European Commission

Better Training for Safer Food

Initiative

TSE/BSE: an introductory overview

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Consumers, Health And Food Executive Agency

European Commission

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Chapter 1. WHAT ARE TSEs

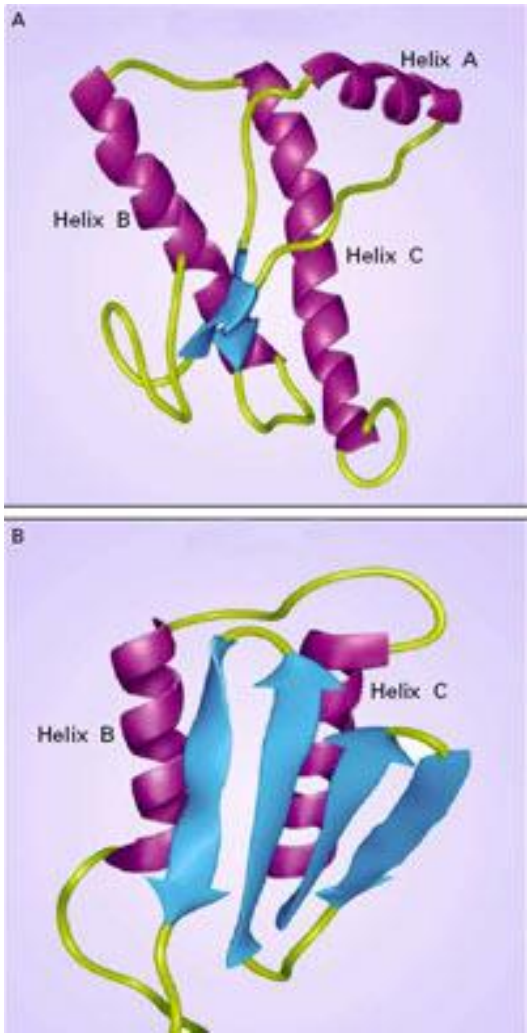
TSEs are neurological diseases, caused by proteinaceous infectious particles, the '**prions**'

Misfolding of physiological form of PrP, PrP^{c} (α-helix), to disease-associated form, **PrP^{res}** (β-sheet)

- Function of PrP still unknown but **PrP^{res} in brain is a consistent infectivity marker**
- Existence of **multiple protein conformations may explain different strains**

PrP **gene sequence may influence susceptibility** to disease (intra- and inter-species)

Infectious, spontaneous (sporadic), hereditary forms of prion diseases occur in animals or humans



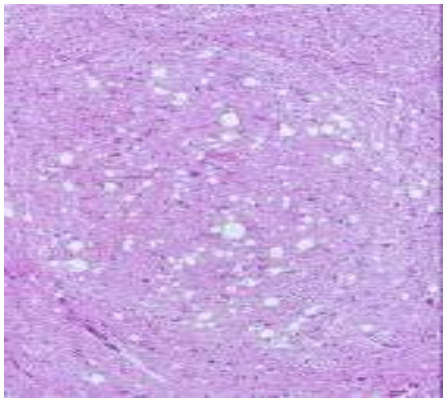
What are TSEs?

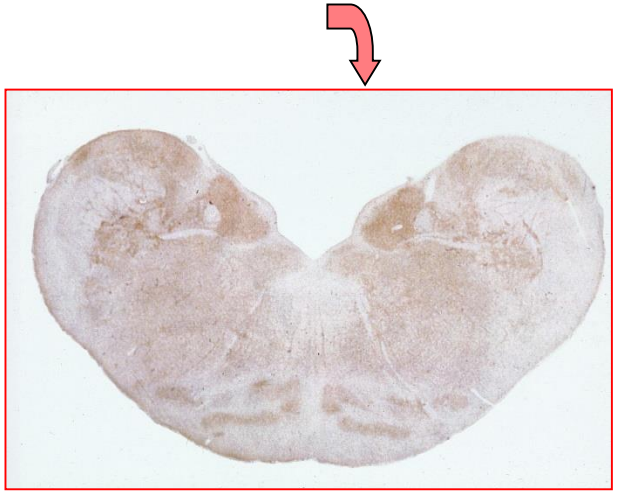
T*ransmissible (or sporadic/genetic)*

S*pongiform lesions in CNS (or not)*

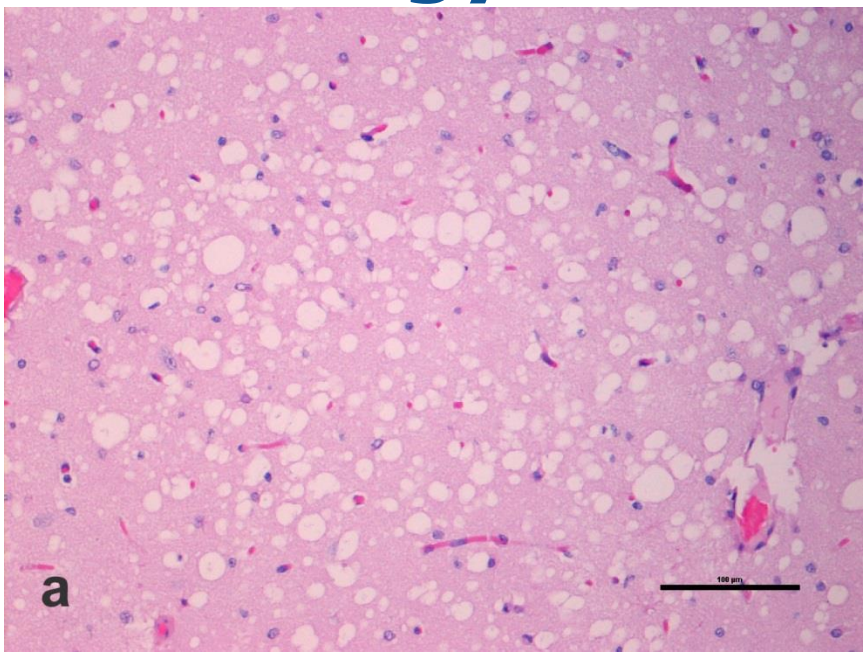
E*ncephalopathy – lesions primarily found in the brain (and/or spinal cord, tonsils, lymph nodes, gut, liver, kidneys, adrenals, eyes…)*

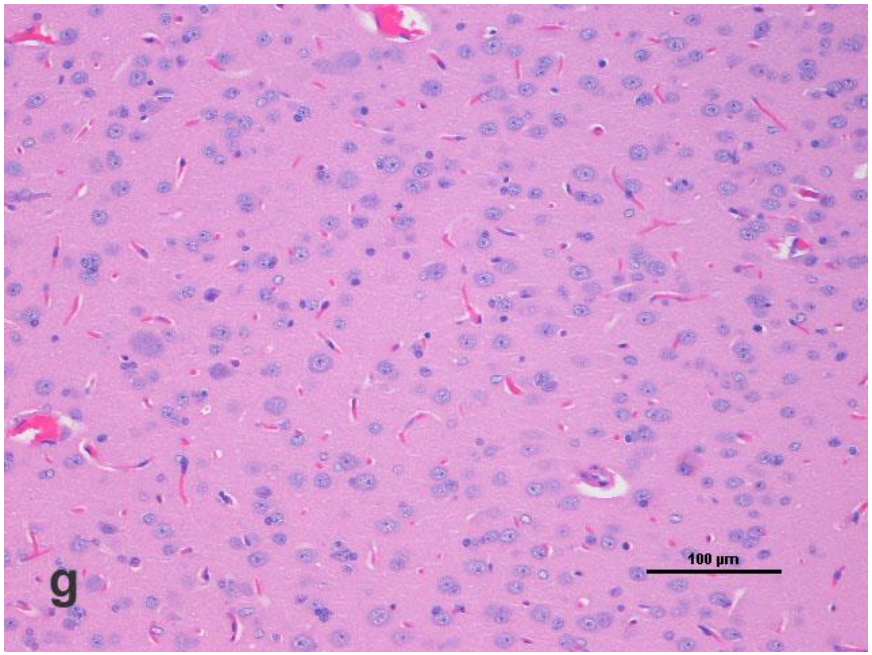
Current definitions now all include the presence of PrP^{res}

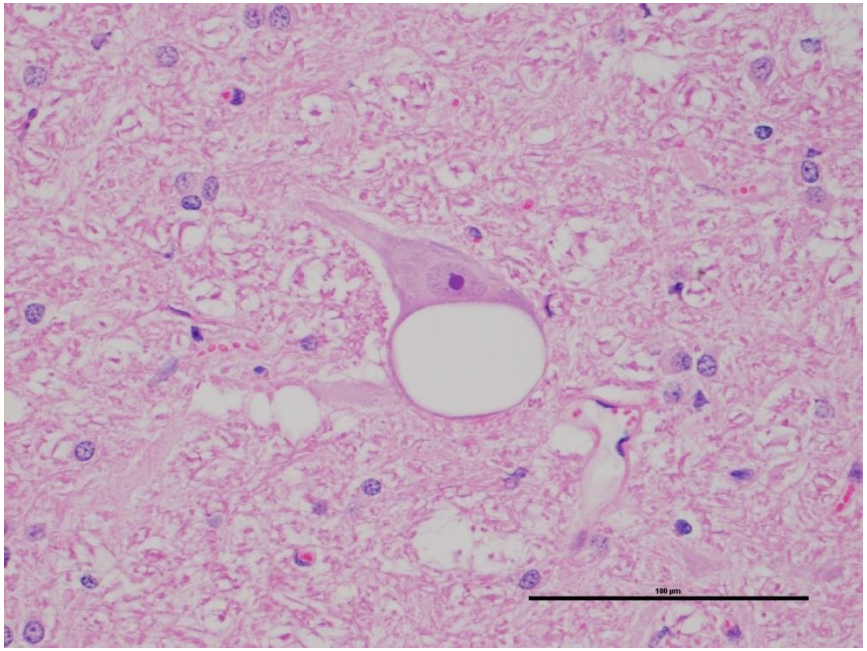


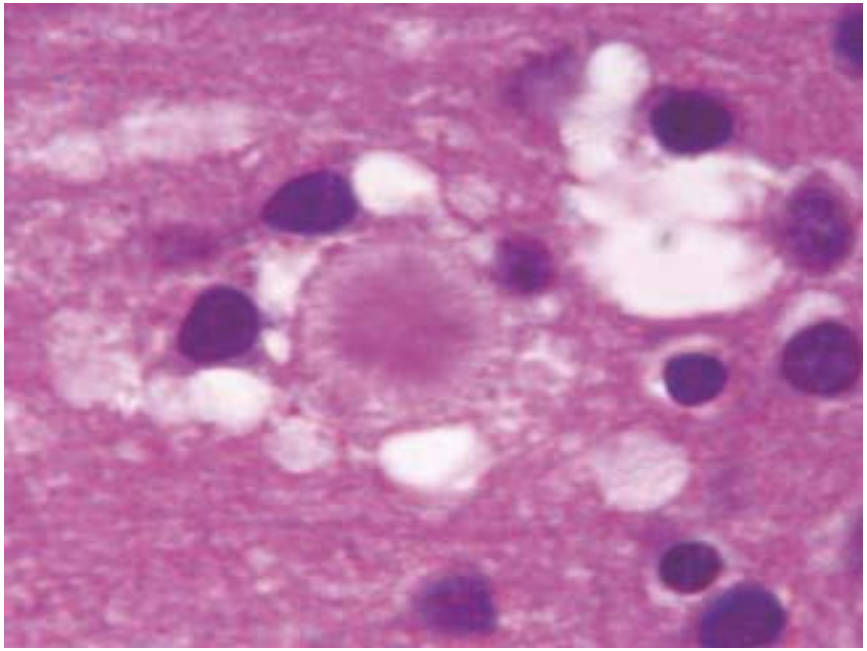


Histology

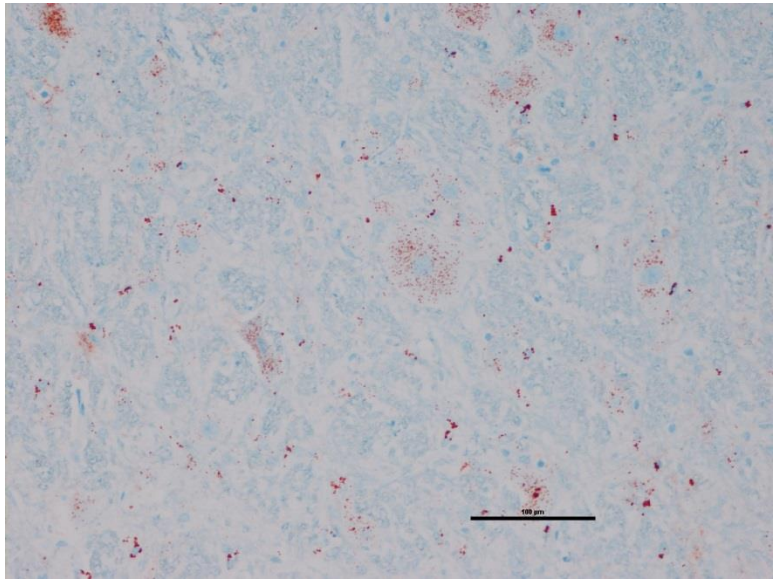


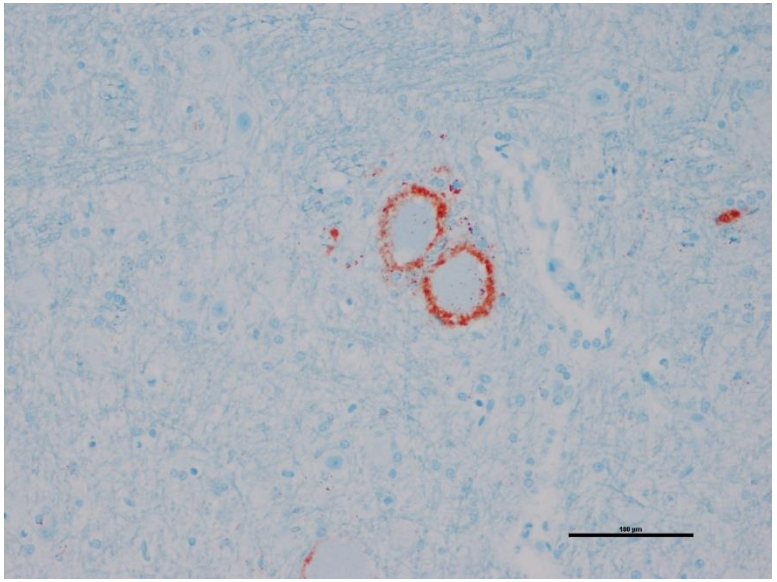


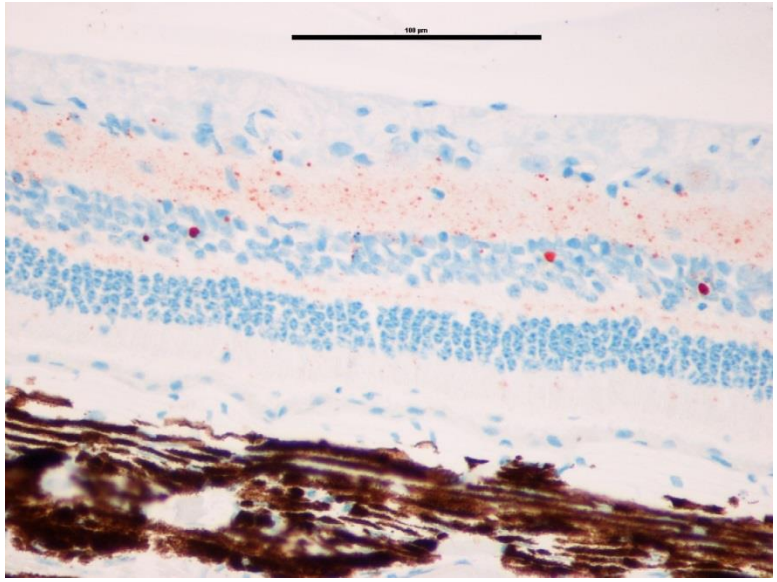


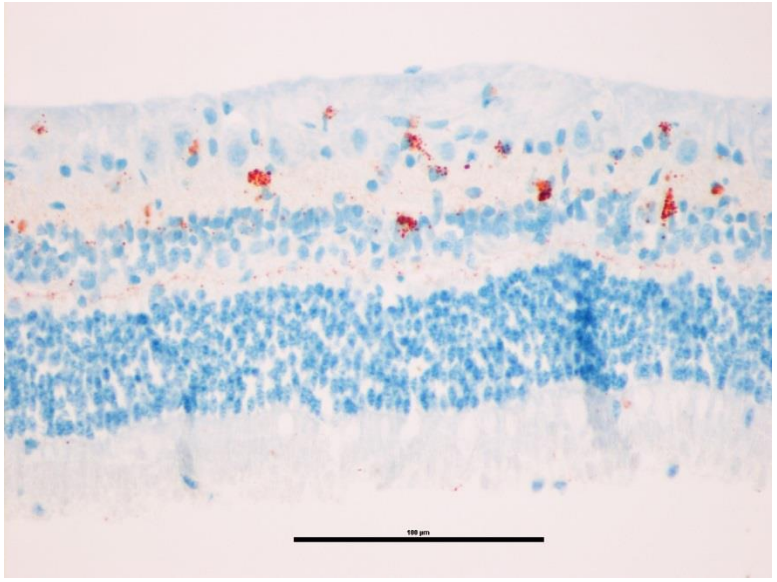


Immunohistochemistry









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Animal Prion Diseases

Scrapie - sheep, goats

Chronic Wasting Disease (CWD) - deer, elk, moose

Bovine Spongiform Encephalopathy (BSE) - cattle

Transmissible mink encephalopathy (TME) - mink

Feline spongiform encephalopathy - large and domestic cats

Spongiform encephalopathy of captive ungulates - exotic hoof-stock in zoological parks

Human Prion Diseases

Zoonotic evidence only for ** Variant CJD or Human BSE, Scrapie and sCJD*, L-type BSE not excluded

Sporadic (spontaneous or link with Scrapie??)

- **Creutzfeldt-Jakob disease (sCJD) (1 per million per year worldwide) (*Scrapie: similar features in Tg Humanized mice)**

Familial (genetic)

- **Familial CJD**
- **Gerstman-Straussler-Scheinker Syndrome (GSS)**
- **Fatal Familial Insomnia (FFI)**

Acquired by transmission

- **Kuru: endocannibalism Papua New Guinea 2700 → SRM+genetic resistance codon 127 (129 V/V for human BSE)**
- **Iatrogenic CJD (neurosurgical instruments, dura mater grafts, HGH) > 405**
- ****Variant CJD (vCJD) or Human BSE 229 patient mortalities (worldwide)**

1 in 2000 carriers (UK)

Alzheimer = prion disease (?) Prusiner 2012

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Transmission Within Species

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COMMON

vertical and horizontal

in utero, fetal fluids, fetal membranes

horizontal

Oral (urine, feces, or blood?)

UNCOMMON

Foodborne
Direct only through bite wounds

Foodborne (MBM)
No direct transmission
from cow to cow

Foodborne, blood,
tissue transplant, HGH, instruments

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Species Barrier Concept

Transmission within a species may occur readily

Barrier between species limits transmission

- Inefficient transmission
- Extended incubation times
- Low or non-existent rate of disease

Serial passage

- Required to overcome species barrier
- Progressive reduction in incubation time
- Increased rate of disease

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PrP Conversion

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BSE/TSE hazard and its characteristics

BSE, is part of a group of transmissible brain affections or prion diseases, characterised by:

Usually, "spongiform" degeneration of neuronal cells.
Occurrence in man and animal, no pathognomonic clinical signs
Usually, a fatal outcome
Long incubation period
No apparent immune reaction.

Characterised by the transformation of normal brain protein (PrP^{c}) into an abnormal protein (PrP^{res}) or *prion* which is routinely used as a marker for infectivity.

256 amino acids, 26-32 kilodaltons

Prion usually resistant to (at a variable degree…):
- **Heat / Ultraviolet light and ionising radiation**
- **Enzymes**
- **Chemical substances**

PRIONS HAVE A VERY HIGH AFFINITY FOR STAINLESS STEEL→ surgical instruments + prototype test for Human BSE using stainless steel (nickel)powder

Inactivation of TSE agents → Also depending on strain!

Physical methods

Irradiation (ionizing, UV, microwave):*little effect;* ***Dry heat****:* 360°C 1h and 600°C 15 min: *partial survival;* ***Autoclaving****:* conflicting data! 132°C 1h: *residual infectivity* ;132°C 90 min: ***inactivated;*** 134°C 30 min: *5.3 logs reduction;* 134°C 18 min: ***inactivated;*** 138°C 1h: *residual infectivity*

→ ***increased temp = increased thermostability?***

Chemical methods

Acids and bases *:* pH 2-10 1h: *little effect;* pH 14 2M NaOH: *5 logs reduction* →combination GD 121°C 1h 1M NaOH: ***complete inactivation;*** ***Alkalyting agents****:* formaline, glutaraldehyde, acetylethyleneimine, bèta-propiolactone, ethylene oxide: *no effect or increased!* ***Detergents*** *:* SDS+boiling or sarkosyl : *some effect;* ***Halogens****:* Sodium hypochlorite (25.000ppm chlorine) 1h: ***effective*** Sodium iodide 2%: *little effect;* **Organic solvents** *:* acetone, chloroform, ethanol, phenol, hexane, perchlorethylene, petroleum: *little effect;* **Oxidizing agents** *:* chlorine dioxide, hydrogen peroxide, peracetic acid*: little effect;* ***Salts****:* sodium periodate, potassium permanganate: *contradictory results;* ***Chaotropes****:* 4M GdnSCN or GdnHCl: *some effect;* ***Proteolytic enzymes****:* pronase and proteinase K: *some effect*

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Hazard Characterisation

In general, there is a dose-response relationship in experimental TSE infectivity (incubation time)

It is assumed that a TSE human-animal species barrier exists, which would affect the efficacy of TSE transmissibility to humans

However, both dose-response relationship in humans and the bovine-human species barrier can only be roughly estimated using animal models

Hazard Identification

What defines a particular TSE Agent?

1. **Biochemical characterisation (orientative)** Western blotting, ELISA
2. **Biological characterisation**: Inoculation in mice (RIII, C57Bl, VM, Tg mice) (not routinely applicable, laborious technique).

→**Transmissibility;**

→**Lesion profile;**

→**Incubation period;**

→**Western blotting characterisation. Topology and quality of PrP^{res} deposition.**

In 1996, a new form of CJD, named variant CJD or human BSE, was identified in humans and it was demonstrated to be caused by the agent that causes BSE in cattle.

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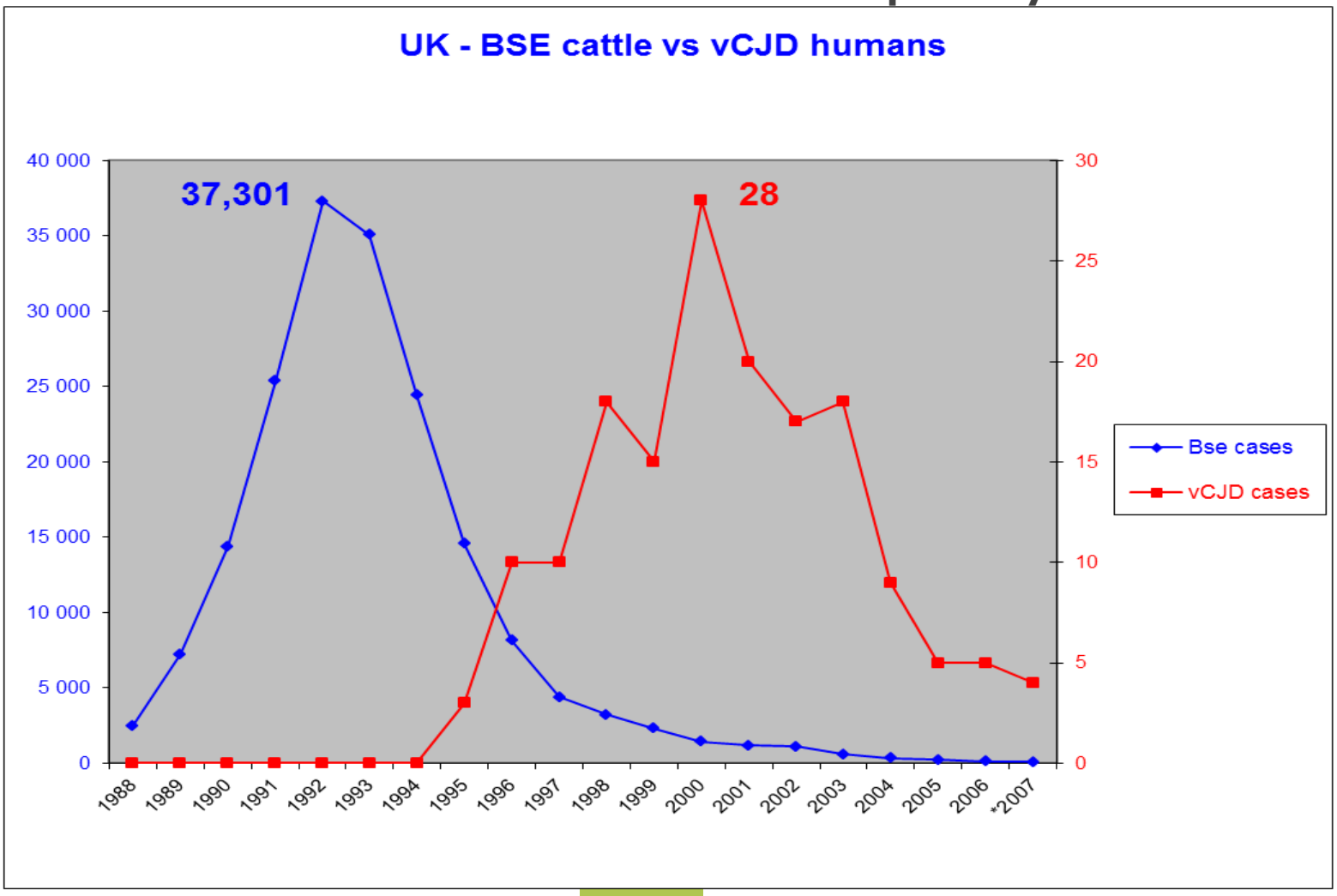
Hazard Identification:

3 million estimated UK cattle BSE →176 UK primary human BSE



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Hazard Identification

The origin of BSE? UNKNOWN…

Conversion of physiological PrP^{C} into the abnormal PrP^{res} or Prion.

Main hypotheses:

Spontaneous occurrence - never proven (L-type?)

Scrapie transmission to bovines - not experimentally shown

1996 Organophospates? (Phosmet)?

1996 Spiroplasma?

2001 Microbacteria from meteorites froling the earth? Cambridge, Wickramasinghe and Hoyle

2003 Acinetobacter? Veterinary and Immunopathology, Wilson et al

2005 Cadavers from the Ganges? The Lancet, Shankar and Satischandra

Only commonly accepted:

it appeared somehow in UK, already in the 70s

it was distributed from there via export of feedstuffs and of infected cattle

Hazard Identification

Transmission of BSE in cattle:

***MBM (never experimentally reproduced) aggregate associated infectivity -> only few animals per herd* : what is the field CoID50 (Experimentally between 1mg and 0.1 mg fresh BSE brain)**

Horizontal transmission - NO!?

Vertical transmission - HOW?

Oral – via feed or via injection of infective tissue

Semen - not

Embryos – unlikely

Other ??

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Chapter 2. THE SCIENCE-BASED EU TSE LEGISLATION AND RELATED LEGISLATION

All EU TSE legislation has to be based on Science

Scientific Opinions of EFSA

The TSE Agent behaves both like a microbiological and a chemical contaminant

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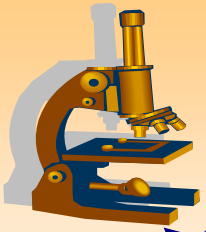
EFSA is not responsible for legislation

Risk assessment



Risk manageme

Risk communication

efsa
European Food Safety Authority

EC

Risk Analysis framework in the EU

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European Commission

European Parliament

Member States

EFSA ("self mandate")

Question?

Opinion

Risk Assessment

Risk Management

- Consumers
- Media
- Industry
- Professionals

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EXAMPLE : ESFA SCRAPIE OPINION; EFSA JOURNAL 2014;12(7):3781 [155 PP.

BACKGROUND

- *Scrapie situation surveyed since 2002*
- *A number of control/eradication measures have been implemented in the EU*
- *This includes breeding programmes for resistance to Classical scrapie (CS) in sheep*
- *Global strategy in force for ten years*
- *European Commission needs better understanding of:*
  - **Epidemiological situation of CS/AS**
  - **Retrospective analysis of the efficiency of control measures applied**
- *Mandate for EFSA opinion in 2012*

- *Opinion adopted by BIOHAZ Panel and published in July 2014*

TERMS OF REFERENCE

1. *On the basis of the results of the TSE monitoring programme laid down in the TSE Regulation,* ***what is the trend since 2002 of the situation of Classical scrapie and Atypical scrapie in sheep and in goats respectively, in the EU as a whole and in the 27 Member States individually?***

   Where no favourable trend can be observed, ***what are the identifiable causes for failure*** *to improve the situation of Classical scrapie?*

2. *Has the* ***evolution*** *of the Classical scrapie situation been* ***statistically different in the MS which have implemented a breeding programme from 2004 to 2011 compared to the MS without a breeding programme*** *in the same period?*

TERMS OF REFERENCE

3. *On the basis of the above analysis**, can a minimum level of frequency of the ARR allele in the sheep population in a MS be defined or estimated above which Classical scrapie can be expected to fade out, in a context where no control and eradication measure is being applied?***

4. ***In a context where no breeding programme is implemented, are the present mandatory measures** in terms of active monitoring, eradication and control of Classical scrapie **effective** to achieve a decline of this disease and its eradication on the long term?*

5. *What **additional measures can EFSA recommend** in view of achieving the eradication of Classical scrapie in the MS?*

SOME CONCLUSIONS – TOR 1

On the trends of Classical scrapie (CS) in sheep:

- *Country-specific temporal trends are heterogeneous, preventing any meaningful interpretation of the overall temporal trend at the EU27-level. […] the results of the analysis allow the classification of the EU27 MSs into four groups:*
  - **CS detected with a statistically significant decreasing trend (Cyprus, France, Ireland, The Netherlands, Slovenia and the United Kingdom)**
  - **CS detected with an observed trend not statistically different from a flat one (Belgium, Czech Republic, Greece, Italy, Romania, Slovakia and Spain)**
  - **CS reported only sporadically (Bulgaria, Germany, Hungary and Portugal)**
  - **no cases of CS in 2002-2012 (Austria, Denmark, Estonia, Finland, Latvia, Lithuania, Luxemburg, Malta, Poland and Sweden)**

On the trends of CS in goats:

- *Statistically **decreasing trends were evident** respectively for France over the entire period (2002-2012) and for Cyprus and the United Kingdom after 2007.*

SOME CONCLUSIONS – TOR 1

On possible causes for failure to improve the situtation of CS:

- *Although it is **not possible to identify causes** that can explain objectively the failure to improve the situation of CS, **some hypotheses** are formulated in the opinion (for both sheep and goats).*

Atypical scrapie (AS) in sheep:

- *Where detected, AS in sheep showed **a similar prevalence over time and space**: no large epidemics were reported and five countries detected AS in sheep only sporadically. Only two countries showed a statistically significant trend, with a reduction in the annual prevalence rates in France and an increase in the United Kingdom.*

AS in goats:

- *AS in goats was reported by five countries, at a very low prevalence and with **no statistically significant trend** in any of them.*

SOME CONCLUSIONS – TOR 2

On the effectiveness of breeding programmes for resistance to CS in sheep:

- *Given the characteristics of each national BP-SC, a deterministic model was used to estimate the ARR/ARR frequency in the general sheep population over time. Subsequently, the outputs of the model were compared with the national CS situations. The results obtained suggest* ***a favourable CS situation being linked to better-achieving BP-CSs.***
- *Cyprus and the Netherlands, countries in which the improvement in the epidemiological situation of CS is clear, applied their BP-CSs to the whole population, without any distinction between population tiers. This approach produced an effective change of the genetic structure of the whole sheep population, but* ***required extensive genotyping efforts****.*

SOME CONCLUSIONS – TOR 3/4

On a minimum ARR allele frequency to observe fade out of CS in sheep:

- *Given the very strong resistance of the homozygote ARR genotype to CS, there is a conceptual parallel between the ARR homozygote proportion of a sheep population and the proportion immunized in a vaccinated population.*
- *Case studies [NL, GB, CY, Sardinia] illustrate the non-universality of the minimum ARR frequency; across the case studies it* ***ranges between 53 % and close to 100 %*** *according to a crude model. The case studies also provide some insight into how the minimum frequency depends on MS-specific parameters.*

On the non-genetic control and eradication measures:

- *Due to the pathogenesis and the epidemiological characteristics of CS, and to the high persistence of the CS agent in the environment, a CS eradication policy that relied solely on detection of infected flocks by post-mortem testing and subsequent depopulation* ***would be unlikely to succeed****.*

SOME CONCLUSIONS – TOR 5

On additional measures to achieve eradication of CS:

Additional/alternative measures to control CS in sheep and goats are recommended in the opinion. These focus on:

- *the improvement of surveillance and control measures and their adaptation to the individual MSs;*
- *the reinforcement and improvement of the policy of breeding for resistance in sheep;*
- *the introduction of breeding policies in goats;*
- *knowledge transfer on scrapie.*

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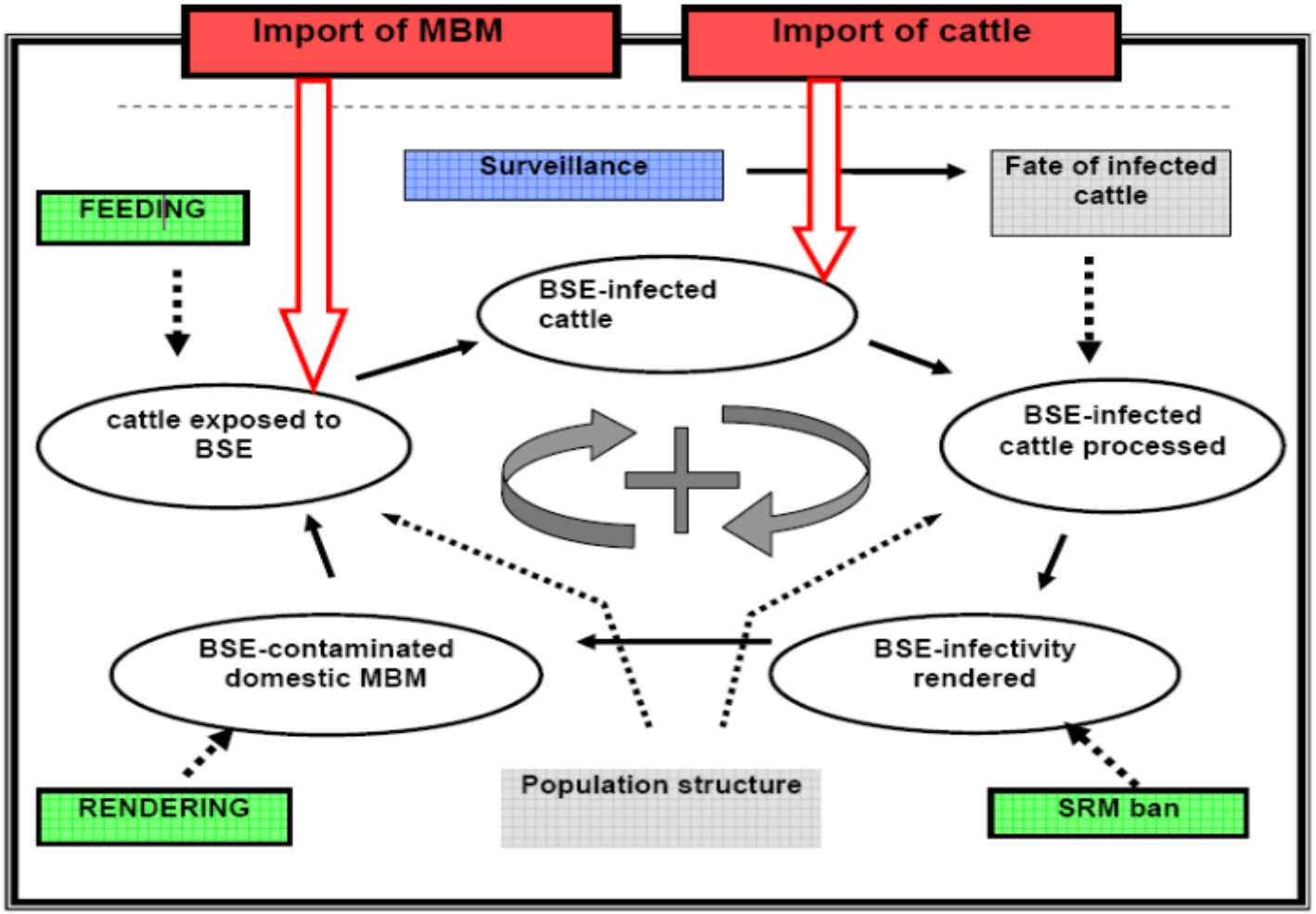
TSE regulation 999/2001

- Sets out parameters for TSE suspects and eradication measures and definition of SRM – category 1 ABPs (Art 8 of 1069/2009)
- Rules for export of SRM set down in TSE rules
- Prohibitions on feeding – complements restrictions in Art 11 of 1069/2009
- TSE roadmap - stepwise amendments relaxing TSE rules e.g. on feeding prohibitions


Import of MBM
Import of cattle
Surveillance
Fate of infected cattle
FEEDING
BSE-infected cattle
cattle exposed to BSE
BSE-infected cattle processed
BSE-contaminated domestic MBM
BSE-infectivity rendered
RENDERING
Population structure
SRM ban

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TSE regulation 999/2001

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Animal By-Products legislation (1069/2009 and 142/2011)

→Boundaries of ABP legislation (1069/2009 and 142/2011) with following legislation:

- **TSE regulation- 999/2001**
- **Food hygiene legislation -852/2004 and 853/2004**
- **Feed legislation – 183/2005 (hygiene) and 767/2009 (placing on market)**
- **Various legislation on cosmetics, medical devices, veterinary / medicinal legislation**
- **Environmental legislation (main focus) - Waste Framework Directive 2008/98 and Waste Incineration Directive 2000/76**

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Chapter 3. EXPOSURE AND ZOONOTIC POTENTIAL OF ANIMAL TSEs

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VARIANT CREUTZFELDT-JAKOB DISEASE OR HUMAN BSE
CURRENT DATA (2015)

Estimation: 1/2000 subclinical infected with BSE (UK)

Confirmed cases: 229 from 12 countries

176 United Kingdom, 27 France, 5 Spain, 4 Ireland, 4 United States, 3 the Netherlands, 2 Portugal, 2 Italy, 2 Canada, 1 Japan, 1 Saudi Arabia, 1Taiwan

COUNTRY	TOTAL NUMBER OF PRIMARY CASES (NUMBER ALIVE)	TOTAL NUMBER OF SECONDARY CASES: BLOOD TRANSFUSION (NUMBER ALIVE)	RESIDENCE IN UK > 6 MONTHS DURING PERIOD 1980-1996
UK	172 (4)	4 (0)	176
France	27 (0)	-	1
R of Ireland	4 (0)	-	2
Italy	2 (1)	-	0
USA	3 (0)	-	2
Canada	2 (0)	-	1
Saudi Arabia	1 (0)	-	0
Japan	1 (0)	-	0
Netherlands	3 (0)	-	0
Portugal	2 (0)	-	0
Spain	5 (0)	-	0

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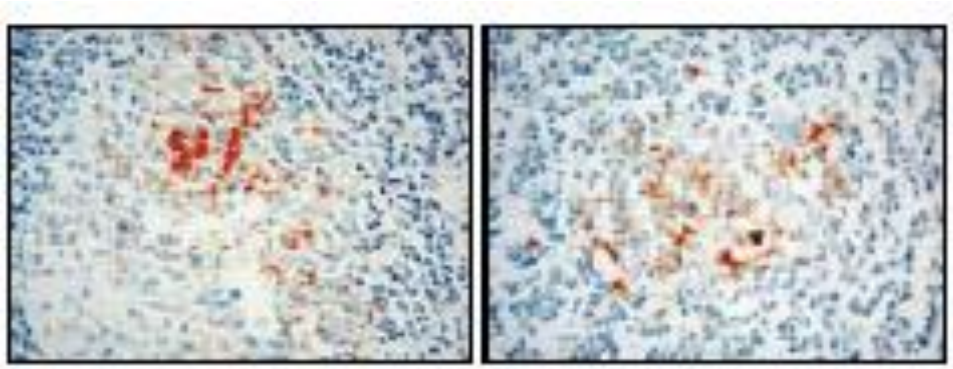
PrP^{res} in UK appendices: implications for prevalence of subclinical or preclinical Human BSE infections



Over 32 000 anonymous appendix samples →one in 2000 people are likely to be carriers.

No particular age group or geographic region affected, no susceptible genotype of patients was identified.

A higher proportion of valine homozygous (VV) genotype in codon 129 of the gene encoding the prion protein (PRNP) compared with the general UK population. This also differs from the 177 patients with Human BSE, all MM (oral exposure BSE infected meat products)

What is real risk carriers pose of transmitting the disease by blood transfusion or surgery?

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Transmission of TSE by blood transfusion

SHEEP

Around 20% transmission of infectivity by transfusion of whole blood or buffycoat cells from BSE and Scrapie preclinical and clinical donor incubating transfused into recipient sheep

HUMANS

- **Infectivity in erythrocytes, leukocytes, and plasma in vCJD or human BSE**
- **Infectivity levels comparable to those reported in various animals with TSEs**
- **In the United Kingdom, 4 vCJD transmissions from 18 donors who later had positive test results for vCJD**

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Exposure

To what extent are consumers exposed to the BSE Agent from ruminants?

Pathogenesis studies serve as the basis for the identification of potentially infectious cattle material (mainly CNS).

Titration of this material can help to quantify the infectious load and thus the exposure risk.

Based on these, the Specified Risk Materials are defined.
Its removal from the human food and animal feed chains is the most efficacious measure to decrease the exposure risk (estimated to be at least 95% of the total infectivity in cattle).

Exposure Assessment

Pathogenesis studies serve as the basis for the definition of SRM
Assumption:100 mg =1 $CoID_{50}$ but recent data 1 - 0.1 mg = 1 $CoID_{50}$

Tissue	Infectivity density ($CoID_{50}$/g)	Weight (kg) per 537 kg animal	Cattle oral ID50 per BSE Case	% of total infective load per animal	Cumulative load
Brain	10	0.5	5000	64.1 %	64.1 %
Spinal cord	10	0.2	2000	25.6 %	89.7 %
Trigeminal ganglia	10	0.02	200	2.6 %	92.3 %
Dorsal root ganglia	10	0.03	300	3.8 %	96.1 %
Ileum	3.20 E-02	0.8	26	0.3 %	99.4 %
Spleen*	3.20 E-02	0.8	26	0.3 %	99.7 %
Eyes	3.20 E-02	0.1	3	0.04 %	99.74 %

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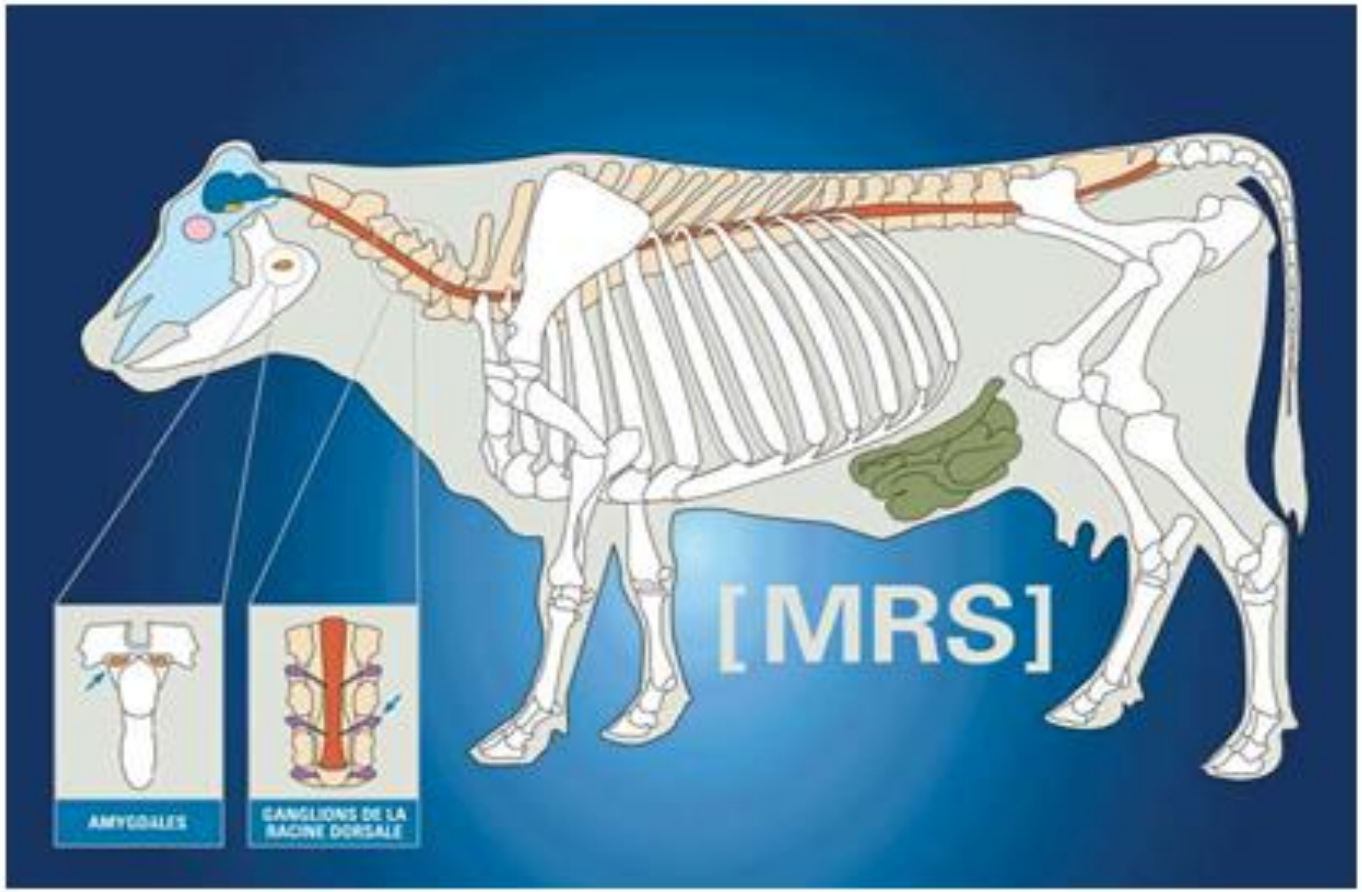

Exposure Assessment

Where lays at present the residual Classical BSE exposure risk?

Clinical BSE cases: Should not enter the human food chain (*antemortem* veterinary inspection).

Pre-clinical BSE cases: Eclipse phase of several years after infection (distal ileum >> sympathic/parasympathic nerves >> CNS): risk mainly from end-stage incubating animals.

Exposure only through potential **cross contamination** during slaughtering process and hypothetical residual infectivity left in lymphoid (and nervous?) tissue.

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Exposure Assessment: Main TSE monitoring uncertainties

Atypical BSE (H or L-type): Efficiency of the current TSE monitoring system? No clinical signs, > 10 years of age, no healthy slaughter testing

The impact of TSE testing policy on TSE monitoring in cattle: Considerations on sensitivity, active and passive surveillance, early detection.

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Exposure Assessment

Food processing is **not** assumed to affect infectivity potential, but can only dilute this (like chemical contaminant).

Consumer habits do **not** clearly address increased exposure in particular sub-populations.

Consumer cooking practices are **not** assumed to affect infectivity if present

Variety of usage of Animal By Products (examples)

OF/SI: BSE/TSE prions in soil and plants relatively stable against protein-denaturing "influences"

Shopping bags–slip agent from animal fat

Tyres & fireworks-animal based stearic acid

Violins & pianos–animal glue

Cosmetics – glycerin

Zoonotic potential of animal TSEs

Zoonotic risk of other TSE Agents?

Scientific data:

Small ruminants TSE Agent transmitted to cynomolgus and marmoset monkeys (i.c.)…

L-Type BSE transmission rate in Tg Hu mice higher than Classical BSE (i.c.)…

CWD transmitted to non-human primates (i.c)…

…The "*Howevers*",

Does the susceptibility of the animal models resemble that of the humans (dose-response and species barrier)?

Does the experimental exposure route resemble the natural human exposure route?

No evidence of epidemiological link!

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Atypical TSEs: unknowns

Origin:scrapie/BSE related/spontaneous?

Timing of the origin?

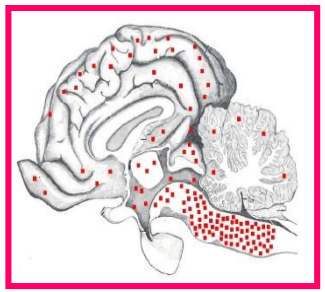
Tissue distribution of infectivity?

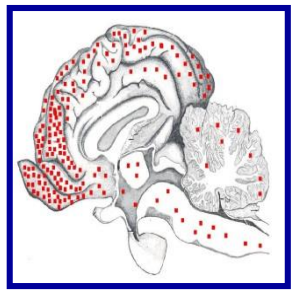
Geographical distribution?

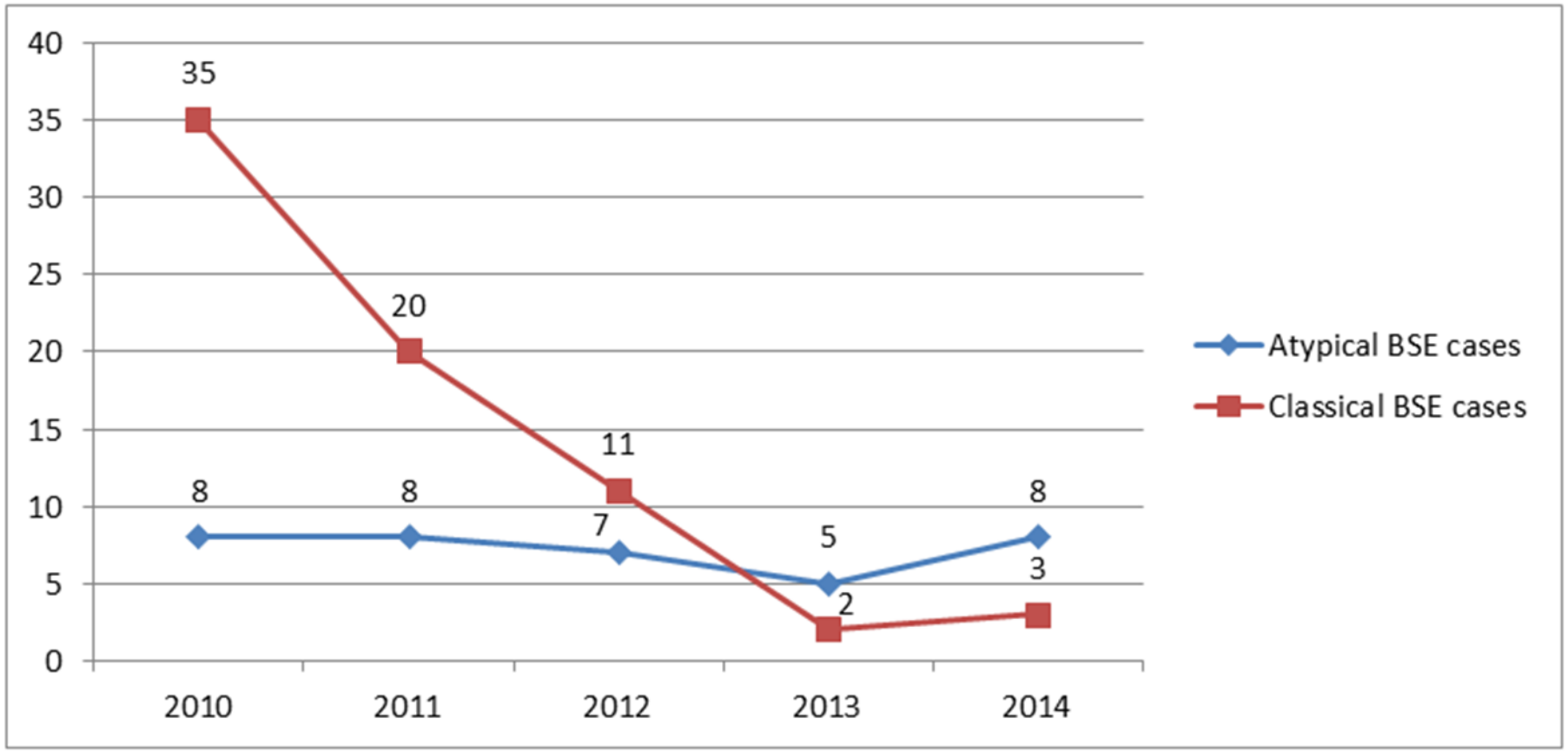
True incidence by country?

Temporal trends in incidence by country?

Phenotype in humans?







Apparent low and stable number of Atypical BSE cases

Chapter 4. TSE ROADMAP 2

Stepwise amendments relaxing TSE rules

EFSA – *BSE* – EC/EU

↓

<u>Science</u> based Risk Assessments (but lot of uncertainties!)

↓

Proportionate Risk Management <u>measures</u>

Possible reduction of the <u>costs</u> for BSE control and surveillance

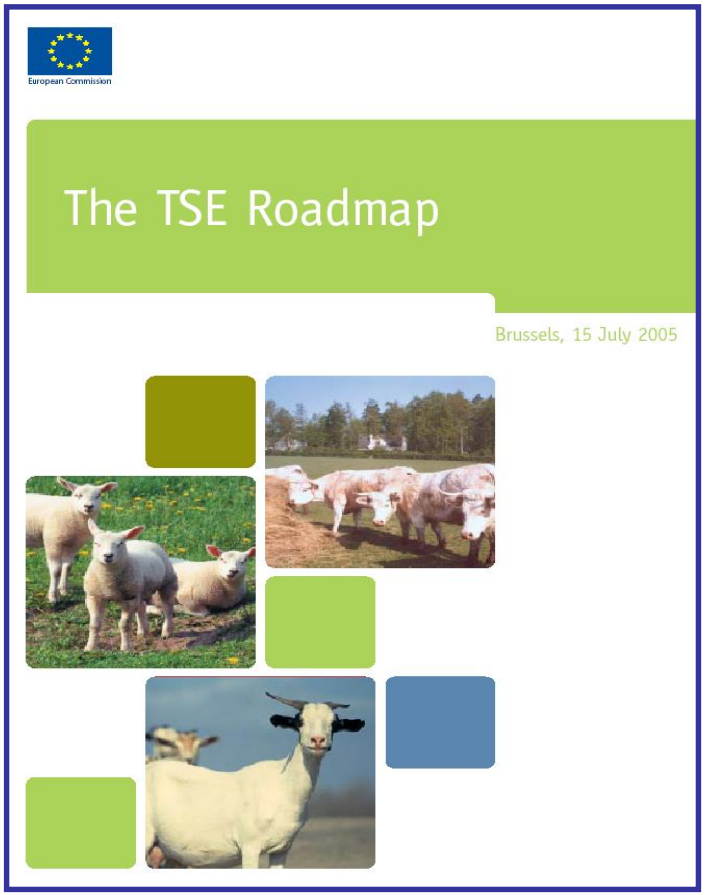
European Commission

BSE *Quo Vadis*?

Consumers, Health And Food Executive Agency

European Commission

EC-DG Sanco - Risk Management



… significant decrease in the number of positive BSE cases in the EU, due to stringent risk reducing measures

…and new developments in science and technology…

…the TSE Roadmaps consider possible amendments to certain BSE measures

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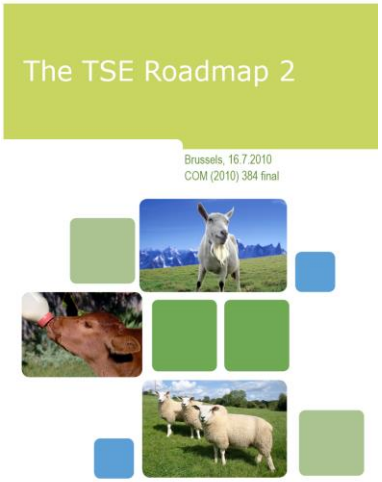
Directorate-General for Health & Consumers

TSE Roadmap 1



- Adopted on 15 July 2005
- Stepwise and **science based** approach
- **Positive trend in BSE epidemic** has continued since then

TSE Roadmap 2

- Adopted on 16 July 2010
- Continue the
- **review of the TSE measures while assuring a high level of food safety**
- Still **stepwise and science based approach (EFSA)**
- Different topics covered: **SRM removal, feed ban, BSE surveillance, TSE measures in small ruminants**

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TSE Roadmap 2: feed ban

- **Strategic goal**: to review certain measures of the current total feed ban when certain conditions are met

- Introducing **tolerance level** for processed animal proteins (PAP) in feed for farmed animals

- **Lifting feed ban provisions** for non-ruminants (pigs, poultry, fish) while avoiding cannibalism

- Conditions: control tools available, channelling

TSE Roadmap 2: Feed ban

Allow non-ruminant pap for non-ruminants

- 1st step (see above) for aquafeed: 1 June 2013 (Reg. (EC) N°56/2013)
- Next steps: pigs and poultry, insects
- Principles: no TSE species, Anti-cannibalism, only cat 3
- What do we need to relax?
  - Solid basic control ABP
  - Species specific analysis
  - Species specific production/products

Controls of the feed ban

2 control methods laid down in Annex VI to Reg (EC) No 152/2009 (as amended by Reg 56/2013 at present only poultry for aquafeed) + SOPs of the EURL-AP: light microscopy and PCR.

- Light microscopy allows to detect particles of animal origin (bones, hairs, scales, feathers, etc.) and to distinguish between terrestrial and fish particles. Does not allow identifying the species.

- PCR allows to detect DNA of ruminants (but does not allow to identify the source of the DNA, e.g. milk vs bovine PAP).

Feed not destined to aquaculture: only LM should be used.

Feed destined to aquaculture: see flowchart

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Possible evolution of the feed ban

Lifting feed ban provisions for non-ruminants (pigs, poultry, fish) while avoiding cannibalism : Regulation 56/2013

<u>Conditions:</u> control tools available for species distinction (PCR methods) + dedicated production lines

Lifting feed ban provisions for ruminants <u>is not</u> envisaged

TSE Road map 2: BSE surveillance

- **<u>Strategic goal</u>**: to continue to **adapt the BSE monitoring system** in bovine animals with a better targeting of the surveillance activity while keeping the capacity to monitor the evolution of the epidemiological situation and to assess the effectiveness of the protective measures in place

- Options: age limit / date of birth / sample size

- Revision only allowed for Member States demonstrating a good epidemiological situation

- OIE compliance

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TSE Roadmap 2: SRM removal

- **<u>Strategic goal</u>:** to ensure and maintain the current level of consumer protection by continuing to assure safe removal of SRM but modify list/age based on new & evolving scientific opinions

- **EFSA opinions**: crucial role but quantitative or semi- quantitative approach needed

- **Alignment with OIE rules** desirable

Proposed roadmap for SRM and Atypical BSE

- To collect additional data in view of increasing knowledge and understanding of atypical BSE

In the meantime:

- To provisionally maintain status quo on removal of SRM
- To reflect on establishing a limited list of SRM applicable in MS with a negligible risk
- To reiterate our request to OIE to work on atypical BSE

SRM: new regulations

1. 6 May 2015: Regulation 728/2015: amended SRM list for bovine intestines: last 4 meters of the small intestine, caecum and mesentery remain SRM / duodenum, colon and small intestine except the last 4 meters no longer SRM.

2. 15 July 2015: Regulation 1162/2015: reduced bovine SRM list for MS with negligible BSE risk : skull, eyes, brain and spinal cords of bovines above 12 months remain SRM / vertebral column, tonsils, intestines and mesentery no longer SRM for animals originating from those MS. SRM list for small ruminants not touched.

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TSE Road map 2: Eradication measures in small ruminants

- **<u>Strategic goal</u>**: to **adapt the current eradication measures** in TSE infected flocks of sheep and goats to bring them in line with the latest scientific knowledge and to develop sustainable tools to control TSE in small ruminant flocks in the EU

- Herd certification

- Measures for Atypical Scrapie

- Genetic resistance in goats

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TSE Road map 2: Challenges

- **Complete elimination of the risk: unrealistic**
  → Proportionality of the measures
- **No complacency**
- **Solid scientific advice: semi-quantitative or quantitative risk assessments taken into account epidemiological situation**
- **Communication towards the consumers**

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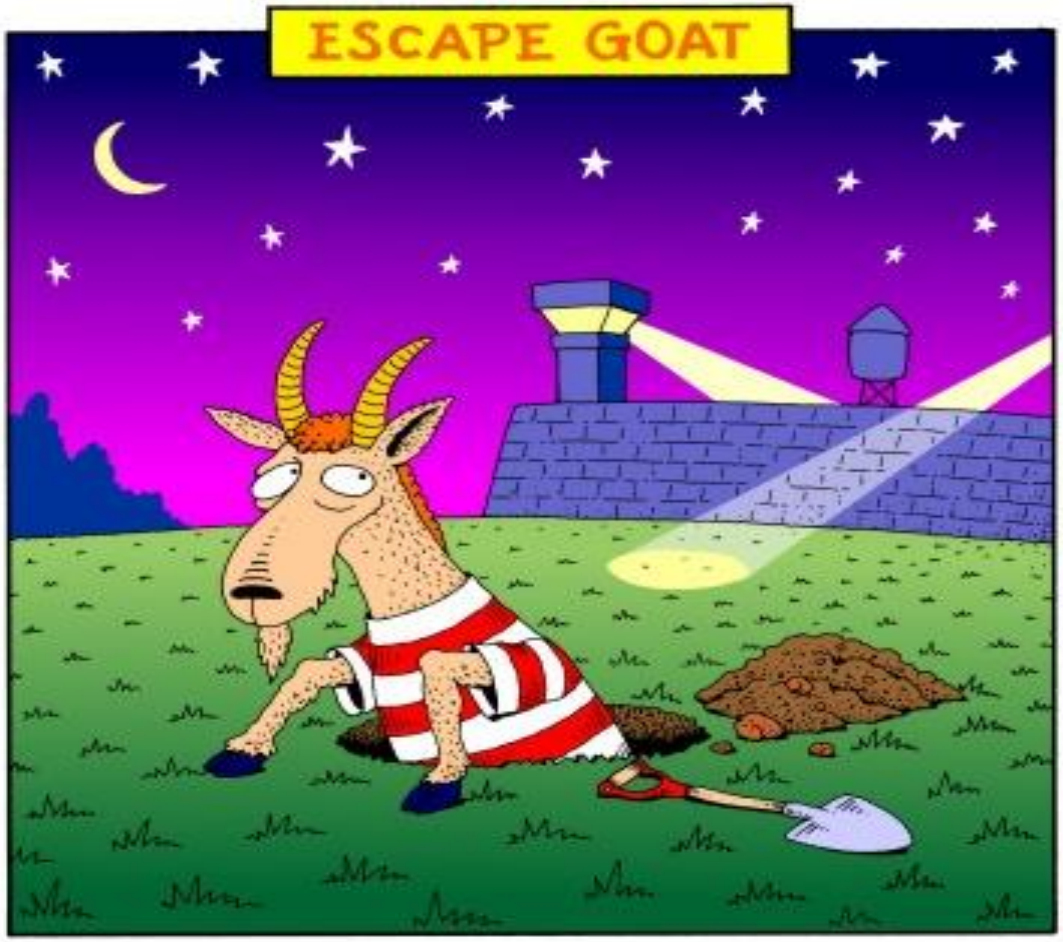
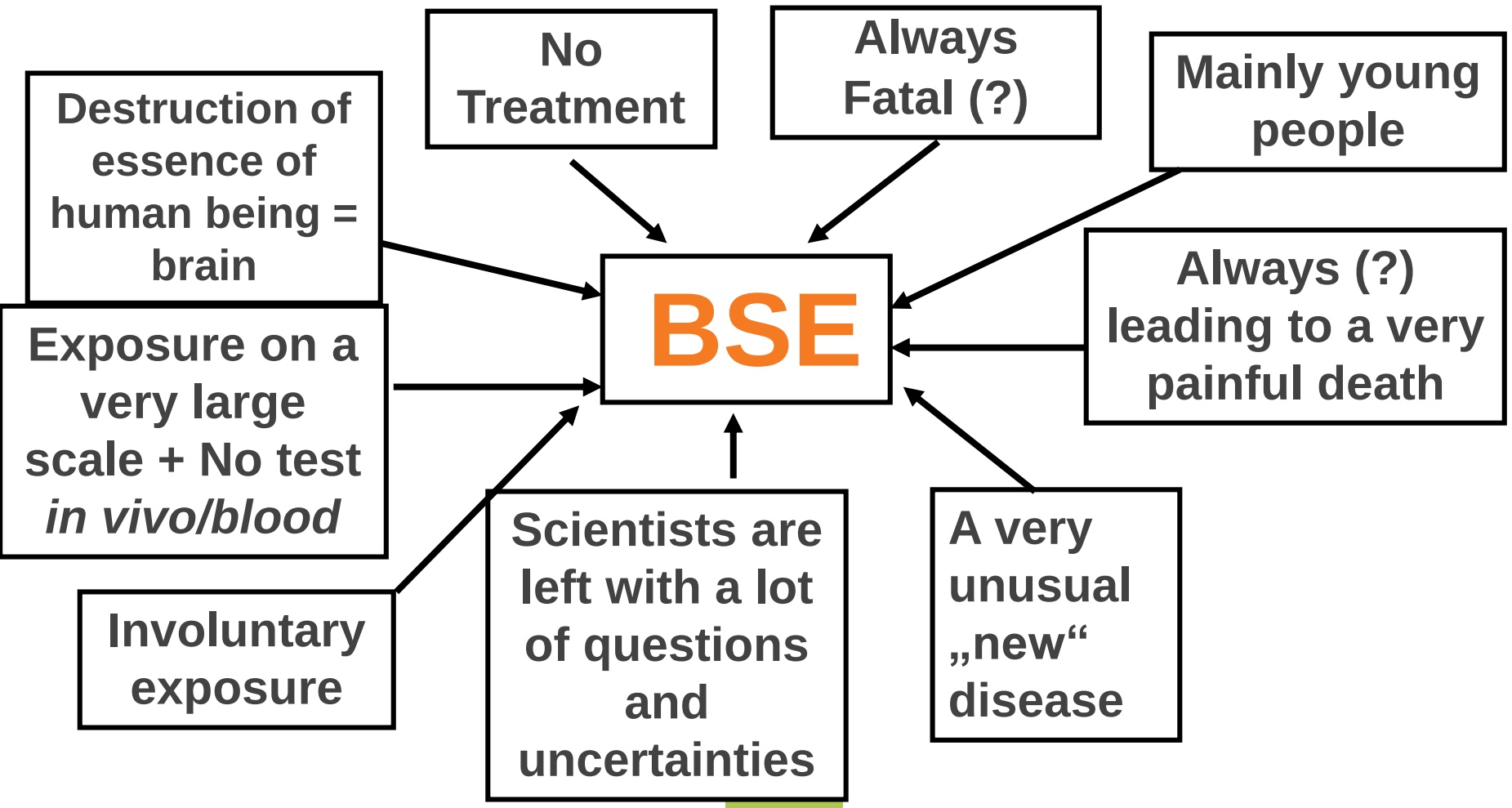
BSE crisis was one of the reasons for the establishment of "Risk Assessment bodies" such as *EFSA and National Food Safety Agencies*



And vigilance remains

BSE: REASONS FOR CONCERN AMONG THE PUBLIC



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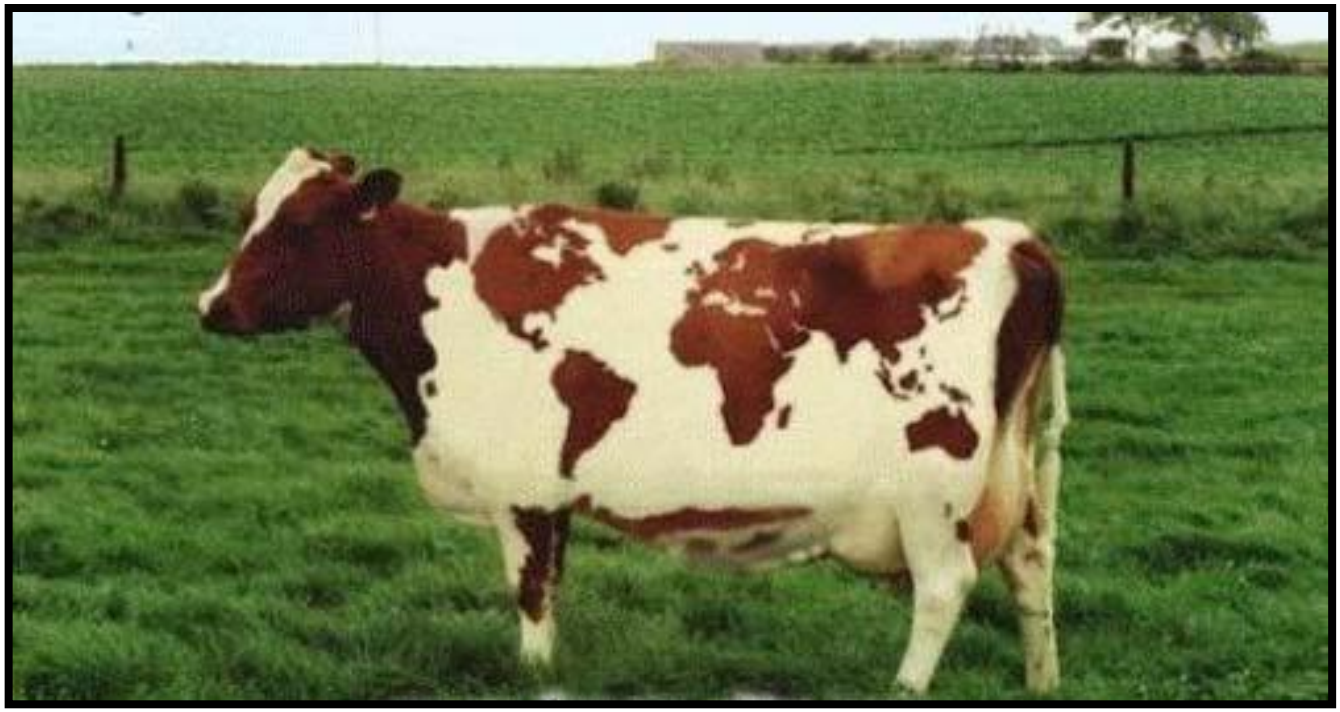
Thank you for your attention



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Time for discussion……

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