OXOID™

culture

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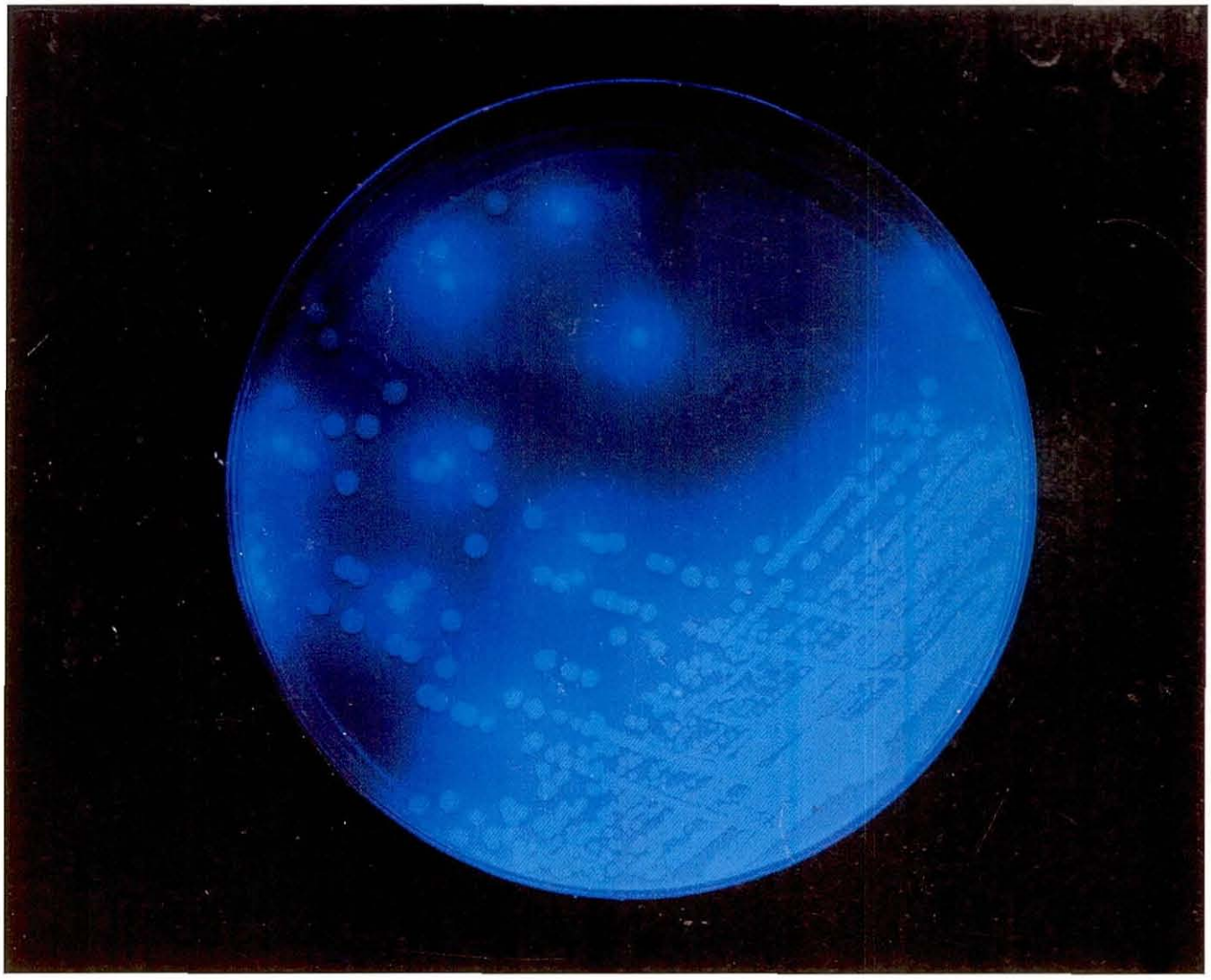


Food irradiation and microbiological safety

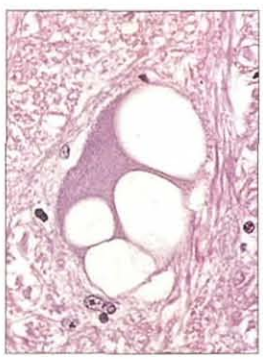
J Farkas, Director, Institute of Preservation and Live Stock Products Technology, University of Horticulture and Food Industry, Budapest, Hungary.

A review of the most important developments in the microbiological safety of irradiated food.

Prions—a new microbiological hazard

RE Spier, Professor of Microbiology, University of Surrey, Guildford, United Kingdom.

An analysis of small proteinaceous infectious particles (prions), the most recent form of microbiological threat to be recognised.



Food irradiation and microbiological safety

J Farkas, Director, Institute of Preservation and Livestock Products Technology, University of Horticulture and Food Industry, Budapest, Hungary.

Irradiation as an effective method of preservation and decontamination of food is largely accepted. However, the microbiological safety of such processed food is still the subject of controversy, although no other food process has been investigated as thoroughly.

Table 1: Typical radiation resistance of some potentially pathogenic non-sporing bacteria in fresh and frozen foods of animal origin.

Bacteria	Decimal reduction dose (D_{10}), kGy[a]	
	fresh food	frozen food
Vibrio parahaemolyticus	0.03–0.12	—
Yersinia enterocolitica	0.05–0.21	—
Campylobacter fetus subsp. *jejuni*	0.08–0.16	—
Shigella spp.	—	0.2–0.4
Aeromonas hydrophila	0.14–0.19	—
Proteus vulgaris	0.2	—
Escherichia coli	0.3–0.4	0.5–0.6
Brucella abortus	0.34	—
Salmonella spp.	0.35–0.8	0.4–1.3
Staphylococcus aureus	0.3–0.6	—
Listeria monocytogenes	0.4–1.0	1.0–1.3
Streptococcus faecalis	0.7–1.0	—

[a] Gy (Gray) is the SI unit of absorbed radiation dose (1 joule/kilogram). 1Gy = 100 rad

Microbiological goals of food irradiation

One of the main goals in the irradiation of food is the control of microbiological spoilage.[1] The objectives of such treatment of perishable food are either to delay the onset of spoilage by substantially decreasing the number of spoilage organisms, or to destroy them to such an extent that the product will be stable (commercially sterile) microbiologically. Food processing by irradiation may be combined with other antimicrobial agents and complementary treatments.

Much information has been published on the radiation resistance of various food-borne micro-organisms and their relative radiation resistances can be found in several reviews.[2-4] There is an obvious difference of inherent resistance between microbial species and between strains of the same species. As with other antimicrobial processes, the response of a microbial cell and hence its resistance to ionising radiation depends (i) on the nature and amount of direct damage produced within its vital target, (ii) on the number, nature and longevity of radiation-induced reactive chemical radicals produced which will damage the organism and on the inherent ability of the cell to tolerate or accurately repair the damage, and (iii) the influence of the intra- and extracellular environment on its resistance. Although the range of microbial radiation resistance is large, it is not as wide as the variation in heat resistance.

In general, Gram-negative bacteria, including the common spoilage organisms of many foods such as *Pseudomonas* spp. and particularly enteric species including pathogens such as salmonellae and shigellae, are generally more sensitive than vegetative Gram-positive bacteria. The spores of the genera *Bacillus* and *Clostridium* are more resistant still. Rarely, very resistant vegetative organisms may be encountered, such as *Deinococcus* (formerly *Micrococcus*) *radiodurans*. These bacteria are able to repair a multiplicity of double-strand breaks in their DNA. The radiation sensitivity of moulds is of the same order as that of vegetative bacteria. Yeasts are more resistant than moulds and as resistant as the more resistant bacteria. Viruses are highly radiation resistant.

Food irradiation feasibility

Radiation treatment may be applied to food with the primary aim of eliminating enteropathogenic, non-spore-forming bacteria from food.[5,6] The primary responsibility for food safety lies with those who handle and prepare food for consumption, and public education is probably the single most important measure to prevent food-borne diseases and unnecessary food losses. However, the importance of processing food products for safety cannot be overemphasised and the control of potential contamination of foods must not create hazards for public health, or alter the acceptability of the food for the consumer.

Extensive publications support the conclusion that appropriate use of ionising radiation is an effective method of improving food safety. It can safely reduce the microbial contamination load without causing undesirable alteration in composition or acceptability of products.[4] Radiation can inactivate organisms in foods that are hermetically sealed in packages or even in the frozen state without thawing. Although freezing of food tends to raise the radiation dose required to reduce the number of food-borne organisms, it also raises the threshold doses which cause 'irradiated' flavours in high moisture foods. However, the exact dose required for each individual application has to be established for fresh or frozen food by feasibility studies and risk analysis. These would include the microbial contamination level, the microbial hazard involved, the efficacy of the radiation treatment and the fate of critical organisms during manufacture, storage, distribution and final preparation of foods.

Decades of research efforts have shown that irradiation of food is an effective method of improving food safety by reduction of pathogenic micro-organisms, particularly in poultry, meat and fish products, as well as radiation decontamination of dry food ingredients. Unfortunately, this work is often overshadowed by discussions in which prejudice and misunderstanding predominate.

From the typical radiation resistance data of the most important pathogenic non-sporing bacteria in chilled and frozen food of animal origin (**Table 1**), 3 to 5kGy is sufficient to decontaminate such products without affecting the sensory and nutritional qualities significantly.[6] Radiation doses of 3 to 10kGy proved to be sufficient to reduce viable cell counts in spices, herbs and enzyme preparations to a satisfactory level.[7] The flavour and other important technological and sensory properties of most ingredients are not affected at these radiation doses which are necessary for satisfactory decontamination.

The microflora surviving irradiation are more sensitive to subsequent food-processing treatments than the microflora of unirradiated products. Radiation decontamination can be applied best as a terminal treatment of packaged products and as such it may result in considerable saving of energy and labour when compared with alternative decontamination techniques.

Increasing awareness of the safety and acceptability of food irradiation as one of the tools for alleviating food supply problems by controlling food contamination and post-harvest losses is mainly due to research results compiled and evaluated by national and international scientific bodies. The Joint FAO/IAEA/WHO Expert Committee on Wholesomeness of Irradiated Food

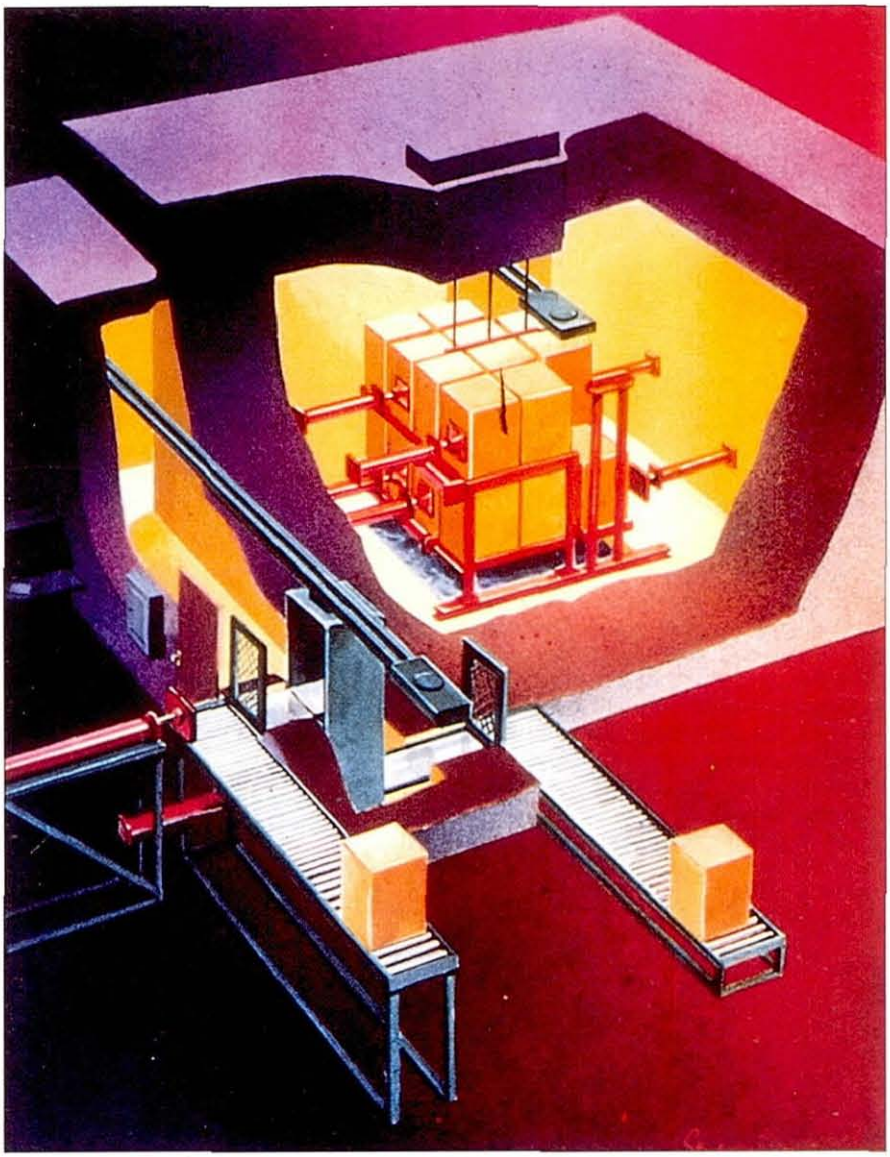


Figure: Cut-away view of a gamma irradiation plant.
Courtesy of Leatherhead Food RA, Surrey.

concluded in 1980 that irradiation of any food commodity, up to an overall average dose of 10kGy, did not present any toxicological hazard or introduce special nutritional or microbiological problems.[8]

Subsequently, a 'Codex General Standard for Irradiated Foods' and a 'Recommended International Code of Practice for the Operation of Irradiation Facilities used for the Treatment of Foods' were developed and they were adopted by the Codex Alimentarius Commission in 1983. Food irradiation is now an emerging technology in several countries and more and more clearances on irradiated food are being issued or expected to be granted in the future.

Microbiological safety of irradiated foods

Although the preservative and decontaminating effects of irradiation of food are accepted, concern is still expressed about the possibility of unseen microbiological consequences from food irradiation at 'non-sterilising' doses.

With high-dose irradiation, aimed at achieving commercial sterility of the food, no public health problems of microbiological origin can be foreseen, providing the process is effective. However, even high radiation doses capable of producing commercial sterilisation in food cannot be relied upon to completely destroy preformed microbial toxins, whether these are present in the microbial cells or in the food. This situation is no different from conventional methods of food processing; for example, staphyloenterotoxins or mycotoxins are not safely inactivated by canning food because of their high thermal resistance.

The main questions concerning microbiological safety of irradiated foods are:

- Can selective changes in the microflora, caused by non-sterilising radiation doses, make known pathogens more likely to occur or increase unfamiliar pathogens?
- Is it probable that 'mutational' (including adaptive) changes might make pathogens more virulent, more harmful, or more difficult to recognise and could new pathogens arise in this way?
- Is it possible that development of radiation-resistant strains might render the antimicrobial irradiation processes *ineffective*?

These problems have been investigated by many laboratories and were the subject of discussions at international meetings of experts.[9] A consultants meeting jointly convened by FAO and IAEA scrutinised the microbiological aspects of food irradiation in 1974. The conclusion was that microbiological safety of irradiated foods is fully comparable with that of foods preserved by other acceptable methods.[10] This conclusion was subsequently endorsed by the Joint FAO/IAEA/WHO Expert Committee on Wholesomeness of Irradiated Food[11] and by a meeting of the Board of the International Union of Microbiological Societies.[12]

Irradiation might cause foods to spoil in a somewhat unusual manner because of the changes in the microflora. Normally, the more resistant and metabolically less active species survive, such as *Moraxella* spp., lactic acid bacteria (especially enterococci) and yeasts. A similar situation, however, has long been shown to exist in cured foods and in certain types of food packaging. Among the more radiation-resistant vegetative organisms, none has been associated with a pathogenic risk in irradiated foods. One should bear in mind, however, the high resistance of spores of a few food-poisoning, spore-forming species. Irradiation up to a dose of 10kGy is unlikely to kill all spores unless they occur in small numbers in food. Therefore, restrictions are desirable for the storage of radiation-pasteurised, non-acidic, perishable, high protein foods. The requirement is to hold them below 3°C. Similar problems of spore survival arise, however, with any non-sterilising food process, e.g. vacuum packaging, smoking of fish or heat pasteurisation, where similar storage temperature requirements should exist.

The effect of irradiation differs from that of heating as far as viruses are concerned because viruses are among the most radiation-resistant pathogens. Therefore, radiation treatment is not likely to be useful to 'clean-up' a virus-infected food. In this respect, irradiation is similar to refrigeration. On the other hand, irradiation produces some inactivation of viruses whilst controlling bacteria. In food processed by heat treatment above 55°C before or after irradiation, the risk from virus infection diminishes considerably because of their high heat sensitivity.

Foods which suffer from mould spoilage, when treated with relatively low doses, of the order of 1kGy, will show a significant reduction of the mycoflora and delay spoilage. Semi-dry grains and pulses which normally support toxigenic fungi if mishandled, could become reinfected after irradiation, but the indications are that irradiation diminishes this problem.

Regarding induction/selection of 'mutants', the general mutational effect of irradiation will be damage, i.e. impairment of normal functions and introduction of new biological demands.[2] Whilst this is likely to make organisms more difficult to grow and be recognised, it is not likely to make them more pathogenic.

It was shown for *Clostridium perfringens* and *Staphylococcus aureus* that the production of enterotoxins is not affected by low dose irradiation treatment. As for fungi, the genera *Aspergillus* and *Penicillium*, with other important toxigenic species, are among the more radiation-sensitive moulds. Although some laboratory studies with heat-sterilised media inoculated with irradiated toxigenic cultures reportedly resulted in an apparent increase of mycotoxin pro-

duction, most of these studies have little relevance to practical food irradiation.[2] In addition, it was demonstrated by several independent experiments that increased toxin production is not related to the introduction of more toxigenic mutants because, in unirradiated cultures, toxigenic fungi may produce more toxin when grown from lower inoculum levels than from dense inocula. Thus, reduction of a high inoculum level, either by irradiation or simply by dilution, resulted in the same increase in toxin production.[13] When mycotoxin studies were carried out under more practical conditions, increased formation of mycotoxins was not observed.[9]

Most attempts to develop and/or select radiation-resistant mutants have generally used the principle of 'growth–irradiation cycles'.[2] However, heat resistance can develop in a similar way. In normal processing circumstances, it is unlikely that similar 'recycling' conditions would exist.

Radiation-induced changes in taxonomically relevant diagnostic characters seem less serious than might be feared. Nevertheless, methods should be specifically evaluated for their suitability to isolate radiation-damaged cells. Media providing the highest colony counts of untreated micro-organisms are not necessarily the most suitable for recovering radiation-damaged cells.[14] Demands on media, by organisms which may be modified by radiation damage, may differ even for species belonging to the same genus. Similar observations have been made with heat-damaged cells.[15]

Conclusion

Food irradiation is an important addition to the methods of control over food-borne micro-organisms and would not present any specific hazards from shifts in the microflora or changes in the characteristics of micro-organisms. Although each individual case should be thoroughly considered, most of the questions concerning microbiological safety of irradiated foods are not unique to irradiation but are common to all food preservation treatments which do not lead to a sterile product. The probability of microbiological risk seems sufficiently low when compared to the expected benefits that the balance of advantage to public health seems to be heavily in favour of irradiation. **It must be stressed, however, that the benefits of irradiation should never be considered as a replacement for poor product quality or for poor handling and storage conditions, i.e. as a substitute for good *manufacturing practice***. As is true with any other food process, gains in microbiological or keeping quality attained by irradiation can be and must be safeguarded by proper control in the food irradiation facilities and by proper care of the product before and after processing. Similarly, potential hazards can be more efficiently prevented by the identification and monitoring of critical control points both in the production process and in the irradiation facilities.

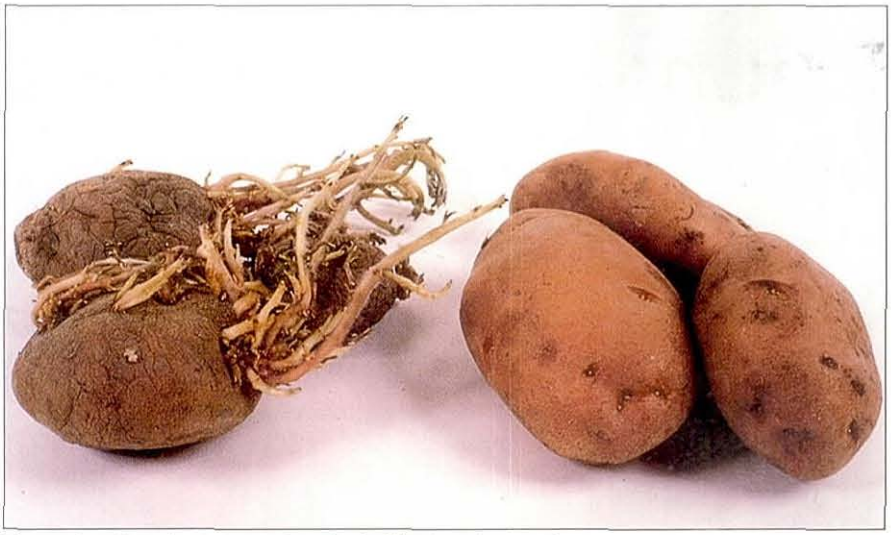


Figure: Irradiated potatoes, showing inhibition of sprouting.
Courtesy of Leatherhead Food RA, Surrey.

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OXOID NEWSLINES

Media for Food Mycology

Oxoid offer a comprehensive range of media for food mycology. There is an increased awareness of the need for specific media for the isolation and enumeration of foodborne fungi. The Oxoid range of media include general purpose media such as Rose Bengal Chloramphenicol Agar and Oxytetracycline GYE Agar, DG18 Agar for xerophilic moulds and AFPA Agar for the isolation of toxigenic fungi.

Further information and details are provided in an Oxoid booklet describing media for food mycology.

Prions—a new microbiological hazard

RE Spier, Professor of Microbiology, University of Surrey, UK.

Small proteinaceous infectious particles (prions) are the most recent form of microbiological threat to be recognised. They appear to lack nucleic acid and, furthermore, their infective association with transmissible encephalopathies overlaps with genetic origins of these diseases.

Prions as causative agents

When an infectious disease is observed, the specific cause is sought using Koch's postulates. Such was the case when sheep exhibited a disease which came to be known as scrapie (**Figure 1**). The material in the sheep's brain was partially purified and was shown to initiate a similar disease in a second animal. Infectious material could similarly be prepared from the brain of the second animal.

This work, however, showed a unique problem. The purified material which carried the disease forward did not contain any nucleic acid. How could such material replicate when it lacks sufficient nucleic acid to code for its replication? There is still contention about the lack of any nucleic acid in the agent but the evidence that prions are nucleic-acid-free is compelling (**Table 1**).

The data shown in **Table 1** indicate that nucleic acid cannot be detected in prions and, whilst it cannot be conclusive, the evidence on radiation inactivation would only allow a <5 base of single-stranded DNA or a 30–45 base pair segment of double-stranded DNA. Infectivity is associated with a glycoprotein which has an apparent MW of 27–30kD. In nature this glycoprotein is associated with lipids and detergent materials so that the physical form which it takes can be amyloid and form strands. Alternatively the glycoprotein can be modified so as to become associated with liposomes, in which case it appears to be in the form of irregular spheres. The infective material is resistant to heat denaturation and can withstand variations in the ambient pH. It is also resistant to some proteases.

Figure 1: A sheep with scrapie.
Courtesy of AFRC & MRC Neuropathogenesis Unit., Edinburgh.

Table 1: Evidence of lack of nucleic acid in prions.

The protein:

1. A protein with a SDS electrophoresis based MW of 27–30kD which co-purified with the infectious agent.
2. Monoclonal antibody affinity chromatography purified material also related the infectious material to a protein throught to be the causal agent of infection.
3. The amount of the 27–30kD protein is proportional to the infectivity of that material.
4. Rabbit antisera raised to the 27–30kD protein neutralised scrapie infection.
5. Denaturation or other modification, such as proteolysis, of the 27–30kD protein destroyed its infectivity.
6. When the 27–30kD protein is incorporated into liposomes and other lipid-containing particles, infectivity is maintained.

Nucleic acid content:

1. *Neutralisation of the protein by ionising radiation indicates that if this takes place by nucleic* acid degradation then the amount of nucleic acid is less than 30–40 base pairs if it is double-stranded and much less if it is single-stranded. Such a small length of nucleic acid is inadequate to code for the 27–30kD protein associated with infectivity.
2. Attempts to neutralise the infectivity of the agent, using nucleic acid hydrolysing enzymes, have been unsuccessful.

Prion-associated diseases (transmissible encephalopathies)

A complication of the infective cause of prion-associated diseases is that, whilst in some cases transmission is clearly by extracorporeal agents, in other cases the diseases could be inherited.

The transmissible *human* encephalopathies include:

i) Creutzfeldt–Jakob disease (CJD), where 85–90% of the cases can be attributed to infection from the use of human cadaver brain sources, e.g. growth hormone, corneal transplants or implantation of cerebral electrodes. The remaining cases involve genetic, vertical transmission.
ii) Gerstmann–Straussler syndrome, where the proportions are reversed and some 90% of cases are genetic in origin.
iii) Kuru, a disease, confined to the Eastern Highlands of Papua New Guinea, consistent with transmission by endocannibalism before 1956 and with an incubation period of 30 years.

The transmissible *animal* encephalopathies include: scrapie in sheep and goats; transmissible mink encephalopathy; chronic wasting disease of mule, deer and elk, bovine spongiform encephalopathy (BSE) (**Figures 2** and **3**).

The animal diseases can be quite useful in checking hypotheses in human diseases. A further help in these investigations is that the infectious agent can be transmitted to both mice and hamsters and, more recently, to a line of mice neuroblastoma cells.[1]

The infective pathway

The cells of normal mammals contain a glycoprotein which has the same amino-acid composition as the infectious agent, providing the agent is derived from an animal of the same species. However, glycosylation of the protein is not necessarily *identical*.[1]

When infectious material is taken from one species of mammal and inserted into the brain of another species of mammal the incubation period of the disease is much

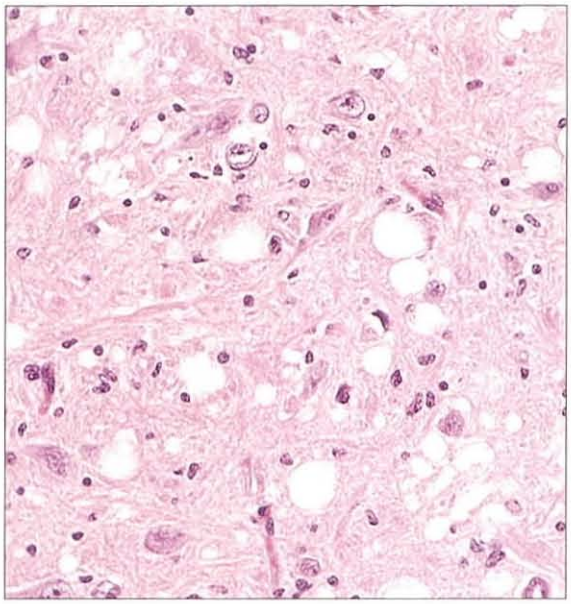

Figure 2: Neurophil vacuolation in BSE affected brain.
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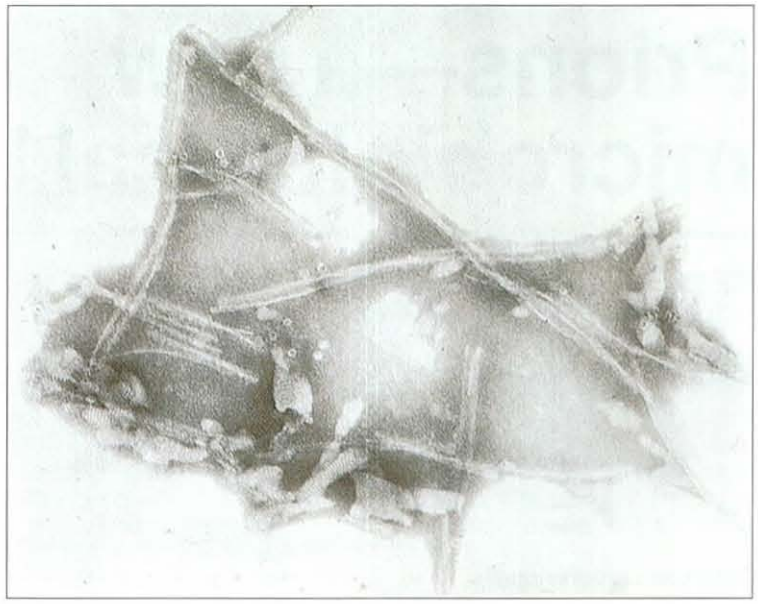

Figure 3: Electron micrograph of negatively-stained BSE fibrils.
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longer than when inserted into the same species.

Intracerebral inoculation gives a shorter incubation period than other methods of infection. Inoculation of neonates causes a more rapid onset of disease than inoculation of weanlings.

Prion protein which is produced naturally in cells (PrP-C) is under the control of a gene which codes for the protein sequence. There is also a second gene involved which affects the incubation period before an infection becomes apparent. PrP-C is known to increase when neuroblasts differentiate into neurons and the nature of glycosylation indicates that this molecule could be involved with cell–cell recognition.

Mutations in the gene which codes for the sequence of amino acids in the naturally produced protein (PrP-C) can lead to the emergence of an encephalopathy without the agency of an infective process.

Terminology: differences and similarities of the proteins

(i) PrP-C is the prion protein naturally produced in the cell.
(ii) PrP-Sc is the infectious prion protein which causes scrapie.
(iii) PrP-X is a general term for infectious prion proteins.
(iv) PrP-C(X) is the natural protein modified into the infectious agent.

In normal cells there is 1mg PrP-C per g of cell. When a cell is infected with the scrapie agent the same amount of PrP-C can be detected but there is an additional 10mg per g of PrP-Sc produced. PrP-C is generally found in the cell membrane and it can be released from that membrane with an identified enzyme. PrP-Sc is found within the body of the cell.

The PrP-Sc material will co-purify with scrapie infectivity whereas the PrP-C material will not.

The half-life of PrP-C in the cell is about 6–8 hours. It is thought that the half-life of PrP-Sc is many times longer.

Both PrP-C and PrP-Sc have two asparagine residues which are glycosylated, although the details of the glycosidic side-chains have yet to be determined. They also have glycosylphosphatidylinositol anchor moieties as part of their molecular structure. They both have an intramolecular disulphide bridge.

Prions and disease—a possible mechanism

The infectious prion protein differs from the normal cell generated prion protein by its ability to alter the way the cell makes the protein so that infectious prion protein material is also produced. This could be achieved by an alteration to the way in which the protein coded by the normal PrP-C gene is post-translationally modified. This change in the post-translational modification is affected by the infectious form of the PrP-C which already has been so modified.

In **Figure 4** the modifying effect could be achieved by causing an alteration to the activity or spatial relationships of the enzymes in the Golgi apparatus or by expressing an enzyme activity in its own right. However, the end result is the production of a protein whose post-translational modifications are like that of the infectious agent rather than the normal protein, irrespective of the amino-acid sequence of the original protein.

In order for the infection to proceed it is necessary for the infectious prion protein PrP-C(X) to leave the cell of its origin and to pass to other cells in the same animal, forming a large number of modified cells and creating the pathogenic state. Furthermore, following the spread of the agent, it is possible for materials from the infected animal to pass to another mammal of the same or different species. Clearly it is unlikely that intracerebral inoculation occurs in nature; rather the usual ports of entry should be considered such as the gut (where the stability of the protein to adverse conditions preserves some low rate of infectivity) or alternatively via a skin abrasion in the mouth or foot or other sensitive area.

The different incubation times of the disease following infection may be explained with a differential treatment of the infectious agent by either the components of the immune system or by the way in which the infecting agent enters the cell. Thus the long incubation period observed when the infectious agent from one species of mammal is inoculated into a different species of mammal can be explained by the prevalence of some of the existing, but not specifically induced, circulating antibody. The antibody can impede either the infectious process itself or the facility with which the infectious material gets into the cell. This could account for the more rapid onset of disease when infectious prions of one species are inoculated into animals of the same species. In these cases there would be fewer reactive antibodies to the autologous material. Similar concepts could explain why neonates are more susceptible than weanlings to infectious material of the same species. That the disease can be both infectious and genetic can be attributed to either the processes described above and/or a new process whereby an alteration in the sequence of the amino acids causes a change in the post-translational modifications so as to generate the infectious type of agent. In which case a new infectious agent will be generated.

It should be noted that when a cell is infected it continues to produce the non-infectious material at the normal rate. This effect suggests that only a portion of the post-translational machinery is affected and it may not all be involved in the processing of the normal prion protein material.

The attraction of the agent to nerve cells may be explained by the particular capability of these cells to take both non-infectious and infectious prion proteins into their membranes. The difference in the infectious material is that it does not stay in the membrane but moves to the Golgi apparatus, expressing its modifying properties at that site.

Conclusion

Prions are clearly a new class of infectious material. They are unlike their closest

equivalent, the viruses, in that they lack nucleic acid as an essential component of their structure. Rather, they take advantage of a gene which seems to be most active in nerve cells and produces an essential protein which is benign and necessary to the functioning of the cell. However, infectious prions seem to have a post-translational modification which is capable of diverting some of the normally produced native protein to the infectious type. The transfer of these infectious-type modification materials from one cell to another, the tropism of the infectious agent for nervous tissue and the way the agent moves between animals have yet to be determined, although the model systems of the methods used by viruses will no doubt provide a fruitful basis for experimentation. There is little doubt that the agent will infect across mammalian species barriers when experimental procedures are used; however, natural infection is many orders of magnitude less efficient than the direct methods of the laboratory. Whilst the possibility of a human becoming infected by ingesting BSE-infected bovine material cannot be ignored, the probability of such an event occuring is so remote that actions taken to avoid such a contingency could have a similar risk of causing damage.

Laboratory experiments have shown that it is possible to neutralise the infectivity of the prion by induced antibodies and it is possible that a vaccine protective against infection with this new agent may be practicable. Whether or not the production of such a prophylactic is cost effective, it is certain that research on prions will show new intricacies of cellular activities which will carry forward the work to understand life.

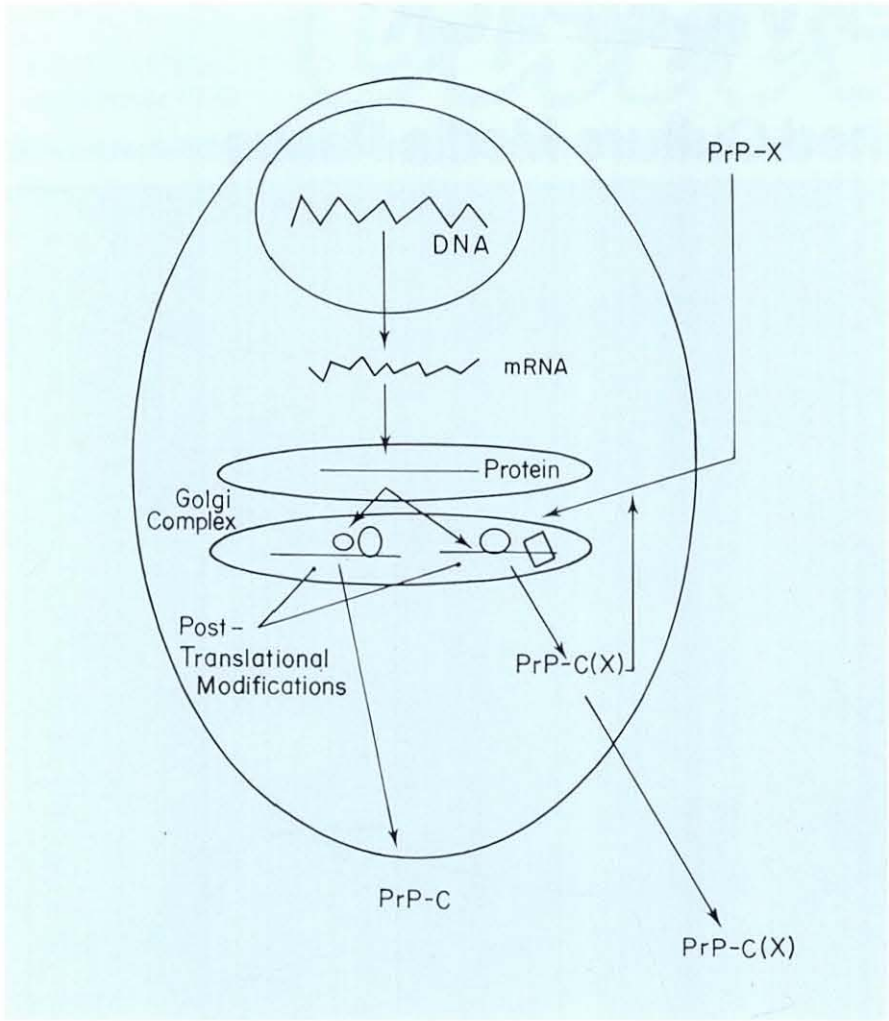

Figure 4: Post-translational modification.

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Front cover picture

The front cover picture from the previous issue of *Culture* represented a scanning electron microscopy of a *Xenopus laevis* oocyte damaged by the cytolytic S-toxin produced by *Clostridium perfringens*.

OXOID NEWSLINES

New Culture Media —Meat Products

Unipath have further extended their Oxoid Culture Media range to include STAA Medium. STAA Medium has been developed for the selective isolation of *Brochothrix thermosphacta* which constitutes a significant proportion of spoilage flora in meat and vaccum packed meat products.

The principle of the medium is based firstly on the ability of *Brochothrix thermosphacta* to resist high concentrations of streptomycin sulphate and secondly the inhibitory action of thallous acetate and cycloheximide on the growth of other background flora found in those meat products.

STAA Agar Base (Code CM881B) is available in packs of 500gms. STAA Selective Supplement (Code SR151E) is available in packs of 10 vials, each to supplement 500ml of CM881B.

For further information please contact Miss N Pope, Unipath Limited, Wade Road, Basingstoke, Hants RG24 0PW. Tel: (0256) 841144. Tlx: 858793. Fax: 0256 463388.

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- No Mess
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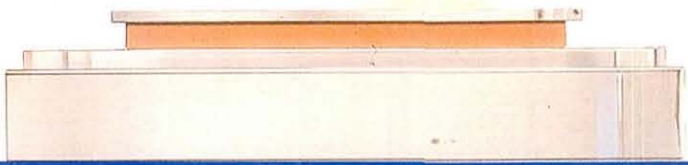

Just when you thought that media preparators had simplified the task of preparing culture media, Oxoid's SERVICE-PACKS have made it even simpler.

Culture media weighed for you and supplied in convenient, easy open containers. Simply snap off the lid and empty the contents into your preparator vessel.

That's what I call service!

OXOID QUALITY CULTURE MEDIA

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