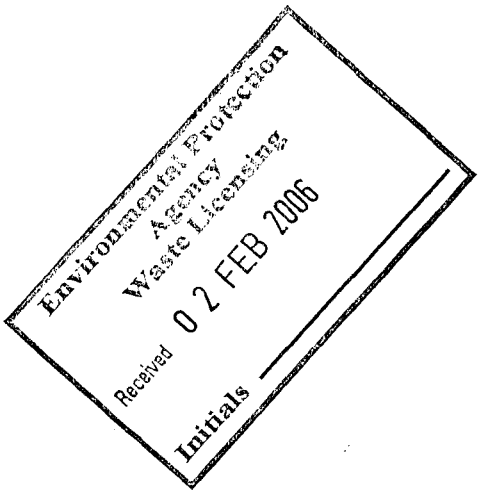

Attachment 05

Composition of Ash & Risk Assessment

1 BACKGROUND INFORMATION

1.1 OVERVIEW

Meat and bone meal (MBM) is produced by cooking discarded animal tissues, removing moisture and collecting the melted fat (as tallow), leaving residual solids which are pulverised to produce MBM. The high protein content of MBM (*circa* 50 %) led to its inclusion in animal diets throughout the world. In the 1980's Bovine Spongiform Encephalopathy (BSE), a novel fatal disease of cattle, emerged in the UK, reaching epidemic proportions very quickly. The role of MBM in the spread of BSE is now well established (Phillips *et al.*, 2000) and restrictions on the use of MBM as a feed supplement have been introduced as part of control measures to combat this disease (European Commission, 1994, 2001a; European Council, 2000).

1.2 MEAT AND BONE MEAL

1.2.1 Introduction

Meat and bone meal (MBM) is produced through the rendering of animal offal and bone waste collected from abattoirs, butchers and meat processors. The rendering process is essentially a series of fat and moisture removal steps which reduces the volume of this material by approximately a factor of four (McDowell, 1997) and stabilises the decay processes in animal waste. The fat removed during the process is refined and sold as tallow (animal fat) to food, feed and pharmaceutical industries. The remaining solid portion of the rendered material is the MBM, a microbiologically sterile powdered product with simple storage requirements, that has been utilised as a protein supplement in animal feeds.

Restrictions have been introduced by the EU to regulate the use of MBM as a feed supplement in response to the BSE crisis. Initially, the restrictions prohibited the inclusion of ruminant derived MBM in ruminant diets. In Ireland there was an additional voluntary but widely practised exclusion of MBM from pig and poultry diets in response to a perceived consumer demand. At present there is a total ban on the use and export of MBM which was introduced in January 2001 as a temporary measure in response to the growing number of BSE cases identified in the EU (European Commission, 2000).

In the absence of a market for MBM as a protein supplement, the rendering industry fulfils a role as a waste management service for the meat industry. The waste management function of rendering is somewhat incomplete, however, because a sustainable disposal outlet / system must then be found to safely utilise / dispose of the output of the rendering plant. Before the onset of BSE, a rendering plant was a self-sustaining and profitable business, purchasing raw material from meat factories, butchers and fallen animal collectors (knackers) for processing and onward sale as a protein supplement with a value of approximately €190 / tonne. Currently there are few outlets for MBM which has fallen in value to €65 / tonne and in recent years, rendering plants have introduced a gate fee to accept and process consignments of meat and animal waste. This situation illustrates the changed environment in which the rendering industry operates today.

In the absence of other waste treatment options, the continued operation of the rendering process is necessary for the sustainable operation of the Irish meat industry. Figures prepared by the Irish Government Central Statistics Office show that for 1998, livestock and livestock products contribute 4.69% of Irish Gross Domestic Product (GDP) and so the importance of maintaining this industry is clear (Central Statistics Office, 2001).

1.2.2 The Rendering Process

As an animal is processed through a meat factory, the carcass is partitioned and those parts of the animal that cannot be utilised for human consumption are separated for other uses. These uses include pet food production and MBM / tallow production. Pet food production is sometimes termed as 'low-risk' rendering while MBM production is termed 'high-risk' rendering. These designations are related to the different microbial load associated with the material dispatched to the two different industries and do not relate to a BSE risk. An abattoir would typically sell stomachs, some organs and various other tissue parts for pet food production while fat trimmed during carcass preparation may be processed on-site in a fat-melting plant for use as food-grade animal fat. The remaining material that cannot be otherwise utilised is sent to a high-risk rendering plant and may include bones, feet, the trachea and parts of the gut. The proportion of animal by-products processed by each of the above options will vary from site to site and over time depending on prevailing economic and logistical factors.

The other input to the rendering plants is sourced from the retail butchery trade where trimmings and other assorted wastes are collected and brought by a waste contractor to the rendering plant.

On entering a rendering plant, the material is dumped either directly into receiving bunkers or onto the floor of the receiving hall. If the material is unloaded onto the floor, the plant operator may take the opportunity to mix the incoming raw materials somewhat to provide a relatively homogeneous feed to the process equipment in terms of meat and bone levels. No attempt is made or required at this stage to segregate the raw material by origin or species. Most rendering plants would receive bovine, ovine and porcine material in proportion to the prevailing agricultural practice of the plant's catchment area. Some plants may take in poultry waste although it is customary for poultry units to operate a closed system and so little poultry material is sent to the rendering plants. The term 'closed system' in this instance indicates that, typically, the waste material produced from the slaughter line of a poultry unit is processed on-site into poultry offal meal (POM) which is then incorporated into feed for use in the chicken rearing houses.

The raw material is conveyed from the receiving area to a series of pre-breakers and crushers which reduce the particle size to below 50 mm, as required by current legislation (European Commission, 1996a). The crushed material is conveyed to steam jacketed vessels and held for the duration of the cooking process. During cooking (approximately 2 hours), the material is agitated, free-flowing fats are allowed to drain from the bottom of the cooking vessel and moisture is removed. Originally, this process was carried out on a batch basis but in recent years, continuous cookers have become standard. The cooked material is then transferred to expeller presses to extract further tallow from the hot material under a pressure of approximately 10 MPa. Finally, the material is cooled and milled prior to bagging or bulk storage at ambient temperature. In 1996, as a response to the BSE crisis, the European Commission introduced an additional step into the rendering process to reduce the risk of BSE infectivity being present in MBM (European Commission, 1996a). This requirement calls for the MBM to be held at 133 °C at a pressure of 3 bar (absolute) for 20 minutes. This hyperbaric heat treatment may currently be carried out at any stage of the process, but usually in Ireland the finished meal is treated prior to milling and storage. The scientific basis for this heat treatment step is

discussed in Section 1.3.5. A flow diagram of the rendering process is given in Figure 1.1.

Physically, MBM may be described as a powdered solid with a distinctive meaty smell and a brown colour. Physical and chemical properties of a typical batch of MBM as produced in Ireland are given in Table 1.1, although it is noted that these may vary depending on the offals processed by the factory on a daily / weekly basis.

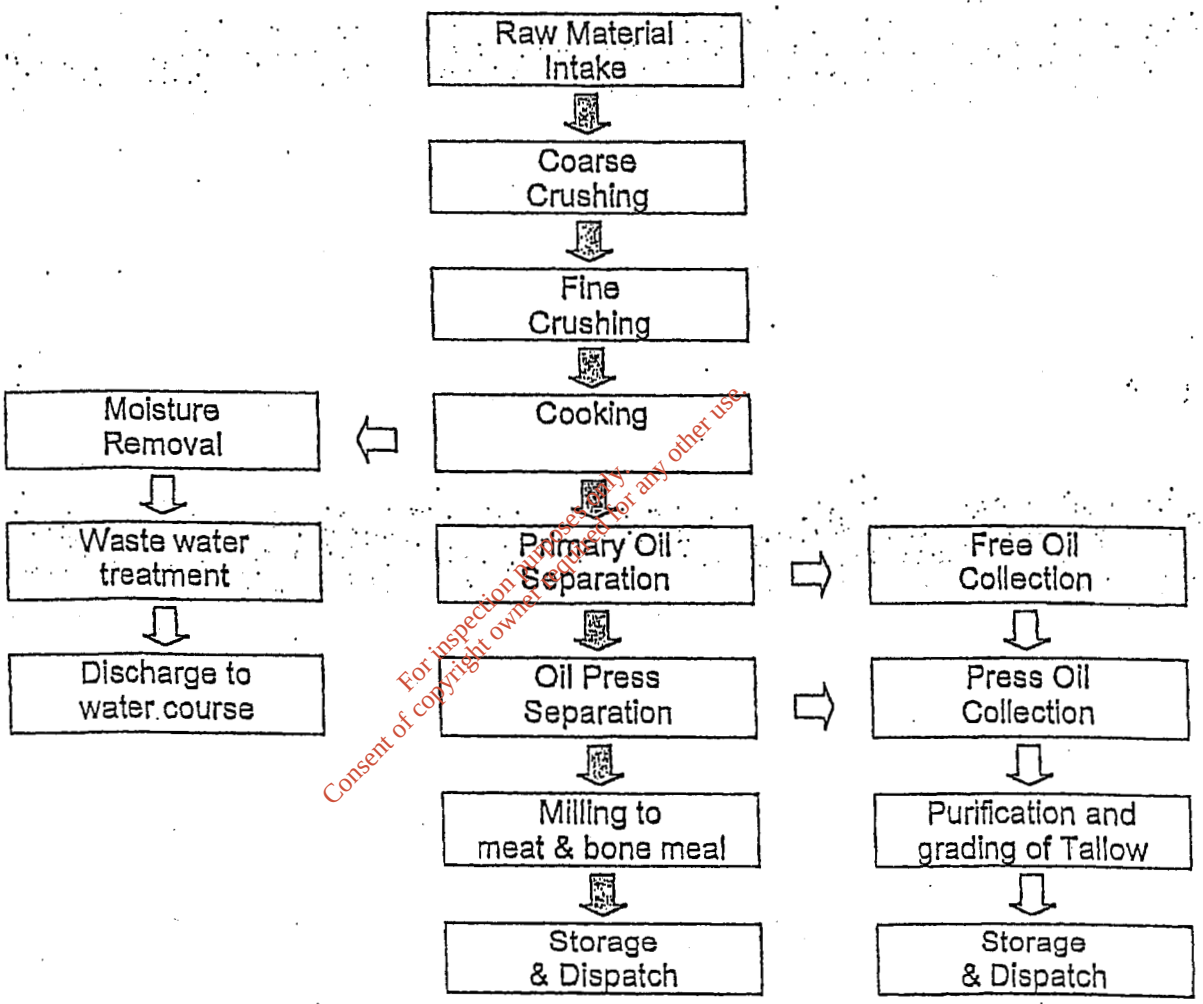

Figure 1.1: Flow diagram depicting the production of meat and bone meal and tallow.

Table 1.1 Typical physical and chemical properties of Irish meat and bone meal (DAF, 1998, Personal Communication)

Property	Value
Density	600 kg/m^3
Moisture	7.0 % w/w
Crude Protein	46.0 % w/w
Crude Oil	13.0 % w/w
Crude Fibre	2.0 % w/w
Crude Ash	30.0 % w/w
Calcium	10.0 % w/w
Phosphorus	5.0 % w/w
Gross Energy	16.2 MJ/kg

As mentioned in Section 1.1, MBM has been widely implicated in the spread of BSE. This situation has focussed much scientific attention on the practices of the rendering industry and on the uses of its products. The origins and epidemiology of the disease is now discussed in some detail.

1.3 Bovine Spongiform Encephalopathy (BSE)

1.3.1 Introduction

First identified in November 1986 in the United Kingdom (UK) (Wells *et al.*, 1987), Bovine Spongiform Encephalopathy (BSE) is member of the Transmissible Degenerative Encephalopathies (TDE)[1] (Fraser *et al.*, 1988; Hope *et al.*, 1988), of which scrapie in sheep and Creutzfeld-Jakob Disease (CJD) in humans are better known examples. It is a progressive degenerative disease of the central nervous system that does not produce fever and elicits no immune or inflammatory response in the affected animal. Typical incubation periods for BSE are 4-6 years (Kimberlin 1993; Stekel *et al.*, 1996) and the disease course varies from less than 2 weeks to 14 months; resulting in death within 4 months after the appearance of clinical signs (Brewer, 1999). Symptoms of BSE include in-coordination, repetitive movements and aggressive behaviour in bovines leading to the common name 'Mad Cow Disease'.

[1] Although these diseases are commonly described as Transmissible Spongiform Encephalopathies (TSE), the term Transmissible Degenerative Encephalopathy (TDE) is preferred, reflecting the histopathological evidence of neurodegeneration in the Central Nervous System (Taylor, 2000).

Disease confirmation can be achieved by a post-mortem histopathological examination of brain tissue which will appear vacuolated and spongy in a diseased animal. Recently, the European Commission has validated a number of rapid tests (European Commission, 2000, 2001b) for use in disease confirmation allowing the implementation of expanded testing regimes. The Enfer test, which was developed in Ireland, is one of these validated tests and is likely to see major use from 2001 onwards as the Department of Agriculture and Food (DAF) implement an active BSE surveillance scheme for the Irish herd based on rapid test methods. The Enfer test is a novel, high throughput chemiluminescent immunoassay, which can be completed in under 4 hours, using a polyclonal anti-PrP antibody for detection. The test itself comprises of a rapid sample extraction procedure, coupled to an ELISA technique, using a polyclonal primary antibody, a Horse Radish Peroxidase-conjugated secondary antibody and an enhanced chemiluminescent reagent (European Commission, 2000, 2001b).

1.3.2 Causative Agent

The precise nature of the causative agent of BSE remains unresolved (Horn, 2001). It is generally accepted, however, that the infectious agent is a mutated prion gene which induces the conversion of normal prion protein (PrP) to the disease associated form (PrP^{res}) (Prusiner, 1997). A prion protein (PrP) is a small self-replicating glycosylated protein molecule found in the brain cell membrane. An infective prion is one which has undergone a conformational change and in the process becomes heat resistant and protease-resistant. The causative agent is not *live* in that it has no associated nucleic acid but the transformation appears to be a physio-chemical phenomenon. When a distorted prion molecule reaches the prions in the brain cell membrane of a host, the distorted molecule is able to act as a template to cause a normal prion protein molecule to adopt a similar distorted shape. The process is then self-replicating as each mutated molecule is able to act as a template to distort another normal molecule. This process causes brain lesions in the affected animal resulting in the classic 'mad cow' symptoms of the disease followed by death within 4 months of the appearance of the initial clinical signs.

Alternative theories have been proposed for the origin and route of BSE infection. The bacterium hypothesis proposed by Ebringer *et al.* (1997) proposes that BSE is an autoimmune disease triggered by exposure of cattle to bacteria containing

molecular sequences which resemble those of, and cause cross-reactivity with, brain tissue. The particular bacteria suggested is *Acinetobacter calcoacetius* which is commonly found in the green offal of cattle and contains proteins similar to the prion protein. The bacterium hypothesis is not generally accepted as a credible explanation for the origin of BSE (Phillips *et al.*, 2000) not least because *Acinetobacter calcoacetius* would be killed by the rendering process. A further theory, the organophosphate theory, links the emergence of BSE with the widespread use of these chemicals in the late 1970's to combat warble fly in cattle (Purdey, 1996). The BSE Inquiry (Phillips *et al.*, 2000) concluded that a direct toxic effect by organophosphates on nerve cells is unlikely although there is a possibility that organophophates may be shown to have a contributory effect in the spread of BSE by affecting the resistance of the animal to the infective agent.

1.3.3 The role of MBM in the spread of BSE

When BSE was first identified in the UK, it was postulated that the inclusion of MBM made from scrapie infected sheep in bovine feed was the source of the BSE epidemic (Wilesmith *et al.*, 1991) implying that scrapie, a disease of sheep, had crossed the species barrier to cattle.

With regard to the origin of the BSE agent, the following points should be considered:

a. During the period 1970-1985, the UK had the largest population of sheep and the third largest population of cattle within the EU. Overall, the UK had one of the highest ratios of sheep to cattle.

b. It is estimated that, in the UK, there are between five and ten thousand cases of scrapie in sheep per year. Comparisons with other countries cannot be made since corresponding data are not available. If the proportion of sheep to cattle carcasses included in MBM reflects the proportions in the standing stocks, then UK MBM would contain a relatively high level of scrapie infected material.

c. This material may have included a BSE strain. Although no scrapie strain yet identified has the characteristics of BSE, the evidence for ruling it out is not strong since it is based on studies of only a small number of scrapie infected sheep. Furthermore, it is known that scrapie strains can on occasion change their characteristics on transmission through a new species. Other possible sources of a TSE agent that may have been included in the MBM contained in the starter

rations of artificially reared calves include a sporadic event in a cow or TSE infected tissue from other sources, such as goats or exotic ungulates.

Taking these considerations together, and recognising that many assumptions are implicit in them, it seems likely that over the years when MBM was widely used in cattle feed an unusual concatenation of events occurred in the UK during the period 1970 to the 1980s. The diet of many calves was changed so that MBM was included in their starter rations. Furthermore, the MBM is likely to have included a relatively high level of scrapie infected material. Changes in rendering processes may have resulted in a small but clinically significant increase in the degree of infectivity of this material in MBM.

The practice of including MBM in the diet of calves for most of the first twelve weeks of their lives began in the mid 1970s in the UK, but does not appear to have been common in continental Europe and the USA. This practice raises the question of whether there is a sensitive period of susceptibility: that young calves are more susceptible than adult cattle to the infective agent (Horn, 2001).

There is some doubt over this theory however as a number of significant epidemiological and pathological differences have been distinguished between the two diseases. These differences are most apparent in the host range - BSE has occurred in a much wider range of species than scrapie. Bruce *et al.* (1997) reported that the brain lesion profile for which that produced by BSE is different to known scrapie strains although Horn (2001) noted that this study involved a relatively small number of scrapie-infected sheep from the UK and does not offer sufficient evidence to exclude an unmodified scrapie agent as the agent of BSE.

The theory reported by the BSE Inquiry suggested that BSE originated from the sporadic mutation of the gene coding for the prion protein (PrP) gene in an animal - most probably a bovine (Phillips *et al.*, 2000). The affected animal developed an abnormal type of prion protein (PrP^{res}) which caused neural degeneration and finally death. The practice of rendering animal waste into MBM for use as a feed supplement would have resulted in number of animals being exposed to the infective agent through the consumption of feedstuffs contaminated with the mutated prions of the original host animal. It was proposed at the BSE Inquiry (Phillips *et al.*, 2000) that the origin of the disease was most likely a random mutation in a single point source

in the Southwest of England. This single animal would have been rendered and the resultant MBM included in feed rations causing a further series of prion modification in animals which again would have been rendered as fallen animals. The original gene mutation - and resultant prion infection - probably occurred in the early 1970's, and a number of 'waves' of the disease then occurred but were not recognised as a new disease (Phillips *et al.*, 2000). The rendering procedures in use at that time were not have been capable of sufficiently reducing BSE infectivity in the raw material - a situation which continued until specific measures were introduced to inactivate BSE infectivity (European Commission, 1996a). The recycling nature of feeding cattle using MBM derived from bovine waste, including fallen animals, created a situation where increasing amounts of infected material were being processed by the rendering plants. This material would have comprised a combination of entire carcasses from the early undiagnosed BSE cases and also the by-products of other, apparently healthy, animals incubating the disease at a sub-clinical level which were slaughtered for human consumption. The disease was thus fuelled by recycling infected BSE carcasses via the MBM route and this cyclical spiral was halted only when an MBM feed ban for ruminants was introduced and fully implemented. It is also possible that an infected animal was used as the tissue source for a veterinary product which then infected a number of animals, but the dramatic effect of the ruminant feed ban in the UK would suggest that feed was the main route of the disease. It is also noted that recent reports have emerged of evidence of a link between BSE and scrapie (Balter, 2001). This research may yet see the 'scrapie hypothesis' gaining acceptance again underlining the evolving nature of scientific knowledge in this area.

Epidemiological investigation suggests that BSE was identified during the third wave, by which time the disease was relatively widespread throughout the UK herd (Phillips *et al.*, 2000). This situation provided the mechanism by which the disease outbreak appeared to reach epidemic proportions extremely quickly.

In the early stages of the MBM feed ban, the ban was ineffective as existing stocks of MBM - which were most probably infectious - continued to be fed to animals and were not destroyed. In addition, it was not appreciated how easily cross contamination could occur in feed plants producing both ruminant and non-ruminant feeds which allowed traces of - probably infectious – bovine rendered material to be included in feedstuffs destined for ruminant diets. After these points were addressed

the epidemic began a downward trend and it would appear that a well-managed feed ban is a critical mechanism for the reduction and control of BSE levels.

In the years prior to the identification of BSE, a number of operational changes were introduced to the rendering industry which are thought to be factors in the emergence of BSE. In tandem with the scrapie origin theory, these changes, notably the switch from batch to continuous processing and the abandonment of solvent extraction procedures were thought to have diminished the ability of the rendering process to reduce scrapie infectivity in MBM (Wilesmith *et al.*, 1991). It was thought that in the UK during the 1970's and 1980's scrapie infectivity was present in MBM at levels sufficient to breach the ovine-bovine species barrier and transfer to cattle. Although no commercial rendering procedure is capable of completely inactivating the BSE agent (Phillips *et al.*, 2000), it is suggested that the new rendering procedures may have led to a ten-fold decrease in the inactivation of BSE by rendering (Horn, 2001). This decrease may have allowed a level of infectivity to remain after rendering that was sufficient to initiate and sustain an epidemic.

1.3.4 Disease Transmission

Transmission of BSE through contaminated feed is the primary route of infection in bovines. The introduction of the ruminant feed ban in the UK quickly reduced the incidence of BSE although new cases of BSE did continue to appear. Incomplete implementation of feed controls and the use of stockpiled feed can lead to the effectiveness of such a ban being reduced – particularly in its early stages. While the majority of BSE cases arose by ingestion, it was recognised that there is a possibility that some vertical and horizontal transmission may occur and a number of studies were carried out to investigate these alternative transmission mechanisms.

Maternal, or vertical, transmission in scrapie, the most researched TDE, has established the existence of maternal transmission and in 1989, a major study was carried out by the UK Ministry of Agriculture, Food and Fisheries (MAFF) to investigate maternal transmission in BSE. In this study, the incidence of BSE in 300 calves from BSE-infected dams was monitored and compared with a control group of 300 calves from non-infected dams. The results of the study suggested that BSE incidence in the offspring of infected dams was 10% higher than the control group. The study could not, however, differentiate between direct maternal transmission and any inherited genetic susceptibility. The 10% rate is generally accepted for maternal

transmission but would not be sufficient to maintain the BSE epidemic in the UK and is a contributory factor alongside oral infection through contaminated feed (Cummins, 2001, Personal Communication; Horn, 2001).

Horizontal transmission of BSE through the herd has been monitored but little animal to animal spread has been observed (Hoinville, 1995). The BSE inquiry concluded that the occurrence of lateral transmission for BSE in cattle has not been conclusively proven (Phillips *et al.*, 2000).

Studies by MAFF (MAFF, 1997a) using deliberately infected calves detected infectivity in animals three months prior to the onset of clinical symptoms implying the existence of a sub-clinical phase of the disease where the animal contains infectivity but has shown no clinical signs. This phenomenon is a cause of some concern for the continued inclusion of MBM as an ingredient in animal feed as it appears that BSE infectivity could be present in apparently healthy cattle sent for slaughter and subsequent consumption by animals and humans.

1.3.5 Inactivation of BSE

One of the major difficulties with the implementation of regulations to control BSE is the lack of knowledge surrounding the disease and its inactivation. Inactivation studies carried out on the causative agents of TDEs, of which BSE is a member, have shown that they are relatively resistant to inactivation (Taylor 1999a, 1993). In order to achieve decontamination rigorous chemical or physical procedures are required. The majority of data on inactivation studies has been collected on scrapie, which is a TDE disease of sheep, first identified over 250 years ago (Bradley, 2001), on which much research has been undertaken.

1.3.5.1 Thermal Inactivation

Thermal decontamination of the scrapie agent is complicated by the existence of several different strains of the same agent, as these strains exhibit differences in their thermostability. In a study carried out by Dickinson (1976) it was shown that scrapie strain 139A was completely inactivated when exposed to gravity displacement autoclaving at 126 °C for two hours, however strain 22A was only inactivated when exposed to the same temperature for four hours. When the trials were repeated using porous load autoclaving it was shown that both of the above

strains of scrapie agent were inactivated at a temperature of 136 °C for four minutes (Kimberlin *et al.*, 1983).

In a gravity displacement autoclave, the air in the sterilisation chamber is displaced as steam fills into the chamber while in a porous load autoclave, a vacuum removes the air from the chamber allowing a rapid influx of steam. This operational difference would appear to have significance in terms of the rapid thermal fixation of the TDE agent and the consequent protection from inactivation during porous load autoclave sterilisation of TDE agents.

More recent studies have shown that BSE agent and two strains of rodent passaged scrapie agent survived exposure to porous load autoclaving cycles of 134 to 138 °C for 18 minutes (Taylor *et al.*, 1994) although the brain macerates used in these studies were much larger (340 mg) when compared with previous studies which only used 50 mg (Kimberlin *et al.*, 1983).

During the studies carried out on porous load autoclaving by Taylor *et al.*, (1994), it was observed that the thermostability of one particular strain of scrapie agent, 22A, was enhanced by an increase in temperature. The explanation given for this is that the brain macerates become smeared and dried onto the surface of the glass containers and it has been shown that the scrapie agent is more resistant to inactivation when infected brain tissue becomes dried onto glass or metal surfaces (Asher *et al.*, 1987). It has also been shown that macerated infected brain tissue survived autoclaving at 134 °C for one hour whereas the same quantity of uncomminuted infected brain tissue became inactivated after eight minutes exposure to the same temperature. It has been suggested that the reason for this is the rapid heat fixation of the PrP^{res} prion protein in the macerated brain tissue which would enhance the thermostability of the scrapie agent (Asher *et al.*, 1987).

Drying of the scrapie agent was found to enhance its thermostability (Asher *et al.*, 1987) although it may be possible that the survival of TDE agents in these experiments could be due to the stabilising effect of moisture removal. In an extreme case Brown *et al.*, (1990) reported partial survival of scrapie following exposure to 360 °C for one hour. The sample had, however, initially been freeze dried, depicting unusual circumstances. The survival of lyophilised infectivity after exposure to 360 °C

has led to some speculation that the effectiveness of incineration for inactivating scrapie-like agents should be questioned.

In studies carried out on the effect of different rendering procedures (Taylor *et al.*, 1995 and Taylor *et al.*, 1997) on scrapie and BSE it was found that only those rendering procedures using pressure for a period of time greater than 18 minutes and temperatures greater than 134 °C were effective in decontaminating the scrapie and BSE agents. No infectivity was found in any of the tallow samples examined.

On the basis of the above experiments the thermal treatment of meat and bone meal using the conditions of 133 °C for 20 minutes at 3 bar pressure (absolute), with a particle size of 50 mm, were adopted by the EU as law - Commission Decision 96/449/EC, (European Commission, 1996a). Similar studies were carried out by Schreuder *et al.*, (1998) to assess the efficacy of variations in the hyperbaric procedures used at the two rendering plants in the Netherlands including the above required EU thermal treatment. The results indicated that by using the recommended EU procedure a reduction in BSE infectivity of 100 to 1000 fold is obtained. These studies, however, implied that where the level of infectivity in a sample is very high the recommended thermal procedure of 133 °C for 20 minutes at 3 bar pressure (absolute), would not be sufficient to completely inactivate the material which is infected. Schreuder *et al.*, (1998) however goes on to say that any risk from high level infectious material would be blocked by the ban on rendering specified risk materials from ruminants. It was acknowledged by the EU Scientific Veterinary Committee (Scientific Veterinary Committee, 1996) that the above recommended process conditions are confirmed to result in a safe product *i.e.* MBM which, when included in feedstuffs, will not induce infection.

1.3.5.2 Chemical Inactivation

It was reported by Mould *et al.* (1965), that scrapie infectivity was not inactivated over the pH range 2-10. Later work by Brown *et al.* (1986) concluded that exposure 1 M sodium hydroxide (pH = 14) for 1 hour inactivated some named strains of scrapie. Recent work by Taylor *et al.* (1999) has shown that BSE can be inactivated by boiling in 1 M sodium hydroxide for 1 minute. This work is being further developed on a larger scale with a view to establishing a commercial facility for the destruction of BSE infected carcasses (Hamilton, Personal Communication, 2000). A recent article

In *Render* magazine reported on the development of a rendering procedure incorporating hot alkali to produce animal meal at lower temperatures to maximise protein digestibility without sacrificing BSE inactivation (Caprella, 2001).

Sodium hypochlorite solutions are the only other decontamination procedures proven to be effective at inactivating BSE infectivity (Taylor, 2000). Research by Kimberlin *et al.* (1983) demonstrated that sodium hypochlorite was effective in inactivating scrapie infectivity and recommended that a solution of 20,000 ppm should be applied for 1 hour. Later studies by Taylor *et al.* (1994) showed no detectable infectivity in BSE infected samples treated with sodium hypochlorite solutions over a range of times and solution strengths.

1.3.6 Human Risks from BSE

Creutzfeld-Jakob Disease (CJD) is a fatal TDE of humans which occurs sporadically at a rate of approximately 1 in 1×10^6. On the 20th March 1996, the UK government announced ten cases of a new variant of CJD (termed vCJD) that had emerged during 1994-95, and were found by the CJD surveillance unit (CJDSU) to exhibit an unusual combination of features. There were no apparent occupational or lifestyle similarities that might suggest a common risk factor. Compared with previously known CJD, the average age was younger, the duration of illness longer, the symptoms different, the electroencephalogram (EEG) brain activity different, and the brain pathology different. The appearance of this novel variant of an established TDE at a time when BSE had been prevalent in the UK for some time raised concerns that BSE may have spread to humans as vCJD. Bruce *et al.* (1997) determined that, based on brain lesion profiles, BSE infectivity and vCJD infectivity carry the same signature which the authors conclude provides strong evidence that the same agent strain is involved in vCJD and BSE.

On 20th March 1996, the UK government's Spongiform Encephalopathy Advisory Committee (SEAC) stated:

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 (SBO) ban in 1989 (SEAC, 1996).

Reviewing that statement three years later, on 18th March 1999, SEAC concluded that vCJD was an acquired prion disease caused by exposure to BSE or a BSE-like agents and stated that it recognised that not all new cases would necessarily relate to exposure before the SBO ban (SEAC, 1999).

In the UK, as of 28th December 2000, some 88 definite and probable cases of vCJD have been identified by post-mortem examination (Department of Health (UK), 2001). The first case of vCJD in the Republic of Ireland was diagnosed on the 11th June 1999 and remains the only case to date (WHO, 2001). There has also been three confirmed cases of vCJD in France (WHO, 2001).

1.3.7 BSE in the UK

1.3.7.1 Introduction

As discussed in Section 1.3.1, BSE was initially recognised in cattle in the UK in 1986 (Wells *et al.*, 1987). When BSE-infected cattle started to die, the carcasses were rendered and the MBM included in protein supplements for bovine feed which caused an amplification effect and precipitated the BSE crisis. The epidemic in cattle in Britain reached widespread proportions; by 1993 more than 1 000 cases per week were being reported. As of the end of 2000, 179 441 infected cows have been identified (European Commission, 2001), involving more than 50% of the dairy herds in the UK (MAFF, 2001). Protein supplements containing sheep and cattle offal were banned in the UK in 1988, but this ban was not strictly enforced until 1991-1992. Given the long incubation of BSE, the epidemic curve did not start falling until late 1993. In 2000, 1 312 new cases of BSE were identified in the UK (European Commission, 2001).

1.3.7.2 Culling Schemes

The UK operates a number of culling schemes which aim to accelerate the eradication of BSE by the slaughter of animals considered to be most at risk of infection and therefore restore public confidence in the safety of British beef and related by-products. A brief description of three of these cull schemes which are related directly to control measures dealing with suspected or confirmed BSE cases and their cohorts is given below.

a) *BSE suspects:* Any cattle which are, in the opinion of a veterinary surgeon, displaying clinical symptoms which might indicate BSE must be destroyed and the carcass sent directly to a designated incinerator for destruction.

b) *The selective cull scheme:* This covers cattle which are believed to have been fed the same feed in the first few months of life as confirmed BSE cases. The cattle are slaughtered and either incinerated in carcass form at a designated incinerator or rendered and stored as MBM for subsequent destruction.

c) *The offspring cull:* This scheme recognises that there is a slight risk of transmission of BSE from cow to calf. Therefore the offspring of female cattle which have been diagnosed as suffering from BSE are traced, slaughtered and sent directly for incineration at designated facilities.

1.3.7.3 Over Thirty Months Scheme (OTMS)

In March 1996, the EU imposed a worldwide prohibition on the export of British beef. In response to this ban, the UK government introduced the over thirty month scheme (OTMS) which, with some exceptions, prohibits the sale of meat for human consumption from cattle aged over 30 months at slaughter. The scheme is intended to restore consumer confidence and provide economic assistance to the beef industry and goes beyond what SEAC believe is strictly necessary to protect public health.

The carcasses of cattle slaughtered under the OTMS are required by European Commission Regulation 716/96 to be incinerated or sent to a rendering plant for processing and then destroyed. In view of the limited amount of incineration capacity available in the UK the Government announced when the Scheme was being drawn up that OTMS waste material would be treated primarily by rendering and the resultant products (MBM and tallow) disposed of by the best practicable environmental option. Approximately 400 000 tonnes are stocked in the form of MBM and 200 000 tonnes of tallow are awaiting destruction (MAFF, 1999).

1.3.7.4 Specified Bovine Offal (SBO)

In 1989, MAFF defined a list of tissues which were designated as specified bovine offal (SBO) and would be removed from bovines at slaughter and destroyed as a control measure against BSE. These tissues harboured the highest levels of BSE

infectivity as determined by MAFF studies and their removal was intended to decrease the amount of infectivity entering UK rendering plants. These plants were at the time producing MBM for use in non-ruminant animal feed rations in the UK and for export to countries without controls on the use of MBM in animal feedstuffs. The general principle of the SBO ban was adopted by the EU as a part of the BSE control measures formulated by the European Commission. The list was expanded to include ovine and caprine material and renamed specified risk material (SRM) which will be discussed in greater detail in Section 1.4 below.

1.4 Specified Risk Material (SRM)

As discussed in Section 1.2.2, a high-pressure sterilisation step was introduced in the rendering process with a view to inactivating the infectious agent. Research into BSE inactivation, reviewed in Section 1.3.6, has shown that this procedure would not be fully effective if very high infective loads were carried in the raw material and so the removal of those parts of an animal that present the greatest risk of infectivity was required. This material, the SRM, is removed and segregated from the other offal at slaughter. The use of SRM and any substance produced from it is prohibited in animal feed and all other food or feed applications. The material is stained with cobalt blue dye on removal to prevent accidental or fraudulent usage and is then processed and stored separately to other animal wastes prior to destruction.

Based on the original concept of the UK SBO ban as discussed in Section 1.3.7.3, the SRM list was compiled by a working group established under the auspices of the European Union Scientific Steering Committee (SSC). The first task of the working group was to use current scientific opinion to assess infectivity in the tissues of susceptible farm animals with a view to establishing the risk to human and animal populations in affected areas. The outcome of this assessment was formally adopted by the SSC and is given in summary form as Table 1.2 below.

Table 1.2: Categorisation of Infectivity in Animal Tissues as published by the European Union Scientific Steering Committee (SSC, 1997).

Category	Organs
High Infectivity	Brain, eyes, spinal cord, dorsal root ganglia, dura matter, pituitary, skull, vertebral column, lungs and ovine and caprine spleens.
Medium Infectivity	Total intestine from duodenum to rectum, lymph nodes, bovine spleen, tonsil, cerebrospinal fluid, adrenal.
Low Infectivity	Nasal mucosa, peripheral nerves, bone, marrow, liver, pancreas, thymus.
No Detected Infectivity	Skeletal muscle, ear, kidney, colostrum, milk, discrete adipose tissues, salivary gland, saliva, thyroid, mammary gland, ovary, testis, seminal testis, cartilaginous tissue, connective tissue, skin, hair, blood clot, serum, urine, bile, faeces

Note: The assessment and allocation of the above tissues to certain categories is based in part on scrapie titres, on the high level of infectivity found in the brain of BSE affected cattle, on the result of mice bioassay tests and on the presumed CJD infectivity of human dura matter and human pituitary gland based on transplant. Some tissues are allocated to higher categories due to the possible contamination during slaughterhouse procedure.

Arising from the information in Table 1.2 and with regard to the variation in the level of infectivity, a suggested list of SRM was defined by the SSC. It was proposed initially that the tissues included in this list, based on the species, age and relative level of infectivity, should be excluded from the human and animal feed chains depending on geographical source. The listing of tissues classified by the SSC as SRM is given in Table 1.3.

Table 1.3: The Scientific Steering Committee's suggested list of specified risk materials to be excluded from human and animal consumption except when derived from a BSE free country with a negligible risk.

Tissue	Species[1]	Age	Basis
Brain	B,O,C	>12 months	Infectivity
Eyes	B,O,C	>12 months	Infectivity
Dura matter	B,O,C	>12 months	Contamination
Pituitary	B,O,C	>12 months	Contamination
Skull	B,O,C	>12 months	Contamination
Spinal cord	B,O,C	>12 months	Infectivity in bovines and theoretical back infection in caprines and ovines.
Dorsal root ganglia	B,O,C	>12 months	Infectivity in bovines and theoretical back infection in caprines and ovines.
Vertebral column	B,O,C	>12 months	Contamination and low infectivity
Spleen	O,C	All ages	Infectivity
Intestine	B,O,C	All ages	Infectivity and contamination
Tonsils	B,O,C	>12 months	Infectivity
Lung	B,O,C	>12 months	Contamination

[1] *Initials indicate species for which list applies; B indicating Bovine, O: Ovine and C: Caprine*

1.5 BSE IN IRELAND

1.5.1 Introduction

In Ireland, *circa.* 125,000 tonnes of MBM is produced annually from 427,000 tonnes of raw material by seven rendering plants. The plants are supervised by DAF staff and since 1 April, 1997, EU directive 96/449/EC which states that material must be rendered at 133°C under pressure of 3 bar for 20 minutes, was applied to these plants. The raw material stream into the plants consists of meat factory and butcher-shop waste. Animal species sent to rendering plants are mainly bovine, ovine and porcine with some poultry material although it is usual for poultry plants to operate an on-site rendering facility producing poultry offal meal (POM) for feeding back to the birds. The levels of different species entering the plant will vary between the seven plants and within each plant according to season and the prevailing level of activity in the meat industry. Discussions with the operators of rendering plants in Ireland did not result in typical species compositions for the incoming material to rendering plants although in 2001 Healy (Personal Communication, 2001) obtained some species characterisation data by way of a survey of the Irish rendering industry as shown in Figure 1.2 below - note at that stage there was a single plant designated to handle SRM.

Until quite recently *circa* 50% of the MBM produced in Ireland was utilised as a feed ingredient on Irish farms being included in ruminant, porcine and poultry rations. The remainder was exported mainly to Eastern Europe and also Asia where it was fed to poultry destined for export markets - including Europe. Since the emergence of BSE and the associated restrictions on the uses of MBM in animal feed within Ireland, export markets have been increasingly sought for this material. The current – albeit temporary - ban on the use of MBM for feed use to all farm animals has resulted in large amounts of MBM being stockpiled around the country awaiting a viable disposal / utilisation outlet.

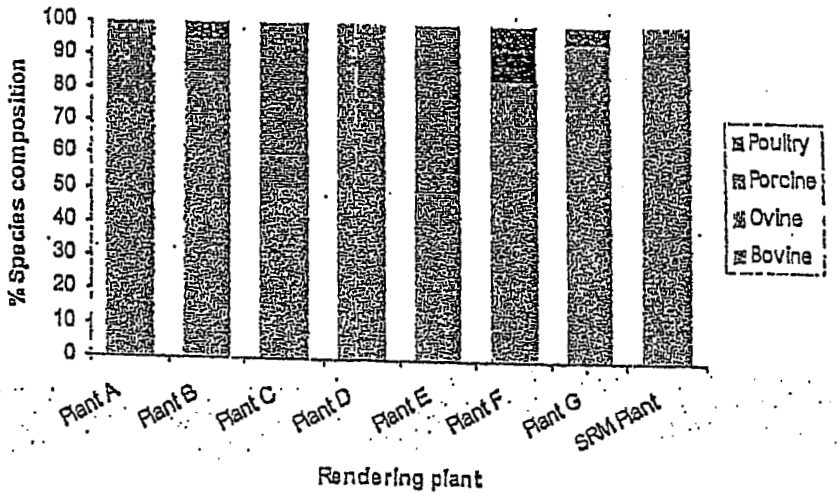

Figure 1.2: Percentage composition by species of incoming raw material to the seven high risk rendering plants in Ireland (denoted as A to G) and the designated SRM rendering plant (Healy, 2001).

1.5.2 BSE Incidence

The numbers of cases of BSE identified in Ireland for each year since 1989 is given in Table 1.4. Griffin *et al.* (1997) described the epidemiology of BSE in the Republic of Ireland from 1989, when the disease was first reported up until the end of 1996. The population at risk each year was estimated at 2.4 million animals. One hundred and eighty nine cases were confirmed between 1989 and 1996. Twelve of these were imported animals. The majority of the cases were between 4 and 6 years of age, which is consistent with a similar incubation period (average 5 years) reported for the UK (Kimberlin *et al.*, 1993). Of the 189 cases, all except one were cows, the remaining animal was an imported bull. There was a higher incidence in dairy herds than in suckler herds and the herd incidence was directly correlated with herd size as was also noted in the UK. The infectivity risk associated with the use of fat from the spinal area in the production of milk replacer for calves should be considered in the incidence of BSE in dairy herds. The use of milk replacer manufactured from fat contaminated with SRM is strongly suspected to be a factor in the spread of BSE in Germany (Honikel, Personal Communication, 2001). The original source of the BSE agent for cattle in the Republic of Ireland has not been conclusively established. However, the available data suggests the following: i) cases that had been imported from the UK had acquired the BSE agent prior to importation into the Republic of Ireland and, ii) contaminated feeds imported from the UK were the indirect source of the agent for a number of the cases.

Table 1.4 The incidence of BSE in Ireland to date (Source: Office International des Epizootes, 2002).

Year of confirmation	Number of cases
1989	15
1990	14
1991	17
1992	18
1993	16
1994	19
1995	16
1996	73
1997	80
1998	83
1999	95
2000	149
2001	246
2002 (to 30/09/02)	254
Total	*1095*

Between 1989 and 1995, 14 to 19 cases were reported annually, while in 1996, 73 cases were reported Griffin *et al.* (1997). The authors list the possible reasons for this increase as being: i) a higher level of detection and reporting than in previous years and, ii) the feeding of imported and native animal feeds which had been contaminated with protein concentrates containing the BSE agent before and during the early 1990's.

Since 1996, the number of cases of BSE reported in Ireland has increased. This would not be expected in view of the introduction of a feed ban which should have removed the infected material from the feed chain. The most probable explanation for this situation is that increased awareness among veterinary surgeons and farmers coupled with the introduction of a compensation scheme resulted in an increased reporting and diagnosis of suspected BSE cases. It is also noted that the feed ban

was not fully enforced until 1996 (Maguire, 1997) and therefore, allowing a disease incubation period of up to five years, the number of cases of BSE could be expected to remain at these levels until 2001.

The figures for BSE incidence in Ireland in 2000 showed a marked rise over previous years and contrary to predictions of disease models (Cummins, Personal Communication, 2001). The most likely cause for this rise is that case numbers had been under-reported in previous years which implies that BSE was more widespread in Ireland than originally thought. An assessment of BSE in Ireland was published, without peer review, by the UK Food Standards Agency based on the assumption that 2000 was the first year that case numbers were fully reported (Food Standards Agency, 2001). This report estimated, using back calculation methods, that up to 22,000 cattle were infected with BSE in Ireland in the period 1985 to 1996.

1.5.3 Control Measures

The following control measures apply where animals are either suspected of being affected by, or have been confirmed as cases of BSE (DAF, 2002):

- Suspect animals are visited by both a veterinary inspector from the local District Veterinary Office, and a veterinary research officer from the Veterinary Research Laboratory. The suspect animal is euthanased, a sample of the brain tissue is sent for testing, the entire carcase of BSE suspect animals are frozen and retained by the Department; pending their ultimate destruction. The herd in question is immediately placed under official restriction and quarantined.

- An inventory of the herd and an initial epidemiological investigation is carried out. The course of the disease is monitored for a short while, following which, where BSE is not ruled out, suspects are slaughtered and the brains taken to the central Veterinary Research Laboratory for examination using both standard histology and immunocyto chemistry.

- If BSE is confirmed, the entire herds are depopulated and compensation for all the animals is paid to the farmer by the State at the full market value of the animals. The slaughter of the animals being depopulated and destroyed is carried out in a special dedicated meat factory. The carcasses are then rendered into meat and bone meal at one specific rendering plant. The collection, transport, slaughter and

destruction of the animals takes place under the direct control of the District Veterinary Office. The meat and bone meal and the tallow are excluded from the human food and animal feed chains, and is stored in secured premises supervised by the Department pending incineration.

- Brain sections of all the adult animals in the herds being depopulated are extracted from the animals being slaughtered and sent to the laboratory for testing for BSE. The brains are examined by histopathology and immunocyto chemistry.

- A full epidemiological examination of the BSE case takes place, including an examination of farm records and a search of the farm to determine if any evidence of potential exposure to meat and bonemeal can be found.

- The affected farm is disinfected with an approved disinfectant (20,000 PPM of available Chlorine) and left vacant for more than 30 days.

- In all cases the progeny of the affected animal and the birth cohorts of the affected animal are traced, purchased at market value, slaughtered, destroyed and rendered in the same manner as depopulated herds.

1.5.4 SRM in Ireland

In February 1997, the Government of Ireland introduced Statutory Instrument 80 regarding the designation and removal of Specified Risk Material (SRM) and in May 1997, European Commission Decision 97/312/EC (European Commission, 1997) approved the implementation of the SRM list for Ireland and a number of other countries. The following portions of animals are currently designated as SRM in Ireland and are excluded from human food and animal feed chains (DAF, 2002)

- the skull, brain, eyes, tonsils and spinal cord of cattle over 12 months and the intestine from the duodenum to the rectum of bovine animals of all ages;
- the skull, brain, eyes, tonsils and spinal cord of sheep and goats that are over twelve months of age or that have one permanent incisor erupted through the gum and the spleen of sheep and goats of all ages.

This material is removed on slaughter, permanently stained and transferred to a dedicated rendering plant. Approximately 900 tonnes/week of SRM material is

delivered to the plants designated to handle SRM, amounting to approximately 54 000 tonnes/annum comprising SRM (from abbatoirs, meat factories and butcheries) and fallen animals. The SRM is rendered and the resultant MBM and tallow are stored pending destruction. The other input to this plant are the cohorts of a BSE infected animal, removed as outlined in Section 1.5.3, which are slaughtered at a designated meat plant in Co. Limerick. This material remains the property of DAF and is processed separately from the SRM. Currently, the MBM and tallow produced from the depopulated herds is exported for incineration in Europe under contract from DAF.

1.6 Disposal of MBM

A safe and controlled strategy for the disposal / utilisation of MBM / SRM is an essential condition for the control of BSE. To be acceptable, the chosen method of disposal must offer a safe, secure and environmentally acceptable solution at a reasonable cost. Currently, Irish SRM-derived MBM is shipped to mainland Europe for incineration at a relatively high cost (approx. €300 / tonne) and economic issues alone cast serious doubt over the long term sustainability of this disposal route. The situation is further complicated by political issues, as the continuing export by Ireland of a domestically produced waste product is known to be unpopular at EU level and is likely to be curtailed in the future.

Every ruminant animal slaughtered for human consumption in Ireland produces a volume of SRM which, by law, must be disposed of in a manner which will exclude this material from the food / feed chain. The mass of SRM in a typical animal weighing 540 kg will amount to approximately 37.05 kg (Cummins *et al.*, 2001). Current scientific opinion is leaning towards extension of the SRM portion of an animal and so the current annual production may increase over time. The continued operation of the Irish meat industry is dependent on the existence of an established procedure for the disposal of SRM and economic and political pressures on the current disposal route impose a requirement for the provision of a facility for the disposal of MBM within Ireland with immediate application for the disposal of SRM derived MBM.

A Task Force on the Beef Industry was established by the Irish Government in November 1998. The remit of the group was to examine and make recommendations on the future of the industry by the drawing up of a strategic plan of action for the

sector covering the complete range of activity from on-farm production to processing and marketing. The final report of the task force on the beef industry in June 1999 (DAF, 1999) stated:

"The Task Force recognises the need for the beef industry in Ireland to have access to appropriate waste handling facilities within the State. It is neither appropriate nor sustainable in the long term that the sector should have to rely on the disposal and destruction of its waste by other countries, either within the EU or beyond. It recognises that in the context of BSE controls, which are likely to last, the handling and disposal of Specified Risk Material (SRM) will be a feature of the industry for the foreseeable future and to this end, a domestic disposal facility will be required. The Task Force recommends that State support should be made available for the establishment of such facilities within the shortest possible timeframe. There are economic and food safety gains to be achieved by this action."

Landfill facilities have been used in the UK to contain MBM and carcasses although current OTMS rules call for thermal destruction of the material. Research has shown that TDE agents will survive interment for periods up to three years without a significant drop in infectivity (Brown and Gajdusek, 1991) and so there is a risk of ground water contamination through the leaching of infectious material through the soil. The use of engineered barriers and a leachate management programme could control this problem to some extent but further problems exist with regard to the possible contamination of surface waters, the distribution of the material by rodents / birds and the current resistance to the use of landfill for waste disposal at a local and government level. In Ireland, the Environmental Protection Agency (EPA) have indicated that it would not be in favour of the disposal of MBM in landfill sites (Macken, 2001, Personal Communication)

1.7 Summary

This chapter provides background on the role of MBM in the spread of BSE along with the BSE control measures introduced by regulatory authorities. These measures included restrictions on the use of MBM in feedstuffs and on which species could be fed MBM produced from another species. The restrictions are likely to continue for the foreseeable future and so a sustainable outlet for MBM is required including use as a fuel supplement.

2 RISK ANALYSIS

It is generally accepted that a safe and controlled strategy for the disposal or utilisation of MBM/SRM is an essential condition for the control of BSE. The ideal disposal method would offer a safe, secure and environmentally acceptable solution with an energy recovery component to offset the logistical and processing costs. The use and behaviour of MBM as a co-fuel in combustion facilities has been researched and shown to be successful (McDonnell *et al.*, 2001).

With regard to human exposure and hence risks associated with the disposal or utilisation of SRM-derived MBM, current scientific literature and opinion was consulted and an exposure assessment was carried out for the proposed operation. Although the clinically infected animals identified on farms are not typically processed through the SRM-derived MBM stream, the level of incidence of BSE will influence the probability of material from animals infected at sub-clinical and pre-clinical levels entering the rendering plant. The risks to humans associated with the combustion of SRM-derived MBM needs to be quantified with a risk assessment. Many elements of risk assessment relating to BSE have been reviewed (Cummins *et al.* 2001). In this research an exposure assessment was conducted as a measure of total societal risk.

The quantitative exposure assessment is consistent with the risk assessment framework described in the report "Application of risk analysis to food standards issues", prepared by the FAO/WHO expert consultation (WHO, 1995). Two distinct stages were taken in the development of the exposure assessment presented in this paper. Initially a deterministic model was designed using fixed worst-case values to provide the basic structure and this model was then further developed into a stochastic (Latin Hypercube sampling) simulation using probability distributions for the most important input parameters.

2.1 MODEL STRUCTURE

The process of risk assessment is divided up into four stages; hazard identification, exposure assessment, dose response assessment and hazard characterisation.

2.1.1 Hazard Identification

Hazard identification focuses on what can go wrong and how it would happen. A hazard is a biological, chemical or physical agent which has the potential to cause an adverse effect. The collection of data relating to the disease (e.g. BSE) is carried out during the hazard identification stage. Epidemiological and surveillance data are collected to quantify the factors which contribute to the survival, mode of transmission and growth of the disease.

2.1.2 Exposure assessment

Exposure assessment evaluates the likelihood of hazards occurring and the implications should they occur. The data collected in the hazard identification stage is used to assess the potency with which the disease can infect taking into account possible critical points which may act as control points to halt the disease, hasten its inactivation or reduce exposure. The pathways by which the disease challenges potential hosts are identified and the initial disease concentration is examined as this may also have an impact when looking at exposure assessment.

2.1.3 Dose-Response assessment

Given the fact that a host has been exposed to the pathogen, what will be the response of a susceptible host be to varying amounts of exposure?. A "dose-response" is used to translate the exposure assessment into a response in terms of infected host animals. The susceptibility/immunity of the host has to be taken into account.

2.1.4 Risk characterisation

An integration of the information generated from all the previous steps is performed in this step. Uncertainty around any parameters can be incorporated to see the effects of these variations. This can point to deficiencies in data or current knowledge and direct future policy decisions and research efforts to address problem areas.

2.2 Model Parameters

The model concentrates on the human exposure from the combustion of SRM-derived MBM as this material contains the majority of the infectivity, hence the exposure associated with MBM (i.e. excluding the SRM portion) will be much less than the exposure assessed here. An overview of the infectivity pathways relevant to this study is presented in Figures 2.1 and 2.2.

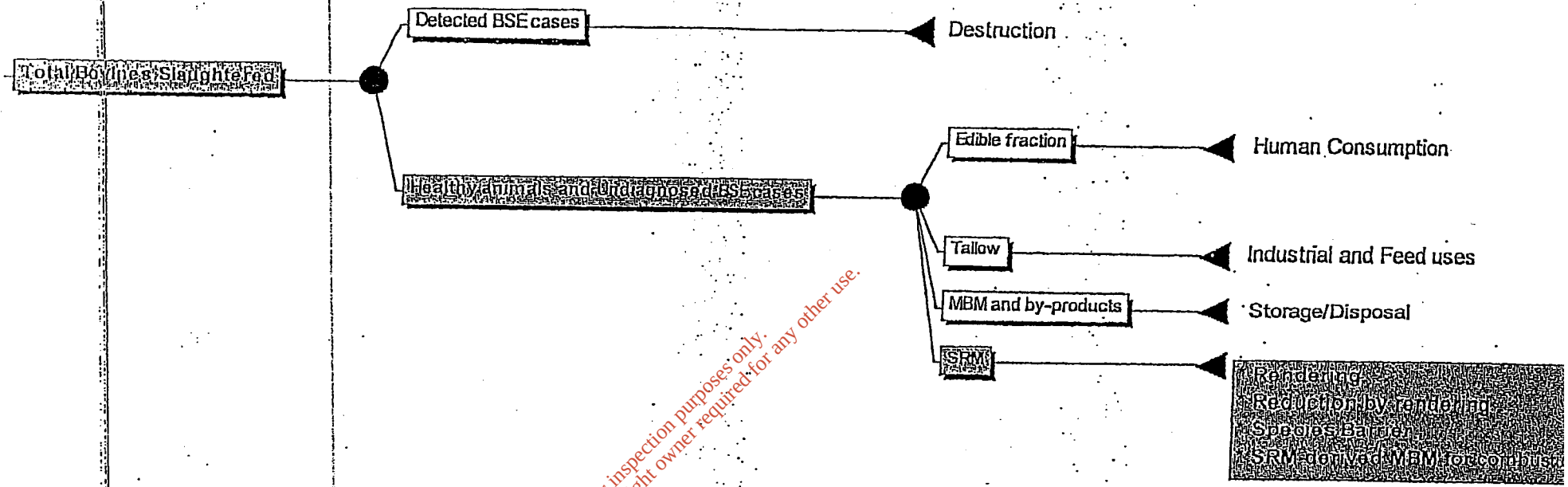

Figure 2.1 Infectivity pathways for SRM-derived MBM for the generation and processing stage

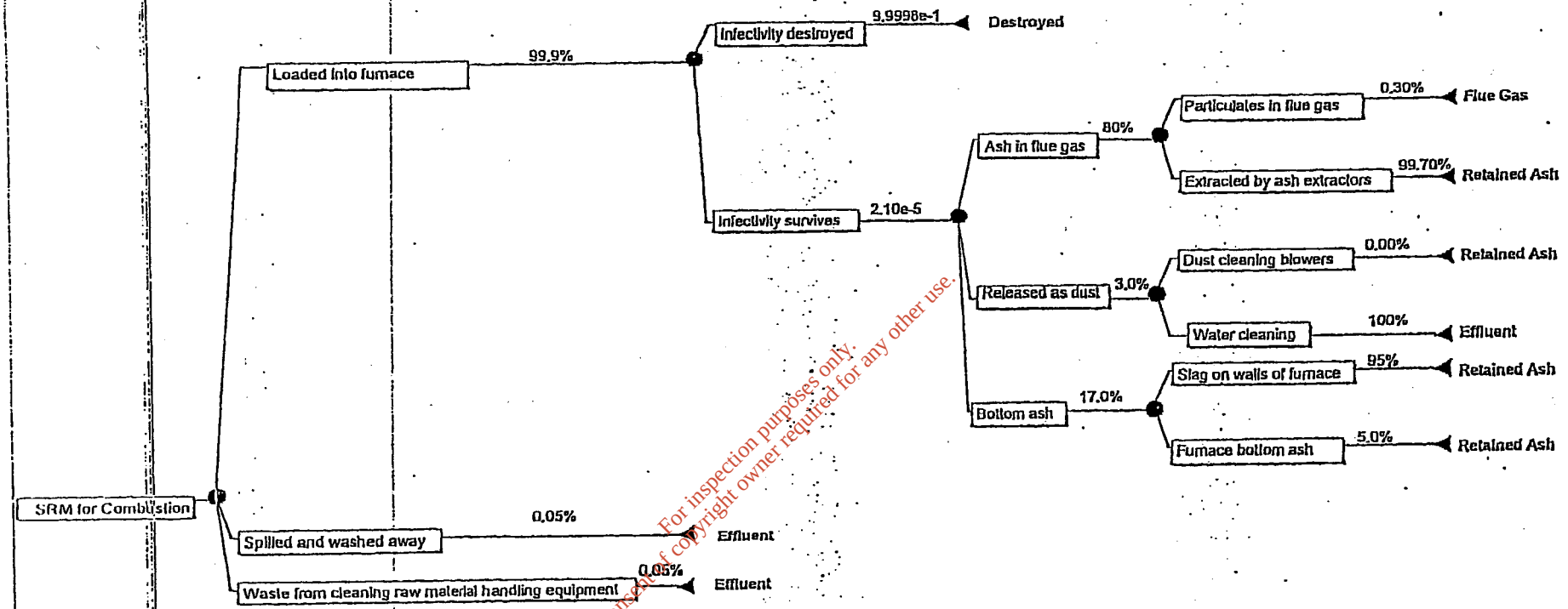

Figure 2.2: Infectivity pathways for the combustion of SRM derived MBM showing their endpoints (i.e. the combustion stage of the model)

The modelling process is split into two stages:

1) Generation and processing stage - This stage concentrates on the generation of the infected SRM taking into account infectivity of different tissues and subclinical BSE (Figure 2.1).

2) Combustion stage - This stage focuses on the basic operations of a combustion facility and looks at the various pathways, taking into account the loading of the SRM into the furnace, the effect of thermal treatments and the efficiency of fly ash extractors (Figure 2.2).

2.3 GENERATION AND PROCESSING STAGE

2.3.1 Infectivity of bovine tissue

The model considers the infectious dose associated with each part of the SRM-derived MBM as measured in ID_{50} units where the ID_{50} value represents the level of infectivity required to induce disease in 50% of exposed animals. The simulation utilised scenarios presented by the Scientific Steering Committee (SSC) (European Commission, 2000a), in which a probability distribution was applied to those tissues with the highest infective load as follows:

- Scenario 1: Infectivity titre in brain, spinal cord, trigeminal nerve ganglia and dorsal root ganglia. A lognormal distribution was used with the geometric mean of 10 cattle oral ID_{50} ($CoID_{50}$) per gramme of material and the 95th percentile at 100 $CoID_{50}$.

- Scenario 2: Infectivity titre in brain, spinal cord, trigeminal nerve ganglia and dorsal root ganglia. A lognormal distribution was used with the geometric mean of 100 $CoID_{50}$ per gramme of material and the 95th percentile at 1000 $CoID_{50}$.

In addition, a probability distribution was modified from data presented by DNV which stated that brain and spinal cord weight is on average 700 g per animal (DNV, 1997). The probability distribution for this figure was normal with a mean of 700 g and a 90th percentile range of 600 g to 800 g. For the purpose of this report the two tissues were split, brain 500 g and spinal cord 200 g, with the same distribution scaled accordingly.

Fixed values for the infectivity and weights of the other tissues used in this simulation were obtained from current scientific opinion data published by the SSC (European Commission, 2000a). An infectivity density for the processing stream was determined using the infective titre distributed in the SRM fraction of each animal which is processed in the SRM stream (this was found to be 37.05 kg of material, European Commission, 2000a). Infectivity density, as used in this paper, is used to define the concentration of infectivity per unit mass of material. There is a high infectivity density for the SRM material as there is a large infectivity associated with a relatively small amount of material i.e. there is little or no dilution effects in this stream.

2.3.2 Sub-clinical infectivity factor

Research suggests that infectivity is mainly confined to the end of the incubation period with a peak when clinical signs appear (Donnelly and Ferguson, 1999; Anderson *et al.*, 1996). This would suggest that sub-clinical animals have substantially less infectivity in their tissues than animals exhibiting clinical symptoms. Research presented by de Koeijer *et al.* (1998) suggested that a factor could be introduced to BSE risk assessments dealing with undiagnosed cases to reflect the lower infectiousness associated with earlier stages of the disease. As a 'worst case' assumption, this sub-clinical factor was set to a value of one i.e. that all infected animals carry the full clinical infective load.

2.3.3 Species Barrier

The species barrier is a term used to describe the natural resistance to transmission when a particular species is exposed to a Transmissible Degenerative Encephalopathy (TDE) of another species. With regard to species barrier, the working group of the SSC reports as follows (European Commission, 2000b):

The size of the species barrier for BSE-in-ruminants to BSE-in-humans is not known. In some risk assessments a barrier of the order of 1000 is assumed. However, the SSC questions these assumptions that it may be large. Until more scientific data is available, the SSC recommends that for risk assessments of human exposure to potentially BSE contaminated products, a species barrier of about 1 should be considered as a worst case scenario and that, in risk assessments the range from 1 to 10000 is considered.

The scenarios used by the SSC (European Commission, 2000a) for tissue infectivity included species barrier distributions that were used in this simulation and were as follows:

- Scenario 1: An adjusted triangular density distribution was used on an arithmetic scale with a mode value of 10^3 and within the range 10^0 to 10^4.
- Scenario 2: An adjusted triangular density distribution was used on an arithmetic scale with a mode value of 10^1 and within the range 10^0 to 10^4.

The triangular density distribution is used as a modelling tool where the range and the most likely value within that range can be estimated. The triangular distribution offers considerable flexibility in its shape while accounting for the uncertainty within the given range (Vose, 2000) and hence is used in this study to take account of the large uncertainly surrounding the true species barrier value.

2.3.4 Probability of infectious material in SRM

This parameter represents the probability that an undiagnosed BSE case enters the abattoir and that the resultant infective SRM is sent to the designated rendering plant with non-infective SRM from healthy animals. The probability of infected BSE material from an undiagnosed BSE case being present in the SRM stream was calculated from figures presented by Anderson *et al.* (1996) which estimated that there were 22,000 sub-clinically infected animals in the U.K. herd. The U.K. herd size in 1996 was approximately 12 million giving a sub-clinical incidence of 1 in 545. In this study it was considered that an undiagnosed incidence level approximately 10 times lower than the U.K. would be representative of the Irish situation i.e. 1 in 5450 (Taylor, Personal communication 1998).

The probability of a particular event occurring at least once is best described with the binomial distribution, the probability of an event occurring more than once during n repetitions is given by the formula:

$$P_{(\text{event one or more times})} = 1 - (1-P_{(\text{singular probability of event})})^n \qquad (1)$$

The binomial distribution has a mean nP and a variance $nP(1-P)$. The Poisson distribution arises as the limiting distribution of the binomial distribution where $nP \equiv$ mean as $n \to \infty$. When P is small it is found that the variance $\equiv nP \equiv$ mean. Thus

Poisson probabilities correspond to binomial probabilities when n is large (>20) and the mean is small (<5) (Chatfield, 1997). This is of practical importance as it is much easier to calculate poisson probabilities than corresponding binomial probabilities. This is especially true when n is large and binomial probability computation becomes difficult due to very large numbers of permutations.

As outlined above the probability of an animal being infected is 1/5450. The parameter n corresponds to the number of animals slaughtered in the year. An average for the years 1997-1999 is used here (1,890,000 slaughterings per year). In this study because n is very large (1,890,000) and P is small (0.0001834) a Poisson distribution may be used instead of the binomial distribution. The mean for the Poisson distribution (λ) can be calculated by the formula:

$$\lambda = n \times P_{(\text{single event occurring})} \qquad (2)$$

hence the mean, λ, in this case is 1,890,000 × 0.0001834 = 347. This is consistent with an estimate of 346 by Donnelly (2001). The probability of a sub-clinical case can therefore be calculated by dividing the number of subclinical cases (generated from the Poisson distribution) by total slaughtering, hence recalculation of the probability of a clinical case can be obtained with each iteration of the model.

2.3.5 Fraction of infectivity remaining after processing

Because of the proteinaceous nature of the Transmissible Spongiform Encephalopathy (TSE) agents they tend to remain with the cellular residues of meat and bone meal during extraction processes, rather than be extracted with the lipids of tallow (WHO, 1995). All MBM must, by law, be processing at 133°C for 20 minutes at 3 bars pressure - Commission Decision 96/449/EC (European Commission, 1996a). Using data from the SSC (European Commission, 2000a) the reduction in infectivity of SRM-derived MBM due to this thermal action was incorporated into the simulation as an adjusted triangular distribution with 10^3 fold reduction as the mode and with 10^6 fold as best and 0 fold as worst reduction (i.e. no effects of processing at all). Currently the SCC considers that the reduction of TSE infectivity as a result of treatment at 133°C for 20 minutes at 3 bars pressure is not less that 10^3 (European Commission, 1998a, 1998b, 1999) and may even be as high as $10^{3.8}$ (European Commission, 1999).

2.4 COMBUSTION STAGE

The assumptions regarding the combustion products and travel pathways for the ash are discussed here.

2.4.1 Material loaded into furnace

The SRM-derived MBM created in the generation and processing stage is now loaded into the furnace at the combustion facility. An allowance is made for the fact that some material may be spilled during the loading process (0.05%) and there may be material attached to the handling equipment. It is assumed that 0.05% of the material may be washed away into the effluent from the cleaning of the raw material handling equipment.

2.4.2 Fraction of infectivity remaining after combustion

It is assumed that a similar action to that used in a previous risk assessment (DNV, 1997) for thermal disposal of material with a possibility of BSE infectivity could be expected. The value used in this model was that given for incineration of MBM with the fraction of infectivity remaining after incineration as 2.10×10^{-05}. This figure was calculated from an assumed 10^6 reduction in infectivity from combustion in controlled conditions with a 10^2 fold reduction in infectivity in abnormal operating conditions. It was further assumed that there was a failure probability of 0.2% and so the overall reduction in infectivity figure was calculated thus:

$$(1/10^6 \times 0.998) + (1/10^2 \times 0.002) \cong 2.1 \times 10^{-5} \qquad (3)$$

DNV (1997) suggested that a normal probability distribution applied to this figure with a 95th percentile at 0.45.

2.4.3 Ash extraction

Ash particles in the combustion flue gas are carried through the furnace where 99.7% of the particulates are captured by fly ash extractors (DNV, 1997), the rest being emitted in the flue gas stream. The ash that remains is released as bottom ash while a small percentage is released as dust. It is assumed that this dust is extracted by cleaning water with the particulates being screened and returned to the bottom ash while a small fraction is emitted with the effluent. The pathways are detailed in Figure 2-2.

2.5 MBM Combustion Risk Model

The input parameters were combined onto a spreadsheet (Microsoft Excel 97) running the @Risk add-on package (Palisade Software, Newfield, USA) and the simulation was performed using Latin Hypercube sampling. Latin Hypercube is a stratified sampling technique where the random variable distributions are divided into equal probability intervals. A probability is randomly selected from within each interval for each basic event. Generally, Latin Hypercube sampling will require fewer samples than Monte Carlo sampling for similar accuracy (Vose, 2000). A summary of the input parameters used for the two scenarios in this exposure assessment is presented in Table 2.1 and Table 2.2 and the inputs used for the combustion facility are given in Table 2.3.

Table 2.1a: BSE input titres and tissue weights used in the exposure assessment model for the combustion of SRM derived MBM

Tissue Infectivity [CoID50]/g	Mean value	Probability distribution applied	Data source
Brain			
Scenario 1	10	Log-normal, log mean 2.30, standard deviation 1.4	European Commission, 2000a
Scenario 2	100	Log-normal, log mean 4.60, standard deviation 1.4	European Commission, 2000a
Spinal Cord			
Scenario 1	10	Log-normal, log mean 2.30, standard deviation 1.4	European Commission, 2000a
Scenario 2	100	Log-normal, log mean 4.60, standard deviation 1.4	European Commission, 2000a
Trigeminal ganglia			
Scenario 1	10	Log-normal, log mean 2.30, standard deviation 1.4	European Commission, 2000a
Scenario 2	100	Log-normal, log mean 4.60, standard deviation 1.4	European Commission, 2000a
Dorsal root ganglia			
Scenario 1	10	Log-normal, log mean 2.30, standard deviation 1.4	European Commission, 2000a
Scenario 2	100	Log-normal, log mean 4.60, standard deviation 1.4	European Commission, 2000a
Ileum	0.32	Fixed value	European Commission, 2000a
Spleen	0.032	Fixed value	European Commission, 2000a
Eyes, rest of head	0.032	Fixed value	European Commission, 2000a
Bone marrow	0.032	Fixed value	European Commission, 2000a
Bone adnexa	0.032	Fixed value	European Commission, 2000a
Other tissues	0	Fixed value	European Commission, 2000a

Table 2.1b: BSE input titres and tissue weights used in the exposure assessment model for the combustion of SRM derived MBM

Tissue Weights [g]	Mean value	Probability distribution applied	Data source
Brain	500	Normal, mean 500, standard deviation 43.42	DNV, 1997
Spinal Cord	200	Normal, mean 200, standard deviation 17.37	DNV, 1997
Trigeminal ganglia	20	Fixed value	European Commission, 2000a
Dorsal root ganglia	30	Fixed value	European Commission, 2000a
Ileum	800	Fixed value	European Commission, 2000a
Spleen	800	Fixed value	European Commission, 2000a
Eyes, rest of head	11600	Fixed value	European Commission, 2000a
Bone marrow	16800	Fixed value	European Commission, 2000a
Bone adnexa	6300	Fixed value	European Commission, 2000a
Other tissues	512950	Fixed value	European Commission, 2000a

Table 2.2: Other input parameters used in the exposure assessment model for the combustion of SRM derived MBM.

Parameters	Mean value	Probability distribution applied	Data source
Sub-clinical factor	1	Fixed value	Worse case value
Species barrier			
Scenario 1	1000	Triangular, minimum 1, maximum 10000	European Commission, 2000a, 2000b
Scenario 2	10	Triangular, minimum 1, maximum 10000	European Commission, 2000a, 2000b
Number of infected BSE cases in SRM Stream.	347	Poisson	See text for derivation
Fraction of infectivity remaining after processing	0.001	Triangular, minimum 0.0001, maximum 0.01	European Commission, 1999, 1998a,1998b
Fraction of infectivity remaining after combustion.	2.01E-05	Normal, 2.01×10^{-5}, standard deviation 3.5×10^{-6}	DNV, 1997

Table 2.3: Combustion facility parameters (Based on data from DNV,1997)

Parameter	Symbol	Mean value	Probability distribution applied
MBM loaded into furnace	MLF	95%	Calculation: MLF = 1-WCE-SWA.
Spilled and washed away	SWA	0.05%	Lognormal, geometric mean 0.0005, standard deviation 1.4
Waste from cleaning raw material handling equipment	WCE	0.05%	Fixed value
Infectivity Remaining	IR	2.1×10^{-5}%	Normal, mean 2.1×10^{-5}, standard deviation 3.5×10^{-6}
Infectivity Destroyed	ID	99.997%	Calculation: ID = 1-IR
Ash in flue gas	AFG	80%	Fixed value
Released as dust	RD	0.03%	Fixed value
Bottom Ash	BA	17%	Calculation: BA = 1-RD-AFG
Ash captured by extractors	ACP	99.7%	Normal, mean 99.7, standard deviation 0.04
Released with flue gas *via* stack	FG	0.03%	Calculation: FG = 1-ACP
Captured by dust cleaning blowers	DB	0%	Fixed value
Captured by water cleaning	WC	100%	Calculation: WC = 1-DB
Slagging factor	SF	95%	Fixed value
Furnace Bottom ash	FBA	5%	Calculation: FBA = 1-SF
Particulates in effluent	PE	0.03%	Fixed value

The simulation was performed using the parameters presented above and each run was comprised of 100,000 iterations. The results are given below (Figures 2.5, 2.7) in the form of a probability distribution of human exposure (measured in human oral ID_{50} units/year). The rank correlation is also provided (Figures 2.6, 2.8) which ranks the various parameters in terms of their influence on the overall result. The relationship between key inputs can be seen in Figures 2.9, 2.10, 2.11 and 2.12. Three end points were looked at in the quantitative exposure assessment, i.e. the societal exposure from particulates emitted through the flue gas, effluent and ash. It is assumed that exposure is proportional to risk, hence any measures taken to decrease exposure will also decrease risk.

2.6 Results and Discussion

The results are presented for simulations in two situations reflecting the infectivity risk in the SRM-derived MBM stream for two scenarios defined by the SSC. The risks calculated are in terms of societal exposure (i.e. the number of individuals in the exposed population is not taken into account). The risks are presented in terms of human oral ID_{50} titre that the exposed population may be subjected to as a result of the combustion of SRM-derived MBM in Ireland. Societal exposure represents the total exposure to a community of people. Assuming a linear dose-response curve (i.e. 1 ID_{50} has a 100% chance of infecting 1 exposed susceptible individual, a very pessimistic assumption) societal exposure can be used as representative of societal risks. The individual risk can be calculated by the following equation: Individual Risk = Societal Risk/population exposed). The mean calculated societal exposure from scenario 1 was 7.57×10^{-6} ID_{50} per year (5^{th} percentile 7.32×10^{-7} ID_{50}/year, 95^{th} percentile 1.39×10^{-4} ID_{50}/year) while the mean exposure associated with scenario 2 was an order of magnitude greater (8.38×10^{-5} ID_{50}/year with 5^{th} percentile 5.76×10^{-6} ID_{50}/year and 95^{th} percentile 2.41×10^{-3} ID_{50}/year). Explaining the significance of the results, using the mean value of 7.57×10^{-6} ID_{50}/year from scenario 1 as an example. Assuming a linear dose-response curve, 7.57×10^{-6} ID_{50}/year will result in to 7.57×10^{-6} human vCJD infections per year from all sources as a result of the combustion of SRM-derived MBM. In order to receive this exposure, and hence risk of contracting vCJD, 1 susceptible individual would have to ingest all fly ash, bottom ash and effluent produced by the plant during a time period of one year while it was burning SRM-derived MBM from 1,890,000 animals (an impossible scenario). The increase in risk in scenario 2 is explained by the increase in the infectivity of the brain and

spinal cord in this scenario. The increase in infectivity is illustrated in the change in the infectivity density of the SRM material as shown in the difference between Figures 2.3 and 2.4. The mean infectivity density for scenario 1 was -9.99 log ID_{50} units per kg of SRM material while the infectivity density for scenario 2 was greater (-8.94 log ID_{50} units per kg of SRM material). The analysis shows that the greatest exposure posed to humans is from infectivity spilled into the effluent stream. In both scenarios the exposure risk from effluent was greater than any exposure risk from the resulting ash or infectivity emitted *via* the flue gas (Figures 2.5 and 2.7). It can be seen from the model that the societal risks presented to human health from the combustion of SRM are extremely small. It is accepted that Creutzfeldt-Jakob Disease (CJD) has a sporadic occurrence of approximately 1 in 10^6 (Zivkovic *et al.*, 2000). Taking the human population of Ireland as 3.8 million, one would expect over 3 sporadic cases of CJD per year. This is compares with an average of 2.5 sporadic cases of CJD per year between 1997 and 2000 in Ireland reported by Eurocjd (2002). The societal risks calculated from Scenario 1 and 2 are significantly less than this background societal risk of sporadic CJD (Table 2.4). Hence the risk from SRM combustion is negligible in comparison to the sporadic occurrence of CJD. Actual individual exposure and hence risk would be much less than the societal exposure values calculated here since not all materials leaving the flue or combustion chamber would result in human exposure.

Table 2.4: Summary of results from risk assessment including uncertainty assessment.

Scenario	Infectivity of brain & spinal cord CoID50/g (mean value)	Species barrier (mean value)	Predicted exposure (mean societal ID_{50}/year)	5th percentile	Median	95th percentile
Scenario 1	10	1000	7.57×10^{-6}	7.32×10^{-7}	6.42×10^{-5}	1.39×10^{-4}
Scenario 2	100	10	8.38×10^{-5}	5.76×10^{-6}	6.89×10^{-5}	2.41×10^{-3}
Scenario 3	10	1	2.18×10^{-2}	2.99×10^{-3}	1.80×10^{-2}	0.301
Scenario 4	100	1	0.188	2.20×10^{-2}	0.162	2.79

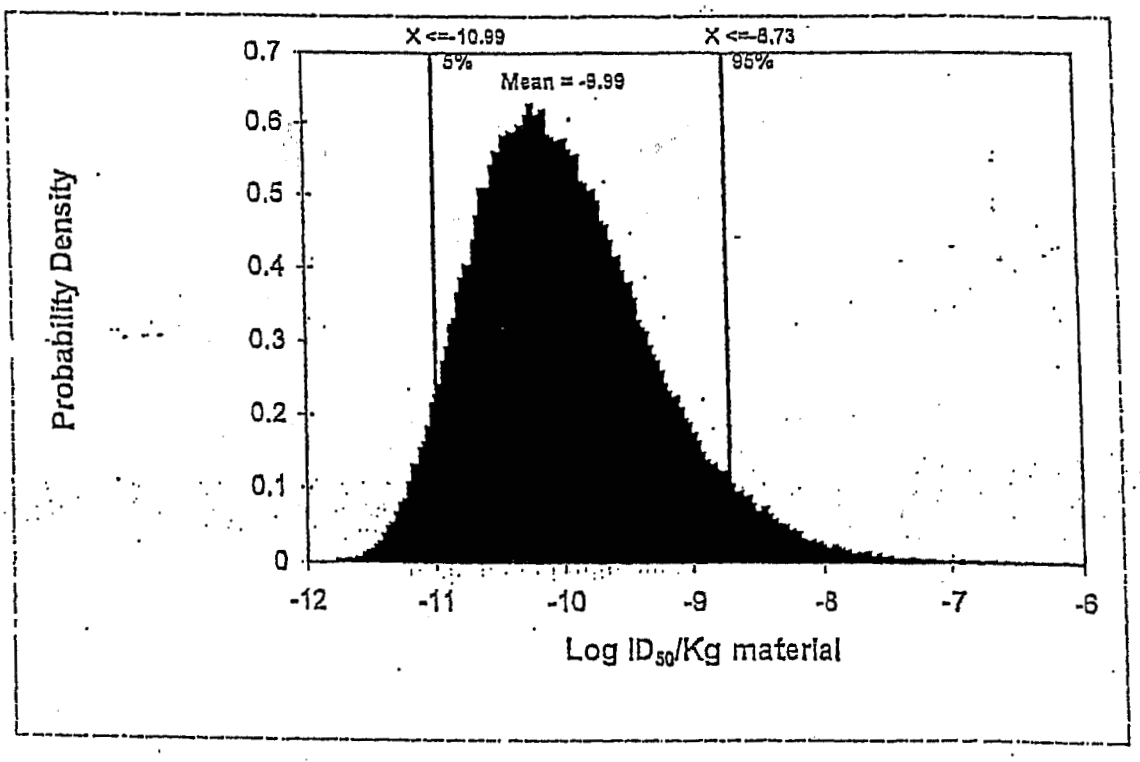

Figure 2.3: Infectivity density of SRM derived MBM for scenario 1 in the model.

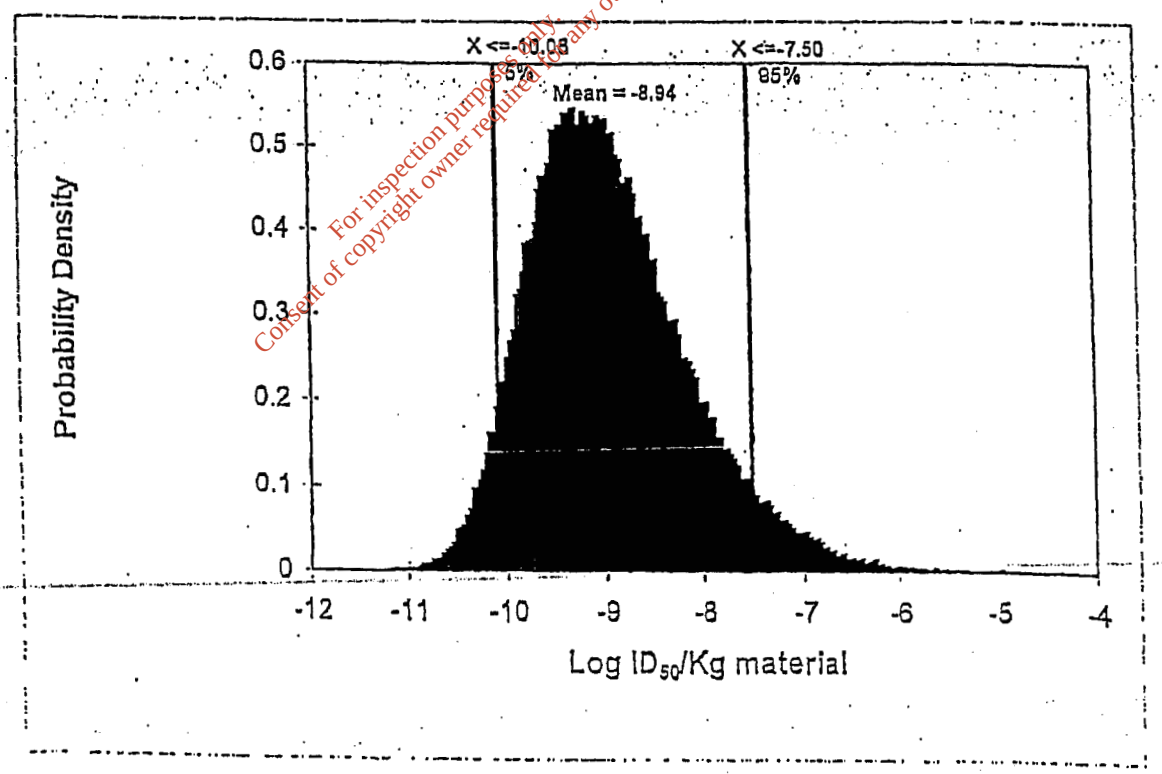

Figure 2.4: Infectivity density of SRM derived MBM for scenario 2 in the model.

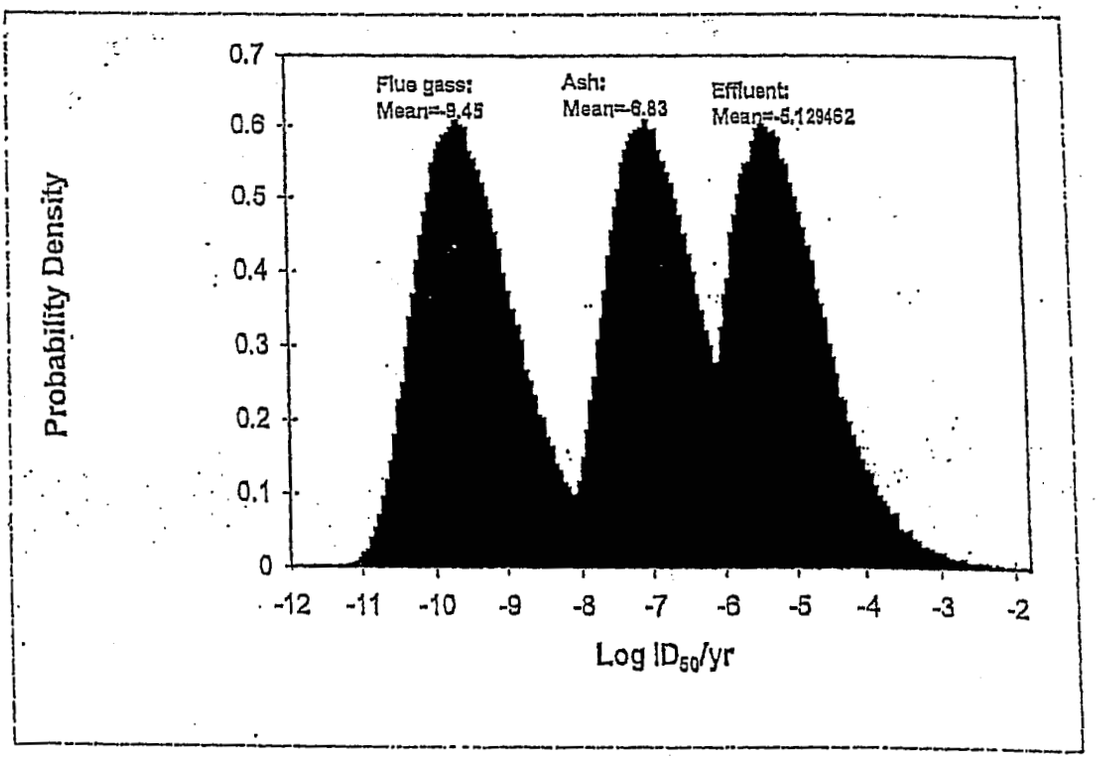

Figure 2.5: Scenario 1: Distributions for infectivity emitted from flue gas, ash and effluent sources resulting from the combustion of SRM derived MBM.

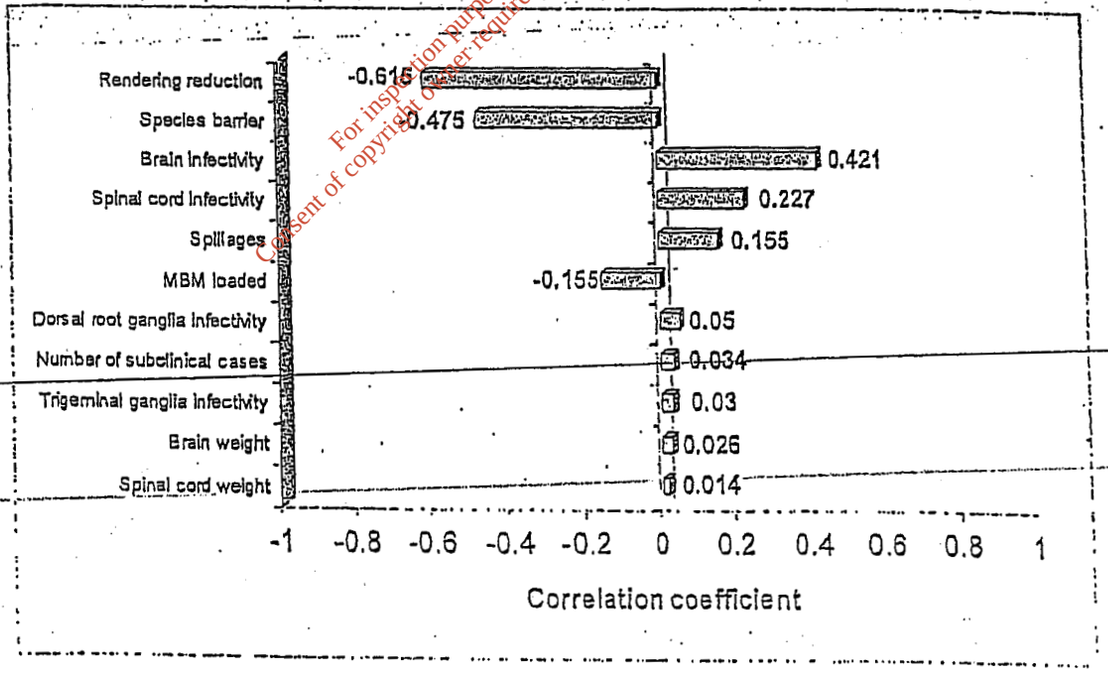

Figure 2.6: Scenario 1: Sensitivity analysis of main input parameters in the model.

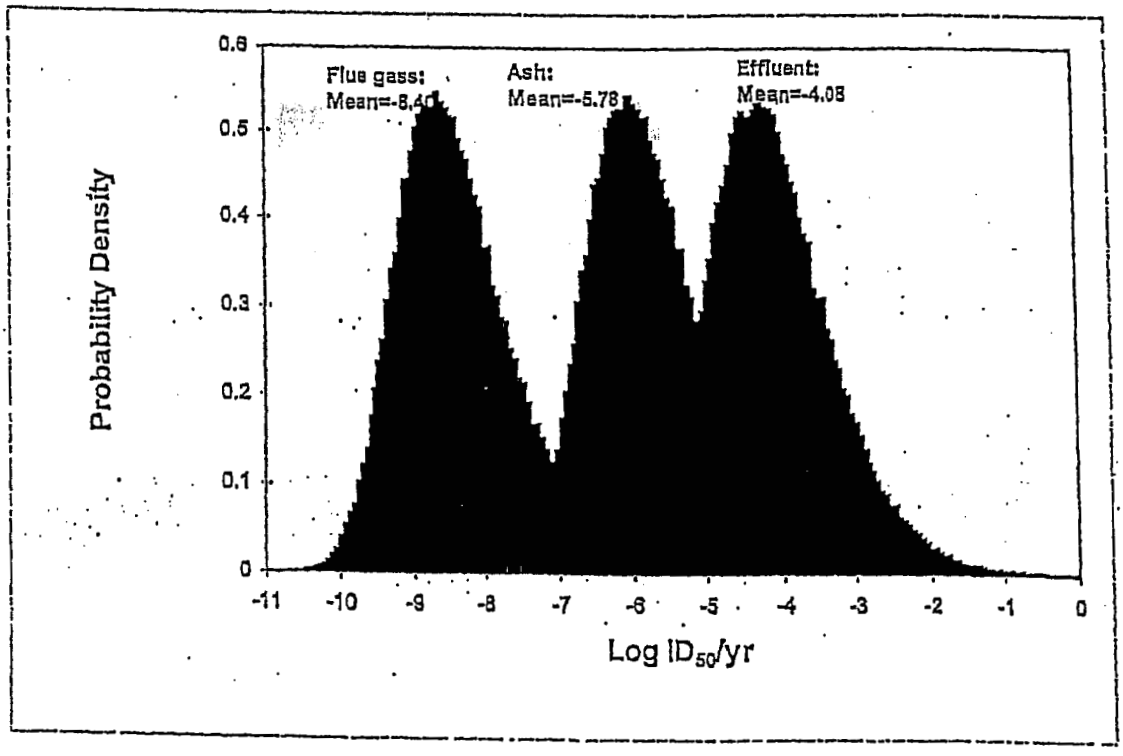

Figure 2.7: Scenario 2, Distributions for infectivity emitted from flue gas, ash and effluent sources resulting from the combustion of SRM derived MBM.

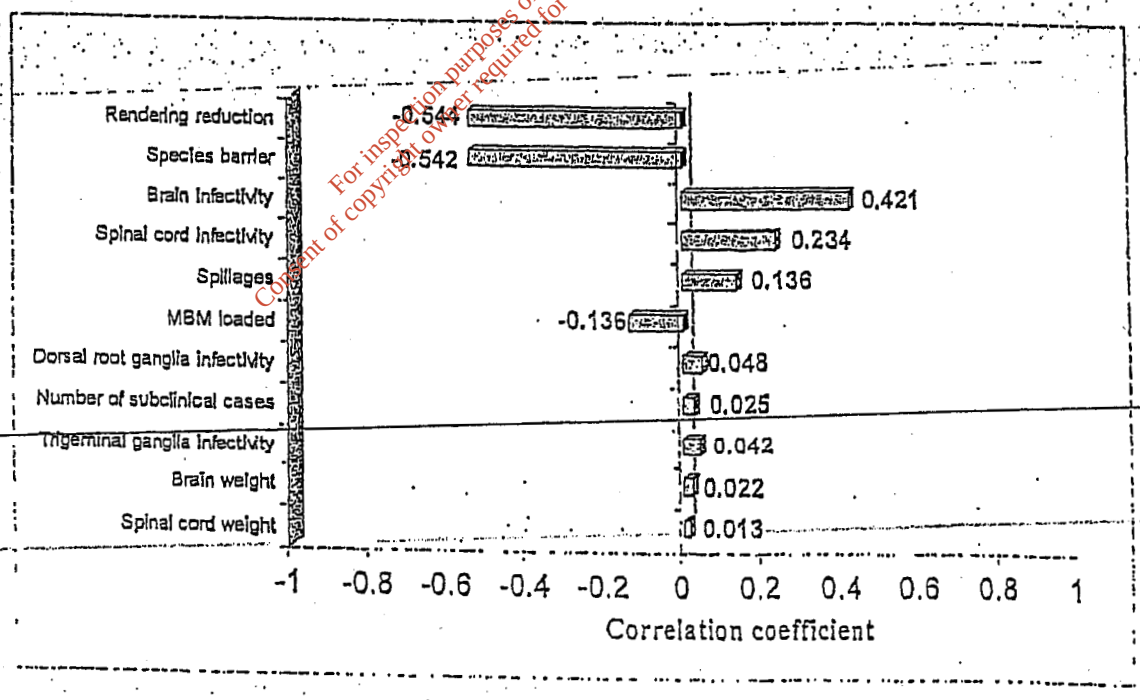

Figure 2.8: Scenario 2, Sensitivity analysis of main input parameters in the model.

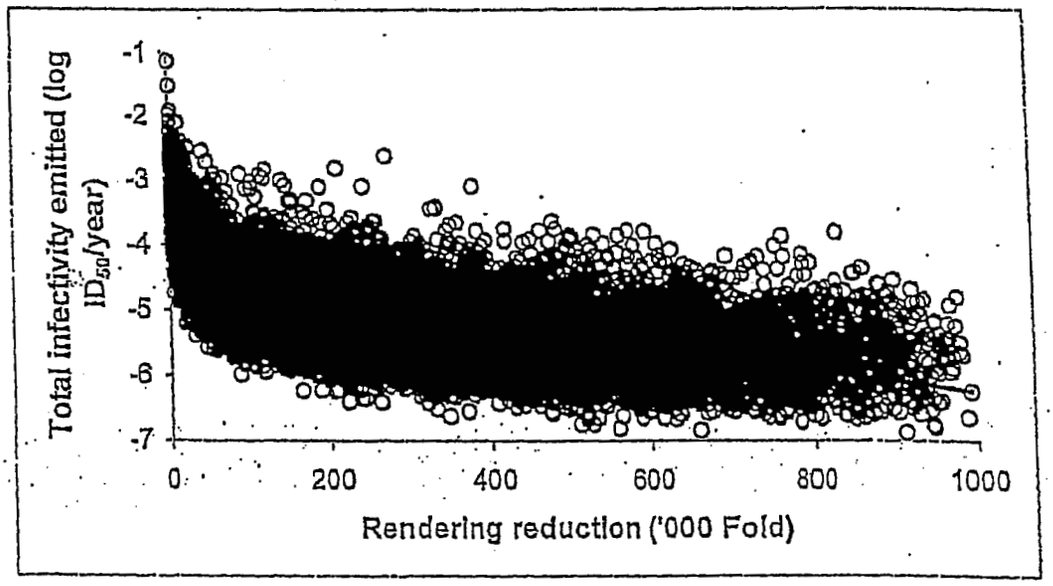

Figure 2.9: Iterations showing the correlation between rendering reduction and total infectivity emitted. The best-fit line is shown.

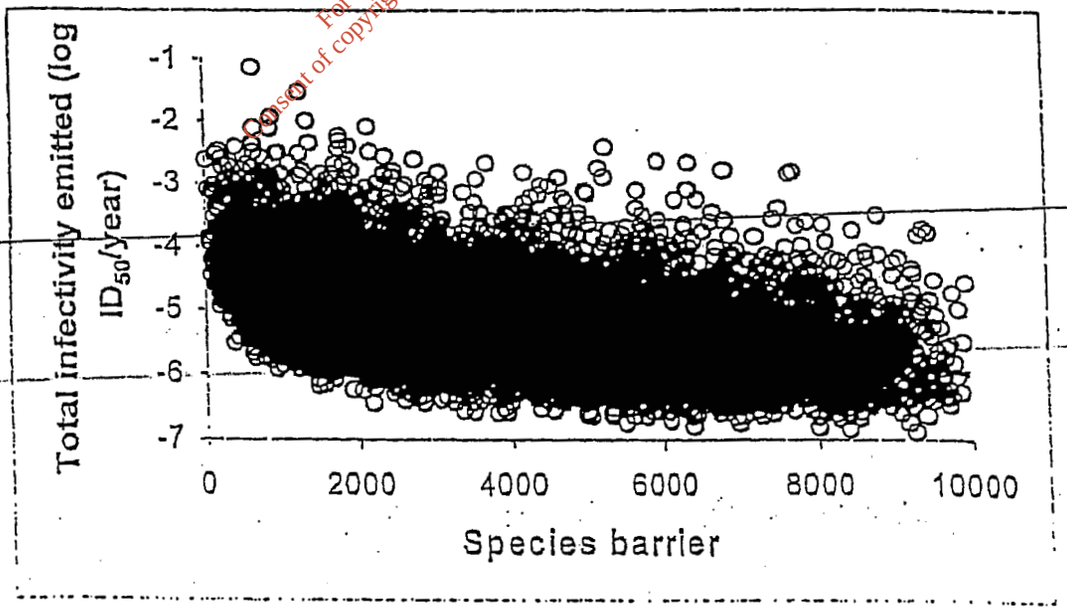

Figure 2.10: Iterations showing the correlation between the species barrier and total infectivity emitted. The best-fit line is shown.

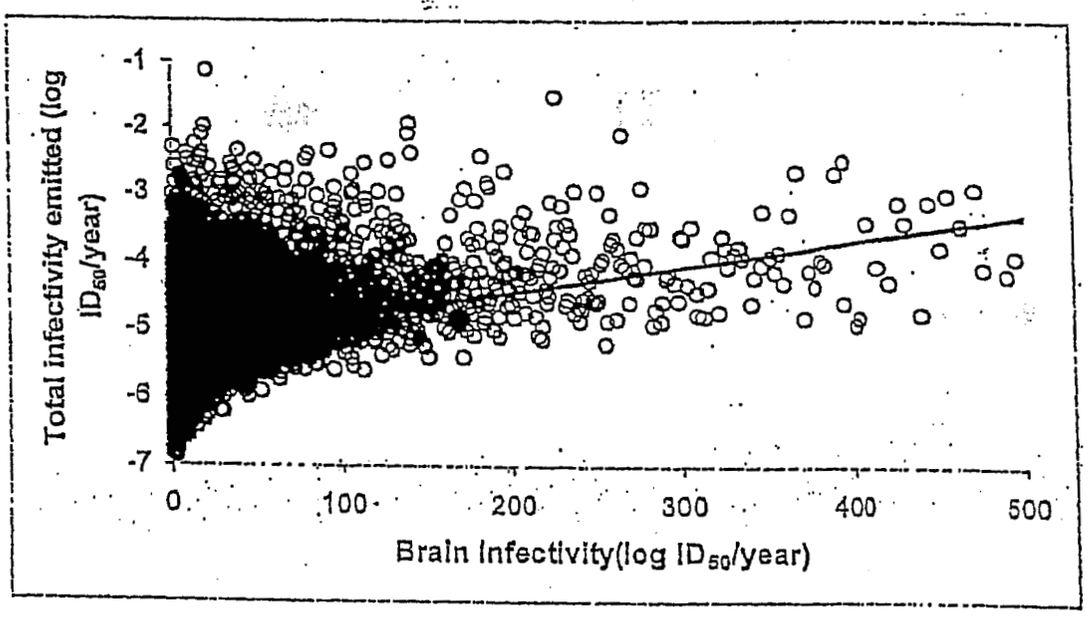

Figure 2.11: Iterations showing the correlation between brain infectivity and total infectivity emitted. The best-fit line is shown.

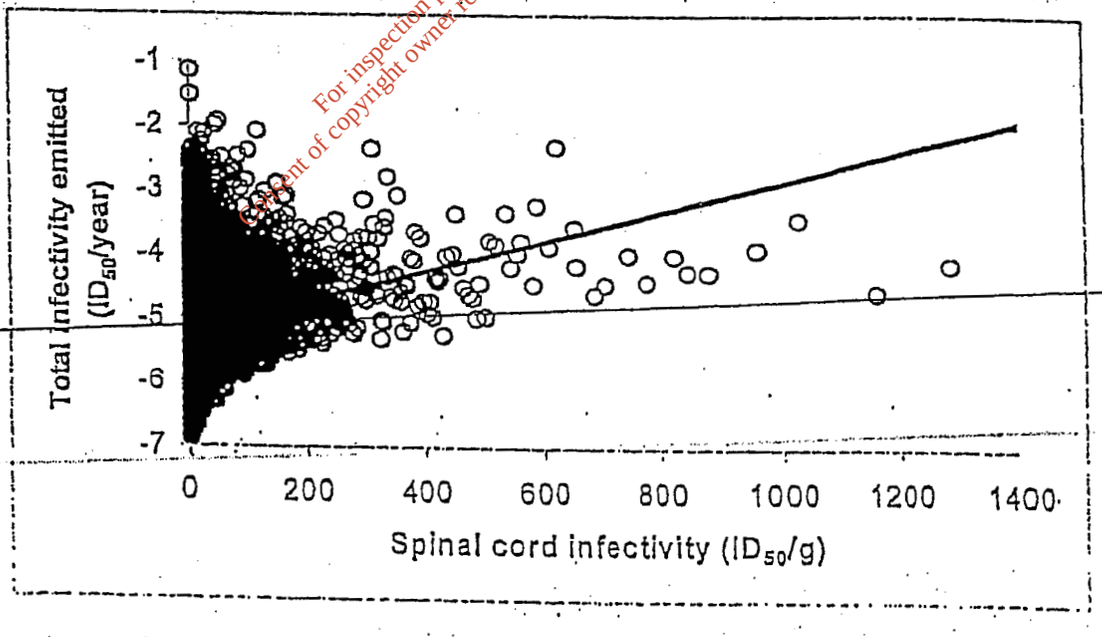

Figure 2.12: Iterations showing the correlation between spinal cord infectivity and total infectivity emitted. The best-fit line is shown.

A sensitivity analysis (measured by the rank correlation) performed for each simulation (Figures 2.6, 2.8) shows that the rendering reduction and species barrier are the most important parameters in terms of human exposure from BSE due to combustion of SRM-derived MBM. The infectivity of the brain and spinal cord were also found to have quite large effects on the overall exposure to humans. The species barrier is an epidemiological factor and although subject of discussion and research, it is effectively out of the control of mankind. The sum of the combined risks from all sources (ash, effluent and flue gas) was combined and summarised in Table 2.4. It should be noted that these calculations represent the societal exposure risk i.e. the potential exposure to the total population, hence the individual exposure and therefore risk to any one individual would be much less than the values calculated here. In the case of materials exiting the flue and effluent streams, there would be dispersion and possible attenuation, so that only a proportion of material released would result in human exposure (i.e. reach a ground level receptor). The Health and Safety Executive (HSE) guideline for an "acceptable risk" for an *individual* person is 1 in 10^6 per year (DNV 1997). In order to exceed this guideline an individual would have to be exposed to the total infectivity emitted from all sources (flue, ash and effluent) resulting from the combustion of SRM-derived MBM from 1,890,000 animals over a period of one year, an unlikely scenario given the volumes of materials involved.

As the species barrier had a large influence on the risk calculations (as determined by rank correlation, Figures 2.6, 2.8) and is outside the control of human influence (unlike parameters such as rendering temperatures, control of spillage's e.t.c), an exposure assessment was also carried out using the parameters as in scenarios 1 and 2 but with the species barrier fixed at a value of 1, i.e. no species barrier - which is probably an unrealistic worst-case scenario. The resulting mean societal exposures were still small with a societal exposure of 2.18×10^{-2} ID_{50}/year for scenario 3 and 0.188 ID_{50}/year for scenario 4 (Table 2.4).

Two factors affecting infectivity which mankind has control over are the processing and combustion conditions. From the sensitivity analysis (Figures 2.6, 2.8) it can be seen that the SRM processing has the greater effect on the final risk calculation. This highlights the importance of adequate processing procedures in minimising exposure and hence risks from BSE.

2.7 Conclusions

From this analysis of the exposure associated with the combustion of SRM-derived MBM it can be concluded that the societal exposure and hence risks are negligibly small. An analysis of the important risk factors is detailed through a sensitivity analysis. The species barrier has a large effect on risk calculations and is outside the control of mankind, but even when the species barrier is removed the exposure calculations are still small (Table 2.4). It should be noted that these exposure calculations represent a societal exposure, the individual exposure would be much smaller as a result of dispersion and possible attenuation *via* various media such as ash and liquid effluent. The quantitative exposure assessment developed provides a means to analyse the relationship between exposure and factors which might be used to mitigate risk. A risk manager is likely to be interested in the sensitivity analysis. The fact that the processing procedures appear to have more of an effect on the final exposure calculations than combustion procedures highlights the importance of having good processing procedures to minimise exposure and hence human risks. Possible interventions can be concluded by identifying the controllable variables which have an important contribution to human exposure. These procedures may include the management of the thermal treatment of SRM-derived MBM. It can be concluded that human efforts for risk mitigation should be focused on reducing the exposure through minimising the fraction of infectivity remaining after processing and minimising untreated spillages.

With the OTM cattle cull scheme now in place in Ireland the production of SRM-derived MBM is set to continue. This research into the exposure risks associated with the combustion of SRM leads the way for further research into the uses of SRM/MBM while minimising the risks. With continuous improvements in available information about the epidemiology and nature of the BSE agent, modelling represents a very useful decision-support tool as it utilises the most current knowledge and allows a number of different scenarios to be tested. The model allows consideration and allocation of resources to potential risk reduction strategies that may be immediately feasible, while at the same time identifying priorities for focused longer-term research to better understand and intervene at critical stages in the process. The results presented here indicate, based on current knowledge about BSE, that with adequate management of both the rendering process to ensure correct heat treatments and combustion temperatures the use of SRM-derived MBM in a combustion facility would have negligible implications on human health.

2.7 CONCLUSIONS

From this analysis of the exposure associated with the combustion of SRM-derived MBM it can be concluded that the societal exposure and hence risks are negligibly small. An analysis of the important risk factors is detailed through a sensitivity analysis. The species barrier has a large effect on risk calculations and is outside the control of mankind, but even when the species barrier is removed the exposure calculations are still small (Table 2.4). It should be noted that these exposure calculations represent a societal exposure, the individual exposure would be much smaller as a result of dispersion and possible attenuation *via* various media such as ash and liquid effluent. The quantitative exposure assessment developed provides a means to analyse the relationship between exposure and factors which might be used to mitigate risk. A risk manager is likely to be interested in the sensitivity analysis. The fact that the processing procedures appear to have more of an effect on the final exposure calculations than combustion procedures highlights the importance of having good processing procedures to minimise exposure and hence human risks. Possible interventions can be concluded by identifying the controllable variables which have an important contribution to human exposure. These procedures may include the management of the thermal treatment of SRM-derived MBM. It can be concluded that human efforts for risk mitigation should be focused on reducing the exposure through minimising the fraction of infectivity remaining after processing and minimising untreated spillages.

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SRM-derived MBM in a combustion facility would have negligible implications on human health.

The greatest individual risk (i.e. the risk associated with the most exposed person) of infectivity was from ingested water with 7.68e-12 human oral ID50 units per year. With the worst case scenario that there is no safe threshold and that risk is directly proportional to dose, the risk of a person being infected is less than 1 in 100 million. The Health and Safety Executive (HSE) guideline for an "acceptable risk" is 1 in 1 million per year. A plot of the mean total infectivity ingested by the most exposed person is given in Figure 2.13. It can be seen total ingestion is controlled by two pathways, consumption of contaminated water and particles emitted from the flue gas within 1000m of the stack. The graph is presented on a logarithmic y-scale. It can be concluded that there is negligible risk associated with each of the pathways.

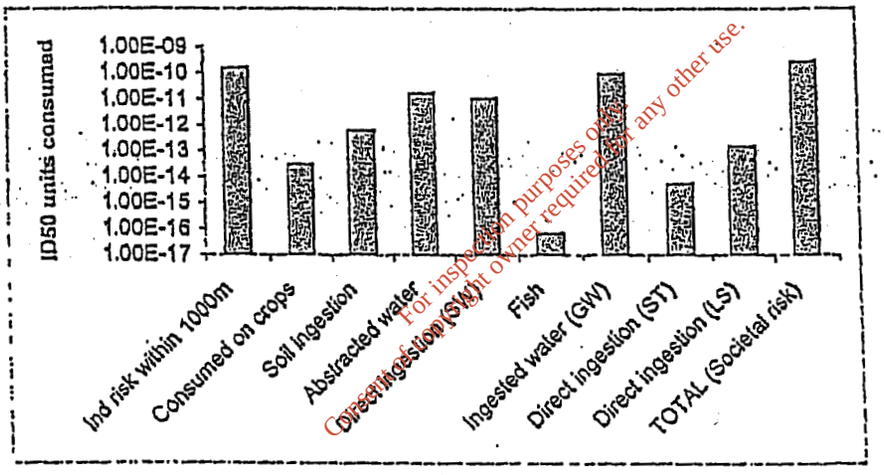


Figure 2.13: Mean individual risk (ID50 units)
(SW = surface water, GW = ground water, ST = sewage treatment, LS = landspreading)

The maximum sources of infectivity are from particles deposited from plume gases (maximum 2.34e-9 ID50 units) and from ingested ground water (1.12e-9 ID50 units). Particles from exhaust gasses have the potential to travel long distances, with the greatest risk from the potential inhalation of these particles. The maximum individual risk to an individual as a result of burning MBM/SRM is given in Figure 2.14 (logarithmic y-scale).

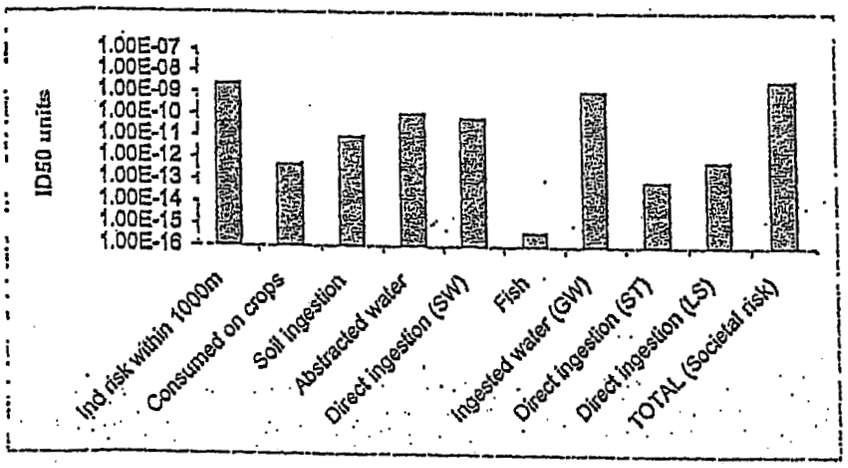


Figure 2.14: Maximum individual risk (Human oral ID50)
(SW = surface water, GW = ground water, ST = sewage treatment, LS = landspreading)

The results from the Monte Carlo simulation are given in Figure 2.15. The vertical bars represent the relative frequency of results from 5000 trials. The plot range is from 1.85e-12 to 6.81e-10. The 95-percentile range is between 1.84e-12 and 4.87e-10 with the median value 1.33e-10.

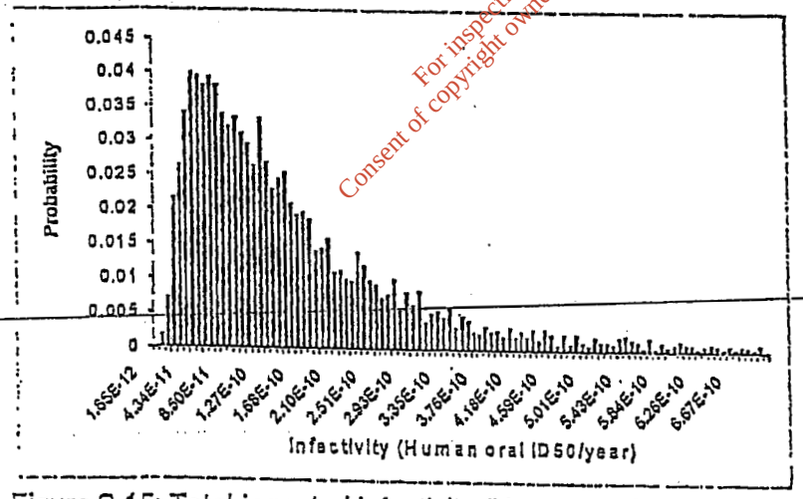
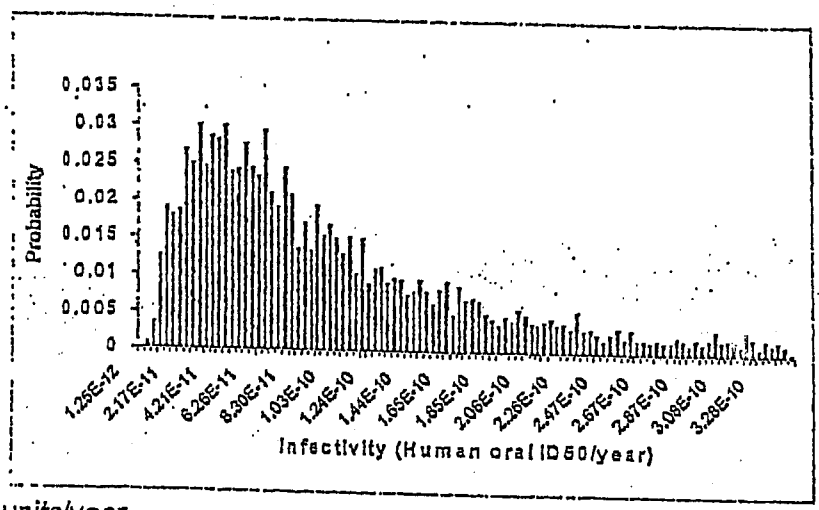


Figure 2.15: Total ingested infectivity (Human oral ID50/year)

The individual risks from the particles emitted from the stack represent one of the greatest risks of infection (Figure 2.16). The risk itself is still negligible but it highlights the need to minimise the emission of particulates by using filters cyclones and

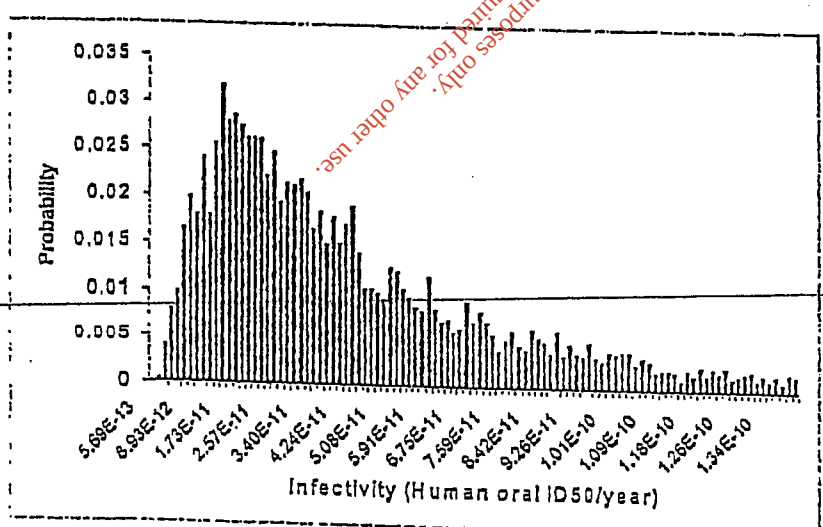
electrostatic receptors. The scale is between 1.25e-12 to 2.34e-9. The 95 percentile range is between 1.25e-12 and 3.42e-10 ID50 units/year with the median 1.54e-11 ID50



units/year.

Figure 2.16: Individual risk within 1000m of stack (Human oral ID50/year)

A plot (Figure 2.17) of risk from ingested water resulting from the Monte-Carlo analysis's given. The scale is from 1.24e-12 to 1.37e-10. The 95-percentile range is from 5.69e-13



to 1.39e-10 ID50 units/year with the median 3.95e-11 ID50 units/year.

Figure 2.17: Probability distribution for ingested water (Human oral ID50/year)

It is concluded that burning MBM (including SRM) from slaughtered animals will result in negligible risks to humans through environmental pathways. If MBM excluding the SRM portion was to be combusted the risks would be even smaller. The uncertainty analysis gives a distribution for each of the pathways at which individuals may be exposed. The societal risk i.e. the total human ingestion of infectivity from burning MBM/SRM is estimated to be 1.52e-11 human oral ID50 units. The risk of any human infections is extremely small. Sporadic CJD occurs at a rate of 1 in a million, the societal risk of being infected by BSE as a result of burning MBM is negligible compared to this. In terms of both individual and societal risks the risks from burning MBM/SRM have been shown to be negligible. The combustion of MBM excluding the SRM portion would be several orders of magnitude lower that the values presented here and hence the overall societal risk would also be much smaller. An uncertainty analysis has shown that the risks may give several order of magnitude greater or smaller than the mean values presented here, but even the most pessimistic inputs into the model give very negligible risks.

It is important to note the risks are dominated by pathways affected by spillages of infected material in addition to particles escaping with the plume gases. This places emphasis on the importance of good housekeeping and material handling practices in combusting the material.

3 REFERENCES

Anderson, R.M., C.A. Donnelly, N.M. Ferguson, , M.E.J. Woolhouse, C.J. Watt, H.J. Udy, S. MaWhinney, S.P. Dunstan, T.R.E. Southwood, J.W. Wilesmith, J.B.M. Ryan, L.J. Hoinville, J.E. Hillerton, A.R. Austin, and G.A.H. Wells. 1996. Transmission dynamics and epidemiology of BSE in British cattle. *Nature* 382: 779-382.

Asher, D. M., Pomeroy, K.L., Murphy, L., Gibbs, C.J. and Gajdusek, D.C., 1987, Attempts to disinfect surfaces contaminated with etiological agents of the spongiform encephalopathies. *Abstracts of the. VII International Congress of Virology*, Edmonton, 9-14 August, p. 147.

Balter, M., 2001, Intriguing clues to a scrapie-mad cow link, *Science*, 292, 827-828.

Bradley, R., 2001, Will scrapie in sheep in Great Britain disappear?, *Trends in Microbiology*, 9, 6, 260-261.

Brewer, M.S., 1999, Current status of Bovine Spongiform Encephalopathy - a review, *Journal of Muscle Foods*, 10, 97-117.

Brown, P, and Gajdusek D.C., 1991, Survival of scrapie virus after 3 years internment, *The Lancet*, 337, 269-270.

Brown, P., Liberski, P.P., Wolff, A. and Gajdusek, D.C., 1990, Resistance of scrapie infectivity to steam autoclaving after formaldehyde fixation and limited survival after ashing at 360°C: practical and theoretical implications. *Journal of Infectious Diseases* 161,467-472.

Brown, P., Rohwer, R.G. and Gajdusek, D.C., 1986, Newer data on the inactivation of scrapie virus or Creutzfeldt-Jakob disease virus in brain tissue, *Journal of Infectious Diseases*, 153, 1145-1148.

Bruce, M.E., R.G. Will, J.W. Ironside, I. McConnell, D. Drummond, A. Suttie, L. McCardle, A. Chree, J. Hope, C. Birkett, S.N. Cousens, H. Fraser, and C.J. Bostock. 1997. Transmission to mice indicate that 'new variant' CJD is caused by the BSE agent. *Nature* 389: 498-501.

Caprella, T., 2001, Aussies Claim Invention of BSE-Free Rendering Process, http://www.rendermagazine.com/February2001/Newsline.html.

Central Statistics Office, 2002, Principal Statistics – Agriculture, http://www.cso.ie/principalstats/pristat6.html.

Chatfield, C. 1997. *Statistics for technology.* London, 3rd edition: Chapman & Hall.

Cummins E., P. Grace, D.J. Fry, K. McDonnell, and S. Ward. 2001. Predictive modelling and risk assessment of BSE: A review. *Journal of Risk Research* 4 (3): 251-274.

Cummins, E., 2001, Personal Communication, Department of Agricultural and Food Engineering, University College Dublin, Ireland.

Cummins, E., Colgan, S., McDonnell K. and Ward, S., 2001, Human risks from the combustion of SRM-derived tallow in Ireland, *Risk*, Under Review.

DAF, 1998, Personal Communication, Department of Agriculture, Food and Rural Development, Dublin, Ireland.

de Koeijer, A.A., J.A.P. Heesterbeek, R.C. Obertheur, B.E.C. Schreuder, and M.C.M. de Jong. 1998. BSE Risk assessment – Calculation of the reproduction ratio for BSE infection among cattle. *Internal report* – ID-DLO, Lelystad, Netherlands.

Department of Agriculture and Food, 1999, Report of the Beef Task Force, Government of Ireland, ISBN 0-7076-6219-2.

Department of Agriculture and Food, 2002, BSE in Ireland – Information Note, http://www.irlgov.ie/daf/Areasofl/BSE/BSE.htm.

Department of Health (UK), 2001, Monthly Creutzfeldt-Jakob Disease Statistics, http://www.doh.gov.uk/cjd/stats/jan01.htm.

Dickinson, A.G. and Taylor. 1988. Options for the control of scrapie in sheep and its counterpart in cattle. *Proceedings of the Third World congress on sheep and beef. cattle breeding*. 1, Paris, Versailles; INRA Publication, 553-564.

Dickinson, A.G., 1976, Scrapie in sheep and goats. In: Slow Virus Diseases of Animals and Man (ed. Kimberlin, R.H.) pp.209-241. Amsterdam: North Holland.

DNV. 1997. Risks from BSE via Environmental pathways. *Report to Environment Agency*. Ref C7243, UK. June 1997. Det Norske Veritas Technica.

Donnelly, C.A. 2001. BSE in the Republic of Ireland (http://www.bsereview.org.uk/data/bse_in_Ireland.htm). Head of statistics unit, Wellcome trust centre for the epidemiology of infectious disease, University of Oxford, UK. Accessed 20/2/2002.

Donnelly, C.A. and N.M. Ferguson. 1999. *Statistical aspects of BSE and vCJD. Models for Epidemics*. London. ed. D.R. Cox, V. Isham, N. Kelding, T. Louis, N. Reid and H.Tong. Chapman and Hall/CRC.

Ebringer, A., Pirt, J., Wilson, C., Cunningham, P., Thorpe, C. and Ettelaie, C., 1997, Bovine Spongiform Encephalopathy: Is It An Autoimmune Disease Due to Bacteria Showing Molecular Mimicry With Brain Antigens?, *Environmental Health Perspectives*, 105, 1172-1174.

Eurocjd. 2002. The European and Allied countries collaborative study group of CJD plus the extended European collaborative study group of CJD. www.eurocjd.ed.ac.uk. Accessed 20 Feb. 2002.

European Commission, 1994, Commission Decision 94/381/EC of 27 July 1994 concerning certain protection measures with regard to bovine spongiform encephalopathy and the feeding of mammalian derived protein, *Official Journal of the European Communities* L 172.

European Commission, 1996a, Commission Decision 96/449/EC of 18 July 1996, on the approval of alternative heat treatment systems for processing animal waste with a view to the inactivation of spongiform encephalopathy agents, *Official Journal of the European Communities*, L184.

European Commission, 1996b, Commission Regulation (EC) No 716/96 of 19 April 1996 adopting exceptional support measures for the beef market in the United Kingdom, Official Journal of the European Communities, L99/14.

European Commission, 1997, Commission Decision 97/312/EC of 12 May 1997 approving the measures to be implemented as regards bovine spongiform encephalopathy in Ireland, *Official Journal of the European Communities*, L133.

European Commission, 1998a. Opinion of the Scientific Steering Committee on the safety of meat and bone meal from mammalian animals; naturally or experimentally susceptible to Transmissible Spongiform Encephalopathies. Adopted on 26-27 March 1998.

European Commission, 1998b. Opinion on the risk of infection of sheep and goats with Bovine Spongiform Encephalopathy agent. Adopted by the scientific steering committee at its meeting 24-25 September 1998.

European Commission, 1999. Preliminary scientific report on: The risks of non conventional transmissible agents, conventional infectious agents or other hazards such as toxic substances entering the human food or animal feed chains via raw material from fallen stock and dead animals (including also: ruminants, pigs, poultry, fish, wild/exotic/zoo animals, fur animals, cats, laboratory animals and fish) or via condemned materials. Presented by the scientific steering committee at its meeting of 18-19 March 1999.

European Commission, 2000, Commission Decision 2000/764/EC of 29 November 2000 on the testing of bovine animals for the presence of bovine spongiform encephalopathy and amending Decision 98/272/EC on epidemio-surveillance for transmissible spongiform encephalopathies *Official Journal of the European Communities*, L305

European Commission, 2000a. Preliminary report on quantitative risk assessment on the use of the vertebral column for the production of gelatine and tallow, submitted to the Scientific Steering Committee at its meeting of 13-14 April 2000.

European Commission, 2000b. Preliminary opinion oral exposure of humans to the BSE agent: infective dose and species barrier adopted by the scientific steering committee at its meeting of 2-3 March 2000.

European Commission, 2001a, Commission Decision 2001/9/EC of 29 December 2000 concerning control measures required for the implementation of Council Decision 2000/766/EC concerning certain protection measures with regard to transmissible spongiform encephalopathies and the feeding of animal protein *Official Journal of the European Communities*, L2.

European Commission, 2001b, Commission Decision 2001/8/EC of 29 December 2000 amending Decision 2000/764/EC on the testing of bovine animals for the presence of bovine spongiform encephalopathy and updating Annex IV of Decision 98/272/EC on epidemio-surveillance for transmissible spongiform encephalopathies *Official Journal of the European Communities*, L2.

European Council, 2000, Council Decision 2000/766/EC of 4 December 2000 concerning certain protection measures with regard to transmissible spongiform encephalopathies and the feeding of animal protein, *Official Journal of the European Communities*, L306.

Food Standards Agency, 2001, BSE in the Republic of Ireland, http://www.bsereview.org.uk/data/bse_in_ireland.htm.

Fraser, H., McConnell, I., Wells, G.A.H. and Dawson, M., 1988, *Veterinary Record*, 123, 472.

Griffin, J.M., Collins, J.D., Nolan, J.P. and Weavers, E.D., 1997, Bovine Spongiform Encephalopathy in the Republic of Ireland: Epidemiological observations 1989-1996. *Irish Veterinary Journal incorporating Irish Veterinary Times*, 50, 10, 593-600.

Hamilton, S., 2000, Personal Communication, WRE Europe Ltd, UK.

Healy, N., 2001, Personal Communication, Department of Agricultural and Food Engineering, University College Dublin, Ireland.

Hoinville L.J., Wilesmith, J.W., and Richards, M.S., 1995, An investigation of risk-factors for cases of bovine spongiform encephalopathy born after the introduction of the feed ban. *Veterinary Record*, 136, 13, 312-318.

Honikel, K.O., 2001, Personal Communication, Bundesanstalt für Fleischforschung (BAFF), Kulmbach, Germany.

Hope, J., Reekie, L.J.D., Hunter, N., Multhaup, G., Beyreuther, J.K., White, H., Scott, A.C., Stack, M.J., Dawson, M. and Wells, G.A.H., 1988, *Nature*, 336, 390.

Horn, G.H., 2001, Review of the origin of BSE, Study commissioned by the Department of Environment, Food and Rural Affairs (UK) in light of the findings of the BSE Inquiry

Kimberlin, R.H., 1993, Bovine Spongiform Encephalopathy - An appraisal of the current epidemic in the United Kingdom. *Intervirology*, 35, 208-218.

Kimberlin, R.H., Walker, C.A., Millson, G.C., Taylor, D., Robertson, P.A., Tomlinson, A.H. and Dickinson, A.G., 1983, Disinfection studies with two strains of mouse passaged scrapie agent, *Journal of the Neurological Sciences*, 59, 355-369.

Maguire, D., 1997, Seventy-six positive feed samples found last year, *Irish Farmers Journal*, 15th February 1997.

Macken, K., 2001, Personal Communication, Environmental Protection Agency, Dublin, Ireland.

McDonnell, K., J. Desmond, J.J. Leahy, R. Howard-Hildige, and S. Ward. 2001. Behaviour of meat and bonemeal/peat pellets in a bench scale fluidised bed combustor. *Energy* 26: 81-90.

McDowell, D. 1997. Rendering of animal by-products in Ireland. IEI seminar-*Disposal of animal by-products in the food and agricultural industries*. Dublin, Ireland 12 June 1997. The Institution of Engineers of Ireland.

Mould, D.L., Dawson, A.M. and Smith, W., 1965, Scrapie in mice. The stability of the agent to various suspending media, pH and solvent extraction, *Research in Veterinary Science*, 6, 151-154.

Office International des Epizooties, 2002, Reported cases of bovine spongiform encephalopathy (BSE) worldwide, http://www.oie.int/eng/info/en_esbmonde.htm.

Phillips, N., Matravers, N., Bridgeman, J. and Ferguson-Smith, M., 2000, *The BSE Inquiry*, The Stationery Office, London.

Prusiner, S.B., 1997, Prion diseases and the BSE crisis, *Science*, 278, 245-251.

Purdey, M., 1996, The UK epidemic of BSE: Slow virus or chronic pesticide-initiated modification of the prion protein - An epidemiological perspective, *Medical Hypotheses*, 46, 5, 445-454.

Schreuder, B., Geertsma, R., van Keulen, L., van Asten, J., Enthoven, P., Oberthur, R., de Koeijer, A., and Osterhaus, A., 1998, Studies on the efficacy of hyperbaric rendering procedures in inactivating BSE and scrapie agents-an interim report. Submission to BSE workshop, April 27th - 29th 1998, Dublin, Ireland.

Scott M.R., R. Will, J. Ironside, H.O.B. Nguyen, P. Tremblay, S.J. DeArmond, and S.B. Prusiner. 1999. Compelling transgenic evidence for transmission of bovine spongiform encephalopathy prions to humans. *Proceedings of the National Academy of Sciences of the United States of America* 96 (26): 15137-15142.

SEAC, 1996, Statement of 20th March 1996, http://www.maff.gov.uk/animalh/bse/bse-publications/level-4-seacstate-20mar96.html.

SEAC, 1999, Public summary of meeting on 18th March 1999, http://www.maff.gov.uk/animalh/bse/bse-publications/seac3-99.html.

Stekel, D. J., Nowak, M. A. and Southwood, T.R.E., 1996, Prediction of future BSE spread. *Nature*, 381, 119.

Taylor, D., Woodgate, S., Fleetwood, A and Cawthorne, R., 1997, Effect of rendering procedures on the scrapie agent. *Veterinary Record*, 141, 643-649.

Taylor, D.M., 1993, Inactivation of spongiform encephalopathy agents. *British Medical Bulletin*, 49, 4, 810 –821.

Taylor, D.M., 1995, Survival of mouse-passaged bovine spongiform encephalopathy agent after exposure to paraformaldehyde-lysine-periodate and formic acid, *Veterinary Microbiology*, 44, 111-112.

Taylor, D.M., 1999, Transmissible degenerative encephalopathies. Inactivation of the causal agents. In: *Principles and Practice of Disinfection, Preservation and Sterilisation*, (3rd edition), Russell, A.D., Hugo, W.B., and Ayliffe, G.A.J. (editors), pp 222-236, Blackwell Scientific Publications, Oxford.

Taylor, D.M., 2000, Inactivation of Transmissible Degenerative Encephalopathy Agents: A review, *The Veterinary Journal*, 159, 10-17.

Taylor, D.M., Fraser, H., McConnell, I., Brown, D.A., Brown, K.L., Lamza, K.A. and Smith, G.R.A., 1994, Decontamination studies with the agents of bovine spongiform encephalopathy and scrapie, *Archives of Virology*, 139, 313-326.

Taylor, D.M., McConnell, I. and Fernie, K., 1996, The effect of dry heat on the ME7 strain of scrapie agent, *Journal of General Virology*, 77, 3161-3164.

Taylor, D.M., Woodgate, S.J., Atkinson, M.J., 1995, Inactivation of the bovine spongiform encephalopathy agent by rendering procedures. *Veterinary Record*; 137, 605-610.

Vose, D. 2000. *Risk analysis, A quantitative guide.* Chichester, England: John Wiley and Sons, Chichester, England. ISBN 0-471-99765-X.

Wells, G.A.H., Scott, A.C., Johnson, C.I., Gunning, R.F., Hancock, R.D., Jeffrey, M., Dawson, M. and Bradley, R., 1987, A novel progressive spongiform encephalopathy in cattle, *Veterinary Record*, 121, 419-420.

WHO, 2001, Variant Creutzfeldt-Jakob Disease (vCJD) – Fact Sheet No. 180, http://www.who.int/inf-fs/en/fact180.html.

WHO. 1995. Report of a WHO consultation on public health issues related to human & animal transmissible spongiform encephalopathies. World Health Organisation, Geneva, 17-19 May 1995. *Document WHO/CDS/VPH/95.145.*

Wilesmith, J.W., G.A.H. Wells, M.P. Cranwell, and J.B.M. Ryan. 1988. Bovine spongiform encephalopathy: epidemiological studies. *Veterinary Record* 123: 638-644

Wilesmith, J.W., Ryan, J.B.M. and Atkinson, M.J., 1991, Bovine Spongiform Encephalopathy: epidemiological studies on the origin, *Veterinary Record*, 127, 199-203.

Zivkocic S., R. Boada, and O. Lopez. 2000. A review of Creutzfeldt-Jakob disease and other prion diseases. *Revista De Neurologia* 31 (12): 1171-1179.