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## E-poster 223 EP- 0276



Epidemiology / Public Health

### Causes and Origin of the Bovine Spongiform Encephalopathy(BSE) Incidence in the United Kingdom?

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#### **Abstract No 0276**

#### **Purpose**

Epidemiological incidence of neurodegenerative diseases in a certain period, was detected only in cattle in the UK, as the BSE. The official statement about the ca 5-years incubation period of the BSE is based on the feed ban (1988) of meat and bone meal (MBM) in the UK cattle, and the BSE incidence significantly decreased(after 1993).

#### **Materials and Methods**

However, it has never been proven MBM feeding in British cows, in the finding of the BSE incidence, in the respective herd of cows ! On the other hand, there are scientific publications from that time (with detailed description of the cows nutrition ) when BSE disease was detected in cows, without the MBM feeding (however, high protein intake). However, if in the 80s was among British cows found long-term Mg-deficiency, then after significantly higher Mg-supplementation should be the incidence of BSE significantly reduced.

#### **Results**

National agency of the Great Britain, which has monitored (1982-1992) the incidence of clinical-subclinical hypomagnesaemia (hypo-Mg); found subclinical hypo-Mg at about 7-15 % of tested cows. Data has been collected monthly from 200- 250 farms, with an average herd size of 110–130 cows.

#### **Conclusion**

Why Mg-deficiency should be related to neurodegeneration and the emergence of BSE? If the lower the Mg<sup>2+</sup> level in the animal tissue cells, the more marked is „ Ca<sup>2+</sup> effect excitotoxicity“ ( neurodegeneration- BSE) , because this condition overactivates glutamate receptors, specifically the N-methyl-D-aspartate ( NMDA) receptor. Prolonged Mg deficiency (subclinical hypo-Mg) leads to an excess of Ca<sup>2+</sup> in animal tissues, and NMDA receptor overstimulation.

**Keywords** : Bovine spongiform encephalopathy, Meat and bone meal, Magnesium deficiency, Neurodegeneration , Magnesium

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## **Causes and Origin of the Bovine Spongiform Encephalopathy(BSE) Incidence in the United Kingdom?**

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### **E-poster 223 EP**

#### **Purpose**

Epidemiological incidence of neurodegenerative diseases in a certain period, was detected only in cattle in the UK, as the BSE. The official statement about the ca 5-years incubation period of the BSE is based on the feed ban (1988) of meat and bone meal (MBM) in the UK cattle, and the BSE incidence significantly decreased (after 1993). However, it has never been proven MBM feeding in British cows, in the finding of the BSE incidence, in the respective herd of cows! On the other hand, there are scientific publications from that time (with detailed description of the cows nutrition ) when BSE disease was detected in cows, without the MBM feeding (high protein intake). However, in the 1980s was among the UK cows found long-term Mg-deficiency.

#### **Materials and Methods**

To evaluate the situation of the Mg- deficiency (1982-92), publications from the journal Veterinary Record were selected. Monitoring in England and Wales was carried out by Department of Veterinary Clinical Studies, University of Edinburgh. One survey in Northern Ireland was conducted by Veterinary Sciences Division, Belfast.

#### **Results**

England and Wales

The national incidence of hypomagnesaemia (hypo-Mg) in dairy cows is available from the Dairy Herd Health and Productivity Service (DHHPS). Data has been collected monthly (1982) from 206 farms, with an average herd size of 110 cows, on disease treatments. Carefully pre-planned blood testing of cows typical of specified lactation groups is carried out three or four times a year, there was a 1 per cent average incidence of clinical hypo-Mg. The highest level was in May, but cases occurred in all months. The incidence of subclinical hypo-Mg which was defined a a

cow having a serum Mg level below 0.78 mmol/litre, was as high as 7 per cent of milkers and 15 per cent of dry cows tested in some months. The number of blood samples looked per month varied from 200 to 2000 (Whitaker and Kelly, 1982). There was a substantial increase in the numbers of cases of clinical hypo-Mg in dairy cows in spring 1984 when 1 per cent were affected in May 1984 and 0.9 per cent in June (250 herds, average size 130 cows). This was two and half times greater than the May average for the previous three years and five times greater than the June average. Interpreted as a similar percentage of the United Kingdom national herd, there probably were 64.000 cases of clinical hypo-Mg in this two month period of 1984 (Whitaker et al., 1985).

Through the agency of DHHPS, which monitors 30.000 cows (1992), it has become apparent that the incidence of clinical and subclinical hypo-Mg in dairy cows seems to be still unusually high. Farmers are reporting losses and a significant proportion of blood samples are showing low Mg levels. With the absorption of available Mg notoriously variable, the average target for daily intake in a lactating cow is 30 g. This is equivalent to 60 g of calcined magnesite of good quality. To achieve this level of daily intake with certainty, Mg needs to be included in feed, thus not leaving any option to the cow. At this time the only choice is often incorporating Mg in concentrates fed at milking (Whitaker et al., 1993).

#### Northern Ireland

513 dairy herds were sampled during the grazing season from March to November 1991, and 1266 suckler herds from March 1991 to February 1992. Serum blood Mg below 0.8 mmol/l was found in 28% of cows (McCoy et al., 1993).

#### **Conclusion**

Why Mg-deficiency should be related to neurodegeneration and the emergence of BSE? If the lower the Mg<sup>2+</sup> level in the animal tissue cells, the more marked is „Ca<sup>2+</sup> effect „excitotoxicity“ (neurodegeneration - BSE), because this condition overactivates glutamate receptors, specifically the N-methyl-D-aspartate (NMDA) receptor. Then after significantly higher Mg-supplementation should be the incidence of BSE significantly reduced (after 1993).

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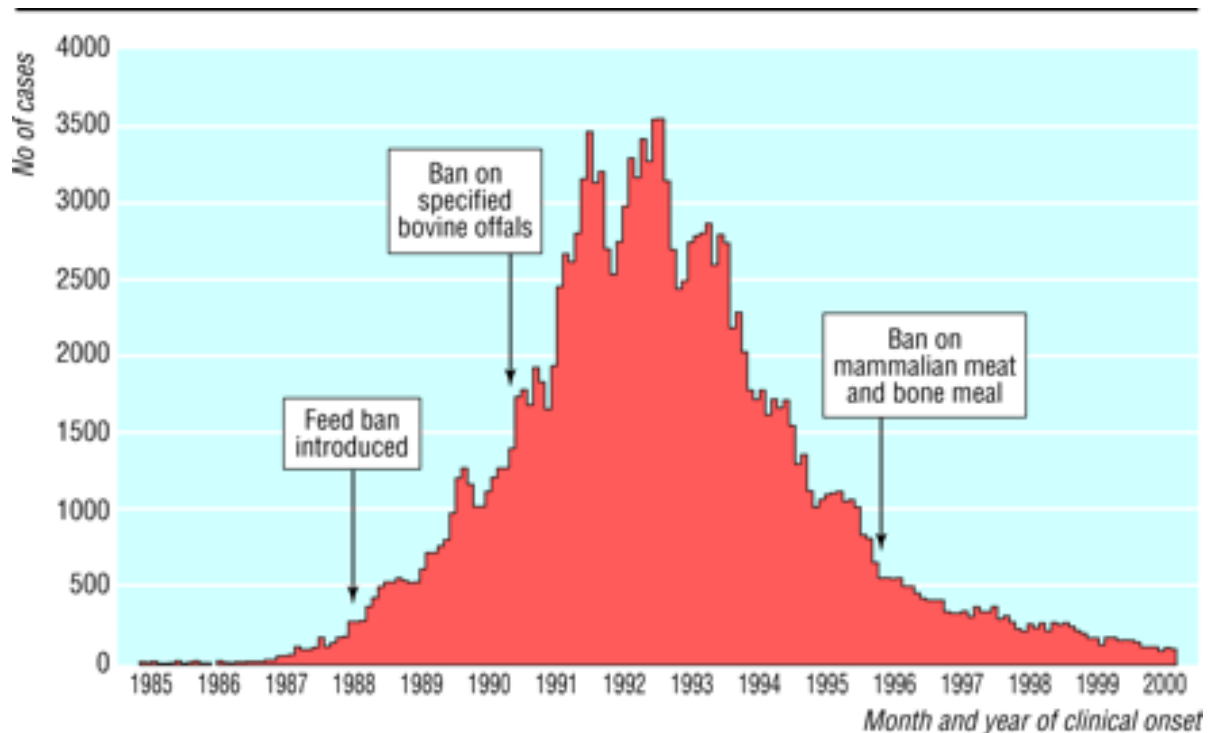
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## E-poster 196 BM- 0356



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**Bovine Medicine**

### **Bovine Spongiform Encephalopathy (BSE) Prevention and Alzheimer's Disease (AD) Connections?**

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#### **Abstract No 0356**

##### **Purpose**

From the literature it is known that in British cows at 80s a higher incidence of subclinical (chronic), hypomagnesaemia was found and a new BSE disease appeared. After 1993 began to significantly reduce the incidence of the BSE. The aim of this study was to determine what changes in the period about 1985- 1995

(magnesium content in feed concentrates) have occurred in British cows, in this context.

## **Materials and Methods**

In order to detect changes in the supply of magnesium in cows (1985-1995), in the form of mineral supplements, data from two British producers were examined.

## **Results**

In the late of 80s were available only commercial Mg-blocks with very considerable variation in palatability, mostly very low Mg-intake. So usually the survey has been; the subclinical hypomagnesaemia was found in about 7-15 % of tested cows. Since the early 90s, it was in Britain gradually implemented, incorporating Mg in concentrates. To achieve the „extra dietary“ requirement level 30 g of daily Mg-intake with certainty, Mg was included in feed, thus not leaving any option to the dairy cow, about the Mg intake. In lactating cows at pasture, more palatable Mg-cobs were used. Subclinical/chronic hypomagnesaemia decreased to about 3-4 % in dairy cows.

## **Conclusion**

Based on this interpretation ( BSE/Mg), should be similarly preventive do about the AD? Recent research has shown that increased magnesium (Mg) in the brain, prevents Alzheimer´s in mice. There the Mg acts as a „natural“ antagonist of NMDA receptors. In addition another, although „synthetic“ NMDA receptor antagonist (memantine) is a drug in AD.

**Keywords** : Neurodegeneration , Magnesium deficiency , NMDA receptors , BSE in United Kingdom, Alzheimer´s Disease

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## **Bovine Spongiform Encephalopathy (BSE) Prevention and Alzheimer´s Disease (AD) Connections?**

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### **E-poster 196 BM**

#### **Purpose**

The neurodegenerative diseases, occurred to a greater extent, only in ruminants (BSE, scrapie, chronic wasting disease), because only in them, magnesium is not absorbed in the intestine, but in the rumen. From the literature it is known that in British cows at 1980s a higher incidence of subclinical (chronic), hypomagnesaemia was found and a new BSE disease appeared. After 1993 began to significantly reduce the incidence of the BSE. The aim of this study was to determine what

changes in the period about 1985- 1995 have occurred in British cows, in this context.

### Materials and Methods

In order to detect changes in the supply of magnesium (Mg) in cows (1985-1995), in the form of mineral supplements, there was the intention, data from two British producers obtained. Unfortunately, this failed to realize, so only published literary data are used in this context.

### Results and Discussion

Mg- deficiency among British cows has been known since the 1960s. In early 1980s (published in Outlook on Agriculture; 1983), it was stated that in most circumstances there is no safe alternative to providing extra dietary Mg, with daily 30 g of available Mg per lactating cow being an average target. However, mostly were available commercial Mg-blocks with very considerable variation in palatability, mostly very low Mg intake. So usually the veterinary survey has been; the subclinical hypomagnesaemia was found in about 7-15 % of tested cows.

Since the early 1990s, it was in Britain gradually implemented, incorporating Mg in concentrates. To achieve the „extra dietary“ requirement level 30 g of daily Mg-intake with certainty, Mg was recommended to include in feed. Thus not leaving any option to the dairy cow, about the Mg intake. In lactating cows at pasture, more palatable Mg-cobs were recommended to use. All other approaches have significant practical drawbacks and have been seen to fail (Whitaker et al., 1993). In other study about the Mg palatability, there is recommended a need for commercially available blocks to be evaluated before being put on the market (McCoy et al., 1994)..

During the 1980s, the occurrence of hypo-Mg in the UK cows was complicated by higher protein intake- high level of ammonia in the rumen and insoluble quante ( $MgNH_4PO_4$ ) formation. A new system has been introduced (ARC, 1980) and the dietary intake crude protein divided into undegradable dietary protein (UDP) and degradable dietary protein (DDP) fractions. However, this ARC publication, as an official proposal for this country, grossly overestimated the requirement of cattle for the protein (Alderman, 1993).

In the 1990s, there from 1993 (Whitaker et al) is the last known national monitoring of Mg- deficiency. Subclinical/chronic hypo- Mg often goes unrecognised, but recently investigations have revealed an annual rate of 3-4 per cent in British lactating dairy cows. At present British experts say; Mg-supplements should be mixed with the diet and provided so that every individual receives the necessary dose.

The best method is to use 60g magnesium oxide (calcined magnesite) per cow per day. The granular or powdered form? The powdered Mg- oxide will deliver a higher rate of Mg absorption in cattle, Mg-experts say.

Absorption and in the body higher Mg- utilization it is also very important in people. Researchers have found (Slutsky et al. 2010) that a new highly absorbable form of Mg called Mg-L-threonate concentrates more efficiently in the brain, rebuilds ruptured synapses, and restores the degraded neuronal connections observed in Alzheimer's

disease (AD). In this context, recently has been confirmed well-known fact, that activation of extrasynaptic NMDA receptors promotes cell death and thus contributes to the etiology of AD, which can be blocked by an AD drug, memantine (Wang and Reddy, 2017).

### Conclusion

Based on this interpretation ( BSE/Mg), should be similarly preventive do about the AD? Recent research has shown that increased Mg in the brain, prevents Alzheimer's in mice. There the Mg acts as a „natural“ antagonist of NMDA receptors. A number of studies suggest that eating certain foods may help keep the brain healthy. Unfortunately, there is no emphasis on the AD preventive effect of eating food, rich in magnesium. Perhaps this knowledge (research) may be blocked by still valid infectious BSE hypothesis (from 1988). So the professional medical community does not question about the correctness of this hypothetical veterinary diagnosis. However, more accurate informations about Mg-supplementation in British cows are needed, from the early 1990s.

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