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Fermentation and pathogen control: a risk assessment approach

Martin Adams [a,*], Robert Mitchell [b]

[a] School of Biomedical and Life Sciences, University of Surrey, Guildford, Surrey GU2 7XH, UK
[b] Environmental Surveillance Unit, PHLS Communicable Disease Surveillance, Centre, 61 Colindale Avenue, London NW9 5EQ, UK

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Abstract

Food fermentation has a long tradition of improving the safety, shelf life and acceptability of foods. Although fermented foods generally enjoy a well-founded reputation for safety, some notable outbreaks of foodborne illness associated with fermented foods have occurred. Microbiological risk assessment (MRA), as it has emerged in recent years, provides the scientific basis for the control and management of risk. Aspects of fermented food processes are discussed under the various stages of risk assessment and data are presented that would inform more detailed risk assessments.

Keywords: Risk assessment; Lactic acid bacteria; Fermented foods; Food safety

1. Introduction

Most fermented foods owe their origin to the fact that processes used in their production are inhibitory to many microorganisms. As a result, fermented products generally have a longer shelf life than their original substrate and their ultimate spoilage is different in character. The antimicrobial effects of fermentation are not confined to spoilage organisms alone and can also affect pathogens that might be present. Thus, traditional food fermentations can take potentially hazardous raw materials, such as raw meat and milk, and transform them into products with both improved keeping qualities and a reduced risk of causing illness. The extent to which fermented foods are safe and how fermentation processes should be conducted to achieve a required level of safety are key questions that are not simple to answer. All approaches to this depend critically upon the quality of the data available. In the past, we have had to rely largely on expert judgement to interpret the available information, but modern microbiological risk assessment (MRA) techniques will enable us to achieve food safety objectives related to fermented foods in a more reliable and consistent fashion.

Microbiological risk assessment (MRA) is essentially a tool for applying our knowledge of microbiological food safety in a logical, systematic, consistent and transparent way to assess food safety risks. It aims to tell us what is the chance that a certain food will cause illness, who will be affected, and what those effects will be. It provides a scientific basis for the management and control of the microbiological risks posed by foods. A generally agreed approach on

* Corresponding author. Tel.: +44-1483-686-492; fax: +44-1483-300-374.
E-mail address: m.adams@surrey.ac.uk (M. Adams).

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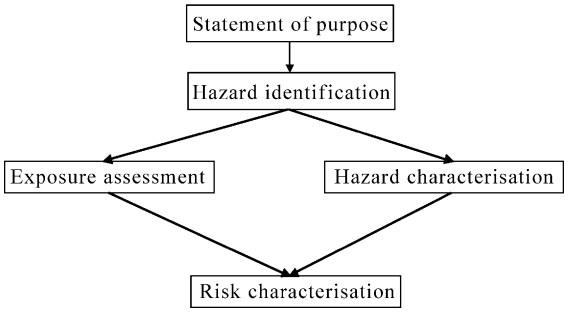

Fig. 1. Microbiological risk assessment.

how this is done has evolved over the last few years and has been well described in a number of recent monographs (Mitchell, 2000; Voysey, 2000; Benford, 2001).

Essentially, the process consists of the stages outlined in Fig. 1. The first requirement is to formulate the problem in a statement of purpose—what is your objective in doing the risk assessment. Once this is done, it is necessary to decide which hazardous organisms will be of concern in the product (Hazard Identification); what will be the intake of hazard as a result of food consumption (Exposure Assessment); what will the effect be on people (Hazard Characterisation); and, finally, what is the overall risk to a given population (Risk Characterisation).

2. Statement of purpose

Fermented foods can be defined as foods in which microbial activity plays an essential role in conferring the required stability, safety and sensory properties to the product. This definition will exclude those products which are often described as fermented but are largely the product of nonmicrobial, enzymic processes such as black tea and the fish sauces of Southeast Asia, but will include those products where the principal microbial activity is nonfermentative, such as tempeh and vinegar. When the virtues of fermentation as a means of preservation are discussed, however, it is almost invariably in connection with those foods where lactic acid bacteria (LAB) play a central role in the production process, and it is these which will be the focus of what follows.

3. Hazard identification

Identifying which pathogenic agents may be transmitted by fermented foods is clearly an important step in the overall risk assessment process. Generally, this will require expert knowledge and the appraisal of data from a variety of sources. The major concern is that a significant pathogen is not disregarded and to avoid this pitfall, attempts have been made to develop structured and systematic approaches to hazard identification (Notermans et al., 1994; van Gerwen et al., 1997).

While we recognise that many pathogens can gain access to a product as a result of contamination during processing and storage, raw materials are often the principal source of hazards. The range of fermented foods is, however, huge (see for example, Wood,1998). They are made using raw materials from all the main food commodity groups—meat, fish, milk, vegetables, fruits, grains and pulses, and there is considerable variation in the unit operations involved in individual processes and how the products are stored and consumed (Nout, 2001). As a consequence, the number of microbiological hazards potentially associated with fermented foods can be correspondingly large. It is possible to edit the extensive lists obtained by examining epidemiological data from outbreaks where fermented foods have been implicated. This information has a decided bias since many fermented foods are produced and consumed in countries that lack highly developed systems for the reporting of foodborne illness. The information we have is therefore largely related to products popular in the developed world, such as fermented milks and meats. Even here, when we are considering a more limited range of products, a substantial list of pathogens can emerge. By way of example, a list of pathogens associated with illness caused by cheese is substantial and includes most common and some less common foodborne bacterial pathogens (Table 1). Clearly, hazard identification for fermented foods as a whole is not a particularly illuminating exercise as virtually all the known foodborne pathogens will be included. There is, however, a hierarchy and some pathogens are more common and therefore pose a greater risk. If we look specifically at fermented sausages then on a worldwide basis, *Salmonella*, VTEC and *Staphylococcus aureus* seem to be the main concerns, although there may be some geograph-

Table 1
Pathogens associated with outbreaks of foodborne illness caused by cheese (data from Nichols et al., 1996)

Pathogen/Syndrome
Bacillus cereus
Brucella melitensis
Campylobacter jejuni/coli
Clostridium botulinum
Clostridium perfringens
Escherichia coli (O27:H20; O124:B17; O157)
Hepatitis A
Histamine poisoning
Listeria monocytogenes
Mycobacterium avium complex
Salmonella
Shigella sonnei
Staphylococcus aureus
Streptococcus pyogenes
Streptococcus zooepidemicus
Tick-borne encephalitis

ical variation in their importance relating to fermentation temperatures used (Taplin, 1982; Adams, 1986; Van Netten et al., 1986; Cowden et al., 1989; Anon, 1995a,b).

4. Exposure assessment

This stage of the risk assessment focuses on the food vehicle and assesses the likely intake of a particular pathogen arising from the food's consumption. To do this requires the estimation of the level of pathogen or toxin in the food at the time of consumption and details of its consumption pattern.

Fermented foods are prime examples of the hurdle or multiple barrier approach to food preservation, where the overall antimicrobial effect seen is the aggregate effect of a number of different factors. The range of these antimicrobial hurdles is illustrated in the case of fermented sausages in Fig. 2 and it is important to note that nonmicrobial barriers such as preservative chemicals, water activity reduction through salting or drying, and inactivation by any heating step will also make important contributions. Interactions between microbial and nonmicrobial preservative factors can significantly enhance inhibition (see, for example, Singh et al., 2001) and the sequence in which these factors are applied can also be important. For example, when an acid stress preceded a low water activity stress, there was a greater lethal effect on *Escherichia coli* than when the stresses were applied in the reverse order (Shadbolt et al., 2001).

Although bacterial fermentation is not necessarily the most important barrier in terms of the inhibition of pathogens, it often receives most attention. A number of antimicrobial factors produced by LAB have been identified and their role has been reviewed periodically (Lindgren and Dobrogosz, 1990; Ouwehand, 1998; Adams, 2001). In recent years, considerable efforts have been devoted to the isolation and study of LAB antimicrobials such as bacteriocins, and this has tended to obscure the fact that the principal inhibitory contribution of lactic acid bacteria during lactic fermentations is the production of organic acid at levels up to and exceeding 100 mM and the consequent decrease in pH. For any inhibition to occur, lactic acid bacteria require a large numerical superiority over any pathogens present. In mixed culture experiments using a nisin-producing strain of the dairy starter *Lactococcus lactis*, *E. coli* was still able to grow for 5 h and increase in numbers by 2 log cycles even when outnumbered by a factor of 10^5:1 by the *L. lactis* (Yusof et al., 1993). In the same work, it was shown that inhibition of the nisin-sensitive, Gram positive pathogen, *S. aureus*, was entirely due to the acid present with no discernable contribution from the nisin produced by the starter.

The antimicrobial activity affecting safety in fermentation is largely directed at bacterial pathogens. Studies using foodborne viruses or their surrogates have suggested that foodborne viral hazard are largely unaffected by the pH and acidity levels occurring in

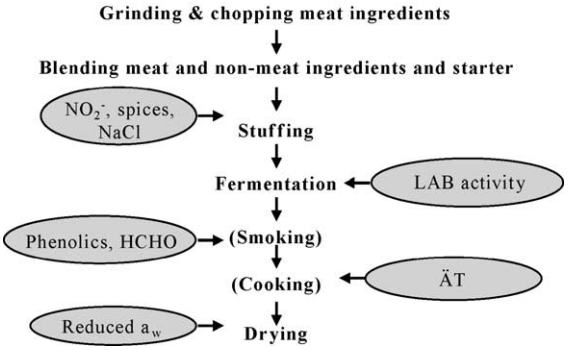

Fig. 2. Antimicrobial hurdles in meat fermentation.

fermented foods (Hermann and Cliver, 1973; Dethmers et al., 1975; Kantor and Potter, 1975; Nout et al., 1989; Wood and Adams, 1992) and there is limited information on the prevalence and survival of parasites in fermented foods (Warnekulasuriya et al., 1998; Perez et al., 2001).

The extent to which the risk of foodborne illness is reduced during the production of any particular fermented product will thus differ depending on the type of hazard being considered, the nature of the raw material and the precise details of the process used. In the case of bacterial hazards, possible outcomes could be that growth is inhibited or that some proportion of the pathogen population is inactivated. Both outcomes can result in a reduced risk and a safer product. With toxigenic organisms, such as *Clostridium botulinum* or *S. aureus*, preventing growth can effectively ensure safety, assuming that initial numbers are well below those necessary to produce a harmful level of toxin. With infectious pathogens, however, slowing or preventing growth may be insufficient to provide an acceptable safeguard, depending on the infectious dose for those consuming the product. Any inactivation of bacterial hazards may not be sufficient to eliminate risk entirely. To what extent this is achieved will depend on the pathogen concerned, its initial numbers and their physiological state.

An obvious way of determining the prevalence of a pathogen in food is to use data obtained from microbiological survey work. Governments, regulatory authorities and companies may all have relevant data from their own surveillance activities relating to hazard levels both in the product and in the raw materials used. One example of the type of information available relates to fermented meats in England and Wales. These were the subject of two independent surveys in 1996; one conducted by the Public Health Laboratory Service (PHLS) and Local Authority Co-ordinating Body for Food and Trading Standards (LACOTS) as part of its participation in the European Community Co-ordinated Food Control Programme (ECCFCP), and the other conducted independently by the Ministry of Agriculture, Fisheries and Food earlier the same year (MAFF, 1997; Little et al., 1998). The results showed remarkably similar findings. VTEC was not detected in a total of almost 3500 independent samples, although *Salmonella* was found at a similar low level in both surveys, 2 out of 2981 samples (ECCFCP) and 1 out of 455 samples (MAFF), and levels of *S. aureus* were greater than 10^2 g^{-1} in 1.3% and 1.1% of samples, respectively. The MAFF survey detected *Listeria monocytogenes* in 3% of samples, but at levels below 10^2 g^{-1}.

Such data can also usefully inform a hazard identification process. They suggest that, in a UK context, where fermented meats are largely imported from continental Europe, there is a much greater risk from *Salmonella* than from VTEC. Some limited supportive evidence for this conclusion is provided by the fact that two of the three reported outbreak of food poisoning associated with fermented meats in the UK were caused by *Salmonella* (Cowden et al., 1989; O'Brien and LeBaigue, 2002).

Surveillance data can be supplemented by the use of predictive models which describe how the conditions prevailing during production and storage will affect growth and survival of pathogens. Knowing the prevalence of a particular hazard in the raw material, it might therefore be possible to predict the levels expected in the product at the point of consumption. Software packages are available and the use of growth and inactivation models in risk assessment has been discussed (van Gerwen and Zwietering, 1998). This is more difficult for fermented foods compared to other food products as most models describe a static situation where conditions such as pH and water activity are constant. In the production of fermented foods, such as cheese or salami, these factors will change during the course of production. So although a model might predict that growth of a particular pathogen is impossible under the conditions prevailing in the final product, these conditions are not attained instantaneously and there could be considerable scope for microbial growth during earlier stages of the fermentation (see below). Where growth has been prevented, survival must be predicted and this area of modelling survival under adverse conditions of pH and water activity is less well advanced. Survival of *E. coli* O157 in the production of uncooked, semi-dry, fermented sausage has been modelled using data from challenge trials (Pond et al., 2001). Though the models correlated well to the data used in their generation, they over-predicted the reduction in *E. coli* numbers when validated using independent data. This would limit their practical use as it is undesirable to employ models which under-predict risk. The

authors did, however, identify factors to account for this and which could be incorporated in more refined models.

While models have their limitations and should be used with some caution, they do serve a particularly useful role in directing attention to where the acid test of challenge trials could be used most effectively. Challenge trials will provide robust data by determining the fate of pathogens inoculated into the raw materials through the actual course of fermentation and storage. This has been the approach adopted in the United States with fermented meats. Following VTEC outbreaks in the US and Australia associated with fermented sausages in 1994–1995 (Anon, 1995a,b), the US Food Safety Inspection Service required producers to validate that their processes achieved a 5 log reduction in viable numbers of *E. coli* O157. This objective assumed a worst-case scenario based on the maximum level of *E. coli* O157 detected on a beef carcass and a requirement that the product contains less than one *E. coli* O157:H7 per 100 g. The coincidence of such a series of improbable events (high level contamination/low infective dose) is unlikely, so products from any process meeting this standard would be considered safe. This approach considerably over-estimates risk and was modified subsequently by FSIS to include other options (Getty et al., 2000). An alternative, probabilistic approach based not on single values such as an average or worst case value but on several values drawn from a probability distribution can provide a more realistic estimate of risk and how that risk can be affected by process changes (Hoornstra and Notermans, 2001).

As a result of the FSIS ruling, a host of challenge trials have been reported and the results from these have been reviewed (Getty et al., 2000). They confirm that if *E. coli* O157:H7 is present in sufficient numbers, it is able to survive the fermentation and subsequent drying to a range of moisture to protein ratios. There are inevitable differences in the precise details of the production processes employed but typically the reduction in viable numbers of *E. coli* O157:H7 is of the order of 2–3 log cycles, though it can be less in some cases. Other conclusions drawn from these data are that the European method of sausage fermentation which generally involves a longer fermentation period at a lower temperature gives a better reduction and that, less surprisingly, high salt, high nitrite and low pH give better reduction. The overall conclusion from this work has been that to achieve 5 decimal reduction reliably, a longer fermentation period or incorporation of a final heat process is necessary.

A broadly similar picture emerges from work conducted on pathogen survival in cheeses. When *E. coli* O157:H7 is present in the milk, its numbers have been shown to multiply during the early stages of production of several cheese types, including hard cheeses, Camembert, cottage cheese and smear ripened cheese (Arocha et al., 1992; Reitsma and Henning, 1996; Ramsaran et al., 1998; Maher et al., 2001; Saad et al., 2001). This is seen less with fermented meats where the presence of curing salts in the initial mix helps inhibit growth. Although numbers of *E. coli* O157:H7 in cheeses generally fall later in the production process and during maturation, they are often still detectable after extended periods of storage (Reitsma and Henning, 1996; Maher et al., 2001). After the final heating stage used in cottage cheese production, *E. coli* initially present in the fresh cheese at 10^7 cfu g^{-1} was undetectable (Arocha et al., 1992). This observation re-emphasises the overriding importance of heating steps noted earlier with respect to fermented meats. Most cheese do not experience the high temperature scalding used in cottage cheese production, but pasteurisation of the milk used in cheese production is an extremely valuable critical control point not available to producers of fermented meats. Data collected by the UK's Institute of Food Science and Technology on published outbreaks of bacterial foodborne illness associated with cheese demonstrated that cheese is not a common cause of foodborne illness, but suggest that when outbreaks do occur, cheese made from correctly pasteurised milk is only very rarely implicated; Table 2 (IFST, 1998). This is reinforced by surveillance data from England and Wales which showed that, from 1437 soft cheeses sampled, significantly more of those produced from pasteurised milk conformed to the PHLS guidelines for ready-to-eat foods (94%) than did raw (unpasteurised) milk soft cheeses (71%) (Nichols et al., 1996). A survey of cheeses made solely from unpasteurised milk, failed to find *E. coli* O157:H7 in any of 801 samples tested and the species *E. coli* was undetectable (<10 cfu g^{-1}) in 83% of samples. However, very high levels of *E. coli* ($>10^5$ cfu g^{-1}) were found in a small proportion (1.4%) of cheeses (MAFF, 2000).

Table 2
Pasteurisation and its association with outbreaks of foodborne illness caused by cheese (data from IFST 1998)

Year	Location	Pathogen	Food	Unpasteurised milk used
1992	England	*Salmonella* Livingstone	cheese	no
1992/1993	France	VTEC	fromage frais	yes
1993	France	*Salmonella* Paratyphi B	goats' milk cheese	yes
1994	Scotland	VTEC (O157)	local farm cheese	yes
1995	France	*Listeria monocytogenes*	Brie de Meaux	yes
1995	Malta	*Brucella melitensis*	soft cheese	yes
1995	Switzerland and France	*Salmonella* Dublin	cheese from Doubs region	yes
1996	England and Scotland	*Salmonella* Gold Coast	cheddar cheese	pasteurisation failure
1996	Italy	*Clostridium botulinum*	Marscapone cheese	no

Clearly, while the value of pasteurisation cannot be ignored, it is possible to produce safe products without it. To do so, however, places even greater emphasis on other aspects of the process such as careful attention to hygiene and temperature control during production to avoid contamination and minimise bacterial growth.

It is not sufficient to have an estimate of the level of a hazard in a food and the variation in that level. Exposure will also depend on the pattern of consumption—the amount of food consumed by individuals, the average serving size, the frequency of consumption and the distribution of that consumption within the population. There may be a whole range of socio-economic, seasonal, regional, ethnic or demographic factors affecting consumption. For example, UK statistics indicate that in the year 2000 total cheese consumption was equivalent to 110 g per person per week, but also revealed consumption to be highest in higher income households, lower in households where there were more children, lowest in the Yorkshire and Humberside area and highest in the Southeast of England (National Food Survey, 2000). Official statistics may have rather a broad brush approach in this regard and commercial data owned by manufacturers and retailers can play a critical role in assembling a complete picture of the consumption pattern for a particular product.

5. Hazard characterisation

In contrast to exposure assessment, which focuses mainly on the food, hazard characterisation is concerned with what the effect of a hazard will be on people. It provides a description of the frequency, nature, severity and duration of illness caused by the presence of the hazard in the food. This can be either qualitative or quantitative. Central to this activity is establishing the relationship between exposure (dose ingested) and the response (harm caused). This is not an easy task as several interacting factors such as the properties of the pathogen, the food vehicle and its consumption pattern, the dynamics of infection and the individual consumer can all contribute to determining whether illness occurs. There is an inherent variability in each of these and this is further compounded by uncertainty in the data available.

For most pathogens a set of characteristic symptoms can be described which are generally associated with the illness they cause. However, these symptoms can occur with a widely varying degree of severity and complications or long-term sequelae such as reactive arthritis from salmonellosis or Guillain–Barré syndrome from campylobacteriosis can also sometimes arise (Mossel et al., 1995; Nachamkin et al., 2000). There is considerable variability between strains of the same organism in their capacity to cause illness, and the previous history of a strain can also affect its virulence. Human susceptibility to infection can differ markedly between subpopulations such as young healthy adults, the very young, the very old, the sick, pregnant women and the immuno-compromised as well as between individuals within subpopulations. This might lead to diffuse outbreaks which are difficult to recognise. The food vehicle can play a significant role in facilitating infection by protecting the pathogen from the effects of the stomach's acidity. In some outbreaks associated with fermented foods such as cheese and salami, the level of pathogen present in

the implicated food has been very low and this may be due to the protective effects of fat (D'Aoust et al., 1985; Getty et al., 2000). Foods that are often consumed on an empty stomach, e.g. at breakfast, could pose a greater threat due to their more rapid transit through the stomach.

In the past, the concept of a minimum infective dose has often been used. The idea that there is a certain threshold below which an organism cannot cause illness may apply to toxigenic pathogens where a certain population may be necessary to produce sufficient toxin to cause illness. In such cases, the risk assessment procedures will resemble those used to assess risk posed by chemicals. With infectious pathogens which multiply in the body, a single organism could in principle initiate an infection. Although the chances of infection from a single organism may be very low, they cannot be neglected entirely, particularly in view of the low doses implicated in some outbreaks associated with fermented foods (Getty et al., 2000). Data on the relationship between dose and risk of infection can be obtained from volunteer feeding trials. These have their own inherent flaws such as the employment of healthy volunteers and the frequent use of nonfood matrices to deliver the pathogen (Kothary and Babu, 2001). They are also not possible with pathogens such as *L. monocytogenes* and *E. coli* O157 for ethical reasons and in these cases outbreak data have to be relied on. Dose–response modelling is currently an active area of research. Use of a number of models has been explored and problems associated with availability of data, treatment of sub-populations and extrapolation to low doses have been identified (see, for example, Coleman and Marks, 1998; Teunis et al., 1999; Teunis and Havelaar, 2000). There is still some way to go in this area. For example, in one study comparing six different models, the estimate of the infectious dose required to affect 1% of the population ranged over nine orders of magnitude (Holcomb et al., 1999).

6. Risk characterisation

The final step in the risk assessment process is a synthesis of the information assembled in the earlier stages of the process to produce an estimate of the probability of illness and its severity in a given population. To be of greatest use informing risk management strategies, it would ideally be quantitative, but can be qualitative, and must also specify the degree of uncertainty attending that estimate.

Given their huge diversity, quantitative risk assessment cannot be applied in any meaningful sense to fermented foods as a whole. A more tightly defined statement of purpose does facilitate this and a quantitative risk assessment has been described covering the risk of human listeriosis from consumption of raw milk soft cheeses (Bemrah et al., 1998). Overall, the epidemiological information indicates that fermented foods have a good safety record, particularly in view of the large quantities consumed worldwide. One estimate has suggested that fermented foods of various types can comprise 30% of our food supply (Knorr, 1998). The available data have, however, also highlighted some notable exceptions where fermented foods have been associated with outbreaks of foodborne illness and these have prompted concern in the industry and regulatory intervention in some cases. Further development of risk assessments in this area offers a powerful and valuable tool in successfully managing food safety hazards, allowing these interesting and appealing foods to be consumed with confidence.

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