RISK: Health, Safety & Environment (1990-2002)

Volume 13 | Number 1 Article 7

March 2002

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Enda Cummins

Pat Grace

Kevin McDonnell

Shane Ward

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Enda Cummins, Pat Grace, John Fry, Kevin McDonnell, & Shane Ward*

Introduction

Bovine Spongiform Encephalopathy (BSE) is a member of the Transmissible Spongiform Encephalopathies (TSEs) of which scrapie in sheep and Creutzfeldt-Jakob Disease (CJD) in humans are well known examples. The recognition of the BSE agent as a possible cause of a new variant of Creutzfeldt-Jakob Disease (vCJD) in humans and confirmation data indicating that a link between vCJD and BSE does exist placed BSE in the public arena.[1] The present rates of vCJD in Great Britain highlight the significance of exposure risk to the BSE agent in man.

The term "risk society" has been used to describe the situation where a risk can have an effect, not on one particular individual, but on an entire community. In our "risk society," a range of potential risks and uncertainties are associated with new technologies and new diseases such as BSE. These risks bring with them worries about human health, industrial competitiveness, ecological disruption, and the potential for

* Mr. Enda Cummins is a Ph.D. student in the Department of Agricultural & Food Engineering at University College in Dublin, Ireland (U.C.D.). He graduated with a B.S. Degree in Agricultural Sciences at U.C.D in 1997. *E-mail: Enda.Cummins@ucd.ie.*

Dr. Pat Grace earned a B.E. at U.C.D. and a Ph.D. at U.C.D. Dr. Grace is a lecturer in the Department of Agricultural & Food Engineering at U.C.D., Ireland.

Dr. John Fry graduated with a B.A. at the University of York, York, England and a Ph.D. at the University of Exeter, Exeter, England. Dr. Fry is a lecturer in the Department of Environmental Resource Management at U.C.D., Ireland.

Dr. Kevin McDonnell earned a B.A. in Agricultural Science at U.C.D. and a Ph.D. at U.C.D. Dr. McDonnell is a lecturer in the Department of Agricultural & Food Engineering at U.C.D., Ireland.

Dr. Shane Ward earned a B.A. in Agricultural Science and a Ph.D. at U.C.D. Dr. Ward is a lecturer and lead research scientist in the Department of Agricultural & Food Engineering at U.C.D., Ireland.

1 Robert G. Will et al., *A New Variant of Creutzfeldt-Jakob Disease in the UK,* 347 Lancet 921 (1996); Moira E. Bruce et al., *Transmission to Mice Indicate that 'New Variant' CJD is Caused by the BSE Agent,* 389 Nature 498 (1997); Andrew F. Hill et al., *The Same Prion Strain Causes vCJD and BSE,* 389 Nature 448 (1997).

trade disputes. In an ever changing society, the ability to manage new health scares is an essential skill for governments and related industries. Government has the job of ensuring safety for consumers, the general public, and protection of the environment. This involves making informed decisions about potential effects on human health, ecology, and the countryside more generally.

BSE, which reached epidemic proportions in Great Britain in the late 1980s, resulted from the feeding of infected meat and bone meal (MBM) to ruminant animals.[2] When an animal is processed through a meat factory, the carcass is partitioned and those parts of the animal that are not utilized for human consumption are separated (largely bone and intestine tissues). The resulting material is rendered in specialized rendering plants by a number of crushing and cooking stages. The resulting solid material is termed MBM while the liquid material is called tallow (essentially animal fat). The rendering of offal from BSE-infected cattle gave rise to a spiralling effect of infectivity in animal feed resulting in increasingly larger numbers of cattle becoming infected with BSE in Great Britain in the early 1980s. It is suggested that the early cycles began because a novel TSE agent originated in the early 1970s. The cause of the novel agent is likely to have been a new prion[3] mutation in cattle or possibly sheep.[4] The recycling nature of the feeding practices at that time gave rise to an amplification effect that produced the BSE epidemic seen in Great Britain in the late 1980s.

This paper develops a systems modelling approach to the risk assessment of BSE transmission in the food/feed chain, which assesses the effectiveness of policy change in curtailing the epidemic.

Risk Assessment

Risk assessment has emerged as a valuable analytical tool for describing the public health consequences of human exposure to environmental contaminants. It is a widely used method of characterizing hazards, and in doing so, allaying or compounding fears

2 John W. Wilesmith et al., *Bovine Spongiform Encephalopathy: Epidemiological Studies*, 130 Veterinary Record 90 (1988).

3 A prion is a small glycosylated protein molecule found in the brain cell membrane. In its abnormal state, the prion becomes heat resistant and protease-resistant.

4 BSE Inquiry, Volume 1, *Findings and Conclusions* ch. 12, Science and Research 219 (2000) (available at <http://www.bse.org.uk/>).

of the hazard while increasing the probability of project success by decreasing the degree of project risk. Risk assessment is a decision-making process that entails the integration of political, social, economic, and engineering information with risk-related data to develop, analyze, and compare regulatory options and to select the appropriate regulatory response to a potential health hazard. The selection process necessarily requires the use of value judgments on such issues as the acceptability of risk and the reasonableness of the control costs. Risk assessment techniques have been used to monitor and analyze the future course of BSE and provide estimates of biological parameters to assess the risks to both animal and human health.[5] Risk assessment procedures have been used in many countries to assess the incidense of BSE infected animals and the resulting risks to humans.[6]

Science faces a fundamental difficulty in assessing the risks, control measures, and costs associated with the emergence of a new and relatively unknown disease. As a result, many uncertainties exist. In light of this, scientists are faced with two possible options: (1) conduct experiments to generate new information; or (2) make informed assessments about the potential effects of the disease. The onus is put on scientists to provide the information required for informed commercial and political decisions. Risk assessment can be used to assist in this process.

During the 1970s, legal and administrative challenges led to the adoption of risk assessment and its ability to highlight the need for policy changes as a means of protecting human health. International trade agreements such as the General Agreement on Tariffs and Trade (GATT) and the North American Free Trade Agreement (NAFTA) have requirements for risk assessment in their sanitary and phytosanitary (S&P) clauses.[7] Such agreements highlight the growing need for risk assessment methodologies in trade situations. Risk

5 Enda J. Cummins et al., *Predictive Modelling and Risk Assessment of BSE: A Review*, 4(3) Journal of Risk Research 251 (2001).

6 Det Norske Veritas (DNV) technical, *Risks from BSE via Environmental Pathways*, Report to Environment Agency Ref C7243 (June 1997).

7 *See* Michael Wooldridge et al., *"I Don't Want To Be Told What To Do By a Mathematical Formula" Overcoming Adverse Perceptions of Risk Analysis*, Proceedings of The Society for Veterinary Epidemiology and Preventative Medicine 37-47 (1996); North American Free Trade Agreement, *Sanitary and Phytosanitary Measures* ch. 7, section B (1993).

assessment is used as a valuable tool in decision making while highlighting control strategies that may be required or any need for policy change. Risk assessment procedures and methodologies have been reviewed in the context of BSE by Cummins et al.[8]

Stages in Risk Assessment

Risk assessments can characterize the fundamental risks and uncertainties associated with the potential impacts of BSE on the food and feed chain. There are four main stages in the risk assessment process. These steps include hazard identification, exposure assessment, dose-response assessment, and risk characterization. The aim is to reduce the probability and impact of the disease by combining the information from each of the four steps.

Hazard Identification

The collection of data relating to the disease (i.e., BSE) is carried out during the hazard identification stage. Epidemiological and disease surveillance data are collected describing the factors which contribute to the disease's survival, mode of transmission, and growth. Hazard identification focuses on what can go wrong and how it would happen.

Exposure Assessment

The data collected in the hazard identification stage is used in assessing the potency with which the disease can infect others taking into account possible critical points which may act as a control point to halt the disease, hasten its inactivation, or reduce exposure. The pathways, by which the disease challenges a potential host, are identified during this stage. The initial dose may also have an impact on disease efficacy; thus, it must be assessed during exposure assessment. Exposure assessment evaluates the likelihood of these hazards occurring and its resulting implications.

Dose-Response Assessment

If a host has been exposed to a pathogen, its response will vary depending on the amount of pathogen it is subjected to. A dose-response is used to translate the exposure assessment into a response in

8 *See* Cummins et al., *supra* n. 5.

terms of infected host animals. The susceptibility/immunity of the host has to be taken into account.

Risk Characterization

The information generated from all the previous steps is incorporated in the risk characterization step. Uncertainty about the value of any input parameter used in the preceding stages can be incorporated here by means of probability distributions. This allows the effects of variations in these input parameters on the risk calculation to be ascertained. This can point to deficiencies in research data or current knowledge and direct future research efforts to correct this. This stage provides scientific basis for policy decisions.

BSE Risk Modeling

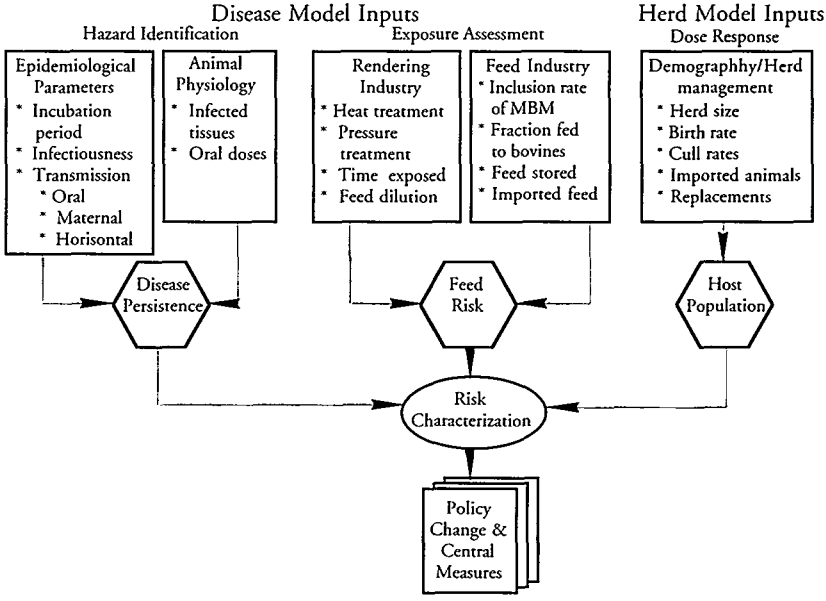
Risk assessment models and analyses have been used as tools to help manage risk and to help make policy decisions. Researchers have put a number of research documents and policy consultation responses forward in relation to BSE. In particular, works by Anderson et al. and Ferguson et al. have been significant to the formulation of BSE control strategies and policy within the European Union and the calculation of risk in terms of the human consumption of contaminated meat and meat products.[9] These policies have had a significant impact on the importing and exporting of goods to and from European countries for the facilitation of trade.

The construction of a model for the purpose of a quantitative risk assessment is essential in supporting or initiating policy decisions.[10] In its simplest form, a risk assessment model of BSE can be broken-up into two inputs: (1) disease model inputs; and (2) herd model inputs. Further analysis reveals that the factors at play and their interaction with one another are quite complex. This highly complex set of factors interact to create an environment for BSE to establish in a herd and contribute to human food risks. The most important factors that need to be taken into account in a risk assessment of BSE are given in Figure 1.

9 Roy M. Anderson et al., *Transmission Dynamics and Epidemiology of BSE in British Cattle*, 382 Nature 779 (1996); Neill M. Ferguson et al., *The Epidemiology of BSE in GB Cattle Herds: II. Model Construction and Analysis of the Transmission Daynamics*, 352 Philosophical Transactions of the Royal Society London B 808 (1997).

10 *See* Wooldridge et al., *supra* n. 7.

Figure 1
Risk Assessment Factors for BSE



The first stage in the risk assessment process (hazard identification) includes the identification of the BSE agent along with any epidemiological and surveillance data. Epidemiological parameters such as the incubation period and the method of transmission will contribute to disease persistence. A longer incubation period will result in a prolonged epidemic while control strategies will take longer before they will have a noticeable effect. The feed risk is very much dependent on rendering and feed industries. Within the rendering industry, the raw material used and the process condition applied contribute to the overall feed risk, while within the feed industry, the inclusion rate of MBM (domestic and imported) and the potential for cross contamination add to the feed risk. The herd structure and dynamics of the host population are important in terms of herd size, age distribution, and animal cull rates. The susceptibility of the host population to a given dose of infected tissue is assessed in the dose response relationship. With BSE, one infected animal has the potential to infect many more animals provided that the oral dose to an

individual animal is sufficient to cause infection. All of these factors lead to the development of a combined risk assessment of BSE incidences. This information is used to facilitate the implementation of disease control policies in an effort to reduce disease incidence and protect human and animal health.

The effect of the control policies is not always obvious from the back calculation techniques used by Anderson et al., Ferguson et al., and Donnelly et al. due to their complexity; while in many instances the techniques used are merely for biological parameter estimations.[11] While the factors detailed in Figure 1 have been used in many risk assessment approaches, the systems modelling approach may be easier for an unfamiliar public to comprehend the main components of detailed risk assessment procedures. To facilitate an easier understanding of the factors at play and the policies implemented in response to the BSE crises, a systems modelling approach has been developed here.

System Modeling Approach to BSE Risk Assessment

A systems model integrates and predicts the effects of a series of individual components which interact to form a coherent system. For the development of a BSE model, the overall system can be broken-down into a number of different components. Examples include the feed industry, rendering industry, slaughter plant, host population, and imports/exports. A systems modeling approach to risk assessment represents an approach that is robust, systematic, and transparent in terms of the assumptions made and systems that are modeled. The following types of systems are proposed in this paper: (1) Closed System Model (Figure 2), where the main cause for concern is from potentially infected animals produced internally with little or no imports to the system; and (2) Open System Model (Figure 3), where the importation of potentially infected animals and animal feed is the main concern as a source of infection.

11 Christl A. Donnelly et al., *The Epidemiology of BSE in Great Britain Cattle Herds: I. Epidemiological Processes, Demography of Cattle and Approaches to Control by Culling*, 352 Philosophical Transactions of the Royal Society London B 781 (1997); *see* Anderson et al., *supra* n. 9; Ferguson et al., *supra* n. 9.

Figure 2
Closed System Model of BSE

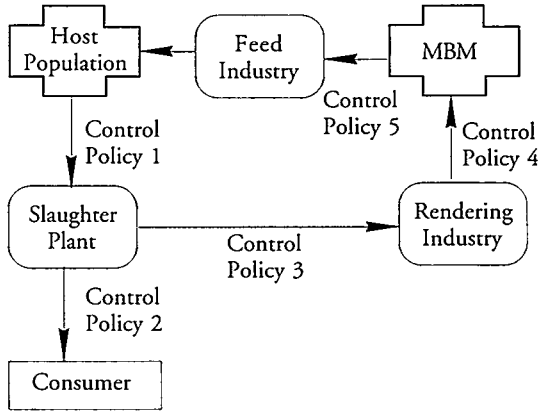
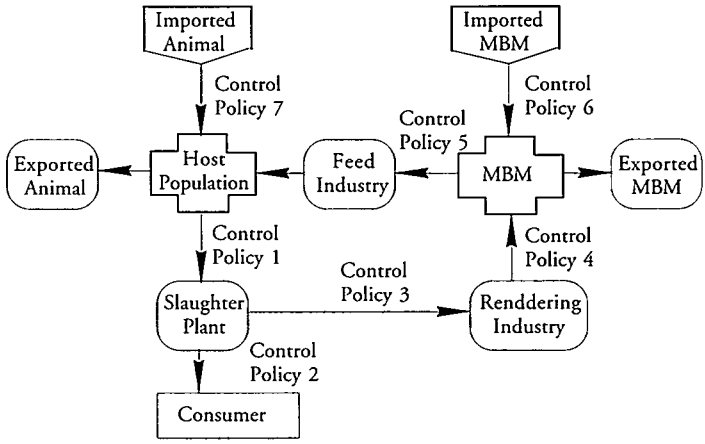


Figure 3
Open System Model of BSE



Control policy 1: Active surveillance measures for the detection, control and eradication of BSE, as of May 1, 1998 (Decision 98/272 April 1998). The introduction of targeted testing for BSE, with a focus on high-risk animal categories, from January 1, 2001. This measure will be reviewed and extended to all cattle aged over 30 months entering the food chain from July 1, 2001 (Decision 2000/374 June 2000).

Control policy 2: The requirement to remove, from the human food chain, specified high-risk materials (SRMs like spinal cord, brain, eyes, tonsils, parts of the intestines) from cattle, sheep, and goats throughout the EU from October 1, 2000 (Decision 2000/418 June 2000).

Control policy 3: The requirement to remove specified high-risk materials from cattle, sheep, and goats throughout the EU from October 1, 2000 from the animal feed chain (Decision 2000/418 June 2000).

Control policy 4: Higher processing standards for the treatment of animal waste (133 degrees Celsius, 3 bars of pressure for twenty minutes) in order to reduce infectivity to a minimum (Decision 96/449 July 1996).

Control policy 5: A ban on the feeding of mammalian meat and bone meal (MBM) to cattle, sheep, and goats, as of June 1994 (Decision 94/381 June 1994).

Control policy 6: Great Britain shall not export, from its territory to other Member States or third countries, bovine animals and bovine products (Decision 96/239 March 1996).

Control policy 7: Great Britain shall not export, to other member states, live cattle born before July 18, 1988 or born females in which bovine spongiform encephalopathy is suspected or has been officially confirmed (Decision 89/469 July 1989).

Details of all these control policies and others relating to European BSE legislation can be located at http://www.europa.eu.int/comm/food/fs/bse/bse 15 en.pdf. In addition, there are country specific measures in force in several European Union member states, especially those with the highest incidence of BSE.

In the Open System Model, there are two possible routes of introduction of infection: (1) infected cattle; or (2) importation of infected MBM. The transportation of animals and their products between communities has long been recognized as having the potential to spread infection.[12] A Closed System Model of BSE in Great Britain, which shows the feedback loop required to initiate a BSE epidemic, is given in Figure 2. It is the simplest representation of what happened in Great Britain to give rise to the BSE epidemic.

While the BSE agent has been identified in relatively few tissues of infected cattle,[13] the available methods of detection cannot rule out the possibility that other tissues may also contain the BSE infection. Furthermore, cattle tissues are used in several products and manufacturing processes which could potentially result in human exposure. Current processing strategies are not able to completely eliminate the BSE agent from food products. Therefore, BSE risk assessment in the food industry rests on knowing the source and BSE incidence of cattle used in food production, the tissues involved, and the consumption patterns of the final product.

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12 *See* Wooldridge et al., *supra* n. 7.

13 Scientific Steering Committee of the European Commission, *Listing of Specified Risk Materials: A Scheme for AssessingRelative Risks to Man* (opinion adopted December 9, 1997).

In the early 1980s, if a subclinical animal was present in the host population and was culled, offal from this animal would have been rendered and converted into MBM and fed back to the host population in the form of a protein rich feed. While this cycle of events gave rise to the BSE epidemic as we know it today, policy makers have attempted to renew public confidence in the food industry by laying down strict control strategies/policies to curtail this disease.

BSE Control Policies

Given the importance of beef in the human diet and the use of bovine tissues in the manufacture of household, industrial, agricultural, and pharmaceutical products, professionals must implement interim control measures until our knowledge of TSE is complete. The main polices that the European Commission has put in place to control BSE and protect consumers can be viewed as a series of control policies. These are then used in the systems models of BSE developed in this paper (see Figure 2 and 3).

BSE Model Structure

The model developed in this paper demonstrates that the policies in place create a number of obstacles should the infection be introduced into the system. The question is how effective are these obstacles? If some of the infection gets through each stage, there may be some degree of risk to human and animal health. The effectiveness of each of these control policies needs to be quantitatively assessed and updated if future scientific information deems there is a deficiency in the system. The efficiency of the cumulative effects of the control policies to eliminate infectivity can be measured by the product of each of the efficiencies of the control policies within the system. For example, within a closed system (e.g., neglecting imported feed) the efficiency of the control policies can be measured by Equations 1 and 2 below.

The equation Animal $Risk_I$ represents a measure of the risk to animals internally in the closed system. It is comparable to the basic reproduction ratio (R_0), which is the fundamental epidemiological quantity determining if an infectious disease will persist in a host population.[14] R_0 is defined as the average number of new infections

14 Roy M. Anderson & Robert M. May, *Infectious Diseases of Humans* (Oxford University Press 1991).

caused by a typical infective individual during its total infectious period in a fully susceptible population. If R_0 remains below 1, an epidemic will not occur. If, however, R_0 is greater than 1 (i.e., one infected animal will infect more than one other animal) a disease outbreak would occur. It is a simple and convenient measurement of risk. The equation Human $Risk_I$ represents a method of measuring the risk to humans in a closed system. It is the average number of new human infections caused by an infected animal.

Equation 1:

$$\text{Animal Risk}_I = CP1*CP3*CP4*CP5 * Y(1)$$

Equation 2:

$$\text{Human Risk}_I = CP1*CP2*Y/B(2)$$

CP1 is the fraction of subclinical cases entering a slaughter plant unrecognized as diseased animals.

CP2 is the fraction of material with the potential infectivity, which enters the human food chain.

CP3 is the fraction of material with potential to infect that enters the rendering industry.

CP4 is the fraction of infection which survives the rendering process.

CP5 is the fraction of MBM which is used for animal feed.

Y is the infection level in an infected animal measured in cattle oral ID50 (i.e., the infectivity required to induce infection in a bovine animal when exposed orally (CoID50/Animal)), and B is the species barrier factor. The species barrier is a term used to describe the natural resistance to transmission when a particular species is exposed to a TSE of another species. It enables a conversion from CoID50 to human ID50.

If an infected animal or MBM is imported, the risk posed is very much dependent on the control policies in place in the source country. This would be of importance in a country where the potential for infection is largely from imported material and is represented by the Open System Model (Figure 3). The risk to humans and animals is dependent on control policies in both the importing and the exporting countries and is represented by Equations 3, 4, and 5. Animal $Risk_{E, MBM}$ (Equation 3) results from the importation of MBM (i.e., live animals are slaughtered and processed in the source country then the MBM material is imported to the destination country). It is the risk from an external (E) source of MBM.

Equation 3:

$$\text{Animal Risk}_{E,\,MBM} = CP1_e * CP3_e * CP4_e * CP5_i * CP6_i * Y(3)$$

Equation 4:

$$\text{Animal Risk}_{E,\,A} = CP1_i * CP3_i * CP4_i * CP5_i * CP7_i * Y(4)$$

Equation 5:

$$\text{Human Risk}_E = CP1_i * CP2_i * CP7_i * Y/B(5)$$

CP6 is the fraction of externally produced MBM imported into the destination country; and

CP7 is the fraction of animals imported from the total herd population.

Subscript e indicates the exporting country control policy, subscript i indicates the importing country control policy.

Animal $\text{Risk}_{E,\,A}$ (Equation 4) results from the importation of potentially infected animals (i.e., live animals are imported from the source country and are slaughtered and processed in the destination country). It is the risk from an external (E) source of animals (A). Human Risk_E is the risk to humans from imported animals.

The control policies, CP6 and CP7, are dependent on the control measures in place in the importing country. For the control policies to be effective (i.e. reducing risk to humans and animals), the risk calculation for human and animal risks should be less than 1. By being less than 1, it indicates that the control policies in place have the effect of decreasing the disease incidence. This indicates that the policies in place are effective in controlling the disease and that the disease would not reach epidemic proportions. This calculation can indicate that the control policies in place are effective and can allow for some degree of ineffectiveness/uncertainty of a particular control policy. While Animal Risk_I is a reproduction ratio, the other calculations (Equations 2, 3, 4, and 5) are variations of this reproduction ratio calculation as applied to a type of system (open or closed) and a species type (human or bovine). They serve as a convenient risk measurement. Ireland can be considered representative of an open system model as it trades with Great Britain and, hence, the greatest source of initial infection was from imported

feed or animals while the greatest risks to Great Britain were from internal sources (i.e., Closed Model System). Parameters from Great Britain and Ireland were used in the model calculations. The input parameters and their sources are given in Table 1.

Table 1
Inputs for Risk Calculations for Great Britain (Closed System) and Ireland (Open System)

Input	*GB-1986*	*GB-1990*	*IRL-1986*	*IRL-1990*
CP1[15]	0.7	0.7	0.7	0.7
CP2[16]	0.011	0.001	0.011	0.001
CP3[17]	0.7	0.07	0.7	0.07
CP4[18]	0.11	0.01	0.11	0.01
CP5[19]	0.2	0.002	0.034	0.013
CP6[20]			0.008	0.
CP7[21]			0.0005	0.00004
ColD50 in a clinical animal[22]	10,000.00	10,000.00	10,000.00	10,000.00
Species barrier[23]	100.00	100.00	100.00	100.00

Results

Great Britain was considered to be representative of the Closed Model System as much of the risk of infection was from within Great Britain. Imports to Great Britain played a minor role in the spread of

15 *See* DNV 1997, *supra* n. 6.

16 Scientific Steering Committee of the European Commission, *Opinion of the Scientific Steering Committee on the Human Exposure (HER) via Food With Respect to BSE* (adopted Dec. 10, 1999).

17 Aline De Koeijer et al., *Calculation of the Reproduction Ratio for BSE Infection among Cattle* (unpublished report from the Institute for Animal Science and Health 1998) (on file with ID-DLO, P.O. Box 65, 8200 AB Lelystad, Netherlands); J.T. Cohen et al., *Evaluation of the Potential Bovine Spongiform Encephalopathy in the United States* (Nov. 26, 2001).

18 *Id.*

19 Scientific Steering Committee of the European Commission, *Report on the Assessment of Geographical BSE Risk of Ireland* (adopted July 2000); *see* Aline De Koeijer et al., *supra* n. 17.

20 *See* Scientific Steering Committee, *supra* n. 19.

21 *Id.*

22 Scientific Steering Committe of the European Commission, *Opinion on Oral Exposure of Humans to the BSE Agent: Infective Doses and Species Barrier* (adopted April 13 & 14 2000).

23 *Id.*

the disease. Ireland was considered representative of an Open System Model as it traded with Great Britain, hence, the greatest source of initial infection was from imported feed or animals. Based on animal survival curves for infected and non-infected animals, it was estimated that approximately 70% of infected animals would be culled before exhibiting clinical signs (CP1 = 0.7). After the introduction of active surveillance, the number of subclinical animals entering the factory would be reduced. However, it is difficult to estimate the extent with which the subclinical numbers are reduced hence CP1 is assumed, pessimistically, to be 0.7 for all years. The main source of infection for humans is exposure from consumption of SRM or of meat products containing SRM. Foods such as sausages, mince, meat stuffed pasta, pate, and beef burgers may have contained SRM while many components of the SRM list have been consumed as gourmet foods in a number of countries.[24] SRM (i.e., brain, spinal cord, trigeminal ganglia, dorsal root ganglia, illeum, spleen and eyes) carries about 99% of the infectivity in a clinical BSE case, hence the exclusion of SRM from the food/feed chain reduces the risk of infection significantly. For the 1986 risk calculation for humans, the risk was calculated on the basis that all the SRM from an animal could have been consumed (i.e., CP2 = 1) while decreasing to negligible amounts in latter years due to the exclusion of SRM. The fraction of material with potential infectivity that enters the rendering industry (i.e., CP3) and fraction of infectivity which survives the rendering process (i.e., CP4) were taken from Cohen et al., who give estimates of the effectiveness of various rendering systems at inactivating the BSE agent.[25] The fraction of MBM allocated to cattle for Great Britain was also taken from De Koeijer et al. In Ireland, CP5 was estimated from data supplied by the Scientific Steering Committee who gives figures for the production of MBM in Ireland (approximately 70,000 tons).[26] Imports of MBM given in the Irish country dossier gave figures between the years 1980 and 1987. In 1987, 2,400 tons were used in ruminant rations giving a CP5 of 0.034 for Ireland. In 1989, the amount of MBM used in ruminant rations was around 960 tons, resulting in the decrease in CP5

24 Scientific Steering Committee of the European Commission, *supra* n. 16.

25 *See* Cohen et al., *supra* n. 17.

26 *See* Scientific Steering Committee, *supra* n. 19.

of 0.013 for Ireland. This decrease is the result of the control policies that were put in place. The total imports of MBM from Great Britain by Ireland ranged from 1,000 to 2,500 tons per annum between 1980 and 1987. However, investigations by the Irish government's Department of Agriculture, Food and Rural Development concluded that no MBM was imported and that numbers given were as a result of tariff code misclassification. Between 1926 and 1986, importation of MBM from Great Britain was subject to license, and very few licenses were issued. For the purposes of the model, 2,500 tons was taken as representative of the quantity imported by Ireland during the 1980s. The total MBM produced by Great Britain was 300,000 tons,[27] giving CP6 equal to 0.008. Since 1989, the importation of MBM from Great Britain was prohibited, hence, a reduction in the risk from imports to negligible amounts.[28] The European Unions Scientific Steering Committee, as a worst case assumption, assumes that 5% (i.e., of twenty animals imported, one could have been infected and this number would be reduced by a multiple of 100 after 1998), of live exports from Great Britain between 1988 and 1993 were infected with BSE.[29] After 1998, of the 2,000 animals imported, one could be infected. Official statistics recorded imports from great Britain as 6,407 cattle in 1988-1990 and 488 cattle in 1991-1993.

In 1986, it was clear that the policies in place in Great Britain were not effective in reducing BSE risk, as indicated by the fact that the calculated risks (i.e., Animal $Risk_I$, Human $Risk_I$) as shown in Table 2, were greater than 1. This indicates the strong potential for further infections of animals and humans in Great Britain. In 1986, on average, the number of animal infections caused by an infected animal in Great Britain would have been ten animals (Table 2). This figure compares well with an estimate of nine animals (upper confidence interval of 10) by Ferguson et al. for the same year.[30] The number of potentially infected humans resulting from an infected animal would have been 70

27 Scientific Steering Committee of the European Commission, *Report on the Assessment of the Geographical BSE-Risk of the United Kingdom* (July 2000).

28 *See* Scientific Steering Committee, *supra* n. 19.

29 *Id.*

30 Neill M. Ferguson et al., *Estimation of the Basic Reproduction Number of BSE: The Intensity of Transmission in British Cattle*, 266 Proceedings of the Royal Society London B 23 (1999).

as shown in Table 2. By 1990, the risk to animals and humans has greatly reduced (0.7) due to the control policies that were put in place. The calculated risk is significantly less than 1, indicating a decrease in the epidemic and consequent decrease in the resulting risk of infection while confirming the effectiveness of the control policies that were put in place to curtail the disease.

Table 2
Risk Calculation Results: Average Number of New Infections Generated From One Infected Animal Given the Controls in Place in Great Britain and Ireland at That Time.

Calculation	*GB-1986*	*GB-1990*	*IRL-1986*	*IRL-1990*
Animal Risk I	10.164	0.001	1.833	0.006
Animal Risk E, MBM			0.015	0.000
Animal Risk E, A			0.0009	0.000
Human Risk I	70.0	0.700	70.000	0.700
Human Risk E, A			0.035	0.000

It is clear that Ireland, which represented an Open System Model, was particularly vulnerable in the early 1980s in terms of risk from imported animals and feed. The potential number of new infections caused by an infected animal was 1.8. Being greater than 1, policy changes were required to ensure this went below 1. The average number of new infections created in Ireland by an infected animal in Great Britain via importing feed containing MBM from Great Britain would have been 0.015 animals while the number of infections in Ireland caused, via an imported animal, would have been 0.0009 animals (i.e., only when cases in the Great Britain were over 1,111 would Ireland be at risk from imported animals). The control policies are shown to greatly reduce the risk in a Closed Model System and an Open Model System in 1990.

Integration of Uncertainty into the Model

Scientific judgments on risks and uncertainties are underpinned and framed by unavoidably subjective assumptions about the nature, magnitude, and relative importance of these uncertainties. These "framing assumptions" can have an overwhelming effect on the results obtained in risk assessments. This partly explains why different risk

assessments on the same issue can obtain widely varying results, even though each has apparently been conducted in accordance with the tenets of "sound science."

The issues of uncertainty are now subject to a well-developed academic field of study and comment. A model could contain inputs and outputs in the form of probabilities or frequency distributions. Implicit in this structure are potential sources of uncertainty and variability. Anand emphasizes the uncertainty surrounding BSE and further emphasizes the lack of research into risk and risk communication throughout the crisis.[31] It is of critical importance when calculating risk that the adoption of assumptions be justified. The existing system for gathering scientific opinion should be complemented by a process for analyzing the subjective framing assumptions used in a risk assessment. Models are based on assumptions. In this assessment, the assumption is made that the same MBM processing conditions existed for Great Britain as in Ireland. The infectious load used in this analysis was 1,000 CoID50 per animal (a fully infected animal has the potential to infect 10,000 other animals with a 50% success rate given that the animals are orally exposed to the BSE agent). This represents the infection level in a clinically infected animal. However, this analysis is used to represent the infection in a subclinical animal. This is a very pessimistic assumption as infectivity grows exponentially in the six months prior to onset of clinical symptoms; hence, subclinical animals have significantly lower infectivity levels and represent a lower risk to human and animal health than an animal exhibiting clinical syptoms.[32] To date, little is known about the ability of the BSE agent to infect humans, a species barrier of 100 was taken in this analysis, but this may be as high as 1000.[33] The assumptions taken are pessimistic and often estimate the upper end of the risks from BSE to human and animal health.

Uncertainty analysis provides a systematic and transparent tool for exploring assumptions in risk assessment procedures. Monte Carlo and other probabilistic analytical techniques provide a relatively

31 Paul Anand, *Chronic Uncertainty and BSE Communications: Lessons from (and limits of) Decision Theory in The Mad Cow Crisis* 51 (Ratzan ed., UCL Press 1998).

32 Christl A. Donnelly & Neill M. Ferguson, *Statistical Aspects of BSE and vCJD: Models for Epidemics* (CRC Press 1999); *see also* Anderson et al., *supra* n. 9.

33 *See* Scientific Steering Committee, *supra* n. 19.

straightforward method to parameterise variability and uncertainty. They fully acknowledge uncertainties and can include a multitude of factors included in a risk assessment. In this way, risk assessment can aid deliberation and reasoned judgment. These methods can assist in bringing to light different ways of interpreting scientific advice in the regulation of risks such as those in relation to BSE.

Conclusions

Implementation of a systematic risk assessment process prepares the food industry and consumers to accommodate new BSE research findings and changing disease patterns towards the goal of protecting human health. The larger political context within which policy developments are situated is how to make decisions in the face of uncertainties while at the same time implementing precautionary approaches under commercial and trade pressures. The System Model Approach developed here highlights the effectiveness of the control policies put in place to curtail the BSE disease. The System Modeling Approach is intuitive detailing the main policies taken in response to the BSE crisis and it facilitates easier communication of the risk factors to a largely confused public. The System Model Approach shows that the policies put in place, if properly implemented, will significantly reduce the risk to human and animal health in both Closed and Open System Models.

It is imperative that decision-makers understand policy formation and rationale in order to anticipate and react to changes which directly or indirectly impact the health-care environment. With past experience of BSE and other food safety issues, it seems reasonable for people to harbor doubts regarding the reliability of regulatory science. The BSE crisis raised the issues of the unnaturalness of some practices within agriculture, the failure of institutions to act to prevent them, the long-term consequences to human, and animal well-being and our inability to avoid the resulting risks.

Risk assessment is an essential tool in increasing our knowledge regarding various food hazards. It helps to identify environmental problems and is frequently vital in helping to solve these problems and influence government decisions. Risk assessment methods need to be

maintained and enhanced. The System Model Approach developed here enables a closer look at policy processes governing food safety and their effects on risks from BSE. The potential for an infected animal to infect other animals was greatly reduced in both Great Britain and Ireland as a result of policy changes. These policy changes also enabled Ireland to protect human and animal health from imports (e.g., MBM and live animals) which may be potentially infected. The model highlights the effectiveness of the control policies put in place in the wake of the BSE crisis. However, only corroboration, by changes in actual incidences, can fully validate adherence to the policies. The model also maintains a central role for scientific information and analysis as it emerges.