Report on : The Risk Born by Recycling Animal By-Products as Feed with Regard to Propagating TSE's in Non-ruminant Farmed Animals. Prepared by a Working Group for the Scientific Steering Committee as an input in the elaboration of the opinion on the same subject adopted on 16-17 September 1999

1. The questions:

The Scientific Steering Committee (SSC) was requested to address the following questions:

- *What evidence is there for and against the possibility of the occurrence of sporadic TSEs in pigs, poultry, fish or other species which are fed animal/fish by-products* [1] *?*

- *What incidence of transmissible spongiform encephalopathies can be expected as a consequence of recycling animal by-products as feed within a species should a case of TSE occur? At what stage (incidence level) would this be detectable?*

The resulting risk assessment should contain a cost benefit analysis and consider the implications for farming and aquaculture practices and changes in this area.

A special Working Group was created to provide inputs to the SSC in its deliberations and the preparation of an opinion on the above questions. The Working Group prepared the present report. It is available on internet as a separate document.

2. Scope

a. The cost benefit aspects and the implications for farming and aquaculture practices and changes in this area, were not addressed by the Working Group as they were not included in the mandate given by the SSC.

b. The present report, on purpose, does not cover the ethical part of the issue of intra-species recycling of animals, as this was not part of the mandate given to the SSC.

c. The present report deals with animal by-products of pigs, poultry, fish and ruminants, not being fallen stock, nor condemned material as defined in the opinion on "Fallen Stock" [2] of the SSC, adopted on 24-25 June 1999.

The Working Group considers that the following ruminant-derived products, when used as a feed or feed-ingredient (additive) should not be considered as being possibly "ruminant intra-species recycled", provided the conditions (including sourcing, pre-treatment, processing and purification) are applied as specified in the corresponding opinions adopted by the Scientific Steering Committee in 1998 and 1999: milk, gelatine from bones or hides/skins, dicalcium-phosphate from bones, hydrolysed proteins from hides and tallow derivatives.

For milk, the Working Group refers to the opinions adopted by the SVC, SSC and SEAC

For the other products, the (chemical) composition and characteristics of the original animal organic molecules can be considered as sufficiently eliminated or modified/reduced into a compound which is also available from other sources and would normally be fed or administered.

However, with respect to TSE risks, the risk assessments presented in the SSC opinions on the safety of these products, including the opinion on "Fallen stock" remain valid. This means, for example, that the raw bone material for the production of dicalcium phosphate or gelatine should not be sourced from animals from high risk countries (except if

they comply with DBES-like criteria), because the production process is not considered to be severe enough to eliminate all infectivity and/or because the final products is likely to always contain impurities which could be a source of infection.

d. As requested in the mandate, the working group addressed the issue of intra-species recycling via (orally consumed) *feed*. However, there are recent scrapie being transmitted to sheep and goats in Italy with a vaccine against *Mycoplasma agalactiae* being implicated as the means by which the animals became infected. (Capucchio *et al,* 1998; Agrimi *et al,* 1999). The vaccine was prepared from homogenised sheep brain and mammary tissue. In the United Kingdom in the 1930s there has been a similar incident with sheep vaccinated against louping ill in which the vaccine was prepared from sheep brain (Gordon, 1946; Greig, 1950). This issue should be addressed separately, but given the high efficiency of certain ways of transmission of TSEs (e.g., parenteral), it seems indicated to recommend extreme prudence and not to extrapolate the conclusions from the working group to other ways of intra-species recycling, *for example* via pharmaceutical products, vaccines, etc.

e. The present report, as requested in the mandate, deals only with the risks related to TSEs. However, the Working Group signals that intra-species recycling should also form the object of an evaluation which puts the issue in the broader context of:

- The risks resulting from the possible future emergence of as yet unknown unconventional agents. The possible existence of "as yet unknown" agents is a fact which should not be ignored as the "BSE crisis" has clearly shown. Their sudden appearance may eventually result in major risks to humans, animals and the environment which should be mitigated as much as possible.

- The feeding of herbivorous animals with animal proteins derived from the same or other species.

- The application of standard procedures in epidemiology and "good disease control practices", for example to control species-specific epidemics by removing susceptible animals of the same species (e.g. depopulation in Classical Swine Fever campaigns).

- The biological mechanisms behind inter-species barriers. If intra-species recycling is applied, the ability to protect the system by the inter-species barriers may be lost. In this context the question should also be addressed whether the standard procedures of handling animal materials and waste (e.g. "133°C/20'/3 bars") modify the effects or importance of the inter-species barrier.

- Many infections are totally or partly species-specific, but infectivity may in some cases adapt to new host species. In this context the possible emergence and propagation, after several cycles of recycling, of micro-organisms that are resistant to the standard recycling/rendering processes could also be mentioned.

3. Background

The main part of animal by-products are slaughter by-products originating from healthy [3] slaughter animals. Large volumes of these by-products are processed into feed constituents. These processed feed constituents represent very often a highly nutritious compound for animal feed production. Inside the EU there is an annual production of about 2.9 million ton animal meal, besides that, large volumes of slaughter by-products are processed directly into petfood and fur animal feed.

Slaughtering and further processing of pork and poultry is in many occasions restricted to specialised plants. The volume of pork, poultry and fish slaughtering in the EU is represented in the table hereafter.

		PORK [1] x1.000.000 tons		POULTRY [2] x1.000.000 tons	
Annually,	live weight	meat produced	live weight	Meat produced [3]	

15 EU countries:	slaughtered	[3]	slaughtered	
1997	20,8	16,3 (78%)	11,2	8,3 (75%)
1996	20,9		10,8	

[1]LEI, The Netherlands, 1998 (personal communication); [2] PVE, The Netherlands, 1998; [3] (personal communication). Meat as it leaves the slaughterhous).

Of the animals slaughtered in the EU certain parts are not intended for human consumption, either because of hygienically standards or as a result of costs being too high for further processing. There exist also considerable differences between the individual slaughter and processing companies as a result of the differentiating specific markets, e.g. several companies produce boneless meat products and thus dispose of large volumes of bones and other small parts of meat. As the market for meat and meat products becomes more and more consumer oriented, increasing volumes of slaughter by-products are not intended anymore for sale to the consumer and reside therefore at the slaughterhouse and, or the meat processing plant. Of the life weight at slaughter the next volumes are disposed of as by-products not intended for human consumption (this includes parts that leave the carcass at slaughter and further meat processing):

	Pigs	Poultry	Fish [1]
% of life weight of an animal at slaughter / catch not intended for human consumption (this includes the gut content)	20 to 40%	30 to 55%	15 to 30%

(Van Sonsbeek *et al*, 1997; [1] large volumes of fish are caught exclusively to produce fishmeal, e.g. in Denmark)

When an average of 30% of the slaughtered weight is used to give an impression about the volume of slaughter by-products not intended for human consumption, but originating from healthy animals, this volume in the EU will be 9 million tons of pork and poultry, equivalent to approximately 3 million tons of animal meal and fat. These by-products are at the moment not only processed at the rendering plants. Large quantities are processed into petfood and fur animal feed. The produced volume of fishmeal in the EU is approximately 0.6 million tons annually (FIN, 1998).

Besides these by-products, slaughterhouses and filleting plants also produce sludge. This is the residue that remains after the treatment(s) of the process water. The volume (on wet basis) of this sludge is in general approximately 30% of the volume of slaughter by-products, but it has a dry matter content of 3 to 8%, whereas slaughter by-products have a dry matter content of approximately 25 to 30%. This sludge originates in general from residues of the animals slaughtered at that particular plant (Fransen *et al*, 1996). Several slaughterhouses and filleting plants have also fat-traps, the residues collected here are either mixed with the slaughter by-products or in the sludge.

In general slaughter by-products are processed into animal feed constituents that can be used in feeding of several species of animals. This implies that intra-species recycling is in general possible and should be considered to be the worst case. The way these risks can be mitigated is dealt with in this opinion.

4. Identification of possible hazards and elements of risk assessment

4.1. *Introduction*:

Within the frame of the present report, the following possible origins of a TSE occurring in ruminants, pigs, poultry and fish, are considered:

- A species-specific TSE agent (either a full species specific or a TSE originating from another species following adaptation in the new host species), present in a given species. The incidence of a larger or smaller part of the cases can be explained by feeding of infected feedstuffs derived from the same species; (for example: BSE in cattle)

- An species-specific TSE agent, present in a given species. The incidence of a case cannot be explained by feeding of infected feedstuffs alone, but must (also) be explained by other reasons, e.g., vertical transmission (for example: scrapie in sheep).

- An non-species-specific (or not yet adapted) TSE agent present in a given species. The incidence of a larger or smaller part of the cases would be explained by feeding of infected feedstuffs derived from another animal species. (This would, *for example*, be the case if BSE infectivity was still present in pigs fed with infected ruminant MBM, but the pigs didn't show any propagation of the agent nor symptoms of any SE.)

The risk that clinical TSE occurs in animals fed with feed constituents derived from animal by-products (from whatever animal origin, including the risk that a TSE is transmitted through animals not (yet) necessarily showing clinical signs and hypothetically *via* healthy looking "silent" carriers), depends upon:

a. the susceptibility of the species to get infected with TSEs through the oral route;

b. the susceptibility of the individual animal (e.g., based on genetic background) to get infected with TSEs through the oral route;

c. the load of infectious particles in feed rations;

d. the species specificity of the infectious particle fed, and

e. the possibilities of TSE-agents to surpass the digestive tract of ruminants, pigs, poultry and fish without loosing infectious properties.

4.2. *Assessment of the available evidence*:

a. Pigs

Several experiments have been reported to investigate the experimental transmission of TSE to pigs. Specifically they include the agents responsible for kuru, BSE and scrapie.

In regard to kuru, Gibbs, Gajdusek and Amyx, (1979) reported the unsuccessful transmission of eight strains of kuru to pigs following parenteral challenge with human brain material. The pigs were kept for 52 - 76 months and no histological evidence of spongiform encephalopathy (SE) was found.

In regard to BSE, parenteral and oral challenge of pigs with brain material from cattle naturally affected with BSE have been described and reported (Dawson *et al*, 1990, 1991, 1994, Animal Health 1996,1997). In the parenteral study, pigs were challenged by the combined i/c, i/p and i/v routes using a total of l g of brain tissue for each pig. Clinical and pathological evidence of spongiform encephalopathy was found in seven of ten pigs. Two died early in the incubation period from intercurrent disease, and in the third pig, sacrificed whilst clinically healthy two years into the incubation period, showed no evidence of SE.

In the oral challenge study, ten pigs were challenged with a total of 4 kg of brain material from cattle with confirmed natural BSE. The material was fed on three occasions at intervals of one to two weeks. No clinical or pathological evidence of TSE was found in the pigs up to seven years post-challenge. Tissues from these pigs have been inoculated into mice. No detectable infectivity was found in neural and non-neural tissues from some pigs killed two years after challenge. The bioassay of tissues from pigs killed at the termination of the experiment is still in progress. None has been reported positive so far (Hawkins *et al*, 1998; and Hawkins, personal communication 1999).

Pigs have also been challenged orally with brain tissue from sheep confirmed to have scrapie in a similar manner

(Animal Health 1996,1997). The experiment is still running but no pig has shown evidence of a TSE disease to date over 63 months from the date of challenge. Bioassay of tissues from pigs killed two years following challenge is still in progress. None has been reported positive so far (Hawkins, personal communication).

In 1997 an incident from 1979 was reported in the USA in which slaughter pigs seemed to show neurological signs and microscopic evidence of encephalopathy (Hansen and Halloran, 1997). In one pig out of 60 examined, neuronal vacuolization and gliosis were found. The affected pig was 6 months old. Subsequent re-examination of the material showed that the lesions were not pathognomonic of those seen in TSE. Also the young age of the animal would argue against the diagnosis of a TSE. The conclusion was that no evidence was provided of the possibility of a previously unrecognized disease or TSE being present in pigs (L.Detwiler, personal communication [4]).

No reports in the world literature describing a naturally occurring TSE in pigs have been found.

b. Poultry (= domestic fowl or chickens)

Chickens have been challenged by parenteral and oral routes with brain material from cattle confirmed to have natural BSE, (Dawson *et al*, 1991, 1994, Animal Health, 1996,1997).

In regard to the parenteral study 12 chicks were inoculated i/c with 50 m l of a 10% saline suspension of pooled brain stem at one day old. A further 1ml was inoculated i/p when the chicks were 2 weeks old. No evidence of spongiform encephalopathy was found at the conclusion of the study. Sub-passage is in progress but no results are yet available (Hawkins, personal communication).

In regard to the oral study 11 birds were challenged with 5g of a pool of brain tissue from two cattle with confirmed BSE on three occasions when the birds were 4, 5, and 6 weeks of age. The material was deposited in the distal oesophagus/crop. No evidence of spongiform encephalopathy was found. Sub-passage is in progress but no results are yet available (Hawkins, personal communication)

Schoon et al (1991a, 1991b) report on a case of spongiform encephalopathy-like clinical symptoms observed in 1986 in ostriches in a German Zoo. Histopathological examination showed vacuolation in the brain. However (1999 personal communications from: Dormont, D., Groschup, M., Heim, D., Hope, J., Matthews, D., Schreuder, B.E.C., Taylor, D.M., Taylor, D.W., Ulvund, M., Vandevelde, M., Vanopdenbosch, E., Wells, G.G.A.), there are no indications at present that these birds suffered from a transmissible prion disease. It has not been shown that the lesions have been associated with PrP accumulation and the disease has not been experimentally transmitted to the same or any other species. No immunochemsitry has been done and there are doubts that the lesions were really scrapie-like. They may have represented some other form of spongy degeneration of the CNS akin to many metabolic diseases described in other species. (According to some pathologists (Wells, G.G.A., pers.comm.), apparently incidental neuronal vacuolation occurs in ostriches, much as is seen in most mammalian species. Wells G.G.A (1999, pers.comm.) examined a single section from the brain of one bird. in the medulla: there was neuronal vacuolation involving mainly a nucleus equivalent to the dorsal parasympathetic nucleus of the vagus nerve in mammals, where the vacuolation was essentially like that seen in scrapie. However, there were features of this which were not entirely typical, particularly a wide variation in the range of size of the vacuoles. Some other neurones in the medulla also showed very fine cytoplasmic vacuolation. Also there was localised vacuolar change within white matter in a different part of the medulla which resembled that produced by a number of metabolic and toxic disorders. It was, therefore, not possible to resolve from this examination the true nature of the disorder.

c. Fish

So far, no evidence for TSE in fish was found. Alderman (1996, communication *via* the UK SEAC secretariat) reports that the Fish Diseases laboratory at Weymouth (UK) has for 25 years been involved in studying the diseases of marine and freshwater fish. During that time the laboratory has not observed any scientific evidence of any condition which might in any way be described as a spongiform encephalopathy in fish, whether of species used to produce fishmeal, or directly for human food, from the UK, other EU member states or from elsewhere in the world. [5]

What precedes is confirmed by Professor Hugh Ferguson of the Institute of Aquaculture at Stirling University (SEAC, 1999, communication to the SSC secretariat). He reports that fish brains are examined quite frequently, and in young fish often as a result of investigations for gill infections [6]. As there are recognised diseases of fish that could cause vacuolisation, fish experts are conscious of concerns about TSEs. Nothing suggestive of a TSE has been found however.

FIN (1999) reports that farmed marine fish feed is mainly composed of fish meal and fish oil, completed with small amounts of vegetable oil and minerals, vitamins etc. Freshwater fish such as trout, carp, etc. are unlikely to receive any fish material other than in the form of fish meal and fish oil. However, according to information obtained from rendering companies, mammalian-derived materials may be used as an ingredient for feeding farmed marine and freshwater fish.

It should further be noticed that a EC funded project FAIR5-CT97-3308 entitled " *Separation, identification and characterization of the normal and abnormal isoforms of prion protein from normal and experimentally infected fish*" has started on 1/3/1998 for three years, with the following objectives:

i. the characterization of the normal isoforms of fish PrP and its coding nucleotide sequence;
ii. an attempt to transmit experimentally TSE material from ovine and bovine to fish;
iii. the setting up of a sensitive and specific diagnostic test for PrP detection in fish tissues;
iv. the evaluation of the uptake and binding of normal fish PrP.

The final outcome should contribute to the assessment of the possibility of transmission of TSE to fish, the evaluation of the potential risk connected to fish derived foods for human and animal, the establishment of analytical protocols for PrP detection in fresh fish food and the comparison of the molecular properties of normal and abnormal isoforms of PrP.

d. Ruminants

A large number of experiments, abundantly reported on in the scientific literature, has shown that cattle and sheep are susceptible to TSE's originating from their own species and that ruminants in general fed with infectious material originating from the same species can be infected with TSE's. Also, experimental evidence (EC, 1998) shows that BSE can be transmitted to sheep (and goats) via the oral route [7].

If a spontaneous TSE occurred in cattle, one might reasonable have expected this to have occurred in detectable levels of a BSE-like disease in a much larger number of countries than presently is the case, and where the rendering systems used are very much alike those used in the EU prior to 1992, that implies not in accordance with the EU rendering directive 90/667. The potential occurrence of spontaneous TSE in cattle has till yet not lead to detectable levels of cattle TSE in most countries, and in countries where TSE in cattle occurs, this has so far not been attributed to spontaneous cases. In fact, most of the BSE incidence in countries where native BSE occurs, is accounted for by feeding of infected or contaminated feedstufs. It must nevertheless be mentioned that, following epidemiological investigations after the occurrence of a case, not all BSE cases can always be brought back to proven feeding practices. (E.Vanopdenbosch, 1998, personal communication)

If scrapie would also spontaneously occur in sheep, it would more likely occur in those sheep with the most susceptible PrP genotypes. However, it is now known that a significant proportion of Australian and New Zealand sheep have such genotypes, but have not developed scrapie (Hunter *et al*, 1997).

e. Pigs, poultry and fish as possible silent carriers

Marsh *et al* (1969) reported the recovery of transmissible mink encephalopathy (TME) infectivity from the spleen of one chicken and from the spleen, caecum, tonsil and bursa of Fabricius of a second chicken of two chickens challenged experimentally by i/v inoculation of fourth passage mink brain with TME. They noted that infectivity administered either intra-cranial, intra-venous, intra-muscular or subcutaneously, persisted for extended periods (30 and 50 days in the case of chickens) in lymphoid tissues of rhesus monkeys, chickens, mice, cats, ferrets, goats and calves that were

studied. No experimental data are available about oral infectivity tests.

Race and Chesebro (1998), reported the results of i/c challenge of mice with hamster scrapie strain 263K that produces no clinical disease in mice, followed by sub-passage from brain and spleen into further mice and into scrapie susceptible hamsters. Infectivity was detected in the spleen and brain tissues by the hamsters, but not by the mice. The authors' view was that the mice had not replicated the agent. They noted that they had not tested to see if the same results were obtained after oral challenge. However, they suggested that food animal species resistant to BSE, such as poultry, exposed to BSE infectivity *via* feed but might show persistent infectivity in their tissues without replication.

Over 80% of pig meat and 80% of poultry meat produced in the EU originates from pigs less than 8 months of age and broilers less than 2 months of age respectively. However, the life expectancy of both pigs and chickens raised for slaughter may be too short to show any signs of SE-s whenever they are infected. Taking account of all our knowledge on prion diseases in animals, it is unlikely that clinical evidence of disease would occur at such a young age. Only adult breeding pigs would be expected to be old enough to exhibit clinical signs if ever a TSE of pigs was found. However, it can be hypothesized that infectivity of extra-neural tissues, particularly lymphoreticular tissues, could theoretically arise in these species exposed to TSE infection *via* feed whether or not replication and neuroinvasion subsequently occurred. The results of the studies using the BSE agent mentioned above do not support the hypothesis that infectivity can be sequestered in the manner described and particularly this is a unlikely event in pigs exposed to the BSE agent by the oral route two years earlier. It is noted however, that these studies used mice to detect any infectivity, cattle would be more susceptible. Furthermore the results of bioassays done at the termination of the porcine studies are still awaited as are those from poultry (see also sections 7.a and 7.b).

However, special attention should be drawn to the eventuality that in pigs under natural conditions the intestinal barrier is would be very efficacious in respect to the development of clinical TSE, but that by feeding infected ruminant MBM and/or intraspecies recycling of pigs with low levels of infectivity, an increasing level of infectivity could be built up in the intestine. Such low levels could only be detected by the most sensitive methods i.e. intracerebral inoculation of calves with intestinal tissue of orally exposed pigs. This would be in accordance with the results of the experiments of Race and Cesebro to detect infectivity in resistant species.

f. Risks related to the content of the gut and to manure (faeces)

The content of the gut and manure represent an increased risk, if the animals were fed with (possibly TSE-contaminated) ruminant material **ev**en if it was previously treated at "133°C/20'/3 bars", because this standard is considered not to eliminate all possible TSE infectivity if the initial titer was high.

If the presence of a BSE risk is not excluded, these materials should therefore be considered as "condemned materials" as described in the SSC's opinion on "Fallen stock" of 24-25 June 1999. Provided they are appropriately processed and if any TSE risk is excluded, they could be recycled into industrial products or fertilizers. However, if a TSE risk exists, they should be disposed of.

5. Conclusions from the Working Group

Concerning the susceptibility of **pigs, poultry and fish** to become infected with TSE's, there is evidence that pigs can become infected with BSE through intra-cerebral inoculation with infectious BSE material. Infectivity could be recovered from poultry inoculated *via* the i/v route with TME. No evidence was found of TSE's in fish. Till date no experiments have shown that pigs, poultry and fish could be infected with TSE through the oral route.

The hypothesis proposed that orally TSE-inoculated non-ruminants without any signs of disease could carry over the TSE-infection through there tissues has till date not been proven.

Concerning **ruminants**, a large number of experiments, abundantly reported on in the scientific literature, has shown that cattle and sheep are susceptible to TSE's originating from their own species and that ruminants in general fed [8] with infectious material originating from the same species can be infected with TSE's. Should a country be free of any animal TSE, epidemiological evidence suggests that the onset of an endemic of a certain TSE based on a spontaneous

native case of TSE is very unlikely.

6. General remark on the safety of derived products

Recycling of animal by-products processed into basic biochemical substances as fat and protein is recognised as an effective way of re-use of valuable materials. When an animal is decomposed through processing into protein, fat and other basic biochemical materials, consumption of this material is not anymore recognised as being intra-species recycling.

Besides the feed-value of these slaughter by-products, effective disposal and processing is of importance to protect human and animal health and to preserve the environment. An effective system that prevents the uncontrolled dispersion of slaughter by-products in the environment is important to preserve human and animal health. Longitudinal integrated safety assurance (LISA) should be implemented based on the HACCP concept to assure the safety of the processed products and to make them available for the market. From environmental point of view protection strategies should be directed towards: firstly a reduction of waste and secondly towards a full re-use of waste. The use of energy and the production of waste water and odour should be implemented in the strategies to comply with these policies.

Intra-species recycling can be acceptable when the material of origin is from epidemiological point of view safely sourced with regard to TSE's and treated accordingly to prevent any spread of conventional diseases.

7. Non-exhaustive list of the consulted literature and documents

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SSC (Scientific Steering Committee of the European Commission):

Scientific opinions :

- Safety of Gelatine, last update,19/2/99
- Safety of Meat and Bone Meal (MBM) from mammalian animals, naturally or experimentally susceptible to Transmissible Spongiform Encephalopathies. 27/3/98
- Safety of Tallow, 27/3/98
- Safety of Dicalcium Phosphate precipitated from ruminant bones and used as an animal feed, 26/6/98
- Safety of Hydrolysed Proteins produced from bovine hides, 23/10/98
- Safety of Organic Fertilizers derived from mammalian animals, 25/9/98
- Risk of Infection of Sheep and Goats with the Bovine Spongiform Encephalopathy agent, 25/9/98
- "Fallen Stock": 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, 23/7/99

Reports of Working Groups

- Report on the safety of meat and bone meal derived from mammalian animals fed to non-ruminant food-producing farm animals, 25/9/98
- Report on the possible vertical transmission of Bovine Spongiform Encephalopathy (BSE),19/3/99.

Opinions of the SSC and related Reports of Working Group are published on the Internet under *http://ec.europa.eu/dg24/health/sc/ssc/outcome_en.html* as soon as possible after the adoption of the opinions by the SSC.

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[1] Intra-species recycling of fur animals is discussed in the SSC opinion on "Fallen stock", adopted on 24-25.06.99

[2] See the "Fallen stock" opinion.

[3] Healthy animals are defined as animals which have undergone an ante mortem inspection by an official veterinarian where it was determined that the animals were not suffering from a disease which is communicable to man and animals and that they do not show symptoms or are in a general condition such as to indicate that such disease may occur and they show no symptoms of disease or of a disorder of their general conditions which is likely to make their meat unfit for human consumption. (Definition as given in Directive 64/433/EEC, laying down the rules for ante mortem inspection)

[4] Based also on the following USA documents: (1) Dr.W.J.Hadlow's Report of 10.04.97 on the microscopic examination of pig brain N° 2709, (2) Dr.J.Miller's comments of 31.03.97 on the incident and (3) H.W.Moon's review of 31 March of the pathology reports of the pigs.

[5] According to Alderman (1996) there are a few recognised diseases of viral and protozoal aetiology which affect nervous tissues of farmed and wild fish which result in pathologies and which, whilst they may be described as encephalopathies, can not in any way be confused with spongiform encephalopathy group of diseases, which include BSE, CJD and scrapie either in their gross, behavioural or pathological characteristics. Such viruses and protozoans are regarded as being extremely host specific and adapted for cold blooded animals.

[6] It is easier to section the entire head, thus including the brain, than to concentrate only on gill.

[7] See also Section 2 Scope, on other ways of transmission.

[8] See also Section 2 Scope, on other routes of transmission.

[9] On the latter sentence, there was no consensus, as some found it misleading: if epidemic BSE originated from

recycling of sporadic BSE, this could have happened everywhere with the right conditions.