any relationship between these diseases and such human diseases as Creutzfeldt-Jacob Syndrome.

Mycotoxins

Other microbiological problems arise from the production of mycotoxins, which are metabolites produced by mould growth in food (or animal feed). In general, mycotoxins are secondary metabolites with low molecular weights and most can survive treatments such as heat and drying, which kill the mould itself. Toxins such as aflatoxin may be involved in liver cancer in less developed countries. In the USA it has been estimated that the average daily intake of aflatoxin B is about 20 ng/kg body weight, although it is not clear what the absolute risk factors are. Foods of particular concern (peanuts and

Table 3
Factors contributing to 1479 outbreaks of food poisoning in England and Wales, 1970–1982

Contributing factors	Number of outbreaks in which factors were recorded (%)
Preparation too far in advance	844 (57)
Storage at ambient temperature	566 (38)
Inadequate cooling	468 (30)
Inadequate reheating	391 (26)
Contaminated processed food	246 (17)
Undercooking	223 (15)
Contaminated canned food	107 (7)
Inadequate thawing	95 (6)
Cross contamination	94 (6)
Raw food consumed	93 (6)
Improper warm holding	77 (5)
Infected food handlers	65 (4)
Use of left-overs	62 (4)
Extra large quantities prepared	48 (3)

Source: Roberts (1988).

cocoa) can be screened by visual inspection (in the case of peanuts) and, although it is not uncommon to find mycotoxin-producing moulds in cocoa, raw cocoa beans do not readily support their production (Llewellyn *et al*, 1978).

Other bacteria
Other bacteria which are already recognised as likely to cause problems in the future include *Aeromonas hydrophila* (Majeed *et al*, 1989) and also *Escherichia coli* 0157:H7, strains of which produce verotoxins and have already caused outbreaks of illness and some deaths in the USA. Little is known about the presence of these organisms in the UK food supply.

PESTICIDES

Alar

With regard to pesticide residues, there has been much publicity recently about Alar, which is actually a plant growth regulator and not a pesticide. This compound behaves as a synthetic plant hormone and is used to assist

apples to set fruit and mature. It helps to minimise the premature drop of the fruit and also to produce fruit of uniform size. In the USA the Natural Resources Defence Council have suggested that between 5500 and 6200 of the current population of American pre-schoolers may eventually get cancer solely as a result of exposure to eight 'pesticides', including Alar.

In fact, the quantitative claims of cancer risk are difficult to sustain and are not universally accepted by toxicologists. The mathematical models used to extrapolate from high dose studies in animals to low dose exposure in man are of very doubtful validity and are not an accurate estimate of risk. The problems relate to such assessments being based on a non-threshold, single-hit hypothesis. In addition, the models do not take account of protective mechanisms and DNA repair processes, nor of metabolic and pharmacokinetic discontinuities which can lead to 'overdose' toxicity at very high dose levels and are irrelevant at very low doses. The Surgeon-General of the US Department of Health and Human Services has said that he believes that it is safe to eat apples treated with Alar although the company producing Alar has now withdrawn it from sale. The risk from the seven other pesticides considered in the Natural Resources Defence Council report is even smaller than that from Alar and its breakdown products.

In general in food toxicology there is a universal tradition of use of the 'threshold' concept and arbitrary arithmetical extrapolation to suggest a 'safe' dose. Unfortunately, the present understanding of and, methods for, quantitative risk assessment are crude, often inaccurate and may even be grossly misleading. It is apparent that further research is needed.

ENVIRONMENTAL CONTAMINANTS

Environmental contaminants include trace elements and organometallic compounds (for example, arsenic, mercury, cadmium, tin and lead) plus organic polychlorinated biphenyls (PCBs) and the halogenated hydrocarbon pesticides. Such contaminants tend to be stable and persistent in the environment and to bioaccumulate in the food chain. They can be transformed with increased toxicity. Some, (such as lead, mercury and PCBs) are of special concern in the case of the foetus, infants and young children because of greater retention as well as susceptibility. Their incidence is difficult to quantify.

Accumulation occurs particularly in fish and their toxicity is usually greater in mammals and especially in primates than in other animals. The US Environmental Protection Agency lists over 43 000 chemical substances in its inventory of chemicals subject to the Toxic Substances Control Act (1979). However, under most conditions of use, these chemicals do not pose as much of a threat as micro-organisms to the safety of the food supply.

NATURAL TOXICANTS

As discussed earlier, contrary to popular perception, the risks to health posed by naturally occurring, biologically active compounds may be greater than those due to pesticides, residues or food additives (Wodicka, 1971b). For example, many higher plants contain potentially hazardous chemicals of natural origin which can survive processing and cooking (Table 4). Ames (1983) has established that human beings ingest such compounds at levels 10 000 times in excess of man-made pesticide residues, and relatively few of these compounds have received a fraction of the toxicological evaluation devoted to synthetic food additives and contaminants.

Table 4
Principal natural toxicants found in the UK diet

Compound	Common source
Cyanogenic glycosides	Legumes, fruit kernels
Lectins (haemagglutinins)	Legumes
Protease inhibitors	Legumes
Glucosinolates	Brassicas
Biogenic amines	Cheese, chocolate, wine
Lathyrogens	Chickpea
Favogenic factors	Fava (broad) beans
Alkaloids	Herbal teas
Glycoalkaloids	Potatoes
Phytoestrogens	Legumes
Saponins	Legumes
Tannins	Widespread
Psoralens	Umbelliferae (carrot etc)
Terpenes	Widespread
Ptaquiloside	Goatsmilk (via bracken)

Source: Roberts (1981).

Effects of dietary changes

In the UK the risk of acute poisoning due to natural food constituents has been limited to a few relatively well known compounds; such outbreaks are rare, although changes in culinary fashions can pose new risks, for example, the recent cases of poisoning from kidney bean lectins (see Bender & Reaidi, 1982). The increased consumer interest in environmental and dietary issues seems likely to lead to greater intakes of vegetables and cereals. Developments in food processing technology will also lead to increased

availability and palatability of such foods and their associated by-products. Further studies in this area are required and the situation needs to be kept under constant review.

Vegetarianism

The following example of increased intake has been noted among vegetarians. For omnivores in the UK, the mean daily intakes of saponins and oestrogenic isoflavone glycosides are about 20mg and <1mg, respectively (Jones, et al, 1989), whereas in vegetarians the corresponding figures are >200 mg and >100 mg, respectively.

There has also been a recent increase in the popularity of potato skins, especially in the form of baked potatoes. This is likely to lead to an increase in the consumption of glycoalkaloids such as chaconine and solanine. Recent work has suggested that these compounds can increase the permeability of gastrointestinal cells to other non-nutritive food constituents. However, the significance of this for man is unknown.

Pyrrolizidialkaloids are also acutely toxic and have led to human fatalities. Since these are present in 'health-foods' such as herbal teas and some plant materials taken as dietary supplements there is the possibility that the prolonged intake of such compounds could present future problems (Ridker, 1987).

Pig-bel

Before leaving the area of the effect of dietary changes there is the interesting possibility of a recurrence of a little known disease amongst vegetarians. This is pig-bel which was common amongst the inhabitants of Papua-New Guinea some years ago. Basically, the bacterium *Clostridium perfringens* type C grew rapidly in the intestine, produced toxin and led to the breakdown of the gut wall and the digestion of the entire abdominal cavity and subsequent death. The reasons for this problem included the fact that the diet was vegetarian which reduced the level of proteases in the alimentary tract; type C toxin is broken down and detoxified by proteolytic activity in the normal gut. In addition, the diet was high in sweet potato containing proteolytic inhibitors, which reduced the activity of any proteases produced. It is an interesting possibility that vegetarians may be more prone than omnivores to the effects of *C. perfringens* and that vegetarians who have occasional lapses into meat eating may be especially at risk.

FOOD ADDITIVES

There is general acceptance among food scientists that food additives should be ranked in the lowest risk category (Anon, 1971; Wodicka, 1973; Hall, 1973a; Roberts, 1976). The US FDA (Food and Drug Administration)

lists about 2800 additives (Lehmann, 1979) including such familiar items as salt, pepper, sugar, mustard and yeast. In addition, the FDA has estimated that there may be more than 10 000 indirect additives which appear in food, for example, as a result of contact with packaging materials. To try and put this into perspective it is worthwhile making a comparison of the levels of natural toxicants in our food. For example, based on an average annual consumption of 119 pounds of potatoes, it has been estimated that we ingest 9700 mg of the alkaloid solanine each year (Hall, 1973b) although the median annual ingestion of direct food additives is about 0.5 mg. The US National Academy of Sciences has reviewed this area (1973).

CONCLUSIONS

The need for vigilance

It is apparent that, as with every other of man's activities, eating can be bad for one's health. Given the rapidly changing methods of food production and the new and unusual diets which become fashionable there is a need for constant vigilance in considering our food. Published statistics and the evidence of scientists and public concern all suggest that microbiological hazards are more important than other hazards in our food. Many microbiological problems are well understood and improved education of food handlers would markedly reduce the number of cases of food-borne disease caused by mistakes in food preparation. The licensing of catering establishments and an increase in monitoring by increasing the number of environmental health officers are also vital if food poisoning is to be reduced.

The need for research

It is apparent that changes in methods of agriculture and in food manufacture have led to the emergence of micro-organisms not previously recognised as important as causative agents of food-borne disease. In the case of organisms such as *Listeria monocytogenes* their importance as agents of food-borne disease is unclear. However, even one death from food poisoning is too many and research is urgently needed to fill the gaps in our knowledge. In particular, rapid methods of detection, enumeration and strain differentiation are required to allow the sources of contamination and the level in both natural and man-made environments to be detected. In addition, the growth characteristics of such newly recognised food poisoning organisms as *Escherichia coli* 0157:H7, *Aeromonas hydrophila* and *Listeria monocytogenes* must be determined if we are to reduce the magnitude of the problems in food safety which are currently facing us.

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