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Foods and microbiological risks

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Scope

This paper considers the significance of foodborne illness of worldwide note, the reasons for significant increases in reported cases in many countries, and what steps could be applied to reduce significantly the incidence of such illness.

Significance of foodborne illness

Illness caused by the consumption of food contaminated with infectious and toxigenic micro-organisms is a major cause of suffering and a very significant cause of death throughout the world (Allen & Kaferstein, 1983). In Africa, Asia (excluding China) and Latin America, it has been estimated that annually there are more than 1000 million cases of gastro-enteritis amongst children under the age of five and up to 5 million deaths, and most are caused by the consumption of contaminated food (Kaferstein & Sims, 1987); in some countries, e.g. Mexico and Thailand, half of the children aged between 0–4 years suffer from Campylobacter enteritis alone. In Europe, morbidity from foodborne illness is second only to respiratory diseases and recent estimates of illness would indicate that in many European countries there are at least 50000 cases of acute gastro-enteritis per million population per year with a figure of 300000 recently suggested for the Netherlands (Guiguet *et al.*, 1992; Notermans & van der Giessen, 1993). Estimates for the USA are even higher and one recent estimate suggests that up to 350000 persons per million population per year suffer acute gastro-enteritis and the majority of this is probably associated with contaminated food (Archer & Kvenberg, 1985). Whilst death rates from foodborne illness are considerably lower in developed than in developing countries, it is increasingly recognized that between 1 and 5% of episodes of acute gastro-enteritis lead to serious, and often chronic, sequelae including rheumatoid conditions (such as ankylosing spondylitis and Reiter's syndrome), nutritional and malabsorption problems, haemolytic-uraemic syndrome (caused by verotoxin-producing strains of *Escherichia coli*, and particularly serotype O157:H7), and other illnesses such as atherosclerosis, and Guillain–Barré syndrome following infection by *Campylobacter* spp. (Archer, 1984, 1987; Archer & Young, 1988; NACMCF, 1993; Smith *et al.*, 1993). The cost of foodborne illness in economic terms is enormous. Thus a figure of 8·4 billion dollars per annum has been estimated for the USA by Todd (1989).

Most countries report salmonellosis and Campylobacter enteritis as the principal causes of foodborne illness, but as might be expected there are some country differences, this somewhat mirroring eating habits. Thus in Japan, raw fish is a major component of the diet and thus the marine micro-organism *Vibrio parahaemolyticus* is one of the main causes of food poisoning. Other interesting country differences can be found amongst the foods causing specific illness. Thus in Europe, botulism from the consumption of fish occurs most frequently in Scandinavian countries, whereas in middle and eastern European countries most cases result from the consumption of meat; vegetables (including plant-based products) are most often involved in the USA (Baird-Parker, 1969).

From time to time there have been reports of the emergence of new pathogens, but in reality they are mostly the recognition of organisms that have probably caused foodborne illness for many thousands of years. However, *E. coli* O157:H7 is probably an example of a new organism as genetic evidence suggests that this organism has evolved relatively recently from an enteropathogenic progenitor. From a rare organism, when first identified as causing foodborne illness in 1982, it has now become a relatively common and important foodborne pathogen (Griffin & Tauxe, 1991).

The increase in numbers of recognized types of foodborne illness-causing organisms from the 1950s to now is illustrated in Table 1; the number of types has nearly trebled in 40 years. Food poisoning is far from under control. For instance in the 5 year period from 1985 to 1989 the reported incidence of food poisoning in many European countries (including the UK) nearly doubled and in most countries there has been a significant increase (WHO, 1992). Much of this increase has been associated with a specific phage type of *Salmonella enteritidis*, i.e. phage type 4 (Baird-Parker, 1991).

Table 1. Types of micro-organisms causing foodborne illness

1950s types	Additions since 1950s
Bacillus anthracis	*Aeromonas hydrophila* and *sobria*
Bacillus cereus	*Bacillus subtilis* and *licheniformis*
Brucella spp.	*Campylobacter jejuni, coli* and *upsaliensis?*
Clostridium botulinum	*Cryptosporidium* spp.
Clostridium perfringens	*Giardia* spp.
Escherichia coli (toxigenic)	Enterovirus e.g. hav srsv
Mycobacterium bovis	*Escherichia coli* (5 types)
Salmonella	inc. enterohaemorrhagic
Shigella spp.	*Listeria monocytogenes*
Staphylococcus aureus	*Plesiomonas shigelloides*
Streptococcus pyogenes	*Streptococcus zooepidemicus*
Taenia spp.	*Toxoplasma gondii*
Vibrio cholerae	*Vibrio fluvialis*
Vibrio parahaemolyticus	*Vibrio vulnificus*
	Vibrio cholerae non-O1
	Yersinia enterocolitica

Reasons for increased incidence of foodborne illness

The reason for the increase in reported illness is probably the result of a combination of factors, including: better reporting; changes in agricultural practices; changes in food marketing and eating habits; identification of new pathogens (including the development of better microbiological methods); and changing population sensitivities. Some of these factors appear more important than others, but it is useful to consider them all, in turn, as we have much to learn about their relative importance.

Reporting

In all countries, there is considerable under-reporting of foodborne illness cases to the authorities and thus even in countries that have well-developed epidemiological surveillance systems such as the UK, the true extent of foodborne illness is largely unknown (Richmond, 1990, 1992). A detailed study of a number of outbreaks of foodborne illness in North America resulted in an estimated ratio of actual to reported cases of 25 to 1 (Hauschild & Bryan, 1980). The deficiencies in reporting systems are well illustrated by a 'Sentinel study' recently carried out in the Netherlands, where patients of designated General Practitioners with symptoms of gastrointestinal illness were asked to fill out a questionnaire, and to submit faecal samples for microbiological examination. It was found that the ratio of actual to reported cases was closer to 100 to 1 and that the relative proportions of organisms causing foodborne disease were quite different to those derived from reports submitted to the authorities (Notermans & van der Giessen, 1993).

In 1990, the World Health Organization (European Region) held a number of Consultations on the reporting of national foodborne illness statistics in an attempt to improve the reporting of foodborne illness, so that more cases can be identified together with information on the source and cause of illness (WHO, 1990). Even when a laboratory investigation is made, and the causative agent identified, the source is only identified in about 20% of infectious-pathogen-caused outbreaks in the UK, and in most countries this figure is even lower; the success rate for sporadic cases of infectious foodborne illness is even lower. The success rate for outbreaks of toxin-caused illness is good with a figure of 85% or more being generally achieved for incidents in England and Wales (Richmond, 1990). Foodborne illness statistics will never be more than a tip-of-the iceberg, because the majority of persons suffering illness do not consult their doctor and they mostly do not need to do so. Sentinel studies will improve our knowledge of incidence and causative agents of sporadic cases, but are less likely to provide more information about sources, or reasons why a foodborne illness occurred. As reporting systems improve, and particularly as more computer-based databases become available, and the data are analysed for trends, and changes in expected numbers and types from historical data are used as an alert mechanism, more sporadic cases will be linked to a common source and investigated. Also, causative agents may be more often identified as detection methods improve, e.g. for viruses, protozoa and such entities as 'viable non-culturable forms' of bacteria. Although reporting systems have improved in recent years this is not usually a major factor for increased reported incidence in countries such as the UK, who have recorded reportable cases of foodborne illness for more than 50 years. Evidence from other sources, such as the Sentinel Practice scheme in the UK, can be used to support this conclusion (Richmond, 1990).

Changes in agricultural practices

There is good evidence that agricultural practices do affect the incidence of foodborne illness-causing micro-organisms in the intestinal tract and on the surface of animals. Also, transportation to the slaughter house and other conditions generating stress will also increase numbers (ICMSF, 1980). In general, intensive farming and associated practices (such as recycling waste products of animal and meat production) intended to improve production efficiency, increase the potential for infection of our food animals with zoonotic micro-organisms. There is a particularly strong correlation between the increasing incidence of human salmonellosis, and more recently Campylobacter enteritis (as mentioned previously, the main cause of gastro-enteritis in many countries) and the increased consumption of chickens (Palmer & Rowe, 1986; Report, 1975–1977; Richmond, 1990; WHO, 1992). The reality of this association is based on a number of facts. Firstly, laboratory and epidemiological studies clearly demonstrate that the main serovars of salmonella found on chicken, and particularly broilers, commonly cause human salmonellosis. There are many examples of this association (Palmer & Rowe, 1986) and the most recent is that of chicken and hen eggs, with

salmonellosis caused by *Salmonella enteritidis* phage type 4 being identified in about 50% of human cases in England and Wales (Report, 1993). Human salmonellosis caused by this phage type occurs in many countries throughout the world, but only in countries where the organism has become established in poultry flocks (Baird-Parker, 1991). Secondly, there is good evidence from surveillance studies that there is a much higher incidence of Salmonella and Campylobacter on poultry than other foodstuffs; incidences of between 30 and 80% on poultry carcasses are regularly reported, with the highest incidence in the summer months (WHO, 1989). Thirdly, in many countries with an established broiler industry, and in countries where broilers are introduced, there is evidence of increased reported cases of salmonellosis and Campylobacter enteritis with increased consumption of poultry in the diet. Finally, in countries where Salmonella contamination in poultry is low, for instance in Sweden, the incidence of salmonellosis ascribable to food products consumed within the country is also low (WHO, 1989, 1992). Despite the clear relationship with poultry consumption and foodborne illness it would be wrong to solely blame the poultry industry, as whilst poultry may be the main source of Salmonella entry into the food chain, the level of contamination on carcasses is usually very low and below that which will cause illness. The conditions that lead to salmonellosis are usually the result of cross-contamination from raw to cooked products in the kitchen and recontamination of the cooked chicken followed by storage at temperatures that allow growth. Undercooking of meat and holding at warm temperatures are also well-recognized risk factors for Salmonella and Campylobacter enteritis and the increasing popularity of barbecues has been associated with increased foodborne illness. There can be little doubt that changes in agricultural practices (and to some extent high speed slaughter and evisceration lines) have contributed significantly to the increased contamination of our meat and this is, in my view, a major cause of the increased incidence of human salmonellosis and Campylobacter enteritis.

Changes in food manufacture and food consumption practices

Over the past 10–15 years, the main trends in food consumption in developed countries have been increased consumption of pre-prepared (convenience) food in the home, and greater consumption of food outside the home, and particularly in fast food restaurants. Over this period there has been an increase in the consumption of chilled and frozen food, and increased consumption of poultry and fish, reduced use of salt (for dietary and health reasons) and less use of chemical preservatives such as sorbate and benzoate for reasons of consumer concern (Gould, 1989). Thus the trend is for foods that are perceived to be more natural, fresher, healthier and more convenient, e.g. can be cooked or heated for consumption in a microwave cooker. Whilst there is no evidence that these particular trends (other than increased consumption of chicken) have led to increased food poisoning, it must be recognized that foods are becoming less microbiologically robust, requiring greater care in their production, distribution and storage.

Various estimates have been made of the involvement of manufactured foods in foodborne diseases and in terms of outbreaks this is small, e.g. less than 5% for England and Wales (Sockett, 1991), and similar figures have been reported for North America (Bryan, 1988). However, it is often argued that this may not reflect the true incidence, because of under-reporting and the fact that most sporadic cases of food poisoning are never traced to a food source. There is some evidence from surveillance studies that much of the food poisoning occurring in the home (and most does) is not because the consumer buys unsafe food but because of poor hygienic practices during storage and preparation of foods for consumption. Hence the drive for better consumer hygiene education and information (Richmond, 1992). However, there is no room for complacency by food manufacturers, as when there are breakdowns in food manufacturing practices, the consequences can be catastrophic. Thus as a result of a processing equipment fault at a milk-pasteurizing plant in Chicago in 1985, there were an estimated 150000 persons ill from salmonellosis (Bean *et al.*, 1990).

There continue to be many reports of illness from foods served in restaurants, hotels, canteens, hospitals, institutions etc., and most of the bad practices that lead to food poisoning are the same as those occurring in the home. However, the consequences of these are very much more serious because of numbers of persons exposed to the risk. Such practices are normally the result of ignorance and sloppy practices resulting from poor management. The requirements for food handlers to be properly trained (Directive, 1993) will undoubtedly continue to improve the handling practices and very significant progress has already been made by the health authorities in the UK. Many of the outbreaks of foodborne illness caused by *E. coli* O157:H7 are the result of under-cooking of hamburger meat. The largest, and most recent of these, occurred in January 1993 in a chain of fast food restaurants in the USA. The cause was undercooking of raw meat, and the result was more than 600 persons ill (mainly children), many hospitalized cases, including 35 cases with haemolytic-uraemic syndrome and three deaths (Tarr, 1993). Thus there is evidence that consumption of food outside the home is a significant cause of illness (outbreaks are increasing) but the jury is still out on manufactured foods and food poisoning in the home. The trend to less well preserved food will potentially increase foodborne illness risks unless better control procedures are applied throughout the food chain.

Newly identified and emerging pathogens

As a result of clinical, epidemiological and laboratory investigations a number of so-called 'emerging' and 'new pathogens' have been identified as food poisoning organisms. The list of organisms implicated in foodborne illness continues to grow and the current recognized foodborne pathogens are listed in Table 1. Some of these newer organisms are clearly associated with unsafe practices

such as the consumption of under-cooked hamburgers and raw milk contaminated with *E. coli* O157:H7 (Griffin & Tauxe, 1991). But some may be associated with consumer demands for fresher food, e.g. there appears to be a relationship between increased consumption of chill-stored chicken and Campylobacter enteritis (Richmond, 1990), and some may be the recognition that persons with certain underlying diseases may be particularly susceptible to illness. For instance, persons with underlying liver disease are highly susceptible to *Vibrio vulnificus* infections and such persons are warned in the USA not to eat raw oysters (Eastaugh & Shepherd, 1989). Particularly acute forms of listeriosis are often associated with persons with reduced immunity (Lovett, 1989).

We are still learning of the significance of some pathogens: the so-called 'viable non-culturable' organisms are potentially important as they may provide (but this is yet to be proven) a means of spreading infectious and resistant forms of sensitive micro-organisms (such as *C. jejuni*) through the environment (Rollins & Colwell, 1986; Jones *et al.*, 1991). The main concern we have with these new pathogens is that whilst with the clear exception of *C. jejuni* they are numerically relatively insignificant causes of illness, they do generally cause higher mortality and give rise to more serious forms of illness than classical foodborne illness-causing organisms.

Susceptibility of populations to infection

It is now well-recognized that there are segments of the population that are particularly at risk from foodborne illness; these are sometime called the YOPIs, which stand for Young, Old, Pregnant and Immunodeficient. This is a very general description, but these groups do include persons that are often highly susceptible to infection and generally suffer much more serious illness than other members of the community. Two particular at-risk groups are cancer patients on immunosuppressant drugs and AIDS patients. These groups suffer infection from a wide range of organisms and the incidence of infection is often much higher than in other sections of the community. For instance, AIDS patients are 300 times more likely to suffer listeriosis than the general public (Mascola *et al.*, 1988). In addition to immunocompetence, there is a whole range of factors that may increase the sensitivity of individuals to infection, including nutritional and physiological factors, concurrent or recent infections and status of the gastro-intestinal tract (Archer & Young, 1988). Many of these problems are associated with the elderly, and with people living longer we must expect foodborne illness to increase even amongst the relatively well-off Western communities. It seems necessary to warn particularly susceptible groups of the potential dangers of infection from some foodstuffs. This can be quite successful. Thus in the UK, doctors were asked to alert at-risk groups to listeriosis, and in particular to target pregnant women as more than one-third of cases were associated with pre- and post-natal infection, and to advise them not to eat such products as soft cheese, pâté and pre-prepared meals. This strategy, together with the identification of particular contaminated food items, and a general tightening of hygiene measures by food manufacturers led to a significant reduction in the incidence of listeriosis in England and Wales – from more than 300 cases in 1989 to 100–140 cases in subsequent years (McLauchlin *et al.*, 1991). There are some indications that our quest for pathogen-free food may lead to increased susceptibility to disease as a result of loss of natural immunity caused by failure to prime the immune system with subclinical numbers of pathogens. Such indications come from studies of rechallenge of human volunteers after initial infection and the finding that infection is more difficult to achieve and symptoms generally less severe (McCullough & Eisele, 1951; Levine *et al.*, 1979; Black *et al.*, 1988) and much evidence that 'Delhi-belly', 'Montezuma's revenge', and other forms of traveller's diarrhoea usually affect only the traveller and not the adult indigenous population. There is also epidemiological evidence that Scandinavians are very susceptible to Salmonella infection and mainly contract Salmonella when abroad. For instance, it is estimated that only 10% of Salmonella infections in Sweden and Finland are contracted at home with 90% abroad; this is claimed to be due to the low level of Salmonella in their home-produced foods (WHO, 1989, 1992).

With aging populations and increased treatment of diseases with immunosuppressant agents and perhaps cleaner foods there is likely to be an increase in foodborne illness unless steps are taken to reduce microbiological contamination of foods and to persuade caterers and consumers to handle food properly.

Foodborne illness risks and their control

Most foodborne illness-causing micro-organisms are zoonotic or geonotic. However, some important foodborne human pathogens such as the hepatitis A virus, *Salmonella typhi* and *Vibrio cholerae* are solely of human origin and contamination with these may occur when the food is growing, e.g. via sewage, or during preparation for consumption, e.g. as a result of poor hygienic practices by a food handler who is a carrier. Thus micro-organisms capable of causing foodborne illness may enter the food chain at any stage.

Microbiological hazards and risks associated with food depend on the following.

- Types and numbers of food poisoning illness-causing micro-organisms, including toxic products of their metabolism, that are present in raw materials or introduced during processing into products or during subsequent handling.
- Effect of any product formulation or processing on their increase, decrease or survival.
- Whether conditions during distribution, and preparation for consumption will further promote contamination, growth or survival.
- Susceptibility of individuals to foodborne illness.

Thus the microbiological safety requirements of a food must take account of, on the one hand, microbiological knowledge concerning the occurrence and fate of potentially hazardous micro-organisms and their toxins that may be present in a food at the point of consumption, and on the other, medical knowledge concerning the numbers of micro-organisms (or amounts of toxin) able to cause illness and the severity of such illness. These two factors form the basis of the risk assessment that industry or a government body would apply in order to decide whether a particular foodstuff is safe and such knowledge is also essential for the control of microbiological and other risks in a food operation. However, as will be indicated in the following paragraphs, risk assessment applied to microbiological agents is complicated because microbes change in numbers through the food chain, and the often idiosyncratic response of humans to foodborne infections (Baird-Parker, 1994).

Risk assessment

The basic steps in any risk assessment are firstly identification and assessment of the hazards. This will require a consideration of the specific microbiological hazards, i.e. the micro-organisms or microbial toxins, that may be of concern in a particular foodstuff, and will be based on knowledge derived from surveys of the types of micro-organisms present in a food, and/or in food raw materials, and information from epidemiological surveillance of foods of an identical or similar nature. Because of the often incompleteness of our information it may well be necessary to make some assumptions concerning the likely presence of micro-organisms in food raw materials and products. Thus a raw product of agricultural origin can be assumed to be infected from time-to-time with any infectious and toxigenic pathogenic micro-organisms and this needs to be taken into account when hazards are assessed. For assessment, the individual and combined effect of any intrinsic, extrinsic and implicit preservation factors that may be applied to the food during its production, manufacture and distribution on growth or survival of identified hazards are considered. We have generally excellent microbiological data concerning how preservation factors affect survival, growth and death of the main foodborne disease-causing micro-organisms (ICMSF, 1980, 1994), and increasingly, mathematical models based on such data are used to predict the fate of micro-organisms in manufactured foods. The most widely available and complete collection of predictive models of foodborne pathogens is Food Micromodel which is the result of a 4 year, £7 million UK MAFF project (McClure *et al.*, 1994). Such models have been extensively tested and have been shown to give good predictions, although some additional but limited testing may be necessary for some foods. The advantages from the use of such models, in terms of speed of response and greater security of safety decisions, far exceeds the cost of their development and use.

The second stage of a risk analysis is to assess the exposure of users of a food to the significant hazards identified at the first stage and aims to assess the probability of illness occurring as a result of consumption of a food. This is a very difficult element of any microbiological risk assessment as in order to determine the likely number of organisms we need to take account of, on the one hand, the distribution of micro-organisms in a food, recognizing that such distributions are often highly heterogeneous, and will change as the food proceeds down the food-chain, and on the other hand, the probability of disease occurring as a result of exposure to different concentrations of organisms. We have limited, and mainly qualitative information, from epidemiological investigations of disease outbreaks and incidents, of factors that will affect the infectivity of foodborne illness-causing pathogens. These include the fat and iron content of the food, exposure of the micro-organisms to heat and cold, other organisms, the way the food is ingested, location of infecting organisms and the host-specific factors listed previously (Archer & Young, 1988; D'Aoust, 1989). However, we have little quantitative data on which to base the dose/response curves to infection that are essential in any exposure estimate. Animal models are not useful to develop such curves, which causes much difficulty, as human feeding studies are not ethically acceptable. For these reasons, formal risk assessment procedures are not used to determine limits for acceptable numbers of microbial pathogens or microbial toxins in food. Most assumptions concerning the levels of micro-organisms that are acceptable in foods are based on limited epidemiological investigation, some (but limited) human studies, and a general consensus amongst experts as to an acceptable number. However, experts often disagree and for some organisms it has proven impossible to reach consensus. Thus there remains no agreement as to what is an acceptable level of *Listeria monocytogenes* in a ready-to-eat food (FAO/WHO, 1993). However, where information concerning the human response to levels of pathogenic micro-organisms is such that a dose/response curve can be generated, and combined with quantitative knowledge of the occurrence in food, some assessment can be made of risk and the need for control strategies (for instance to reduce the incidence of contamination) calculated. Such a risk assessment has been done on the risk of viral infections from shellfish with somewhat concerning results (backed up by illness statistics) of the probability of illness from eating some raw molluscan shellfish in the USA (Rose, 1993).

The HACCP system

When assessing risk, industry will always want to fail safe. Therefore, industry that understands microbial risks and their control will use the philosophy that the best means of preventing risky or microbiologically hazardous products is by the design of a food, such that any foodborne illness-causing organism that is potentially of concern is eliminated, or controlled to a level that on the basis of the best epidemiological evidence is safe.

For the design of microbiologically safe products a procedure called the Hazard Analysis Critical Control

Point (HACCP) system is internationally used. The HACCP system enables the specific microbiological hazards associated with the production, manufacture, distribution and use of a particular food to be identified in an objective, systematic and comprehensive manner and the precise means of eliminating and controlling an identified hazard to an acceptable (safe) level to be defined. The principles and procedures for applying the HACCP system are well documented (Technical Manual, 1992; FAO/WHO, 1993; ILSI, 1993; WHO, 1993). More recently these procedures have been improved by use of modern risk assessment techniques. Thus, the best current forms of HACCP use multidisciplinary teams of experts applying a structured approach to hazard analysis, often based on HAZOP, which considers the consequences of failure to control a raw material, specific piece of equipment or operating practice on the potential for one or more significant microbiological hazards to occur (Mayes & Kilsby, 1981).

A hazard analysis on a product will involve examination of the effects of the following on the microbiological hazards associated with a food product *at* the point of consumption. Raw materials (sources and usage); processing and procedures (including formulation); processing equipment; processing parameters and operating procedures; packaging conditions and storage; and distribution conditions and usage of the products. The significance of each microbiological hazard is assessed, taking account of the risks and severity of each hazard. Hazards that are judged to have no significance such as those of low severity and/or risk are eliminated so that effort can be concentrated on hazards that pose a significant risk. This assessment is done stepwise for each raw material, and for each step in food operation.

The next stage of the hazard analysis (this is a combination of hazard identification and assessment) is to identify the Critical Control Points (CCPs), which are defined as steps at which effective control can be applied to prevent, eliminate or reduce the risk of occurrence of a hazard to a safe level; steps include any stages in the growing, production, manufacture, distribution and use of a food. For this analysis a decision tree may be used to structure the procedure (Mayes, 1992; WHO, 1993). A CCP may include the growing, harvesting of raw materials; the control of product formulation, or a piece of process equipment, as well as control of distribution conditions, specification of procedures used for storage and preparation for consumption. For each CCP, target requirements for control are defined (including any tolerances) and a monitoring procedure identified such that any deviation from a critical limit is identified.

The final stage of HACCP is concerned with identifying procedures for applying corrective action if a critical limit is exceeded, and documentation on all aspects of HACCP analysis including procedures to verify that the HACCP system is working effectively. There are a number of perceived benefits of applying the HACCP system, including more effective use of resources by concentrating effort on procedures that must be controlled, and more timely response to problems by installing an effective control system (Baird-Parker, 1990). The regulatory authorities increasingly accept HACCP as the best means of assessing the safety of food and its use has been incorporated in regulations in the USA, Canada, Australia, the European Community and many other countries.

Foodborne illness: the way forward

Despite much progress in our knowledge and understanding of the disease processes of many of the organisms that contaminate our foods, together with better knowledge of methods for controlling micro-organisms in food, and much stricter regulations applied to food manufacturing, distribution and storage, we are not winning the battle against the many diseases spread by foods, even in the technically advanced Western countries. The facts before us are, that with the exception of listeriosis (where significant reductions of 50 % or more have been achieved during the past few years), the number of cases of reported foodborne illness continue to rise at an alarming rate in the UK and elsewhere.

For instance in the UK, reported cases have risen from 12000 in 1978 to nearly 65000 in 1992. As mentioned previously, the source of many of the micro-organisms causing illness are food animals, and not infrequently the statement is made by trade and government bodies that raw animal products contain unavoidable, but undesirable microbiological contaminants. This implies that we have given up trying to control contamination at its source and try to deal with the consequences of contamination further down the food-chain. This is substantially correct, and although it is well-recognized that the elimination and control earlier in the food-chain is likely to be most effective, it is also likely to be too costly and difficult to achieve because of the large numbers of potential sources of contamination of the live animal (Johnston, 1991). However, we must seriously consider the possibility of eliminating the major food pathogens from our raw meats, ideally at the farm level, as once the animal enters the food-chain the potential for microbial growth, cross-contamination and re-contamination is high, and we lose control as reflected by the food poisoning statistics. A recent analysis of 1320 outbreaks of foodborne illness in England and Wales indicated that 80–90 % of these were associated with meat and poultry, with poultry being responsible for more than 50 % of the outbreaks (Roberts, 1986). The figure for poultry has considerably increased since this survey was completed (in 1982) and is now probably 70–80 % (if eggs are included).

The most striking example we have of effectively dealing with the problem of disease transmission from animal to humans is those procedures that have been applied by the dairy industry to control diseases from milk. This has combined a strategy of elimination of bacteria causing diseases in farm animals and humans, such as tuberculosis and brucellosis, at the farm level by a process of slaughter and vaccination, followed by pasteurization of the milk

before distribution to destroy any infective vegetative micro-organisms that may have contaminated the milk. In 1983, following an extensive outbreak of milk-borne salmonellosis, the Scottish health authorities introduced compulsory pasteurization of milk sold through retail outlets, and more recently totally banned the consumption of raw milk. This has eliminated milk as a cause of food poisoning in Scotland (Cohen *et al.*, 1983). Despite its success the consumption of raw milk is still permitted in England and Wales and there is still significant illness, including salmonellosis, Campylobacter enteritis and, most recently, *E. coli* O157:H7 infection, despite strict hygiene measures on farms and labelling (Richmond, 1992). The freedom of the individual is an important part of our democracy but can we really continue to allow individuals to expose themselves, their families and others, to potentially serious diseases resulting from contaminated food – particularly where measures are available to prevent such illness?

How can we achieve the success with milk with other foods? One approach being investigated is the introduction into food animals of genes that create conditions that block the attachment of specific organisms to the gastro-intestinal tract. The possibility of using this approach to prevent Salmonella contamination of chicken is being explored by the Agricultural and Food Research Councils Institute of Animal Health. However, this is a very long-term research project and there is no evidence that it will be successful. For organisms that are infective, such as certain phage types of *S. enteritidis*, a more certain approach is the use of vaccines. Some progress has been made, but vaccines have not been widely applied as a preventative measure outside animal disease control and there are some difficulties. However, with good knowledge of virulence factors and protein engineering we should be able to design effective vaccines for whole ranges of disease transmitted through animals to humans. One area that is being urgently researched in the USA is the development of a vaccine to prevent *E. coli* O157:H7 infection of cattle.

The cost structure and competitive nature of animal production allows little scope to introduce any prevention measures that increase production costs and despite costing the UK taxpayer many £millions every year (Sockett *et al.*, 1986) there seems little incentive to apply preventative measures. Surely the increased evidence of serious acute and chronic disease arising from foodborne illness will be a spur for action? Industry and the authorities have spent the money to control the listeria threat; will the emerging threat of organisms such as *E. coli* O157 bring about change too?

After heat, the most cost effective and certain method of elimination for Salmonella and other vegetative pathogens from raw foods is by the use of gamma irradiation (WHO, 1988). We are as sure as we possibly can that irradiation is safe at levels approved for food use, but equally we are aware of current consumer concerns, and we need to continue to convince the consumer of the health benefits of irradiated food. I believe that irradiation of raw meats will become more widely used in the next 5 years; it is already widely used for some chicken products in Europe. Possible alternatives to irradiation are the use of ultra-high pressure (Knorr, 1993) or pulsed electric fields (Castro *et al.*, 1993), but these probably have more limited and specific use.

Concluding remarks

I would like to conclude by considering three areas of research that will particularly assist in our need to reduce the incidence of foodborne illness.

Microbial interactions

We have little knowledge of how micro-organisms behave in foods, or indeed on animal surfaces, and most of our knowledge of how micro-organisms respond to inimical environments is by extrapolation from monoculture systems in the laboratory. However, it is becoming clear from the recent work by molecular biologists and microbial physiologists that micro-organisms have a series of mechanisms whereby they can adapt rapidly to particular environments, enabling them to colonize diverse substrates and to adapt to a wide range of hostile conditions. One common mechanism is that external stimuli stimulate a transmembrane protein (a transmitter or sensor) with cytoplasmic and extra-cytoplasmic domains. The extra-cytoplasmic domain senses the environment and transfers a signal to a receiver which via phosphorylation or other means leads to specific binding to the genome, DNA supercoiling, alterations in RNA-polymerase specificity etc., which result in major changes in gene transcription or translation and hence gene expression (Parkinson, 1993). We need to understand the mechanisms of such signal transduction, or other types of gene switching, it we are going to be able to better control micro-organisms in foods and understand the disease process (Bliska *et al.*, 1993). We also need to continue to understand how micro-organisms react to stress, such that we can find ways of interfering with the diverse mechanisms whereby they develop resistance to physical and chemical treatments. This is relevant to the mechanisms of preservative resistance as well as understanding how micro-organisms survive acid-shock during their passage through the stomach (Foster, 1991). We know that temperature has a major regulating effect on the virulence factors of many pathogenic micro-organisms and that pH, osmotic shock, E_h etc. will trigger a number of responses including increased resistance to heat.

The disease process

The processes whereby foodborne pathogens initiate infection, and how infection progresses to the disease state, are poorly understood, particularly at the molecular level. In particular, we need to identify the key virulence factors in order to target disease prevention and to develop specific RNA probes for the detection of virulent strains of pathogenic micro-organisms; we also need to understand more about the susceptibility of individuals to

foodborne disease and particularly to sequelae (Archer & Young, 1988; Smith *et al.*, 1993).

Quantitative risk assessment procedures

I have already touched on some of the difficulties of applying conventional risk assessment procedures to foodborne illness-causing pathogens and we may conclude that this is impossible. However, we need to attempt to establish dose/response curves for pathogens that are at least relevant to major sectors of the population, although we must recognize that there are probably major differences between minimum infective doses for those 'at risk' individuals and most persons in a population. We also need to continue to design foods according to sound principles of prevention and for this purpose predictive models of growth/survival based on good understanding of the physiology of the microbial cell, i.e. mechanistic based, will enable us to predict microbial growth, survival and death will further assist us in developing HACCP and quantitative risk assessment procedures.

Epilogue

We will only achieve safe food by close working together of such disciplines as epidemiologists, biologists, clinicians, microbiologists, molecular biologists, engineers and physical scientists. There is much to do, and the members of the Society for General Microbiology have a key role in promoting the application of good science to prevent foodborne illness.

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