infectivity but what remains is likely to be lower at the point of consumption in sausages enveloped in natural small ruminant casings than it would be from an equivalent weight of bone-in sheep meat form the same animal.

TSE risk reduction from the small intestine

This is an achievable objective but it is not possible to accurately quantify what the level of reduction would be, not least because no natural cases of BSE exist in small ruminants and because there is no knowledge of the titre of infectivity in any part of the intestine. Any estimates of the risk reduction are therefore speculative and subjective. Some guidance can be given from the starting titres in scrapie but there is no knowledge of the infectivity left in natural casings from scrapie-infected sheep. The best judgements can come from studies showing the intestinal tissues that are removed when intestines are processed into casings. These are as follows:

Complete removal of the whole ileum and part of the jejunum.

Complete removal of the serosa, outer longitudinal muscle, Auerbach's nerve plexus and inner circular muscle.

Removal of manure.

Removal of the mucous membrane (epithelium and lamina propria and its contents).

Research

There are a number of serious deficits in knowledge that do not allow a quantitative risk analysis to be undertaken.

There are no reported data on the titres of infectivity in the different parts of small ruminant intestines or in the casings made from them (or indeed any tissues) including in experimental BSE.

There is insufficient knowledge about the contribution made by GALT on the one hand and enteric nervous tissue on the other to the total infectivity.

No studies have been reported about the deposition of PrP in neurones of the autonomic nervous system in other body organs such as the heart for example, which are still allowed for human consumption.

There is also insufficient knowledge about the contribution that M cells, intestinal dendritic cells and tingible body macrophages (as distinct from FDC) make to the total infectivity in the intestine.

Following processing of an intestine to make a casing it is not known what is the reduction in FDC which is a critical point as these are the cells in the intestine known to contain prion protein in an infected animal.

There are insufficient data on the effect of stunning methods in small ruminants and the generation of brain emboli and whether or not they can enter into the systemic circulation.

There are no current data available to show that hand versus machine processing of small ruminant casings produces an equivalent result. This deficit is being corrected by an industry funded study at the University of Utrecht.

Conclusions

Natural casings prepared by the European and international natural casings industry are products that have had a TSE risk-reduction process applied to them.

It is impossible to completely remove TSE infectivity from a TSE-infected intestine during the processing to make a natural casing.

It is possible to reduce the intestinal infectivity considerably by removal of the ileum and by the normal methods of natural casing manufacture. Approximately 50% of any infectivity in the nervous tissue is likely to be removed by the latter process and an unquantifiable amount of the intestine in other cell types (e.g. of lymphatic tissues) in the rest of the casing. Nevertheless this amount is likely to be substantial, perhaps estimated overall to be in the region of > 80%. Some parts of the length of the casing may be completely decontaminated and others not.

Cooking may reduce infectivity still further also by an unquantifiable amount.

By contrast to natural casings, meat sold on the bone has no risk reduction process applied to it and heat applied to it during cooking would only secure the same temperature as achieved in the sausage casing in the superficial layers. TSE infectivity would exist in the carcass lymph nodes at levels comparable to those in intestines from the same age and genotype of animal. Infectivity might also exist in peripheral nerves and in bone marrow.

Collectively this report indicates that if there is a TSE risk in intestines the risk would be lower from natural casings than from the intestine.

In the EU, in 2002 if all the rules are enforced there should be a virtual absence of any infectivity in the gut contributed by feed. Risks from this source could be regarded as negligible.

There are reasons to be concerned that the continuing use of penetrative stunning of sheep might contaminate other tissues (with brain material) sold legally to the consumer in certain circumstances. These risks could be important if BSE occurred in sheep.

It is accepted that there are likely to be other ways to protect the consumer from exposure to the BSE agent in sheep which is not part of this report. For example, by permitting only tissues including bone-in meat and natural casings from certain ages of animal or from certain *PrP* genotypes into the human food chain.

At its meeting of 12-13 September 2002, the Scientific Steering Committee (SSC) examined the most recent data on the safety with regard of BSE, of sheep casings (including that presented in the above report) and concluded that there is no additional or new evidence justifying the possible inclusion of sheep casings in the list of specified risk materials.

Relevant SSC opinions (see annex II):**12, 15.**

THE SAFETY OF RUMINANT MILK

By R. Bradley and D. Heim

Introduction

Milk, in the context of this report, refers to the unadulterated and untreated product derived from cattle in conformity with current milk hygiene regulations. The risk of cross-contamination with TSE-infected material is virtually impossible.

Colostrum is the first milk derived from a cow after calving. This is not normally permitted in the human food supply.

Milk is a source material for preparing other food like cream and cheese, and starting materials like lactose and galactose that are used in medicinal and biological products.

Epidemiological studies

If colostrum and/or milk were infected then it would be most likely represented as a form of maternal transmission.

- In Human TSE: no mother to child transmissions of kuru, sporadic CJD or vCJD have been reported.

- Animal TSE

  The collective evidence points toward minimal involvement of any form of vertical/maternal transmission of BSE in cattle. Virtually all calves receive colostrum but only beef suckler calves generally receive milk from their dam so the widely differing incidences of BSE in dairy and beef herds does not support either colostrum or milk being a vehicle of transmission of BSE. No cases of BSE have been reported in the offspring of over 2000 cases of BSE outside the UK. The incidence of BSE in the offspring of confirmed cases of BSE in the UK is the same as the incidence in the epidemic as a whole. Data on the expected number of cases of BSE from feed exposure alone and the observed number of cases shows no excess in the latter in any year. No cow to calf disease transmissions has been reported in association with BSE infected beef suckler cows from any country though a specific study in Great Britain has only been able to follow a relatively small number of animals to conclusion. In a cohort study there is no evidence that BSE can occur in the absence of a feed-borne source of infection.

There is no firm epidemiological evidence to support the view that colostrum or milk carry infectivity in sheep and goats with scrapie.

In TME of farmed mink there are no indications for maternal transmission. Maternal transmission of Chronic Wasting Disease (CWD) may occur but if it does it is relatively uncommon and the mechanism is unknown.

Transmission studies

To date, no parent to offspring transmissions have been reported in any animal species (including primates) used in experimental studies.

- Human TSE

  Milk from humans affected with kuru has been inoculated into chimpanzees by multiple parenteral routes but no disease has resulted in over 30 years.

  In 1992 in Japan, colostrum from a 38-year-old pregnant woman with sporadic CJD was reported to be infected when injected i/c into mice. However, this original report is not supported following subsequent study.

- Animal TSE

  Oral, intraperitoneal and intracerebral challenge of mice with mammary gland and pooled milk from cows with confirmed BSE resulted in no transmission. Milk pools from cows with confirmed BSE , collected at early, middle and late lactation, were inoculated or fed to susceptible mice. No neurological illness or TSE-like pathology resulted in any mouse.

  No detectable infectivity has been found in the mammary gland of non-pregnant ewes with scrapie, or in the colostrum of high-risk ewes at parturition following i/c inoculation of susceptible mice. Similarly no infectivity was detected in the mammary gland or milk from three goats with clinical scrapie kept in contact with sheep from the same farm.

  In a separate study no detectable infectivity was found in colostrum or milk from sheep or goats naturally affected with scrapie following i/c or sub-cutaneous, or oral challenge of mice, but experimental detail is lacking.

  No detectable infectivity was found in the mammary gland of farmed mink with TME following bioassay by the i/c route in mink.

Critical comments of some of the above studies

The Scientific Steering Committee noted that, in the absence of any infectivity studies on certain tissues including milk and colostrum by i/c inoculation of the homologous species in bovines, ovines and caprines, and in the absence of all the necessary experimental and epidemiological data as detailed in the report, precise estimates of the TSE risks cannot be made.

Research

Currently there seem not to exist plans to inoculate bovine milk i/c into cattle. However, a new study is in progress that seeks to determine if prion protein can be detected in fractions of milk derived from cattle experimentally challenged orally with high (100g) or low (1g) amounts of brain from affected cattle. The earliest that interim experimental results could be available would be the spring of 2003.

In regard to sheep milk a small-scale study is presently running at the NPU (N Hunter, personal communication, March, 2001) with scrapie susceptible lambs removed from their mothers and hand reared. No results are yet reported.

Recent reports of the experimental transmission of scrapie and of BSE to susceptible genotypes of TSE-free sheep following i/v transfusion of about 400ml of blood (or buffy coat) from sheep incubating experimental BSE or natural scrapie raises concern that low levels of infectivity could exist in white cells that also form a component part of milk in this species. The experiment is not yet concluded.

Conclusions

The evidence available to date does not point at milk or colostrum representing a possible TSE risk.

However, the SSC supports the recommendation that for precautionary reasons the milk, colostrum or milk products from suspect BSE cases should not be offered for consumption.

Should BSE become probable or be found in small ruminants then a reassessment has to be undertaken.

Relevant SSC opinion (see annex II): **25.**

PART II D

QUANTITATIVE RISK ASSESSMENT

A FRAME FOR THE QUANTITATIVE ASSESSMENT OF THE RESIDUAL BSE RISK IN CATTLE-DERIVED FOOD PRODUCTS

By D. Dormont, M. Doherr and Ph.Verger

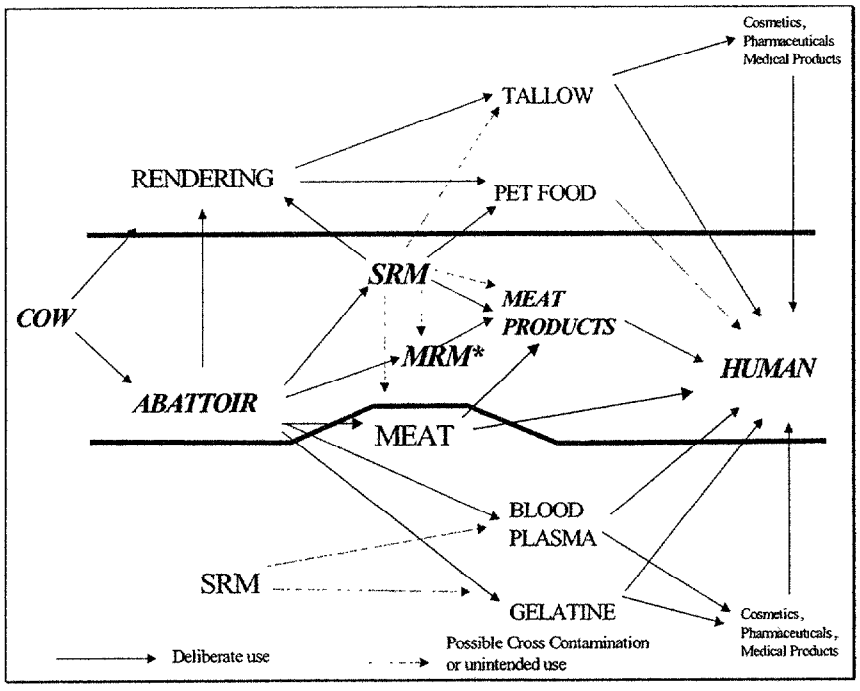
Whether a product is used as food/feed, a cosmetic product, or a medicinal product or medical device will determine the route and the length of possible exposure. The routes can be oral, intravenous, topical, and/or inhalatory and the nature of the product will also determine whether or not there may be a repeated exposure.

The human exposure risk will depend on the following main factors:

- the likelihood that an animal infected with BSE enters the human food chain;
- the amount and distribution of infectivity in that animal;
- the ways in which the various tissues that could contain infectivity are used in the food chain;

The possible routes of exposure of humans to BSE infectivity are multiple (**Figure**).

Figure: Potential Routes of Exposure to Infective Cattle Tissues



Deterministic and stochastic approaches towards BSE risk quantification

For the assessment of the residual BSE risk in ruminant-derived products entering the food chain, two approaches can basically be followed to quantify the residual BSE risk of animal derived products:

a. In the ***deterministic approach,*** a single value is attributed to each parameter in the assessment. This value corresponds to the most likely value this parameter (commonly) takes or is assumed to take.

   The deterministic modelling approach permits the risk manager to rapidly estimate (by linear extrapolation) the risk under alternative conditions (e.g. higher/lower tissue infectivity levels; larger/smaller batch sizes; complete/incomplete specified risk materials removed; etc.). However, the rigidity of the deterministic approach may result in unrealistic scenarios as, for example, the likelihood is almost zero that all values, assumptions and scenarios combined will at once be "average", best" or worst".

b. In the ***probabilistic approach***, the above problem is at least partly avoided. The model is run many times and for each of the model runs, *combinations* of risk values for each parameter are selected at random.

   A major advantage of the probabilistic approach is that it helps to understand interactions between parameters whilst taking into account uncertainties and that therefore it is a most useful tool in decision-making. One should however be aware that the assumed probability distributions in reality reflect at once both the scientific uncertainties in certain areas[35] and the fact that certain field conditions follow a statistical distribution[36].

When using the results of quantitative BSE risk assessment it should be kept in mind that not only a range of values may have been attributed to a given parameter but also that other uncertainties still accompany the BSE problem. By its nature quantitative assessments of the residual risk in consumer products lead to numerical figures which are indicative of the extent of expected events and should be understood more like orders of magnitude ranges than exact predictions. In addition, the level of uncertainty increases as the number of scientific unknowns increase.

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[35] For example: is the species barrier 1, 10, 100, 1000 or 10000? Are the minimal infective doses 1000, 100, 10 or 1 mg?

[36] For example: the risk reduction during production will not always be identical for all plants and within a given factory, but is likely to be distributed around this value. In this respect, it should be noted that the

Summary overview of the input data needed for quantitative BSE risk assessments.

A comprehensive quantitative assessment of residual BSE risk posed by cattle derived products requires information for the following input variables:

1. The **processing risk**.

   The probability that an infective bovine is slaughtered for food (or the "Processing risk") is the most relevant parameter for the human exposure risk.

   It is the **prevalence** of BSE positive animals that become slaughtered for food and determines the probabilities that at least once per year a BSE+ animal is slaughtered but not detected and that a production batch contains material from an infected animal.

2. **The possible infectious load of the cattle by-products**

   The tissue infectivity distribution and typical tissue titres of the BSE agent will determine the infectious load of the animal materials used for the production of a food or derived product. Details are provided in the contribution from G. Wells and H.A. Kretzschmar on *Pathogenesis, tissue infectivity distribution and specified risk materials* **(Part II.A).**

   The age of the infected animal that is slaughtered and processed influences the infective load and its distribution between the tissues of the animal. On the basis of the available knowledge, it is possible to define three age categories of cattle, which have different potential levels of infectivity, mainly as a function of their age at slaughter. Depending on the category, the infectivity that could enter the food chain will differ, both in quantity and with regard to the specified risk tissues:

   Veal Calves (less than 1 year). The level of infectivity in the CNS tissues of these animals can be considered to be negligible. However, there may be infectivity in the intestines, in particular the ileum.

   Cattle older than 1 year, but less than 30 months. These animals could, if infected at birth, show some level of infectivity, though it would be very unlikely to be the same as in a clinical case of BSE. The CNS is not necessarily highly infective, even if the animal was infected very early in life.

   Cattle older than 30 months. If infected early in their life, these animals may show infectivity levels close to those of clinical BSE-cases, even if no clinical signs are

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TSE validation studies carried out by GME involved only processes that could be considered to apply generically to all of the GME member companies that produce gelatine.

apparent. It is clearly evident from the Swiss surveillance of fallen stock and the UK surveillance of cattle over 30 months i.e. those excluded from the food chain under the Over Thirty Months Scheme (OTMS), that apparently healthy but nonetheless infected animals do enter the human food chain in countries where BSE is prevalent in the cattle population. In this category of bovines, the level of infectivity will be high and the CNS is certain to be highly infective.

3. **The typical size of the batch of raw materials** entering the production chain

The **typical batch size** of the raw materials is an important parameter, because it determines the probability that a given batch is BSE+. The number of animals included in such a batch depends on the amount of materials obtained from each slaughtered bovine and the proportion of raw products from other sources than cattle bones (i.e. gelatine from cattle or pig hides).

Defining the batch size for risk assessments purposes depends upon whether the production process is continuous or in batches. For certain products (e.g. melted fats) one can assume that during the production process or during the storage afterwards, a thorough mixing takes place and that infectivity (if any) present in parts of the raw material entering a process is diluted over the end-batch as a whole. But for other processes the raw materials may remain in the production process as discrete amounts or are only partly mixed and the possible presence of residual infectivity (if any) may be limited to a given fraction of the end-batch. In the latter case, the dilution effect is lower and limited to the size of the discrete amounts of raw material that entered the production chain.

Infected animals per batch. As long as the BSE-cases remain geographically scattered, the number of exposed consumers would be proportional to the number of processed BSE-infected animals and the average exposure dose would remain rather constant. If the BSE-density is so high that more than one infective animal could enter a single batch of production, the number of consumers exposed would remain stable while the dose per exposed individual would increase proportional to the number of infected animals entering the batch.

4. **Processing conditions.** The effects of processing determine whether or not any of the risks present are reduced significantly.

Processing conditions influence the level of infectivity in the product. For example, certain production processes for gelatine reduce the infective load at least a 50,000-fold. However, normal cooking and industrial food processing are unlikely to significantly affect the level of infectivity.

5. Serving size or the **amount**[37] **consumed per intake**. Together with the batch size, the serving size influences the dose of exposure and the number of persons exposed. This value also determines the number of possibly infectious doses that may be included in a batch of raw materials.

6. The **modalities of use / application**. Whether a product is used as food/feed, a cosmetic product or a medicinal product or a medical device will determine **the route of possible infection** which can be oral, intravenous, topical, etc. They will also determine whether or not and over how long a period there may be **repeated exposure**.

7. The **condition and genotype of the user**. It may be assumed that certain population groups including for example children, elderly and immuno-compromised people, are more susceptible to possible infection than others. For TSEs, immuno-compromised people may be more susceptible because there is clear evidence of the involvement of the lymphoreticular system in the pathogenesis of **vCJD** (and other **TSEs**). With regard to the genotype of **vCJD**-affected individuals, all of those tested so far have been shown to express methionine homozygously at codon 129 of the **PrP** gene. Considering that this allelic frequency occurs in only around 38% of the human population, it has been listed as one of the risk-factors for developing **vCJD**. However, insufficient time has elapsed to know whether or not other codon 129 genotypes will be equally (or even more) susceptible but have longer incubation periods. Experimental data from animal studies on TSEs also show that genes other than **PrP** have an influence on susceptibility and incubation periods.

   In practice only items 1) through 5) can be taken into account and the route of exposure is assumed to be oral. For the inclusion of items 6) and 7) additional information may be needed which is currently unavailable. Moreover, they relate to fields of application for which special grade products are needed anyway (e.g. for the production of certain special grade gelatines).

Worst case assumptions, risk assessment and risk management.

A comment that is frequently made when it concerns the choice of scenarios for risk assessment is that they (almost always) have the tendency of being based exclusively on worst case scenarios and do not adequately reflect normal field practice. It needs, however, to be pointed out that risk assessment exercises are in the first instance intended to facilitate risk management decisions. For example, a risk scenario assuming that all

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[37] The term "amount" is preferred above "dose". (It is easy to determine the amount consumed but the dose requires a knowledge of titre too which is more difficult. In general the range of variation in amount is likely to be small whereas the variation in titre could be large in the worst scenario.)

good manufacturing practices and the legislation are correctly implemented will/may assure the producer and consumer that a product is safe provided these conditions are fulfilled, but may be of little help for the risk manager who has to propose risk reducing scenarios on the basis of "what if a certain condition is not fulfilled" scenarios.

Numerical combinations of the various worst case values (for example: dose + species barrier) for risk assessment purposes into one "multi-worst case figure" would eventually result in an unrealistic scenario. It is therefore recommended that *realistic* worst case scenarios are used to assess the human exposure risk.

Infectious dose and species barrier.

Quantitative assessment of the risks must take into account the size of the species barrier between cattle and humans, the infection source, the dose of agent, the estimation of the minimum infectious dose and the potential effects of cumulative exposure to low doses of infected materials, the route of exposure and its efficacy in establishing TSE agent infection, the pathogenesis of TSEs and the genetic susceptibility of humans. In this context the following should be mentioned:

The size of this cattle-to-humans species barrier is not known, neither is the dose response relationship for humans. A practical approach is therefore to present the level of exposure in terms of consumption of defined amounts of the BSE agent, measured in Cattle Oral Infective Doses (CoID) and to assume, as a worst cases scenario, the absence of a species barrier. (It needs to be emphasised that the $CoID_{50}$ is only an indicator and should not be confused with the Human Oral Infective Dose ($HoID_{50}$), which is not known.)

Population risk

Risks from exposure to amounts of infection below the minimum infectious dose cannot be determined in the current state of the scientific knowledge. Whether the dose/response relationship in the low dose range (for low levels of potential residual infectivity in products after appropriate processing and handling, i.e. after appropriate sourcing, removal of SRMs, processing, avoidance of cross-contamination, etc.) is linear, or follows for example a sub-linear dose-response relationship, or a Poisson distribution, does not immediately affect the outcome of the assessment as such (in terms of absolute numbers of people at risk), but may affect the perception of the risk in management terms. In the first case, a whole (sub-)population is theoretically exposed to a same, but low level of residual infectivity. In the second case, a major part of a population will not be exposed at all to any infectivity, because it is concentrated (aggregated) and localised in a smaller number of consumption units. However, the part of the population that is exposed, will more likely get infected because the infectivity level is relatively higher.

In terms of risk assessment, the risks resulting from (low) residual infectivity should at present - until further evidence is available - be calculated as if a population as a whole is exposed and assuming a linear dose-response curve down to the low dose range[38] This is a conservative assumption and would mean, *as an example*, that a product containing an evenly distributed residual infectivity of 10^{-3} ID50/g and given to each of 1 million individuals, may result in 500 individuals being infected[39]. The accumulative infectivity scenario has to be considered as valid for risk assessment of the effects of repeated sub-infectious doses, provided the interval of administration is not too long (see further).

Multiple exposure

The dose response relation is not known and little if anything is known about the (cumulative) risk resulting from a repetitive use - possibly during a prolonged period - of a product such as a food, feed, cosmetic, medicinal product or medical device, containing sub-lethal infectivity levels.

An accumulative infectivity scenario may also be valid, provided the interval of administration is not too long (probably less than about 2 to 3 days) and that the repeated doses are sufficiently high so that an infective dose is reached in steady state (the repeated individual doses must be higher than the capacity of an individual to inactivate the infectivity during the interval of administration. From laboratory results it appears that the clearance period is approximately 24-48 hours in rodent models; beyond that, macrophages are again capable to take up their clearance function. Further research in this area is needed.

In terms of risk assessment, repeated exposure would thus increase the risk both in absolute number of cases and the likelihood that exposure would result in effective infection.

Routes of exposure to specified risk materials in food

The routes of exposure to SRMs can be summarised as follows:

1. Consumption of Specified Risk Materials as such

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38 The dose response relation is not known. Whether the dose/response relationship in the low dose range (for low levels of potential residual infectivity in products after appropriate processing and handling, i.e., after appropriate sourcing, removal of SRMs, processing, avoidance of cross-contamination, etc.) is linear or follows for example a sublinear dose-response relationship, does not immediately affect the outcome of the assessment as such (in terms of absolute numbers of people at risk) but may affect the perception of the risk in management terms.(See report)

39 This is just an example; elements such as the weight of the inoculum and of the infectious material or the weight of the bovine material to which the individual was exposed to., are not taken into account.

SRMs are consumed as such. It is known that brain and spinal cord (amourette in French) were consumed in this way, as well as ileum and all the small intestine (andouillette in French) from young veal (< 6months). Even spleen and eyes might occasionally have been eaten. Trigeminal ganglia and dorsal root ganglia are not consumed as such, although there will be some direct consumption of DRG (and possibly spinal cord) from cuts of meats served on the bone and including part of the vertebral column (e.g. T-bone steak, rib of beef).

2. Consumption of Specified Risk Materials after transformation

SRM are transformed and integrated into food products in such a way that it is not detectable by the consumer. The inclusion of materials derived from SRM into food products may happen intentionally or by contamination.

Intentional inclusion of SRM. The use of brain or spinal cord in "paté" or sausages is an example of the intentional use of SRM. Other SRM may also be included into food products as direct ingredients.

Contamination of edible products with materials derived from SRM. Contamination is always possible if the inclusion of SRM is technically possible and does not create quality problems.

3. Estimation of the Exposure Level and of the number of persons exposed.

In order to estimate the expected number of people that would be exposed to an infected dose, several critical factors have to be considered. Some of them are related to the sources, others to the routes.

Relevant SSC opinions (see annex II): **3,5, 59, 60**.

ANNEX I: CHRONIC WASTING DISEASE

AND TISSUES THAT MIGHT CARRY A RISK FOR HUMAN FOOD AND ANIMAL FEED CHAINS

By J. Brugère-Picoux

Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy (TSE) or "prion disease" of certain species of native North American deer: mule deer (*Odocoileus hemionus)* and white-tailed deer (*Odocoileus virginianus)* and Rocky Mountain elk (*Cervus elaphus nelsoni*), occurring in captive, farmed and free living populations. This is the only TSE known to affect free-ranging wildlife species. Its reported occurrence other than in North America (NA) is confined to a single case in an elk imported into Korea from NA.

Historical perspective of occurrence

The clinical syndrome of wasting and death in **captive cervids** was first recognised in the late 1960's in mule deer, in wildlife facilities in Colorado and Wyoming, USA. Neuropathological studies showed that it was a spongiform encephalopathy. By 1982 the disease had also been described in elk in the same facilities. The first case of CWD in Canada was diagnosed in farmed elk in1996 and is thought to have been introduced with elk imported from the USA in the late 1980s.

CWD in **free-ranging cervids** in the USA was first diagnosed in an elk in 1981 and in a mule deer in 1984. Since 2001, surveillance of wildlife in Canada has detected 7 cases of CWD in wild deer.

Host range & transmissibility

Only three species of Cervidae are known to be naturally susceptible to CWD. Experimental transmission studies have not yet been able to show transmission of CWD from deer to cattle. Genetic studies show phylogenetic differences in PrP sequence between Cervidae, Bovidae and humans suggesting an appreciable species barrier for possible transmission of CWD to cattle and humans. Molecular studies and epidemiological observations support this apparent resistance of certain phylogenetic families.

Experimental studies to transmit CWD have been conducted, mostly by intra-cerebral (IC) inoculation, providing information on susceptibility by the most efficient means of interspecies transmission, but not on interspecies susceptibility by natural routes of transmission (e.g. oral exposure).

Epidemiology

There is considerable evidence that CWD is both infectious and contagious but specific details of natural transmission remain to be determined. Epidemiological data have shown that CWD is readily spread by lateral transmission in cervid populations. Indirect transmission via environmental contamination may also play a role in the natural dynamics of CWD and rapid increase in prevalence within captive herds suggests this form of transmission may be locally, quite efficient. There is less evidence for the existence of maternal transmission and alone it is unlikely to sustain epidemics of CWD. The occurrence of CWD is not explained by feed-borne exposure associated with rendered ruminant meat and bone meal (MBM) as has been shown for BSE.

Although hypotheses as to a possible origin of CWD have been suggested (e.g. scrapie), there is no evidence in support of any of the theoretical sources.

Pathogenesis

In CWD affected deer and elk there is a very widè tissue distribution of PrP^{CWD}, resembling that of scrapie and BSE agents in tissues in TSE-susceptible sheep and this is in contrast to the distribution pattern of agent in BSE of cattle. However, tissue distribution of PrP^{CWD} is not identical for deer and elk. In the latter species detectable levels in peripheral tissues are apparent later in the incubation period. This widespread peripheral distribution of PrP^{CWD} early in the incubation period (at least in deer) presents significant, if not insurmountable, difficulty with respect to the potential for the removal of specified risk materials (SRM) in CWD. The significant involvement of the alimentary tract suggests a potential for progressive shedding of PrP^{CWD} and the agent through the disease course.

Diagnosis

Clinical signs of CWD in deer and elk are not specific but consistently include progressive weight loss. Behavioural changes include decreased interactions with other animals, listlessness, lowering of the head, drooping ears, blank facial expression and repetitive walking in set patterns. In elk, behavioural changes may also include hyper-excitability, nervousness, ataxia and head banging. Free ranging CWD affected elk may loose fear of humans. In deer and elk polydipsia and polyuria also commonly occur. The clinical disease is progressive and always fatal. Caretakers familiar with individual animals often recognise subtle changes in behaviour well before serious weight loss occurs. The clinical course of CWD varies from a few days to approximately a year, with most affected animals surviving from a few weeks to three or four months.

The **incubation period** range in naturally occurring CWD is not known. Evidence of pre-clinical infection has been found in deer fawns and elk calves from about 6 months of

age. The youngest naturally infected mule deer diagnosed with clinical disease suggests 16 to 17 months as an approximate minimum incubation, whereas the earliest diagnosis of CWD in elk was in a 24 months old animal.

Differential diagnosis include mineral deficiencies leading to neurological signs listeriosis, copper deficiency, etc. and disorders leading to loss of body weight e.g. fading elk syndrome. Aspiration pneumonia, presumably caused by ptyalism and difficulty in swallowing, may lead to misdiagnosis of the condition if histological and/or immunohistochemical examinations of nervous or/and lymphoid tissues are not carried out.

The **post-mortem diagnostic examination** often reveals gross lesions of emaciation, reflecting the clinical signs. Aspiration pneumonia, which may be the actual cause of death, is also a common post-mortem finding in animals affected with CWD. On **microscopic examination** of clinically affected animals, spongiform lesions are observed in the central nervous system. The earliest detectable lesions in the brain, in the parasympathetic vagal nucleus in the medulla oblongata at the obex, suggests that this is the most important site to examine for surveillance of clinically normal animals. Immunohistochemical (IHC) detection of the disease specific protein marker PrP^{CWD} is used to test brain tissue. PrP^{CWD} also accumulates in tonsillar and other lymphoid tissues at an early stage of the pre-clinical disease (in deer but not in elk). Tonsillar biopsies may be used in pre-clinical diagnosis for detecting CWD in live deer.

Surveillance

In both USA and Canada surveillance programmes include passive and active surveillance strategies. Target animals include those showing clinical signs, particularly, emaciation, discovered by, or reported to, wildlife agencies and those obtained from surveys of normal deer and elk killed by hunters or agency personnel, or road kills.

Surveillance of **free-ranging cervids** for CWD in USA has been ongoing in Colorado and Wyoming since the late 1970s and has since been extended to additional states. CWD has been diagnosed in free-ranging mule deer in Wyoming, Colorado, Nebraska, South Dakota and New Mexico and in free-ranging elk in Wyoming, Colorado, and South Dakota. It has been found in free-ranging white-tailed deer in Wyoming, Colorado, South Dakota, Nebraska, Wisconsin, and Illinois. In Canada, surveillance for CWD since April 2001 has found CWD in wild deer in Saskatchewan.

Surveillance in USA for CWD in **farmed Cervidae** began in 1996 and CWD positive herds have been diagnosed in South Dakota, Nebraska, Colorado, Oklahoma, Kansas, Montana and Minnesota. Also, CWD has been diagnosed in farmed white-tailed deer in Wisconsin. In Canada, surveillance of farmed cervids for CWD since 1997 has detected CWD-infected farms in Saskatchewan (elk) and in Alberta (elk and white tailed deer).

Control strategies

Control measures include prevention of introduction, notification of the disease, control or ban on movements, quarantine, screening/testing, eradication of affected herds, cleansing and disinfection of farms before re-population, compensation and measures to prevent/stop spread from free range to farmed cervids(or vice versa). Because of the commercial aspect of game ranching, animals have frequently been moved across the US and Canada and there is also natural movement of deer and elk across state lines. However, recently, laws have been made to prevent the movement of captive animals across state lines. Surveillance data do not as yet provide information on accurate figures of the prevalence of the disease in NA and the risk factors are not well understood. Some control measures for farmed deer are in place. However, movement of free-ranging deer provides a major difficulty for control strategies.

Following screening, herd certification may be an option. However, given limited knowledge on the incubation of the disease and its variation in clinical presentation it is likely to take a period as long as 5 years of surveillance of all juvenile and adult mortality before a farmed herd may be certified free of CWD.

There has been significant **economic impact** on the NA farmed cervid industry but the total effect is difficult to quantify. A high cost has been involved in compensation of Canadian farmers after eradication on CWD positive farms in addition to the cost of quarantine of farm and grassland following CWD. In Canada, elk are raised for the production of antler velvet and meat and for trophy/hunting and about 70% of velvet antler was formerly exported to South Korea. Some trading partners closed their markets to Canadian cervids and cervid products, including semen, embryos and velvet.

Surveillance for TSEs in cervids in Europe

There is no published information on the possible occurrence for TSEs in cervid species on the European continent available. Research and surveillance programs on CWD in farmed or wild Cervidae have not existed until recently and therefore there are no data on which to draw conclusions about CWD in the Cervidae populations of Europe.

Food and feed safety and human and animal risk

Although CWD is not similar to BSE in terms of epidemiology, in that there is no evidence of natural spread of the disease to phylogenetic families other than the Cervidae, it still may pose a theoretical risk for animal and human food safety.

Food safety and human health

There is no evidence that CWD can be transmitted to humans consuming meat or handling infected cervids or their products, however this possibility cannot be ruled out.

Phylogenetic differences in PrP sequence suggest an appreciable species barrier for possible transmission of CWD to humans. However, since the basis of the transmission barrier in relation to the TSE is complex and not solely a function of PrP sequence of donor and recipient it remains theoretically possible that the CWD-agent could infect humans.

The World Health Organisation recommends that people not consume animal products from any animal infected with a TSE disease and public health policies in Canada and the US are consistent with this direction.

In NA health officials advise hunters not to consume meat from animals known to be infected with CWD. In addition, they suggest hunters take simple precautions when field dressing deer or elk taken in areas where the disease is found.

Feed safety and animal health

The FDA has recently provided new guidance to state public health and agriculture officials throughout the US in which is stated that material from CWD positive animals or animals at high risk for CWD is not permitted to be used as an ingredient in feed for any animal.

In Canada there are no mandatory controls on rendering carcasses and offal from cervids other than those tested positive for CWD or animals that have been exposed to test positive animals. However, the Canadian Renderers' Association has a voluntary ban on the rendering of cervids. Canada prohibits the feeding of ruminant derived proteins to ruminants.

Risk of spread to Europe

Available information indicates that there is negligible trade in live cervids originating in NA to the EU but there are indications of imports of small annual tonnage of edible products from game. Some licences for exports to EU-countries were granted to private persons for hunter-related trophies (skin, antlers). Data provided by Eurostat confirm data provided by the USA and Canada. It is unclear what, if any, trade exists in antler, embryos or semen from cervids between NA and EU countries.

Current surveillance activities in Europe should be encouraged in order to provide more detailed base line data as the current studies are assessed as insufficient to detect a CWD infection in cervids should it be present.

Conclusions

A theoretical risk for prion transmission to humans consuming products of CWD affected-cervids of all ages in countries where CWD exists cannot be excluded. Similarly

also, such transmission risk to domestic animals cannot be excluded. There is therefore a scientific basis on which to exclude tissues from animals that carry a CWD risk, from human or animal feed chains.

However, the early and widespread involvement of tissues in CWD infected animals practically prevents exclusion of any tissues in defining a SRM list, or setting any lower age cut-off as has been defined for cattle in relation to BSE.

Available information indicates that imports of live cervidae from NA to EU and trade in meat products from cervid species are negligible, but it is important to ensure that no transfer of risk takes place through trade of live cervids or derived products.

At present, there are no scientific data that CWD is occurring in Cervidae elsewhere than in those countries from which it has been previously reported. However, systematic TSE surveillance of cervid populations has either been absent or has only just started in European countries. Until results of such surveillance become available no conclusion can be drawn with regard to the occurrence of CWD or similar TSE in the cervid population of Europe.

Recommendations

Given that the possible risks of exposure relate to the tissues of cervids from NA, reinforced protection of the cervid population and animal and public health in Europe could be considered.

Moreover, systematic surveillance is essential to establish the probability of occurrence and incidence of CWD in the cervid populations of Europe. Because of the complexity of conducting such surveillance on a statistical basis throughout the EU, initial research should address the susceptibility of European cervid species to TSEs. Furthermore, a surveillance programme, which might initially target the examination of cervids dying in or culled from zoological collections and fallen stock in farmed cervid populations, prior to decisions on the screening of free-ranging cervids, is recommended.

Relevant SSC opinions (see annex II): **39.**

ANNEX II

LIST OF OPINIONS AND REPORTS RELATED TO TSE/BSE, ADOPTED BY THE SCIENTIFIC STEERING COMMITTEE BETWEEN NOVEMBER 1997 AND APRIL 2003.

Web Address: http://europa.eu.int/comm/food/fs/sc/ssc/outcome_en.html

General aspects

1. **Opinion on** Organophosphate (OP) poisoning **and hypothetical involvement in the origin of BSE. Adopted on 10-11 April 2003.**
2. Opinion on hypotheses on the **origin and transmission** of BSE. Adopted on 29-30 November 2001.
3. Opinion on **oral exposure** of humans to the BSE agent: infective dose and species barrier. Adopted on 13-14 April 2000.
4. Preliminary opinion on **oral exposure** of humans to the BSE agent: Infective dose and species barrier. Adopted on 2-3 March 2000.
5. Opinion on the **Human Exposure Risk (HER)** via food with respect to BSE. Adopted on 10 December 1999.
6. Opinion on the possible **vertical transmission** of Bovine spongiform encephalopathy (BSE). Adopted on18-19 March 1999.
7. Report on the possible **vertical transmission** of Bovine Spongiform Encephalopathy (BSE) - Submitted on 18-19 march 1999.
8. Opinion on possible links between BSE and **Organophosphates** used as pesticides against ecto- and endoparasites in cattle. Report and opinion adopted on 25-26 June 1998.
9. Opinions adopted on **19-20 February 1998.**

Tissue infectivity distribution and Specified Risk Materials

10. Opinion on BSE risk of the **bovine autonomic nervous system.** Adopted by the on 6-7 March 2003).
11. Update of the Opinion on TSE **Infectivity distribution in ruminant tissues.** Initially adopted on 10-11 January 2002 and amended on 7-8 November 2002.
12. Complement to the SSC opinion of 4-5 April 2002 on **safe sourcing of small ruminant materials** (with special reference to the safety with regard to BSE risks of sheep intestines and casings). Adopted on 12-13 September 2002.
13. Opinion on the implications of the recent papers on **transmission of BSE by blood** transfusion in sheep. Adopted on 12-13 September 2002.

14. Amendment on opinion on the safety of **bovine embryos**: Amendment to opinion of 18-19 march 1999 on the possible vertical transmission of Bovine Spongiform Encephalopathy (BSE). Adopted on 16 may 2002.
15. Report on the safety of **sheep intestine and natural casings** derived therefrom in regard to risks from animal TSE and BSE in particular. Report prepared for 7 may 2002.
16. Opinion and report assessment of the human BSE risk posed by **bovine vertebral column** including dorsal root ganglia. Adopted on 16 may 2002.
17. Opinion on the safety of **animal rennet** in regard to risks from animal TSE and BSE in particular. Adopted on 16 may 2002.
18. Opinion on the safety of **calf-derived rennet.** Adopted on 04-05 April 2002.
19. Statement on prions in **muscle.** Adopted on 04-05 April 2002.
20. Opinion on stunning methods and BSE risks (the risk of dissemination of **brain particles into the blood** and carcass when applying certain stunning methods). Adopted on 10-11 January 2002.
21. Report on stunning methods and BSE risks (the risk of dissemination of **brain particles into the blood** and carcass when applying certain stunning methods). Prepared for 13 December 2001.
22. Opinion on TSE **infectivity distribution in ruminant tissues** (state of knowledge, December 2001). Adopted on 10-11 January 2002.
23. Preliminary opinion and report on stunning methods and BSE risks (the risk of dissemination of **brain particles into the blood** and carcass when applying certain stunning methods). Adopted on 6-7 September 2001.
24. Opinion on adipose tissue associated with the **digestive tract** of cattle, sheep and goats: an appreciation of possible TSE risks. Adopted on 28-29 June 2001.
25. Safety of **milk** with regard to TSE: State of affairs. Adopted on 28-29 June 2001.
26. Opinion on the Implications of the Houston *et al* paper in The Lancet of 16 September 2000 on the **Transmission of BSE by blood transfusion in sheep**. (The Lancet, Vol. 356, pp 999-1000; 955-956; 1013). Adopted on 26-27 October 2000.
27. Opinion on **specified risk materials of small ruminants** (Follow-up to the SSC opinion of 24-25 September 1998 on the Risk of Infection of Sheep and Goats with BSE agent). Adopted on 13-14 April 2000.
28. Opinion on the safety of **ruminant blood** with respect to TSE risks. Adopted on13-14 April 2000.
29. Listing of **Specified Risk Materials**: a scheme for assessing relative risks to man - Opinion of the Scientific Steering Committee adopted on 9 December 1997. Re-edited version adopted on 22-23 January 1998.

BSE in sheep

30. Opinion on **safe sourcing** of small ruminant materials. Adopted on 04-05 April 2002.
31. Suggested strategy to **investigate the presence of BSE in small ruminants.** Adopted on 04-05 April 2002.
32. Opinion on the safety of small ruminant products should **BSE in small ruminants** become probable / confirmed. Adopted on 18-19 October 2001.
33. Opinion on **pre-emptive risk assessment** should BSE in small ruminants be found under domestic conditions. Adopted by the Scientific Steering Committee at its meeting of 8-9 February 2001.
34. Report and Opinion on the Criteria for **diagnosis** of clinical and pre-clinical TSE disease in sheep and for differential biochemical diagnosis of TSE agent strains. Adopted on 13-14 April 2000.
35. The policy of **breeding and genotyping** of sheep, i.e. The issue whether sheep should be bred to be resistant to scrapie. Adopted on 22-23 July 1999.
36. **Surveillance** of TSEs in sheep and goat in relation to the risk of infection with bovine spongiform encephalopathy agent and related actions to be taken at EU level. Actions to be taken on the basis of (1) the September 1998 SSC Opinion on the Risk of infection of sheep and goats with the BSE agent and (2) the April 1999 SEAC Subgroup report on Research and surveillance for TSEs in sheep. Adopted on 27-28 May 1999.
37. Statement on the SEAC Subgroup report on **Research and surveillance** for TSEs in sheep, released in April 1999. Adopted on 22-23 April 1999.
38. Opinion on **the risk of infection** of sheep and goats with Bovine Spongiform Encephalopathy agent. Adopted on 24-25 September 1998.

TSEs and BSE in food animal species other than ruminants

39. Opinion on **Chronic Wasting Disease** and tissues that might carry a risk for human and animal feed chains. Adopted on 6 – 7 March 2003.
40. Report on **Chronic Wasting Disease** and tissues that might carry a risk for human food and animal feed chains. Adopted on 6-7 March 2003.
41. Opinion on the feeding of wild **fishmeal** to farmed fish and recycling of fish with regard to the risk of TSE. Adopted by the Scientific Steering Committee at its meeting of 6-7 March 2003.
42. Opinion on the potential requirement for designation of specified risk materials in **pigs.** Adopted on 6-7 March 2003.
43. Opinion on **necrophagous birds** as possible transmitters of TSE/BSE. Adopted on 7-8 November 2002.

Safety of products

44. Opinion on the safety of **tallow derivatives** from cattle tallow. Adopted on10-11 April 2003.
45. Updated opinion and report on the safety of **dicalcium phosphate (DCP) and tricalcium phosphate (TCP)** from bovine bones, used as an animal feed additive or as fertiliser. Submitted on 6-7 March 2003.
46. Updated Opinion on the safety with regard to TSE risks of **gelatine** derived from ruminant bones or hides. Adopted on 6-7 March 2003.
47. Updated opinion on the safety with regard to TSE risks of **gelatine** derived from ruminant bones or hides. Adopted on 5-6 December 2002.
48. Updated opinion on the safety with regard to TSE risks of **gelatine** derived from ruminant bones or hides. Adopted on 12-13 September 2002.
49. Opinion on **peptides from pig mucosa**: risks with respect to TSEs. Adopted on 21-22 February 2002.
50. Updated opinion on the safety with regard to TSE risks of **gelatine** derived from ruminant bones or hides from cattle, sheep or goats. Adopted on 28-29 June 2001, editorial changes adopted on 6-7 September 2001.
51. Updated opinion on the safety with regard to TSE risks of **gelatine** derived from ruminant bones or hides from cattle, sheep or goats (Including amendments to the scientific report attached to the opinion of 21 January 2000). Adopted on 28-29 June 2001.
52. Revised opinion and report on: The safety of **tallow** obtained from ruminant slaughter by-products. Adopted on 28-29 June 2001.
53. Opinion and report on safety with respect to the TSE risks of **collagen** produced from ruminants hides. Adopted on 10-11 May 2001.
54. Opinion on the safety of **organic fertilisers** derived from ruminants animals. Adopted on 10-11 May 2001.
55. Opinion and report on the safety of **dicalcium phosphate** precipitated from ruminant bones and used as an animal feed additive – Update adopted 27-27 October 2000.
56. Statement of the Scientific Steering Committee on its Report and Scientific Opinion on mammalian derived **meat and bone meal** forming a cross-contaminant of animal feedstuffs. Adopted on 26-27 October 2000.
57. Considerations on the safety of **amino acids from human hair hydrolysate** used in cosmetic products for topical application, with regard to Transmissible Spongiform Encephalopathy risks. Adopted on 25-26 May 2000.
58. Updated Report and Scientific Opinion on the safety of **hydrolysed proteins** produced from bovine hides. Adopted on 25-26 May 2000.

59. Preliminary report on **Quantitative Risk Assessment** on the Use of the Vertebral Column for the production of Gelatine and Tallow. Submitted on13-14 April 2000.

60. Opinion on **Quantitative Risk Assessment** on the Use of the Vertebral Column for the production of Gelatine and Tallow. Adopted on 13-14 April 2000.

61. Scientific report and opinion on the safety of **gelatine** - Updated on 20-21 January 2000.

62. Report and opinion on the evaluation of the "133°/20'/3 bars heat/pressure conditions" for the production of **gelatine** regarding its equivalency with commonly used industrial gelatine production processes in terms of its capacity of inactivating/eliminating possible TSE infectivity in the raw material. Adopted on 21-22 January 1999.

63. Report and Opinion on the safety of **gelatine**. Adopted on 21-22 January 1999.

64. Evaluation of the *ELLCO-FOOD* process for the production of **gelatine** regarding its equivalency with commonly used industrial gelatine production processes in terms of its capacity of inactivating/eliminating possible TSE infectivity in the raw material. Report adopted on 22-23 October 1998 and updated on 10-11 December 1998.

65. Report and Scientific Opinion on the safety of **hydrolysed proteins** produced from bovine hides. Adopted on 22-23 October 1998.

66. Scientific Opinion on the safety of **organic fertilisers** derived from mammalian animals. Adopted on 24-25 September 1998.

67. Report and Scientific Opinion on mammalian derived **meat and bone meal** forming a cross-contaminant of animal feedstuffs. Adopted on 24-25 September 1998.

68. Updated scientific report on the safety of **meat-and-bone meal** derived from mammalian animals fed to non-ruminant food producing farm animals. Submitted on 24-25 September 1998.

69. Report and opinion on the safety of **dicalcium phosphate** precipitated from ruminant bones and used as an animal feed additive. Adopted on 25-26 June 1998.

70. Opinion on the safety of **tallow** derived from ruminant tissues. Adopted on 26-27 March 1998.

71. Opinion on the safety of **meat and bone meal** from mammalian animals, naturally or experimentally susceptible to Transmissible Spongiform Encephalopathies. Amended version adopted on 26-27 March 1998 (and updated on 3 April 1998).

72. Opinion on the Safety of **Gelatine.** Amended version adopted on 26-27 March 1998 (and updated on 3 April 1998).

73. Preliminary Opinion on the safety of **gelatine**. Adopted on 19-20 February 1998.

74. Preliminary Opinion on the safety of **tallow**. Adopted on 19-20 February 1998.

75. Preliminary Opinion on the safety of **meat-and-bone meal**. Adopted on 19-20 February 1998.

Risk reduction strategies

76. Opinion on the additional safeguard provided by different **culling schemes** under the current conditions in the UK and DE. Adopted on 11 January 2002.
77. Report on the additional safeguard provided by different **culling schemes** under the current conditions in the United Kingdom and Germany. Prepared on 10-11 January 2002.
78. Updated opinion on **sourcing of ruminant materials from GBR I** countries for medical devices. Adopted on 29-30 November 2001.
79. Opinion on **Sourcing of from GBR I Countries** (Sourcing of Ruminant Materials from GBR I Countries for Medical Devices). Adopted on 6-7 September 2001.
80. Opinion on the scientific basis for **import bans** proposed by Austria with regard to BSE risks in Germany and France. Adopted on 29-30 March 2001.
81. Opinion on the questions submitted by EC services following a request of 4 December 2000 by the **EU Council of Agricultural Ministers** regarding the safety with regard to BSE of certain bovine tissues and certain animal-derived products. Adopted on 12 January 2001.
82. Opinion of the Scientific Steering Committee (1) on the scientific basis for **import bans** proposed by 3 Member States with regard to BSE risks in France and the Republic of Ireland; (2) on the scientific basis for several measures proposed by France with regard to BSE risks; (3) and on the scientific basis for banning animal protein from the feed for all farmed animals, including pig, poultry, fish and pet animals. Adopted on 27-28 November 2000.
83. Opinion on BSE-related **culling in Cattle.** Adopted on 14/15 September 2000.
84. Scientific Opinion on the conditions related to **"BSE Negligible risk (closed) bovine herds"**. Adopted on 22-23 July 1999.

Surveillance and rapid tests

85. Opinion on the **field trial evaluation** of two new rapid BSE post mortem tests Results achieved using the LIA Test (Prionics) and the aCDI Test (InPro) in the field trial. Adopted on 6 March 2003.
86. Opinion on a programme for the **evaluation of rapid post mortem tests** to detect TSE in small ruminants. Adopted on 7-8 November 2002.
87. Opinion on design of a **field trial** for the evaluation of new rapid BSE post mortem tests. Adopted on 22 February 2002.
88. Opinion on requirements for **statistically authoritative BSE/TSE surveys.** Adopted on 29-30 November 2001.

Recycling of animal by-products and animal waste

89. Opinion on "The proposal for controlled use of ruminant **SRMs as feed for fur animals** in Finland". Adopted on 27 October 2000.

90. Opinion on the risk born by **recycling animal by-products as feed** with regard to propagating TSE in non-ruminant farmed animals. Adopted on 17 September 1999.

91. Report on the risk born by **recycling animal by-products as feed** with regard to propagating TSE in non-ruminant farmed animals. Adopted on 16-17 September 1999.

Disposal of animal by-products and animal waste

92. Opinion on **six alternative methods for safe disposal** of animal by-products. Adopted on 10-11 April 2003.

93. Opinion and report on a treatment of animal waste by means of high temperature (150°C, 3 Hours) and high pressure **alkaline hydrolysis**. Adopted on 10-11 April 2003.

94. A framework for the **assessment of the risk** from different options for the safe disposal or use of animal by-products which might be contaminated with microbiological agents including TSE. Adopted on 10-11 April 2003.

95. Opinion on the use of **small incinerators** for BSE risk reduction. Adopted on 16-17 January 2003.

96. Opinion on **open burning** of potentially TSE-infected animal materials. Adopted on 16-17 January 2003.

97. Opinion on the use of **burial** for dealing with animal carcasses and other animal materials that might contain BSE/TSE. Adopted by the Scientific Steering Committee Meeting of 16-17 January 2003.

98. Updated opinion and report on a treatment of animal waste by means of high temperature (150°c, 3 hours) and high pressure **alkaline hydrolysis.** Adopted on 16 may 2002 and revised on 7-8 November 2002.

99. Opinion and report on the treatment of animal waste by means of high temperature (150°c, 3 hours) and corresponding high pressure **alkaline hydrolysis.** Adopted on 16 may 2002.

100. A framework for the **assessment of the risk** from different options for the safe disposal or use of meat and bone meal (MBM) and other products which might be contaminated with TSE's and other materials. Adopted on 28-29 June 2001.

101. Note on the safe **handling, transport and temporary storage of meat-and-bone meal** which may be contaminated with a BSE agent or other pathogens. Adopted on 26-27 October 2000.

102. Preliminary and incomplete notes on the **safe handling, transport and storage of MBM** and other bovine derived materials which may be contaminated with the BSE agent or other pathogens - Comments compiled on 25-26 May 2000.

103. Opinion 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, rats, laboratory animals and fish) or via condemned materials. Adopted by the Scientific Steering Committee at its meeting of 24-25 June 1999.

104. 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. Submitted on 24-25 June 1999 (Containing updates, 13 July 1999).

BSE epidemic in the United Kingdom

105. Opinion and report on BSE in United Kingdom's **cattle born after 31 July 1996** [BARBs]. Adopted on 10-11 April 2003.

106. Opinion on the six **BARB BSE cases in the UK since 1 August 1996** (the six BARB BSE cases born and confirmed in the UK after 1 August 1996: Is there a need to review the opinions of the Scientific Steering Committee with regard to the UK date-based export scheme and other TSE-related risks?). Adopted on 29-30 November 2001.

107. Opinion on Bovine Spongiform Encephalopathy in a second UK animal **born after 1 August 1996** (Case confirmed in Northern Ireland). Adopted on 29-30 March 2001.

108. Opinion on monitoring some important aspects of **the evolution of the Epidemic of BSE** in Great-Britain. Update providing an epidemiological commentary on BSE projections for Great Britain (GB) and on surveillance, as well as on the occurrence of "Born After the Real Ban - BARB" cases. Adopted on 7-8 December 2000.

109. Report on monitoring Some Important aspects of **the evolution of the Epidemic of BSE** in Great-Britain, Update providing an epidemiological commentary on BSE projections for Great Britain (GB) and on surveillance, as well as on the occurrence of "Born After the Real Ban - BARB" cases. Submitted on7-8 December 2000.

110. Report and Scientific Opinion on **export from the UK of bone-in veal.** Adopted on 14-15 September 2000.

111. Opinion on the UK decision to **lift the ban on the consumption of meat on the bone.** Adopted on 13-14 April 2000.

112. Opinion on the Scientific Grounds of the Advice of 30 September 1999 of the **French Food Safety Agency** (the Agence Française de Sécurité Sanitaire des Aliments, AFSSA), to the French Government on the Draft Decree amending the Decree of 28 October 1998 establishing specific measures applicable to certain products of bovine origin exported from the United Kingdom. Adopted on 28-29 October 1999 (edited following a written procedure (30.10 - 15.11.99) and re-edited on 9-10 December 1999.

113. Summary report based on the meetings of 14 and 25 October 1999 of the TSE/BSE *ad hoc* group of the Scientific Steering Committee on the Scientific Grounds of the advice of 30 September 1999 of the **French Food Safety Agency** (the Agence Française de Sécurité Sanitaire des Aliments, AFSSA), to the French Government on the Draft Decree amending the Decree of 28 October 1998 establishing specific measures applicable to certain products of bovine origin exported from the United Kingdom. Adopted and edited following on 26.10-16.11.99.

114. Opinion on Monitoring some important aspects of the **evolution of the Epidemic of BSE in Great-Britain** (Status, April 1999). Adopted on 27-28 May 1999.

115. Opinion on the safety of bones produced as by-product of the **Date Based Export Scheme.** Adopted on 22-23 October 1998.

116. Report on the UK **Date Based Export Scheme** and the UK proposal on Compulsory Slaughter of the Offspring of BSE Cases. Adopted on 8-9 December 1997 (Re-edited version adopted on 22-23 January 1998.

117. Final Opinion on the **UK-Date Based Export Scheme**. Adopted on 19-20 February 1998.

Geographical BSE risk: methodological aspects.

118. Opinion on the Geographical BSE risk for sheep and goats (GBR-S): adaptation of the cattle GBR methodology to small ruminants, in case BSE in small ruminants would become probable or evident under field conditions. Adopted on 7-8 November 2002.

119. Update of the opinion on the Geographical Risk of Bovine Spongiform Encephalopathy (GBR). Adopted on 7 November 2002.

120. Opinion on the geographical BSE-risk (GBR) and its evolution over time in the European Union Member States (adopted by the SSC at its plenary meeting of 21/22 February 2002. Endorsed on 7 July 2002.

121. Updated opinion on the Geographical Risk of Bovine Spongiform Encephalopathy (GBR). Adopted on 11 January 2002.

122. Final opinion on the Geographical Risk of Bovine Spongiform Encephalopathy (GBR). Adopted on 6 July 2000.

123. Opinion of the Scientific Steering Committee on a method for assessing the Geographical BSE-Risk (GBR) of a country or region. Up-dated on January 2000.
124. Opinion on a method to assess the Geographical BSE-Risk (GBR) of Countries or Regions. Revision adopted on 22-23 April 1999.
125. Preliminary-opinion on a method to assess the geographical BSE-Risk of Countries or Regions. Adopted on 10 December 1998.
126. Opinion on BSE risk. Adopted on 26-27 March 1998.
127. Opinion on defining the BSE risk for specified geographical areas. Adopted on 23 January 1998.
128. Preliminary Opinion on BSE risk. Adopted on 19-20 February 1998.
129. Final Opinion on the contents of a "Complete dossier of the **epidemiological status** with respect to TSEs". Adopted on 19-20 February 1998. Adopted on 19-20 February 1998.

Geographical BSE risk (GBR): Opinions on the GBR of countries

130. Opinion on the Geographical risk of bovine spongiform encephalopathy (GBR) in **Paraguay**. Adopted on 10-11 April 2003.
131. Opinion on the Geographical risk of bovine spongiform encephalopathy (GBR) in **Uruguay**. Adopted on 10-11 April 2003.
132. Opinion on the Geographical risk of bovine spongiform encephalopathy (GBR) in **Brazil**. Adopted on 10-11 April 2003.
133. Opinion on the Geographical risk of bovine spongiform encephalopathy (GBR) in **Argentina**. Adopted on 10-11 April 2003.
134. Opinion on the Geographical risk of bovine spongiform encephalopathy (GBR) in **Chile**. Adopted on 10-11 April 2003.
135. Opinion and report on the Geographical risk of bovine spongiform encephalopathy (GBR) in **Costa Rica.** Adopted on 10-11 April 2003.
136. Opinion on the Geographical risk of bovine spongiform encephalopathy (GBR) in **Belarus**. Adopted on 10-11 April 2003.
137. Opinion on the Geographical risk of bovine spongiform encephalopathy (GBR) in **FYR Macedonia**. Adopted on 10-11 April 2003.
138. Opinion on the Geographical risk of bovine spongiform encephalopathy (GBR) in **Estonia**. Adopted on 10-11 April 2003.
139. Opinion on the Geographical risk of bovine spongiform encephalopathy (GBR) in **Lithuania**. Adopted on 10-11 April 2003.
140. Opinion on the Geographical risk of bovine spongiform encephalopathy (GBR) in **Cyprus**. Adopted on 10-11 April 2003.

141. Opinion on the Geographical risk of Bovine Spongiform Encephalopathy (GBR) in **Singapore**. Adopted on 06 March 2003.

142. Opinion on the Geographical risk of Bovine Spongiform Encephalopathy (GBR) in **New Caledonia**. Adopted on 06 March 2003.

143. Opinion on the Geographical risk of Bovine Spongiform Encephalopathy (GBR) in **Greece**. Adopted on 06 December 2002.

144. Opinion on the Geographical risk of Bovine Spongiform Encephalopathy (GBR) in the **Principality of Andorra**. Adopted on 06 December 2002.

145. Opinion on the Geographical risk of Bovine Spongiform Encephalopathy (GBR) in **New Zealand**. Adopted on 07 November 2002.

146. Opinion on the Geographical risk of Bovine Spongiform Encephalopathy (GBR) in **Israel**. Adopted on 13 September 2002.

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ANNEX III

COMMUNITY LEGISLATION ON BSE SINCE MID-1997, REFERRING TO SCIENTIFIC OPINIONS

Legal text *	Contents
D 97/534/EC of 30 July 1997	Prohibition of the use of SRM (mainly brain, eyes and spinal cord)
D 97/866/EC of 16 December 1997	Postponement to 1/4/1998 of the date of application of D 97/534/EC (SRM)
D 98/272/EC of 23 April 1998	Epidemio-surveillance for all animal TSEs
Rec. 98/477/EC of 22 July 1998	Information necessary to support applications for the evaluation of TSE status
D 98/653/EC of 18 November 1998	Total ban on dispatch of live cattle and all cattle products from Portugal (Portugal embargo)
D 98/692/EC of 25 November 1998	Amendment of the UK embargo - Principles of the second step towards lifting the ban under the Date-based Export Scheme applicable in the entire UK
D 1999/129/EC of 29 January 1999	Amendment of D 94/381/EC – Hydrolysed proteins
D 1999/534/EC of 19 July 1999	Conditions for the production of MBM and tallow (Repeals D 96/449/EC)
D 1999/881/EC of 14 December 1999	Postponement to 30 June 2000 of the date of application of D 97/534/EC(SRM)
D 1999/724/EC of 28 October 1999	Health rules on gelatine
D 2000/374/EC of 5 June 2000	Amendment of D 98/272/EC – Introduction of rapid post-mortem test in monitoring for BSE
D 2000/418/EC of 29 June 2000	Prohibition of the use of SRM (Repeals D 97/534/EC)
D 2000/764/EC of 29 November 2000	Amendment of D 98/272/EC – Reinforcement of the surveillance
D 2000/766/EC of 4 December 2000	Temporary ban on use of MBM
D 2001/2/EC of 27 December 2000	Amendment of D 2000/418/EC – Extension of the list of SRM (bovine intestines)

Legal text *	Contents
D 2001/9/EC of 29 December 2000	Conditions for feeding certain animal proteins
D 2001/25/EC of 27 December 2000	Prohibition of the use of dead animals in the production of animal feed
D 2001/165/EC of 27 February 2001	Amendment of D 2001/9/EC – Hydrolysed proteins
D 2001/233/EC of 14 March 2001	Amendment of D 2000/418/EC – Extension of the list of SRM (vertebral column)
D 2001/270/EC of 29 March 2001	Amendment of D 2000/418/EC – Imports from third countries
D 2001/384/EC of 3 May 2001	Amendment of D 2000/418/EC – Extension of the list of derogating third countries
R 999/2001 of 22 May 2001	Prevention, control and eradication of certain TSE
R 1248/2001 of 22 June 2001	Amendment of R 999/2001 – Surveillance and testing
R 1326/2001 of 29 June 2001	Amendment of R 999/2001 – Transitional measures
R 270/2002 of 14 February 2002	Amendments of R 999/2001 – SRM, surveillance, animal feeding and placing on the market of ovine and caprine animals and products thereof
D 2002/670/EC of 20 August 2002	Amendment off D 98/256/EC – Adaptation of some DBES conditions
R 1494/2002 of 21 August 2002	Amendment of R 999/2001 – continuing of BSE testing in fallen stock
	Deletion of the restrictions on trade of bovine embryos – Clarifications of some SRM rules
R 1774/2002 of 3 October 2002	Health rules concerning animal by-products not intended for human consumption
D 2002/1003/EC of 18 December 2002	Survey of prion protein genotypes in sheep breeds
R 260/2003 of 12 February 2003	Culling rules for scrapie
D 2003/100/EC of 13 February 2003	Breeding programmes for sheep