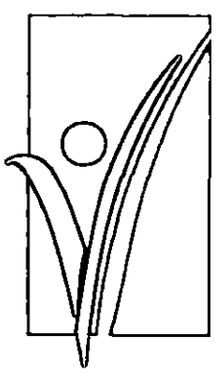
First Annual Report 1995/1996

Report to the Minister for Health



FOOD SAFETY ADVISORY BOARD

Members of the Food Safety Advisory Board

Dr Fergus Hill (Chairman)	Dublin Region Public Analyst Eastern Health Board
Mr James Duggan	Principal Officer, Department of Health
Mr Raymond Ellard	Chief Environmental Health Officer Department of Health
Prof John Flynn	Consultant Bacteriologist University College Hospital, Galway
Dr Cliodhna Foley-Nolan	Specialist in Public Health Medicine Southern Health Board
Ms Ailish Forde	Assistant Director, IBEC
Mr Colm Gaynor	Director of Veterinary Services Department of Agriculture, Food & Forestry
Prof Michael J Gibney	Department of Clinical Medicine Trinity College Dublin
Prof John Hannan	Faculty of Veterinary Medicine University College Dublin
Dr James Kiely	Deputy Chief Medical Officer Department of Health
Ms Mary McCarthy Buckley	Faculty of Food Science & Technology University College Cork
Ms Maura Nolan	Food Division Department of Agriculture, Food & Forestry
Ms Brid O'Connor	Assistant Director of Consumer Affairs
Mr Sean O'Donoghue	Sea Fisheries Control Manager Department of the Marine
Dr Emer Shelley	Specialist in Public Health Medicine Department of Public Health Eastern Health Board
Dr James J Sheridan	Head, Meat Technology Department The National Food Centre
Mr Michael Mulkerrin Acting Secretary	Sir Patrick Dun's Lower Grand Canal Street, Dublin 2

FOOD SAFETY ADVISORY BOARD

FIRST ANNUAL REPORT
1995/1996

Report to the
Minister for Health

Sir Patrick Dun's
Lower Grand Canal Street
Dublin 2

Telephone: (01) 662 5486
Fax: (01) 662 5490
E-Mail: fsab@ioL.ie

ISBN 0-7076-3834-8

P.48664 2,000 4/97 Spl. Brunswick Press Ltd.

CONTENTS

Part 1	**Page**
Establishment of the Food Safety Advisory Board	4
Food Additives, Contaminants and Irradiated Food Committee	5
Microbiology Committee	6
BSE - Maternal Transmission Working Group	7
Finance Committee	8
Legislation Committee	8
Nutrition Committee	10
Food Safety - Dr Fergus Hill	13
Food Safety and Categories of Risk	13
Foodborne Diseases of Microbial Origin	14
Surveillance Programmes	15
Pilot Study on Foodborne and Zoonotic Diseases	16
Food Scares : A – BSE in Cattle	24
B – Phthalates in Baby Milk Scare	26
Annex I	
Report of Food Safety Advisory Board to the Minister for Health on Bovine Spongiform Encephalopathy (BSE)	28
Annex II	
Report of Working Group to the Minister for Health on Maternal Transmission	36

ESTABLISHMENT OF THE FOOD SAFETY ADVISORY BOARD

The Food Safety Advisory Board was established by the Minister for Health under the powers conferred on him by sections 3 to 6 of the Health (Corporate Bodies) Act, 1961 (No. 27 of 1961) on 22 June 1995. The Board held its inaugural meeting on 18 July 1995.

Two of the original members resigned - Ms Mary Kearns, Office of the Director of Consumer Affairs and Dr Vivion Tarrant, Director of The National Food Centre. They were replaced by Ms Brid O'Connor, Assistant Director of Consumer Affairs and Dr James J Sheridan of The National Food Centre.

The Board also met on the following dates:-

6 September 1995
4 October 1995
1 November 1995
6 December 1995
7 February 1996
13 March 1996
3 April 1996
1 May 1996
5 June 1996
3 July 1996
11 September 1996
2 October 1996
6 November 1996
4 December 1996

The following Committees were established by the Board:-

Finance
Food Additives, Contaminants and Irradiated Food
Legislation
Microbiology
Nutrition

Prof Michael J Gibney has agreed to Chair a Committee on Scientific Co-operation which will enable the Board to deal with issues that arise under Council Directive 93/5/EEC.

A Startup Committee has also been formed to oversee the setting up of facilities for the Board. From June 1995 to mid 1996 the Board operated from accommodation provided by the Department of Health. Premises were acquired in mid 1996 on a lease basis from the Eastern Health Board at Sir Patrick Dun's, Lower Grand Canal Street, Dublin 2.

FOOD ADDITIVES, CONTAMINANTS AND IRRADIATED FOOD COMMITTEE

Dr Fergus Hill (Chairman)	Dublin Region Public Analyst Eastern Health Board
Mr Hugh Boyle	Food Unit Department of Health
*Mr Raymond Ellard	Chief Environmental Health Officer Department of Health
Dr James Fleming	Agricultural Inspector Department of Agriculture, Food & Forestry
Dr Frank Kenny	Senior Superintending Veterinary Inspector Department of Agriculture, Food & Forestry
Ms Joyce Lambe	Nutriscan Ltd Trinity College Dublin
Dr Nora O'Brien	Lecturer in Nutrition University College Cork
Dr Michael O'Keeffe	Teagasc, The National Food Centre, Dublin 15

The Committee met on the following dates:-

19 January 1996
10 June 1996
5 November 1996

*Mr Ellard joined the Committee on 5 November 1996.

MICROBIOLOGY COMMITTEE

* Prof John Hannan (Chair)	Faculty of Veterinary Medicine University College Dublin
Mr Raymond Ellard	Chief Environmental Health Officer Department of Health
* Prof John Flynn	Consultant Bacteriologist University College Hospital, Galway
Mr Michael Fallon	Senior Superintending Veterinary Inspector Department of Agriculture, Food & Forestry
Dr James J Sheridan	Head, Meat Technology Department The National Food Centre
Dr Colin Hill	Department of Microbiology University College Cork
Dr Fergus Hill	Dublin Region Public Analyst Eastern Health Board
Dr Cliodhna Foley-Nolan	Specialist in Public Health Medicine Southern Health Board
Dr James Kiely	Deputy Chief Medical Officer Department of Health
Mr Sean O'Donoghue	Sea Fisheries Control Manager Department of the Marine

* Prof Hannan retired as Chair of the Committee in September 1996 and Prof Flynn was appointed to this position.

The Microbiology Committee met on the following dates:-

24 November 1995
22 January 1996
5 March 1996
2 April 1996
29 April 1996
27 June 1996
16 July 1996
30 September 1996
25 November 1996

One of the main tasks undertaken by the Microbiology Committee was to compile a report to the Minister for Health on Bovine Spongiform Encephalopathy (BSE) in April 1996. This report appears at pages 28 - 35.

The Committee would like to acknowledge the assistance given by the following:-

Mr Albert Costelloe	Deputy Chief Veterinary Officer Department of Agriculture, Food & Forestry
Dr Catherine Keohane	Consultant Histopathologist Cork University Hospital
Mr John Nolan	Superintending Veterinary Inspector Department of Agriculture, Food & Forestry
Mr Dan O'Reilly	Director, Central Veterinary Laboratory Department of Agriculture, Food & Forestry
Dr P J O'Reilly	Senior Superintending Research Officer Department of Agriculture, Food & Forestry
Prof Joseph Quinn	Faculty of Veterinary Medicine University College Dublin
Dr Mark Rogers	Department of Zoology University College Dublin
Mr E D Weavers	Senior Research Officer Department of Agriculture, Food & Forestry

Working Group on Maternal Transmission

The Committee also examined BSE - Maternal Transmission and reported to the Minister for Health on 31 July 1996. The report appears at pages 36 - 40. The Working Group on Maternal Transmission had the following membership:-

Prof John Hannan (Chair)	Faculty of Veterinary Medicine University College Dublin
Dr Fergus Hill	Dublin Region Public Analyst Eastern Health Board
Mr Raymond Ellard	Chief Environmental Health Officer Department of Health
Prof John Flynn	Consultant Bacteriologist University College Hospital, Galway
Dr John Devlin	Deputy Chief Medical Officer Department of Health
Mr Albert Costelloe	Deputy Chief Veterinary Officer Department of Agriculture, Food & Forestry

Working Group on Genetically Modified Organism (GMOs) and Novel Foods

The Microbiology Committee established a Working Group to examine issues relating to GMOs and Novel Foods. The Group held its first meeting on 14 November 1996. Dr Colin Hill, Department of Microbiology, University College, Cork has agreed to Chair the Group.

FINANCE COMMITTEE

Mr James Duggan (Chair)	Principal Officer Department of Health
Ms Ailish Forde	Assistant Director IBEC
Dr Fergus Hill	Dublin Region Public Analyst Eastern Health Board
Dr James Kiely	Deputy Chief Medical Officer Department of Health
Mr Sean O'Donoghue	Sea Fisheries Control Manager Department of the Marine

The Finance Committee met on the following dates:-

23 October 1995
1 March 1996

LEGISLATION COMMITTEE

Ms Ailish Forde (Chair)	Assistant Director IBEC
Mr Hugh Boyle	Food Unit Department of Health
Mr Raymond Ellard	Chief Environment Health Officer Department of Health
Mr Ibrahim El-Moslemany	Development and Q.A. Manager CPC Foods (Ireland) Limited, Dublin
Ms Brid Farrell	Food Division Department of Agriculture, Food & Forestry
Mr Michael Hickey	Golden Vale Foods Plc, Charleville
Dr Fergus Hill	Dublin Region Public Analyst Eastern Health Board
Mr John Langan	The National Food Centre

Mr Brendan Moylan	Enterprise Programme Division Department of Enterprise & Employment
Ms Brid O'Connor	Assistant Director Office of the Director of Consumer Affairs
Mr Sean O'Donoghue	Sea Fisheries Control Manager Department of the Marine
Mr Kevin Smyth	Poultry Division Department of Agriculture, Food & Forestry

The Committee met on the following dates:-

29 November 1995
24 January 1996
4 April 1996
25 June 1996
12 September 1996
4 November 1996

Compendium of Irish Food Law

The Legislation Committee formed a working group to deal with this issue.

NUTRITION COMMITTEE

Dr Emer Shelley (Chair)	Specialist in Public Health Medicine Department of Public Health Eastern Health Board
*Mr Jim Beecher	Deputy Chief Inspector Department of Agriculture, Food & Forestry
Mr Hugh Boyle	Food Unit Department of Health
Dr Ralph Counahan (R.I.P. 20 April 1996)	Consultant Paediatrician Waterford Regional Hospital
Dr Albert Flynn	Department of Nutrition University College Cork
Dr Mary A T Flynn	Department of Biological Sciences Dublin Institute of Technology
Prof Michael J Gibney	Department of Clinical Medicine Trinity College Dublin
Dr John Kearney	Institute of European Food Studies Trinity College Dublin
Prof Cecily Kelleher	Professor of Health Promotion University College Galway
Ms Bríd'n McIntyre	The National Food Centre
Ms Ursula O'Dwyer	Consultant Dietitian Department of Health
Ms Kathryn Raleigh	IBEC
Ms Vivien Reid	Department of Preventive Medicine/Cardiology, St Vincent's Hospital, Elm Park, Dublin 4
Dr Helen M Roche	Department of Clinical Medicine Trinity College Dublin
Dr Fergus Hill *(ex officio member)*	Dublin Region Public Analyst Eastern Health Board

*Mr John O'Mahony, A.I., Dairy Inspectorate, Department of Agriculture, Food and Forestry replaced Mr Jim Beecher as a member of the Nutrition Committee in September 1996.

The Committee met on the following dates:-

5 December 1995
17 January 1996
24 April 1996
18 September 1996
9 October 1996
27 November 1996

The Committee formed the following Working Groups:-

Food and Nutrition Policy

Research and Surveillance

Technical Issues

Memberships are as follows:-

Food and Nutrition Policy

Dr Emer Shelley (Chair)	Specialist in Public Health Medicine Department of Public Health Eastern Health Board
Dr Ralph Counahan (R.I.P. 20 April 1996)	Consultant Paediatrician Waterford Regional Hospital
Dr John Kearney	Institute of European Food Studies Trinity College Dublin
Ms Lulu McGann	Food Unit, Department of Health
Ms Brídín McIntyre	The National Food Centre
Ms Geraldine Nolan	Department of Health Promotion University College Galway
Ms Ursula O'Dwyer	Consultant Dietitian Department of Health
Ms Kathryn Raleigh	IBEC
Ms Vivien Reid	Department of Preventive Medicine/Cardiology St Vincent's Hospital, Elm Park, Dublin 4

Research and Surveillance

Dr Emer Shelley (Chair)	Specialist in Public Health Medicine Department of Public Health Eastern Health Board
Mr Hugh Boyle	Food Unit, Department of Health
Ms Sharon Friel	National Nutrition Surveillance Centre University College Galway
Dr Mary A T Flynn	Department of Biological Sciences Dublin Institute of Technology, Kevin Street, Dublin 8
Prof Cecily Kelleher	Department of Health Promotion University College Galway
Ms Joyce Lambe	Nutriscan Ltd, Trinity College Dublin

Technical Issues

Prof Michael J Gibney (Chair)	Department of Clinical Medicine Trinity College Dublin 2
Dr Albert Flynn	Department of Nutrition University College Cork
Prof Patrick Morrissey	Department of Nutrition University College Cork
Mr Paddy O'Donovan	Avonmore Plc, Kilkenny
Dr Helen M Roche	The Trinity Centre for Health Sciences St James's Hospital, Dublin 8

At it's meeting held on 3 July 1996 the Board decided that the structures established to handle nutrition issues should be reorganised.

Sub Committees have since been established to address the following:-

(i) Infant Feeding Policy which met on 19 October, 7 November and 12 December 1996

(ii) Folic Acid & Neural Tube Defects which met on 23 October, 7 November and 5 December 1996

(iii) Food and Nutrition Policy for the Elderly.

FOOD SAFETY

Dr Fergus Hill

Chairman, Food Safety Advisory Board

The Food Safety Advisory Board was established by the Minister for Health on 22 June 1995 under the Food Safety Advisory Board (Establishment) Order, 1995 (S.I. No. 155 of 1995). In accordance with article 20 of this Order, the Board is obliged to submit an annual report. This report covers the period from 22 June 1995 to 31 December 1996.

The Board's terms of reference require it to advise on issues relating to food safety, nutrition, zoonotic diseases, food law, scientific co-operation in the area of food, control systems for food processors and food outlets as well as any other matters referred to it by the Ministers for Health; Agriculture, Food and Forestry and the Marine. The Board has sixteen (16) members and is representative of a wide spectrum of interests concerned with food. Members include nutritionists, food scientists, industry, consumer and Government representatives.

FOOD SAFETY AND CATEGORIES OF RISK

Food Safety can be considered under four main headings:

1. Materials that afford a real hazard to health (based on scientific data)

2. Materials perceived as hazardous but not supported by adequate scientific facts

3. Materials that pose a potential hazard to a minority of sensitised consumers, e.g. allergenic substances in peanuts

4. Materials that may possibly constitute a hazard but where the issue remains to be definitely determined

Coping with technological risk has been described as the major challenge of the twenty first century. As living standards rise, people seek to improve their quality of life and that of future generations without sacrificing the protection of health and safety and the environment.

The making of judgements of these issues is constrained by inadequacies in the availability of information about the extent of risks and their distribution. Risk assessment is coming increasingly into prominence as a means of injecting rationality into the debate. The techniques use a variety of information sources, from hard scientific facts, through hypotheses of possible but often untestable future effects to empirical evidence of public perceptions. It leads to a reasoned estimation of the risk attached to a particular activity or event and to possible alternatives and informs the decisions on the optimum course of action to be taken in the light of the considerations. Such assessments involve identifying what might cause harm in a particular situation (the hazard): estimating the likelihood that the hazard will actually be realised and its consequence for a specialised population (the risk) and assessing what action (if any) is appropriate to reduce the risk, to achieve equity in the distribution and proportionality of the cost of its control.

In practice, risk assessment is a mixture of science and judgement, within a policy framework. It is a composite of many established disciplines which may include toxicology, statistics, biochemistry and microbiology.

Limitations of Current Methods

There are inherent limitations in the use of epidemiology and animal based experiments to predict risk to humans. To detect defects in small numbers of animals it is generally necessary to use high doses; this may distort the effect of the substance. There are unavoidable problems in extrapolating from animals to man and from high doses to low doses. For example, there may be a significant difference in the ways humans and animals metabolise substances, and also substantial differences in metabolic processes within the human population. Some individuals may also exhibit idiosyncratic reactions which are not predictable for traditional animal testing. The role of substances in the causation of allergies is such an example.

New Options

Modern scientific techniques offer new approaches to risk assessment, for instance, invitro toxicology using human tissues, molecular modelling and computer simulation. There is a need to build on these approaches together with the opportunities now being presented by advances in molecular biology to allow the European Union develop a better risk strategy.

A very important responsibility of the Food Safety Advisory Board is to attempt to narrow the gap between public perception and truth in matters concerning food safety.

Despite public preoccupation with risks associated with pesticides, additives and veterinary residues generally, the largest risks to health in Ireland from the diet stem from over-nutrition. Over consumption of saturated fats has been associated with cardio-vascular disease and obesity also carries its numerous adverse consequences for health.

Ireland continues to have a serious problem with alcohol misuse. Almost one-quarter of admissions to psychiatric hospitals are related to alcohol. The degree of alcohol misuse among young people is causing growing concern, and alcohol remains a key factor contributing to road accidents.

The third largest risk to health in Ireland from the diet is given by foodborne diseases of microbial origin which are on the increase in Europe and the United States.

FOODBORNE DISEASES OF MICROBIAL ORIGIN

The increase and frequency of foodborne infections and intoxications are due to several factors - the explosive growth of mass breeding and mass fattening of animals since World War II, the mass production and processing of food, mainly of animal origin coupled with an increasing international trade in food and animal feed. Also the migration of millions of people (tourists, immigrant labourers, refugees) on a hereto unforeseen scale has resulted in the spread of enteric pathogens. Lifestyles are changing and there is an enormous increase in convenience foods and a trend away from the traditional food preparation practices in the home.

Salmonellosis at present appears to be the most important causal agent of foodborne diseases, although campylobacter is increasing in recent years in the United States, the United Kingdom and in the Netherlands. Poultry and red meats play a major causative role in human salmonellosis. Contamination of animal feed has been recognised as a primary source of infection in animals and has led to a great number of clinically healthy salmonella carriers. It has been proved that a polluted environment including surface water and effluents, insects and birds and rodents create a cycle of infection in which animal and human carriers as well as diseased people and animals play an important role.

Secondary contamination during production, processing and culinary preparation of foods may play an even greater role than primary contaminated foods which are insufficiently heated, such as minced beef and hamburgers. Several outbreaks have been reported which make it clear that contaminated surfaces, kitchen utensils and human hands play a significant role in cross contamination, particularly of already cooked and ready to eat foods, such as poultry, meat and meat products.

Campylobacter has now surpassed salmonella as regards being the causal agent in diarrhoeal diseases. Poultry, meat and milk products are implicated. The occurrence of Listeria monocytogenes is ubiquitous. Foods of animal origin and vegetables have been involved. Dairy products and especially soft cheeses seem to be a major source for human listeria infection so far. Also, vegetables have been found to be contaminated with L.monocytogenes resulting in human disease after consumption of raw vegetables and salads. Mortality rate from L.monocytogenes can be alarming. Approximately one-third of all patients suffering from Listeriosis, mainly persons with low immunity die. Luckily the number of patients is relatively low. During the 1960's about 10 cases per year occurred in the UK, but by 1985 this had increased to over 140, the death rate being about 46%. The disease most often affects pregnant women where the incidence of miscarriage or stillbirth is high. The other serious consequences of Listeriosis is meningitis. During the first half of 1985 there were 86 cases of Listeriosis in California, 45 of these occurred in pregnant women, resulting in 13 stillbirths. There were 29 deaths in all. The source of infection in California was a Mexican style cheese. Pasteurisation equipment at the dairy plant consisted of two adjoining containers, one filled with pasteurised milk and the other with raw milk. It was discovered that there were small pin holes in the wall separating the two tanks, thus facilitating contamination of the pasteurised milk.

SURVEILLANCE PROGRAMMES

In the sixth report (published 1995) of the World Health Organisation Surveillance Programme the main objectives of the Surveillance Programme for Control of Foodborne Infections and Intoxications are given as:-

1. to identify the causes of foodborne diseases and to delineate factors contributing to the spread of these diseases.

2. to make available and distribute relevant surveillance information.

Table 1 gives a list of causal agents to be included in the W.H.O. Programme. Each country is asked to report as many foodborne diseases as justifiable and possible, but it is recognised that initially many countries may be able to report only on diseases caused by a limited number of agents.

Over 120 zoonoses have been identified to-date, and a list of these is given in **Table 2**. The list was published by Bell, Palmer and Payne, in 1988.

Table 3 gives a list of the most important food poisoning organisms in Europe as given by Granun *et. al.*, in the International Journal of Food Microbiology 1995, pages 129 to 144.

PILOT STUDY ON FOODBORNE AND ZOONOTIC DISEASES

During the year the Board engaged Dr Maeve Burke M.P.H., M.R.C.G.P., M.I.C.G.P., to carry out a Pilot Study giving information of foodborne and zoonotic diseases in Ireland. A short list of priority diseases was agreed. These were:-

- Salmonella
- Shigella
- Campylobacter
- Listeria
- E.coli 0157:H7.

The Pilot Study identified numerous sources of data on zoonoses and foodborne disease, which include primary data from health boards and laboratories, and secondary sources from Government Departments, statutory bodies and third level institutes. A total of 25 sources were identified of which 16 were contacted during the study.

Dr Burke reported that there is no national foodborne and zoonotic disease surveillance system in place. The functions of such a system are the collection and use of epidemiological information to control disease. All people contacted wished to collaborate and showed good will towards the proposed national system. Dr Burke reported a deficiency in record linkage, both within and between Government Departments on data relating to food, human and animal disease.

The Food Safety Advisory Board is actively considering Dr Burke's report with a view to putting into place a national surveillance system for foodborne disease.

Tables 4 and **5** are taken from Report No. 12 of the Food Safety Advisory Committee on foodborne illness and give information on the common food poisoning organisms and on factors that contribute to outbreaks of food poisoning.

Table 6 is taken from the National Standards Authority of Ireland (NSAI) draft Irish Standard "Guide to Good Hygiene Practice for the food processing industry in accordance with the Council Directive 93/43/EEC on the Hygiene of Foodstuffs".

TABLE 1

Causal Agents to be included in the W.H.O. Programme

Bacteria including their toxins:

Bacillus cereus
Clostridium botulinum
Clostridium perfringens
Salmonella typhi and Salmonella paratyphi A.B.C
Salmonella (other than *S. typhi* and *S. paratyphi)*
Shigella
Staphylococcus aureus
Vibrio cholerae and related vibrios
other bacteria, e.g. Brucella, Campylobacter, Escherichia coli
Francisella tularensis, Mycobacteria, Vibrio parahaemolyticus
Yersinia enterocolitica

Parasites and protozoa:

Cysticercus/Taenia
Echinococcus
Trichinella
other parasites, *e.g. Entamoeba histolytica, Giardia, Toxoplasma*

Viruses and rickettsia:

Hepatitis A
Rotavirus
other viruses, *e.g.* Echovirus, Polio virus
Coxiella burnetii

Toxic animals:

Fish, *e.g.* scombroid poisoning
Shellfish, *e.g.* paralytic shellfish poison
other animals

Toxic plants: mushrooms, *e.g. Amanita toxin*
other plant poisons

Mycotoxins: Aflatoxins
other mycotoxins

Chemical contaminants and residues:

heavy metals, *e.g.* copper, lead, mercury, tin zinc
organochlorine compounds, *e.g.* polychlorinated biphenyls
organophosphorus compounds
polybrominated biphenyls

So-far unknown etiological agents:
There will also be incidents in which the etiological agent is not identified at all; this is to be expected, as not all foodborne disease agents have been identified and/or can be easily isolated.

TABLE 2

The Zoonoses (Bell, Palmer, Payne [1988])

African trypanosomasis
American Trypanosomasis
Ancylostomiasis
Anisakiasis
AnthraxArgentine Haemorrhagic fever
Ascariasis
Asian ixodo rickettsiosis

Babesiosis
Banzi fever
Bolivian Haemorrhagiv fever
Boreliosis
Botulism
Boutonneuse fever
Brucellosis
Bunyamwere fever
Bussuquara fever
Bwamba fever

California encephalitis/
La Crosse encephalitis
Campylobacterosis
Capiallarisis
Cat scrape disease
Chikungunya fever
Chlamydiosis
Clonorchiasis
Clostridial diseases
Colorado tick fever
Cornebacterial diseases
Cowpox
Crimean Congo Haemorrhagic fever
Cryptosporidiosis
Cysticercosis and taeniasis

Dengue
Dioctrophymosis
Diphyllo Buthriasis
Dipylidiasis
Dugbe viral fever

Echinococcosis
Echinostomiasis
Epidemic encephalitis
Erlichiosis
Eeysipleoid
Esophagostomiasis

Fascioliasis
Filariasis
Flea-borne typhus fever
Foot and Mouth disease

Gastrodiscoidiasis
Giardiasis
Glanders
Group C bunyaviral fever

Haemorrhagic colitis
Haemorrhagic fever with renal syndrome
Herpes virus simiae infection
Histoplasmosis
Hymenolepiasis

Influenzae

Japanese B Encephalitis

Kemerova virus infection
Kyasanur forest disease

Lassa fever
Leishmaniasis
Leptospirosis
Listeriosis
Louping ill
Lyme disease
Lymphocytic choriomeningitis

Marburg disease
Mayaro and Sindbis
Meliodosis
Monkeypox
Mucambo fever
Murray valley encephalitis
Mycogacterial infection
(opportunist)

Newcastle Disease

Omsk Haemorrhagic fever
Opisthorchiasis
Orf
Oropouche owassan fever

TABLE 2 continued **The Zoonoses (Bell, Palmer, Payne [1988])**

Paragonimiasis
Paravaccinia
Pasturellosis
Piry fever
Plague
Powassan fever

Q Fever
Queensland tick typhus

Rabies
Rickettsialpox
Rift Valley Fever
Ringworm
Rocio viral encephalitis
Rocky mountain spotted fever
Russian spring summer encephalitis

Salmonellosis
Scabies
Schistosomiasis
Scrub fever
Semliki Forest virus infection
St Louis encephalitis
Streptobacillary fever
Streptococcosis
Strongyloidiasis
Swine Vesicular disease

Tahynia virus infection
Tanapox
Thelaziasis
Toxocariasis
Toxoplasmosis/
Congenital Toxoplasmosis
Trichinosis
Tuberculosis (Bovine)
Tularaemia

Vesicular stomatitis

Wesselbron disease
West Nile disease

Yellow Fever
Yersiniosis

Zika fever

Bolded = Study Priority Diseases

TABLE 3

The most important food poisoning organisms in Europe

Organism	Infective Dose	Incubation Time	Symptoms [a] (in the order they usually appear)
Salmonella spp.	10-10^6	7h-21d	V,D,F,A,DH
E. coli (ST)	10^5-10^8	4-6h	D,F
(LT)	10^5-10^8	16-18h	D,F
(0157:H7)	10	1-7d	BD,A,DH
Shigella spp.	10^2-10^5	1-7d	D,F,A,BD,DH
Yersinia enterocolitica	10^7	2-7d	(V),D,A,F
Campylobacter spp.	10^3-10^5	2-5d	F,BD
Vibrio cholerae	10^8	2-5d	D,A,DH
Aeromonas spp.	10^6-10^8	6-48h	D,A,DH
Staphylococcus aureus	Toxins in foods	1-6h	V,A,(D)
Listeria monocytogenes	10^2-10^8	days	-
Bacillus cereus			
(diarrhoeal)	10^5-10^7	1-6h	D,A,DH
(emetic)		6-12h	V,A
Clostridium botulinum	Toxin in foods	12-36h	V,A,(D) neurological disturbances
C. perfringens	10^8	6-16h	D,A,(DH)

[a] V: Vomiting; D: Diarrhoea; BD: Bloody diarrhoea; F: Fever; A: Abdominal pain; DH: Dehydration (danger of).

ST = heat stable enterotoxin
LT = heat labile enterotoxin

TABLE 4

Principal Food Sources of the Common Food-Poisoning organisms

Agent	Food Sources
Campylobacter jejuni	Raw poultry, meat, raw or inadequately heat-treated milk, untreated water.
Salmonella	Raw meat and poultry, raw milk, eggs.
Clostridium perfringens	Meats, poultry, dried foods, herbs, spices, vegetables.
Staphylococcus aureus	Cold foods, (much handled during preparation), dairy products, especially if prepared from raw milk.
Bacillus cereus and other Bacillus spp.	Cereals, dried foods, dairy products, meat and meat products, herbs and spices.
Escherichia coli	Many raw foods.
Vibrio parahaemolyticus	Raw and cooked fish, shellfish and other seafoods.
Yersinia enterocolitica	Raw meat and poultry, meat products, milk and milk products, untreated water.
Listeria monocytogens	Meat, poultry, dairy products, vegetables, shellfish.
Viruses*	Raw shellfish, cold foods prepared by infected foodhandlers.

*For example, small round structured viruses, parvovirus, hepatitis virus.

TABLE 5

Factors that contributed to outbreaks of Food-Poisoning in England and Wales, 1970-1982

Factors	% of Outbreaks* in which Factors Recorded
Preparation too far in advance	57
Storage at ambient temperature	38
Inadequate cooling	32
Inadequate reheating	26
Contaminated processed food	17
Undercooking	15
Contaminated canned food	7
Inadequate thawing	6
Cross-contamination	6
Raw food consumed	6
Improper warm holding	5
Infected food handlers	4
Use of left overs	4
Extra large quantities prepared	3

*1,479 outbreaks studies.

TABLE 6

Examples of processing steps which are critical to food safety

Processing Step	Critical Controls
Pasteurisation	Time/temperature; Product composition; Post pasteurisation, contamination.
Canning	Time/temperature/pressure; Product composition; Heat penetration; Head space in can; Vacuum in can; Seam integrity; Contamination of cooling water Post-cooling contamination.
Cooking	Time/temperature; Product weight; Core temperature; Product composition.
Chilled Food	Time that the temperature of food is between 5^0C and 63^0C. Contamination from the cooling media; Exposure to adverse temperature conditions after cooling i.e. from storage to consumption.
Freezing Food	Rate of chilling and freezing; Contamination from chilling or freezing media; Temperature abuse after freezing.
Asceptic Packaging	Time/temperature/heat treatment; Sterility of packaging machine and lines from heat treatment to packaging; Sterility and integrity of packages.

FOOD SCARES

A. BSE IN CATTLE

The BSE (Bovine Spongiform Encephalopathy) scare continued during the year. BSE can be classified as an infectious prion disease. Prion diseases are neurodegenerative disorders of humans and animals. Various different forms are manifested in infectious, sporadic and inherited disorders. The prion is a synonym for the infectious agent of BSE or CJD but the prion protein PrP^{c} still is a harmless glycoprotein of relative molecular mass 35,000, that is normally found on the surface membrane of cell in brain and other tissue.

Prions are composed largely, if not entirely of an abnormal isoform of the prion protein PrP^{C} designated PrP^{Sc}. The synthesis of PrP^{Sc} is a post translational process involving the refolding of alpha helical region of PrP^{c} into β sheets. Prion diseases appear to be disorders of protein conformation.

Pauling and Corry in 1951 proposed the two periodic polypetide structures called the alpha helix and the β pleated sheet (beta pleated sheet).

PrP^{Sc} can be distinguised from PrP^{c} by its resistance to protease digestion, insolubility after detergent extraction etc.

The alpha helix is a rodlike structure. The tightly coiled polypeptide main chain forms the inner part of the rod, and the side chains extend outward in a helical array.

The β pleated sheet differs markedly from the alpha helix in that it is a sheet, rather than a rod. A polypetide chain in the β pleated sheet is almost fully extended rather than being tightly coiled as in the alpha helix. Amino acid differences in PrP^{c} are responsible for the species barrier.

TRANSMISSION TO HUMANS

In March 1996 the Cruetzfelt Jacob Disease Surveillance Unit in Edinburgh reported that a new human disease, a variant form of CJD had been identified in 10 patients in the UK over the previous 16 months. Since then, one additional case had been reported from the UK and one from France. In contrast to typical cases of sporadic CJD this variant form affected young patients (average age 26.3 years) with a relatively long duration of illness (average 14.1 months). On 2 and 3 April 1996 the W.H.O. convened a consultation which recommended strengthening world wide surveillance of variant CJD, especially outside the European region, where it has already been implemented, and countries in other regions are beginning to put such a system in place in collaboration with W.H.O.

In December 1995, the UK public was assured that the risk of transmission of BSE to humans was highly unlikely, and only theoretical. The discovery of the CJD variant changed all that.

The Spongiform Encephalopathy Advisory Committee on 20 March 1996, stated that "On current data, and in the absence of any credible alternative the most likely explanation at present is that these cases are linked to exposure to BSE before the introduction of the specified bovine offal ban in 1989. This is a cause for great concern". But in the Press Release issued the same day by Stephen Dorrell, Secretary of State for Health, the statement was also made that "there remains no scientific proof that BSE can be transmitted to man by beef". As Professor Stephen Palmer, Director, Welsh Combined Centres for Public Health, states in Euro Health, Volume 2, June 1996, "All we know is that nine of the ten cases ate beef or beef products in the last ten years. Nothing else links them to BSE, and subsequent correspondence, interestingly from European Scientist suggest that the strength of evidence to link the CJD cases to BSE may well have been overstated. Time will tell".

Unfortunately, at this point in time science does not provide a firm basis for decision making. As Meslin, Stohr and Heymann of W.H.O. state: "risk assessment, public perception, economic interests, social and cultural values are equally important. Advancing our knowledge through research will permit the best possible decisions to be taken not only to ensure that national economies dependent on the beef industry can be maintained and developed, but more importantly to secure consumer confidence and subsequent public health."

The Ministry of Agriculture, Fisheries and Food (MAFF) on the 13 August 1996, issued a statement concerning BSE and the safety of milk. They concluded "There is no scientific evidence that milk from cows with BSE does harbour BSE infectivity, and there is evidence to suggest that feeding on milk (as opposed to colostrum) does not increase the risk of maternal transmission of BSE. On top of this, there are practical considerations which would reduce to a negligible amount, any potential exposure of humans to milk from BSE affected cattle". Taking all of this evidence together it is possible to conclude that milk is perfectly safe.

Writing in the scientific journal "Nature" Collinge *et. al.*, (1995) have reported that transgenic mice expressing both human and mouse PrP when challenged with CJD developed spongiform encephalopathy but produced only human PrP^{Sc} and when challenged with BSE developed spongiform encephalopathy but produced only mouse PrP^{Sc}. This is reassuring, but still not definitive evidence that BSE does not transmit to humans. A second study using similar challenges of transgenic mice expressing only human PrP is in progress. The experiment will not be complete for up to 2 years.

Collinge *et. al.*, published in "Nature" on 24 October 1996 their findings on tracing the passage of individual prion strains within and between species, and they suggest that variant CJD resembles BSE rather than acquired or sporadic CJD.

Writing in "Nature" on 29 August 1996, Anderson *et. al.*, of the Centre for the Epidemiology of Infectious Diseases, Department of Zoology, University of Oxford concluded that the BSE epidemic is likely to fade close to extinction by the year 2001, in the absence of culling.

A report to the Minister for Health on BSE was forwarded in April 1996, from the Food Safety Advisory Board. A report from a Working Group on Maternal Transmission was forwarded to the Minister on 31 July 1996. Each of these reports is reproduced further on.

B. PHTHALATES IN BABY MILK SCARE

Sharman *et. al.*, in 1994 reported that Di-(2-ethyhexyl) phthalate (DEHP) is one of the most commonly employed plasticizers worldwide for a wide variety of applications. It is readily released to the environment through volatilisation and leaching from plastics and other sources, and its widespead usage coupled with its stability has lead to DEHP being present as a ubiquitous environmental contaminant. As a consequence, it is also found in human and animal tissues.

In March 1996, the Food Safety Information Bulletin published by the UK Ministry of Agriculture, Fisheries and Food published information about small levels of phthalates in all brands of infant formulae tested. The Report concluded "the levels of phthalates found in infant formulae would not be expected to have any effects on infants. This advice takes account of the current available information including the recent studies suggesting that some phthalates may weakly mimic the effects of naturally occuring oestrogens".

The media picked up the Report in May 1996 with headlines such as "Gender Benders" found in all Baby Milk Brands and the Department of Health in London issued a report strongly advising that parents should continue to feed their babies with infant formula which they were currently using.

Dr Richard Sharpe, Britain's most respected expert in sex changing chemicals described the controversy as needlessly alarmist. "Infants are not at any significant risk from formula baby milk powder because of the presences of low levels of phthalates which may have weak oestrogen activity".

On 6 and 7 June 1996, the EU Scientific Committee for Food (SCF) at the request of the Commission advised on the public health implications of the presence of phthalates in infant formula on the basis of information provided by the UK and by other member states. The information provided by the UK concerns results of an investigation into the contamination of infant formulae with various phthalates and of related findings in the Fatty Food Groups of total diet samples collected as part of the routine UK diet surveys. Germany and Austria also provided results of investigations into the presence of phthalates in certain food products on their respective markets but they did not include infant formula. The German data covered baby foods for children already weaned while the Austrian data covered a wide variety of food products with more detailed data on milk and its products.

The Committee concluded that the UK figures show that the estimated intakes of individual phthalates were all well below the relevant tolerable daily intakes or temporary tolerable daily intakes which it had set for these substances. The SCF concluded that the estimated intakes of individual phthalates are all well below their respective tolerable daily intakes or temporary tolerable daily intakes. Highest estimated intake of benzyl butyl phthalate reported in the UK analytical data is well below the temporary TDI of 0.1 mg/kg body weight. It is also ten times below the level where effects were reported in the Sharp et al., study.

The latter study suggested that some phthalates may weakly mimic the effects of naturally occuring oestrogens. The Committee considered it important to follow up the findings of

Sharp *et. al.*, but in the meantime was of the opinion that the levels of benzyl butyl phthalate found in infant formulae are unlikely to pose a risk to health.

For di butyl phthalate (DBP) the highest estimated intake is below the temporary tolerable daily intake of 0.05 mg/kg body weight. The Committee concluded that the levels of DBP found in infant formulae do not pose any risk to health. The Committee also recommended that given that phthalates are ubiquitous in the environment, consideration should be given to the environmental aspects by appropriate bodies.

Phthalate levels in 23 infant formulae purchased in Ireland were found to be unlikely to pose a risk to health.

Annex 1

BOVINE SPONGIFORM ENCEPHALOPATHY (BSE)

FOOD SAFETY ADVISORY BOARD

Report to the
Minister for Health
April 1996

Bovine Spongiform Encephalopathy (BSE) is one of a number of transmissible spongiform encephalopathies which affect animals. Members of this group of encephalopathies are linked by common features such as prolonged incubation periods often extending to years, progressive neurological signs ending in death and the production of characteristic spongiform changes in the brain.

BSE is a fatal disease of the nervous system of cattle and was first confirmed as an entity in Great Britain in 1986 and in Ireland in 1989. It is caused by a transmissible agent which has been detected in brain and spinal cord of naturally affected animals and in retina and distal ileum of experimentally affected animals but not in other tissues from clinically affected animals. It shares characteristics with a group of transmissible spongiform encephalopathies both human and animal (see **Table 1**).

Transmissibility in this context is defined in terms of experimental transmission and should not be taken to signify that the disease is proven to be transmitted to animals of the same or different species under natural conditions.

TABLE 1

Transmissible Spongiform Encephalopathies

Disease	Occurring in	Transmissibility demonstrated in
Kuru	Man	1966
Creutsfeldt-Jakob Disease (CJD)	Man	1968
Gerstmann-Straussler-Scheinker Syndrome (GSS)	Man	1981
Scrapie	Sheep/Goat	1936
Transmissible Mink Encephalopathy (TME)	Mink	1965
Chronic Wasting Disease (CWD)	Mule Deer	1983
Bovine Spongiform Encephalopathy (BSE)	Cattle	1988
Feline Spongiform Encephalopathy	Cat	1992
Spongiform Encephalopathy (SE)	Elk Nyala Greater Kudu Gemsbok	n.d.* 1992 1992 n.d.*

* = n.d. experimental transmission not (yet) demonstrated.

WORLD-WIDE OCCURRENCE OF BSE

Between November 1986 and May 1995, 148,200 cases of BSE were confirmed in 32,285 herds in Great Britain. During the same period 1,564 cases were confirmed in 1,074 herds in Northern Ireland (however the first case was not confirmed until 9 November 1988). The incidence in the Republic of Ireland since 1989 has ranged between 14 and 19 cases annually. To date, 124 herds have been affected and the disease incidence in cows, in-calf heifers and bulls has over this period ranged between 0.00060% and 0.00074% (see **Appendix I**). The annual incidence in the total population of cattle during that period has averaged at 0.0002%. A low prevalence of BSE has also been reported from Canada, Denmark, Falkland Islands, Germany, France, Italy, Oman, Switzerland and Portugal. In some of these countries the disease occurred in cattle imported from the UK, while in others the disease has occurred in native-born cattle. The number of suspect cases reported weekly in Great Britain has declined since 1993. This indicates that the ban on using ruminant protein for ruminant feeds which was introduced in July 1988 is having an effect. Furthermore, the use of brain, spinal cord, tonsils, spleen, thymus and intestines - from duodenum to rectum inclusive - in feed for non-ruminant animals and poultry has been prohibited since September 1990.

Epidemiological evidence indicated that the primary cause was likely to have been the use of commercial cattle feed concentrates which contained meat and bone meal derived from sheep presumed to be infected with scrapie. It is possible that feed containing meat and bone meal intended for pigs and poultry could be mistakenly fed to cattle or that feed intended for ruminants could have accidentally been contaminated in mills or feed-stores. As yet, there is no evidence of maternal transmission of the infection in cattle and epidemiological evidence suggests non-occurrence. Experimental trials started in the UK in 1989 are due to run until 1996. The results of these will help to prove whether maternal transmission occurs. The youngest animals will then be seven years of age and will be slaughtered and the brains examined histologically.

The Republic of Ireland has taken appropriate actions to minimise the risk to cattle, thereby reducing the risk of exposure to the agent. These include:

(a) Governmental action to make BSE a compulsory notifiable disease

(b) The establishment of an active BSE surveillance programme

(c) A ban on the feeding to ruminants of protein products originating from ruminants

(d) Complete destruction of all infected cattle

(e) The slaughter and exclusion from the food chain of all cattle in herds in which a clinical case was diagnosed and the removal from the food chain of such animals.

TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

BSE, Creutzfeldt-Jakob Disease (CJD) and kuru have a common neuropathological feature of spongiform change and show an array of distinctive amyloid plaques in the central nervous system. The agents responsible have not been completely characterised, although their composition seems to be very similar, with a peptide as a major component. There are three conflicting interpretations, the agent being termed either a prion, a virion or a filamentous virus.

The prion hypothesis appears to have most credence. The precursor prion protein (PrP^c) of spongiform encephalopathy is a membrane-anchored glycoprotein that is present in most organs and cell types including neurons. Its normal biological role is still not clear. PrP^c is encoded by a single copy gene on chromosome 20 in humans (PrP gene). A key process in the development of the disease is the conversion of this prion protein (PrP^c) to a proteinase resistant protein (PrP^{sc}) variously named scrapie associated protein or proteinase resistant protein or scrapie amyloid protein. The mechanism by which this conversion occurs is currently the subject of much research.

Prusiner's hypothesis is that the PrP^{sc} molecule makes contact with a normal PrP^c molecule in the membrane protein of the brain and induces it to unfold into the aberrant conformation. Molecules so formed go on to do the same to other PrP^c molecules, thus creating further replicas from normal protein. The agent shows amazing resistance to heat, chemical agents and radiation. It is not completely killed by boiling or immersion for long periods in formalin. The precise mechanism of template induced polymerisation of normal PrP^c by altered protein molecules is still not known. Although the amyloid protein of spongiform encephalopathy is chemically dissimilar to that of non-infectious disorders, a common characteristic of both types of amyloidoses is the conversion of a normal precursor protein into β-pleated sheets that aggregate as insoluble amyloid fibrils.

CREUTZFELDT-JAKOB DISEASE

The National Surveillance Programme for CJD has continued in the UK since 1990. The number of sporadic cases of CJD has risen from 26 in 1990 to 46 in 1994 and this is in part related to increased ascertainment in the elderly.

In Ireland, examination of death certificates which identify CJD as the cause of death reveals 17 such cases since 1980. While it is not possible to calculate population incidence from these figures, the actual numbers are extremely low and it is reasonable to infer a low incidence of this condition in this country.

Through the BIOMED 1 programme of the EU, epidemiological surveillance programmes in a number of European countries have been co-ordinated in order to share common methodologies, diagnostic criteria and case-control questionnaires. In 1993 and 1994, the incidence of CJD in France, Germany, Italy, the Netherlands and the UK was remarkably similar. In the countries in which there have been serial surveys, there has been an apparent rise in the incidence of CJD with time, almost certainly due to improved case ascertainment. The overall conclusion of the study is that there is no conclusive evidence of any change in CJD that can be attributable to BSE.

RISK FACTORS FOR CREUTZFELDT-JAKOB DISEASE

The major change in the epidemiology of CJD since 1990 has been an increase in the number of cases of iatrogenic CJD that have been identified. Worldwide, there were 62 cases of CJD in human growth hormone recipients and 20 cases of CJD in recipients of human dura mater grafts. The occupational history of individuals dying of CJD has been studied since 1990 in the UK. A case-control study has provided no evidence of increased risk in relation to specific occupations. Statistical analysis of relative risk is extraordinarily difficult because of the small number of patients, the small "at risk" populations and the different denominators that can be used in statistical analysis. Approximately 12% of all cases of CJD are associated with mutations of the prion protein gene. In the majority of these cases there is a positive family history of a neuro-degenerative disorder and in many there is a family history of CJD itself.

A recent report from the CJD Surveillance Unit in Edinburgh described 10 cases of a distinct variant of CJD, all aged under 45 years with an onset of illness in the last two years. Following careful review, they state that "the most likely explanation at present is that they are linked to exposure from cattle infected with BSE before the Specified Bovine Offal ban in 1990".

WHAT IS THE RISK OF BSE SPREADING TO HUMANS?

There is no scientific evidence that BSE can be transmitted to man by beef. However, the recent report from the CJD Surveillance Unit in Edinburgh raises the possibility and is cause for concern. The risk of BSE causing illness in man would depend on the amount of the agent to which man is exposed, the route of such exposure and the species barrier effect. Studies in mice on the infectivity of tissues from confirmed cases of BSE have shown that to date only brain, spinal cord, retina and distal ileum have detectable infectivity. Other tissues such as milk and muscle showed no detectable infectivity.

The route of BSE exposure is important in risk assessment. For example, in mice it has been shown that the intracerebral route is 100,000 fold a greater risk than when the agent is consumed as a food. Research recently published on experimental work using transgenic mice expressing human prion protein is suggestive of the inability of bovine aberrant PrP to affect normal human PrP. Prior to BSE, many species have been shown to be experimentally susceptible to one or other of the transmissive agents associated with scrapie, kuru and CJD. Susceptibility to Transmissible Spongiform Encephalopathies (TSE) depends on the PrP gene which appears to be present in all mammals. However, for hundreds of years, humans have eaten scrapie infected sheep (including brains) and researchers have failed to connect this with CJD. Also, CJD occurs in Australia yet there is no scrapie in Australian sheep. On the other hand the sheep scrapie agent may have changed slightly, thus infecting cattle. Brains and spinal cords would be dangerous materials, but not meat which is non-infectious. Cooking would not completely inactivate the infectious agent.

While we can reach conclusions about BSE from studies on scrapie, kuru and other TSEs, in the case of BSE the risk of human infection remains, although a remote one.

CONCLUSIONS

The incidence of BSE in Ireland is extremely low (e.g. in 1995, 16 cases in the national herd of over 7 million cattle). At present, there is no scientific evidence that BSE can be transmitted to man by eating beef. Recently however, it has been postulated that a distinct variant of CJD in the UK, in the absence of any more credible alternative, may be linked to the consumption of bovine products which, prior to 1990 may have been contaminated by infected brain or spinal cord. There is no evidence that milk can be infected with the BSE agent.

As the disease in cattle is currently understood, the implementation of the following measures (a, b and c below) together with the extremely low level of BSE agent in cattle in Ireland and the extensive use of grass in their diet, protects the consumer of beef produced in the Republic of Ireland from exposure to the agent of BSE.

The elimination of the disease in cattle is a priority as it would of course remove any chance of exposure to the agent but until this happens the following measures should be implemented:-

(a) The slaughter-out of affected herds and the disposal of the carcases in a manner that will protect humans and animals from the agent;

(b) Rigorous ante-mortem examination of cattle, particularly those of an age more prone to be clinically infected with BSE, so that animals showing clinical signs of disease are identified and excluded from food and feed;

(c) The current practice of the exclusion from food and the safe disposal of bovine brain, spinal cord, retina and the Peyers patches of the distal ileum should be continued. Mechanically recovered meat from bovine vertebrae should not be used in food or feed;

Notwithstanding present prohibitions, the use of mammalian meat and bone meal in the feed of non-ruminant animals should, as a matter of urgency, be examined by the Department of Agriculture, Food and Forestry.

RECOMMENDATIONS

It is recommended that:-

(i) Measures should be taken to monitor the incidence of CJD.

(ii) Research into both the epidemiology and diagnosis of BSE should be supported.

APPENDIX I

SUMMARY OF CONFIRMED BSE CASES IN IRELAND

ANNUAL INCIDENCE

Up to 21 March 1996, 124 cases of BSE have been confirmed

1989	15 cases (of which 5 imported)
1990	14 cases (of which 1 imported)
1991	17 cases (of which 2 imported)
1992	18 cases (of which 2 imported)
1993	16 cases (of which none were imported)
1994	19 cases (of which 1 imported)
1995	16 cases (of which 1 imported)
1996	9 cases to date.

Year	Cows, In-Calf Heifers and Bulls (Census)	Cases	Disease Incidence
1989	2,313,000	15	.00064%
1990	2,323,000	14	.00060%
1991	2,328,000	17	.00073%
1992	2,430,000	18	.00074%
1993	2,410,000	16	.00066%
1994	2,563,200	19	.00074%
1995	2,514,700	16	.00064%
1996	2,636,900	9	

Herd Incidence

National Herd Size: 154,474 herds. To date 123 herds and 1 bull have been affected.

Origin of cases

Imported: 12

Native animals: 112

Herd Type

84 dairy herds
19 suckler herds
20 mixed (dairy and suckler) herds
1 bull

Herd Characteristics

Herd Size	Total number of animals	Number of cases
	<50	29
	50 - 99	35
	100 - 149	21
	150 - 199	12
	200 - 299	17
	>300	10

Breed Distribution

103 Friesian
6 Hereford
4 Holstein
3 Aberdeen Angus
3 Charolais
2 Shorthorn
2 Limousin
1 Simmental

Sex Distribution

123 cows
1 bull

Geographical Distribution

124 cases identified as follows:
32 Cork
17 Donegal
11 Limerick
10 Galway
6 Meath
5 Carlow, Cavan, Sligo, Tipperary
3 Kerry, Kildare, Laois, Waterford, Westmeath
2 Longford, Wexford
1 Clare, Dublin, Kilkenny, Leitrim, Louth, Monaghan, Offaly, Roscommon, Wicklow

Herd Depopulation Policy

124 herds affected
1 animal per herd

To 29 February 1996 a total of 16,485 in-contact animals have been depopulated (slaughtered out) at a cost of IR£11,568,997.

Annex II

BOVINE SPONGIFORM ENCEPHALOPATHY (BSE)
MATERNAL TRANSMISSION

FOOD SAFETY ADVISORY BOARD
Working Group

Report to the
Minister for Health
31st July 1996

BSE UPDATE JULY 1996

RECENT EVENTS REGARDING MATERNAL TRANSMISSION OF BSE

For background information, see **Appendix I.**

Maternal transmission of Bovine Spongiform Encephalopathy has always been recognised as a possibility. There is now some emerging evidence that this may occur at a low level. An estimate of such transmission based on epidemiological studies is reported to amount to 8% of total cases of BSE (Anderson, 1996). The equivalent results of a large scale experimental study are awaited but levels in the range of 1 - 5% have been mentioned (Spongiform Encephalopathy Advisory Committee, 1996).

In theory, the possible routes for maternal transmission include:

1. trans uterine
2. placenta
3. placental fluids
4. milk including colostrum

To date, no infectivity has been found in any of the tissues that would explain the exact route(s) by which maternal transmission occurs.

Milk and milk products, even in countries with a high incidence of BSE are considered safe. There is evidence from other animal and human spongiform encephalopathies to suggest milk does not transmit these diseases (W.H.O., April 1996).

The Group re-affirm the conclusions in the April Report (**Appendix II**) and emphasise that the elimination of the disease in cattle continues to be a priority. The current slaughter-out policy of affected herds as well as the trace back and slaughter of cohort animals in other herds is endorsed.

Although transmission from cow to calf is low, measures should be taken to isolate any suspect cases which are giving birth, to dispose of placenta safely and to cleanse and disinfect the isolation accommodation in order to minimise the risk of transmission.

APPENDIX I

Bioassay in susceptible laboratory species such as mice is at present, the only practical way of detection and measuring BSE infectivity in a large number of samples.

The oral route has been shown experimentally to transmit BSE. However, compared to parental exposure, ingestion is a relatively inefficient route of transmission. In studies of BSE the amount of affected cattle brain required to produce the disease in mice was calculated to be 200,000 fold greater by the oral route than by intracerebral injection.

Transmission of spongiform encephalopathies is dependent on the size of the infective dose.

Clearly, there would be little or no risk of infection as a result of oral exposure to tissues which contain no detectable infectivity as determined experimentally using the most efficient routes of inoculation.

Transmission experiments in mice have been undertaken with a wide range of tissues from confirmed cases of BSE. So far, BSE infection has been transmitted, by feeding or by injection only by brain, cervical and terminal spinal cord and retina. Experiments with mice that were fed milk and mammary gland, placenta, lymph nodes or spleen, have failed to transmit the disease within the natural lifespan of the animals, or even to establish subclinical BSE infection of the lymphoreticular system. Furthermore, mice exposed parenterally to the tissues listed in Table 1 did not succumb to disease within their natural lifespan. A more recent experiment using milk derived from cattle with BSE in early, mid and late lactation and either inoculated or fed to susceptible mice has revealed no evidence of infectivity. There is a cow to mouse species barrier in these studies. However, all calves receive colostrum and beef calves are suckled for up to six months of age.

In BSE, no detectable infectivity has been found in any male or female reproductive tissues, or in placenta or embryos by bioassay in susceptible mice. Furthermore, uterine flushings have been similarly tested and have shown no detectable infectivity. There is a cow to mouse barrier in these studies. However, cattle have been challenged oro-nasally with placenta derived from clinically affected, confirmed cases of BSE and no disease has yet resulted more than 75 months after challenge. Embryos collected from a large number of clinically affected confirmed cases of BSE by International Embryo Transfer Society protocols have been transferred into 347 recipient heifers imported from New Zealand and kept in quarantine. No BSE has resulted in any recipient cow or offspring, the oldest of which is now over 4 years of age. The study will not be complete until 2001.

The risk of infection with BSE arises only from exposure to certain tissue of infected animals or products prepared from those tissues.

In naturally infected cattle exhibiting clinical signs of BSE and confirmed to have the disease post mortem, infectivity has been found only in the brain, cervical and terminal spinal cord and the retina and no infectivity was detected in 50 other tissues.

Detectable infectivity following high dose, experimental, oral challenge with brain from confirmed BSE cases has been found in the distal ileum of calves of 10, 14, 18 and 22 months of age, conversely, there is a range of tissues from cattle in which no detectable infectivity is expected to occur at any time, even in clinically affected animals.

These tissues include:-

- Carcase meat
- Milk
- Hides
- Skins
- Semen
- Embryos washed in accordance with the protocols of the International Embryo Transfer Society

TABLE 1

Tissues from clinically affected cattle with no detectable infectivity by parenteral inoculation of mice grouped by anatomical system

Cerebrospinal fluid	Spleen	Oesophagus
Cauda equina (of spinal cord)	Tonsil	Reticulum
Peripheral nerves:	Lymph nodes:	Rumen (pillar)
N. sciaticus (proximal)	Prefemoral	Rumen (oesophageal groove)
N. tibialis	Mesenteric	Omasum
N. splanchnic	Retropharyngeal	Abomasum
		Proximal small intestine
		Distal small intestine
		Proximal colon
		Distal colon
		Rectum
Clotted blood	Testis	Ovary
Buffy Coat	Prostate	Uterine caruncle (Pregnant cow)
	Seminal vesicle	Placental cotyledon
	Epididymis	Placental fluids:
Foetal calf blood	Semen	Amniotic fluid
Serum		Allantoic fluid
		Embryos
Midrum fat	Liver	
Musculus (M.)	Kidney	
semitendinosus	Heart	Udder
M. longissumus	Pancreas	Milk
M. diaphragma	Lung	
M. masseter	Trachea	
Bone marrow		
Skin		

APPENDIX II

Extract from FSAB Report: April 1996

The elimination of the disease in cattle is a priority as it would of course remove any chance of exposure to the agent but until this happens the following measures should be implemented:-

(a) The slaughter-out of affected herds and the disposal of the carcases in a manner that will protect humans and animals from the agent.

(b) Rigorous ante-mortem examination of cattle, particularly those of an age more prone to be clinically infected with BSE, so that animals showing clinical signs of disease are identified and excluded from food and feed

(c) The current practice of the exclusion from food and the safe disposal of bovine brain, spinal cord, retina and the Peyers patches of the distal ileum should be continued. Mechanically recovered meat from bovine vertebrae should not be used in food or feed

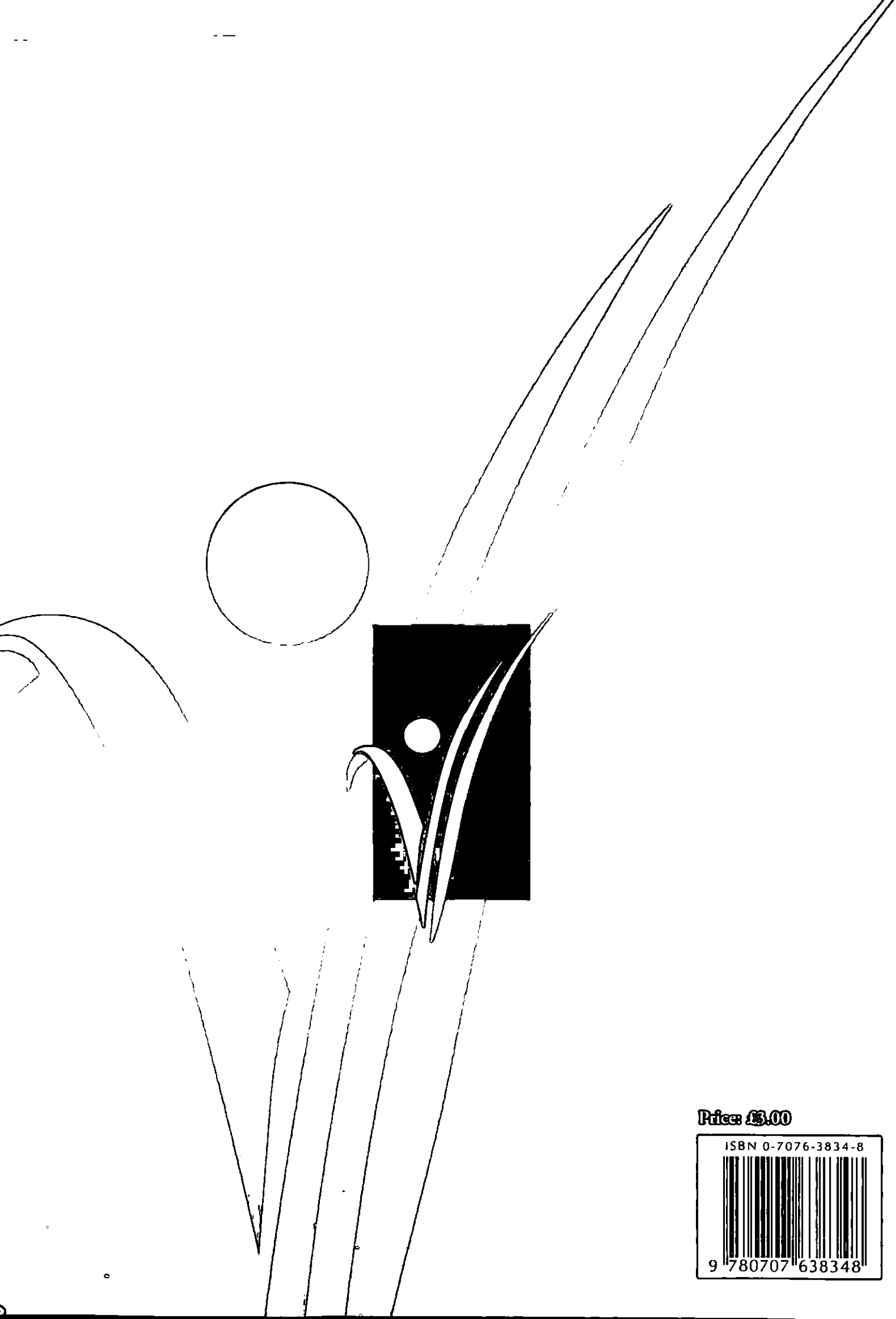
Notwithstanding present prohibitions, the use of mammalian meat and bone meal in the feed of non-ruminant animals should, as a matter of urgency, be examined by the Department of Agriculture, Food and Forestry.

RECOMMENDATIONS

It is recommended that :-

(i) Measures should be taken to monitor the incidence of CJD

(ii) Research into both the epidemiology and diagnosis of BSE should be supported.



Price: £3.00

ISBN 0-7076-3834-8