HLASNY,J.; Infectious diseases derived from meat and bone meal (MBM); epidemics that never was? Vyzkum v chovu skotu (Agrovyzkum Rapotin), 61, 2019 (2); 23-29

My knowledge about meat and bone meal (MBM) feeding is based on findings from a long-term experience in the field conditions. After five years (1969- 1975) as a veterinary surgeon (large animals) I worked as a veterinary inspector for farm animals nutrition, in South Bohemia (1975- 1990).

On many occasions I found, that cows have ceased to receive the mixed feed, which was manufactured and intended for poultry, when contains MBM. It did so in error in the mixing feed company. In 1991, as assistant professor from the Brno Veterinary University, I worked almost for a year, with an international team at West Virginia University. There I from the journals found that in Britain new disease bovine spongiform encephalopathy(BSE) occurs. To my great surprise, British vets diagnosed it (Veterinary Record, December1988) as the cause of the disease MBM, which was in Britain reportedly fed in cattle for several years.

It was suggested that exposure began in 1981/82 and that the majority of affected animals became infected in calfhood. Then I said, it is interesting; in Czechoslovakia cows do not eat MBM and British cows yes?

1988- 2000; First epidemiological field BSE studies in the United Kingdom (UK) and ten years later, experimentally initiated BSE, without MBM feeding

In 1987/88 there 156 confirmed BSE cases in 145 cattle herds (with at least one confirmed case), and „hypothetical“ was concluded, that BSE has an origin in MBM feeding, when majority of cows become infected as the newborn calves. Based on the computer simulation models, see only 1-2 cases BSE/ herd. So, not a classical infectious epidemy, without experimental confirmation of MBM in cows-calves feeding . This first epidemiological study was constructed in intention determine the hypothesis that BSE is caused by a transmissible agent (Wilesmith et al., 1988).

NOTE; In this field study, which became the basis for infectious "BSE – scrapie – vCJD" hypothesis (to date!), there no information about MBM feeding. In addition, newborn calves would die, when MBM was fed !

At the same time (1988- 1990), in Northern Ireland – no feeding MBM in cows was found, examined by Wilesmith´s team. They concluded that the findings were consistent with the current hypothesis, that affected cattle had been exposed to a scrapie-like agent via cattle feedstuffs containing ruminant-derived protein. However, a preliminary investigation of the potential sources of infection for cattle in Northern Ireland did not provide any conclusive evidence (Denny et al. 1992).

Tha same was found, when fourteen cases of BSE were diagnosed on the basis of clinical examination in a closed herd of British Friesian cows, during a 9-month period from October 1987 until June 1988. No protein of animal origin had been fed to either heifers or cows, in this herd during the past 5 years and there had been no direct contact with sheep. The herd consisted of 500 cows, the average lactation yield was 5500 litres in 305 days (Winter et al., 1989).

Ten years later, the BSE disease was tested in dairy cows,"nutritonal experiment" performed in England, see three publications (Dewhurst et al., 2000; Moorby et al., 2000; Moorby et al., 2000a). This experiment was conducted using diets and other conditions typical of north western Europe, under well defined conditions of husbandry and nutrition, without MBM feeding! The effect of altering the amount of protein and energy over the final 6 wk of the dry-period diet, and during the first 21 wk of the subsequent lactation was investigated, in 47 dairy cows. Perennial ryegrass silage was used ad libitum plus a concentrate with high crude protein **(CP)** level (22.5%). High levels of plasma urea-N were found during lactation and also during dry period. However, after the collection of the last blood sample (21 wk of lactation), six of the 47 animals developed clinical signs of BSE. So, after long-term (28 weeks) dietary protein surplus (18- 20% of DM) was fed, when only 15% of protein in feed ration was needed, and ca 13 percent of dairy cows developed BSE! Such a high percentage of BSE disease was never found, under normal conditions in none of British cow herd.

NOTE; The findings about BSE in the UK, confirm my experience, that cows (cattle) do not eat MBM , even if they have been diagnosed with BSE!

1988- 2012; The occurrence of BSE in countries of European Union

After 2012, the incidence of BSE in the world decreased significantly (according to OIE statistics), with no BSE detected in the UK in 2016, there only one BSE case was detected in France and Spain.

Through the end of 2012; 184,621 cases of BSE (mostly to 2000; 180,845 cases) had been confirmed in the United Kingdom (UK), in more than 35,000 herds, BSE peaked in 1992 (37,280 cases).

In 2001, the EU introduced compulsory testing on BSE. As the table shows, until then was greater incidence of BSE detected only in the UK, Ireland, Switzerland, Portugal, France. Conversely, from 2001 there is a beginning of BSE incidence in other EU countries, see in particular Spain.

European countries; about the highest incidence of BSE

	to 2012 BSE cases	**to 2000 BSE cases**	**"BSE peak" year/ cases**
UK	184, 621	180,845	1992/ 37 280
Ireland	1,653	442	2002/ 333
Portugal	1,082	159	1999/ 159
France	1,015	80	2001/ 274
Spain	785	0	2003/ 167
Switzerland	464	335	1995/ 68
Germany	412	6	2001/ 125
Italy	144	2	2001/ 48
Belgium	133	19	2001/ 46
Netherlands	80	8	2002/ 24
Poland	73	0	2005/ 19
Czech Republic	30	0	2005/ 8

The table also shows that in addition to UK and Ireland; also in Switzerland in the mid-90th years, has been the high incidence of BSE. In addition; were also cows in Switzerland fed infected MBM, originating from the UK?

1995 - 1996; The discovery of the human form of BSE, new variant CJD (vCJD)

In 1995 and early 1996, a small number of cases of CJD with a remarkably early age at death (29 years) were identified in the UK. With an unusual clinical and pathological phenotype for CJD, including extensive deposition in the brain of florid plaques. British scientists have found two cases of sporadic CJD in teenagers and in a dairy farmer published in The Lancet (October, 1995). However, scientists emphasized that it is necessary to have definitive experiments, to establish whether BSE can transmit to humans. So, it is necessary act quickly, there is a risk (for the government), threatens reparative legal proceedings (Almond et al.,1995). Therefore American scientist Paul Brown wrote (25 November 1995) in British Medical Journal (BMJ) as follows; „With respect to the four farmers, it is also true that each had at least one infected cow in his herd, raising the possibility of contact infection from the cows or even inhalant infection from the contaminated meat and bone meal feed that caused their illness… Finally, there does not seem to be any need for new governmental hearings, committee meetings, or parliamentary debates about what more might be done because the precautions taken some years ago to eliminate potentially infectious products from commercial distribution were both logical and

thorough. We are left looking at possible present consequences of past events over which we now have no control, and we can only hope that the affair will be happily resolved. At least we do not have to face the spectre of reparative legal proceedings, which in this case would amount to a class action suit for anxiety brought by the entire British population against its own government" (ALMOND et al., 1995).

After this "published" threat, pointing to a possible punishment for the British government, the neurologist R.G. Will (National Creutzfeldt-Jakob Disease Surveillance Unit in Edinburgh) wrote the following letter to British neurologists, in just four months (journal Lancet; March 21, 1996);

"In the past few weeks we believe we may have identified a new clinico-pathological phenotype of CJD which may be unique to the United Kingdom… COULD YOU PLEASE NOTIFY THE CJD SURVEILLANCE UNIT OF ANY SUCH CASE WITH THIS CLINICAL OR NEUROPATHOLOGICAL PROFILE, WHETHER OR NOT THE PRESENTATION IS IN THE YOUNGER AGE GROUP? COULD YOU ALSO CHECK YOUR RECORDS AND NOTIFY ANY SIMILAR CASES THAT MAY IN RETROSPECT FIT WITH THIS CLINICOPATHOLOGICAL PROFILE? Four of the recently identified cases were confirmed by brain biopsy. If you are considering brain biopsy in any suspect cases of CJD it is essential to follow the Department of Health guidelines which state that neurosurgical instruments used on any case of CJD must be destroyed and not reused… The identification of a form of CJD that might be casually linked to BSE will result in widespread anxiety and concern..."

Then, even faster (in a few days), on April 6, 1996, in the scientific journal Lancet (Will et al. 1996) was published a "discovery" of a new disease (new variant of CJD) when it was found that a total of 10 people (in words; ten people!) could be infected with beef. Immediately afterwards, in May 1996, not only in Britain but throughout the world began to spread the news of the vicious and incurable disease transmitted to humans.

1996- 2006; Many scientists have tried to legalize BSE / vCJD infection. However, the predicted "vCJD epidemic" disappeared; so began research about blood transfusion and surgical instruments infection

In the UK after finding BSE in 1986 (in 1987, 446 cases) in the previous period the population probably has consumed hundreds (thousands?) of BSE affected animals (including nerve tissue). However, after about 30 years later, the infections of new variant CJD (vCJD) in the UK disappeared, when in 2000 there this disease peaked (28 cases). How can be this "phenomenon" explained?

The incidence of vCJD disease was (UK) from 1995 following; 1995 (3 cases vCJD);1996 (10);1997(10);1998 (18);1999 (15); 2000 (28); 2001 (20); 2002 (17); 2003 (18); 2004 (9); 2005 (5), 2006 (5); 2007 (5); 2008 (2); 2009 (3); 2010 (3); 2011 (5); 2012 (0); 2013(1); 2014-15 (0); 2016 (1); 2017-19; zero cases vCJD); TOTAL (176 cases of vCJD). In the all world media, there is published number of 178 cases, in fact it was only 123 neuropathology confirmed cases.

Next supporting evidence (gradually to 2003) about BSE and vCJD transmission is mostly related to description in high-ranking journals (Lancet, Nature, Science, PNAS…), In 2010, two articles were published (journals; Pract. Neurol. and Lancet) with a question mark, see the following article titles; Variant CJD: where has it gone, or has it? Variant or sporadic Creutzfeldt-Jakob disease? These last two articles (2010) suggest that the entire scientific saga of BSE transmission to humans has questionable foundations, and that both CJD diseases are not infectious, because they occur only sporadically. In addition, this BSE/ vCJD saga originated and finished in the scientific journal Lancet.

Until 2003, about 20 publications in scientific journals were written on this topic, the most frequent authors were; RG Will, Cousens SN, Ironside JW, PG Smith and RS Knight. However, last mentioned Professor Knight apparently contributed to another "discovery", concerning the transmission of the disease (vCJD) by blood transfusion. However, this could be a confusion of diagnosis (change CJD to vCJD diagnosis ?), as a “detective story”.This follows from the text of the article in The Telegraph (Lambert, 2010) as follows;

“ Judy Kenny´s husband Deryck, died of vCJD on October 24 2003. He was the first person recorded to die from the disease contracted via a blood transfusion in the UK... In November 2003, Judy received a phone call from the hospital to say that Deryck’s death was due to the sporadic form of CJD. But then Prof Richard Knight, consultant clinical neurologist at the National CJD Surveillance Unit in Edinburgh, telephoned her and asked to meet. He revealed that Deryck’s death was due to vCJD – and that he had probably contracted the disease from contaminated blood given in a transfusion during his prostatectomy...”

And again very quickly (February 7, 2004) his “discovery” was published (again in The Lancet) as follows; “One of these recipients was identified as developing symptoms of vCJD 65 years after receiving a transfusion. However, the age of the patient was well beyond that of most vCJD cases „ (Liewelyn et al., 2004). Almost immediately after the vCJD disease was first reported in 1996, concerns were raised about the possibility of transmission between british humans through blood transfusion (Ponte, 2006). The risk was purely hypothetical in nature, as there was no

evidence of transfusion transmission having taken place. The first probable case of transfusion-transmitted vCJD would not be identified until late 2003 , the second in 2004 . And yet, many nations implemented regulations aimed at reducing the risk of such transmission while the risk was still hypothetical in nature (Ponte, 2006). So the American scientist is skeptical about blood transfer, when both cases were again published in Lancet. The second case was even less conclusive because it is a case of preclinical vCJD in a patient who died from a non-neurological disorder 5 years after receiving a blood transfusion. There should still be a known a third case, on February 9, 2006, announced by the UK Health Protection Agency. However, it is unknown from literatury sources. So, like the panic of transmitting BSE to humans, the panic of transmitting vCJD by blood transfusion, ended in both cases..

After the discovery of a new infectious vCJD disease, the scientists concerned began to interest if the disease could be transmitted by surgical instruments. Concerns that surgical instruments may transmit vCJD have been raised by the finding of PrPSc (scrapie prion protein) not only in nervous, but also in lymphatic tissue (1997-99; published mostly by Ironside JW, Collinge J, Hill AF). Instruments used for tonsillectomy or appendectomy on unrecognized vCJD sufferers could become contaminated with the agent.

In 2001 some scientists „hypothetical“ found, that prions are readily and tightly bound to stainless steel surfaces and can transmit scrapie to recipient mice after short exposure times. This system mimics contaminated surgical instruments and will allow an assessment of sterilisation procedures.

However, ten years later the same scientists (Weissmann, Collinge), in a starting new discovery, have shown for the first time that abnormal prions, that can cause fatal neurodegenerative disease, can suddenly erupt from healthy brain tissue (Edgenworth et al., 2010). Their study offers experimental proof that prions can in fact originate spontaneously, and shows that this event is promoted by contact with steel surfaces. Co-author of this study, Julie Edgenworth stated: "One theory for our observations is that the metal acts as a catalyst to promote the creation of prions from the normal prion protein present in brain tissue…"

NOTE; Ten years have been haunted by this "hypothetical" surgical transmission of infection, and even now, after almost another 10 years, this fear persists.

2001- 2006; Three experts strongly opposed BSE infection

Despite continued investigation the origin of BSE is not certain. BSE does not resemble any strain of scrapie. The whole idea of `strains' is based on prion disease in mice. Mice have no known naturally occurring prion disease.Therefore, the disease the mice get is not BSE and it is not variant CJD. Therefore, the temporal relationship between BSE and variant CJD only coincidentally supports the notion that BSE caused variant CJD, and as such is not strong evidence. The evidence other than this

comes from research using mouse models and analysis of subtypes of abnormal prion protein. This supporting evidence is related to papers published in high-ranking journals (1996-1999). The temporal relationship between BSE and vCJD (1990s) only coincidentally supported the notion that BSE caused vCJD, and as such is not evidence. Indeed, BSE has just appeared spontaneously (Brown, 2001).
Note; Professor David Ronald Brown, research scientist notable for his work on prion diseases. He served as a member of SEAC, the British government advisory board on BSE and related diseases.

In 1996 a new variant of CJD was described and tentatively linked to BSE as a possible cause. What was initially a speculation has now evolved into orthodoxy among the medical profession in the United Kingdom if not the whole of Europe. In this paper I examine the evidence for a causal link between new variant CJD and the BSE prion and argue in favour of the alternative hypotheses that the variant is not caused by the prion and is not new (Venters, 2001).
NOTE; Physician George A. Venters is a UK consultant in public health medicine.

Another German scientist Ronald Scholz, opposing BSE/ vCJD infection theory, says that there is no sound biochemical basis for believing the prion to be an infective agent which, if it enterS the digestive system, can cause damage to the brain. In his view, therefore, the alleged oral transmissibility, either within or across species, has no proper foundation. He points out that the procedure cited as demonstrating transmissibility, i.e. injecting material from the brain of diseased animals directly into the brain of another, is not a valid model of infection and certainly does not prove that the disease can cross between species. Moreover, he points out that no experiment of controlled feeding with MBM has been published. The author having examined the science related to prions concludes that there must be grave doubts about the hypothesis's validity (Scholz, 2006). .

NOTE; Professor Roland Scholz, physician and biochemist, research and teaching in the field of metabolism biochemistry, including dietary and metabolic diseases, formerly active at the Institute for Physiological Chemistry, Physical Biochemistry and Cell Biology at the Ludwig-Maximilians-Universität Munich.

2006- 2012; Is prion protein really infectious or is merely a secondary marker of the presence of the scrapie agent?
To determine the mechanisms of intestinal transport of infection and early pathogenesis, of sheep scrapie, isolated gut-loops were inoculated (Jeffrey et al., 2006). While their published research confirms that under laboratory circumstances prion protein can be absorbed across the gut, it also shows that this is unlikely to

occur in real life. In addition, the results show that the places in the gut that do take up these disease-associated proteins are different from the locations where infectivity is known to be amplified. This research questions whether prion protein is really infectious, and it suggests that prion protein is merely a secondary marker of the presence of the scrapie agent. If that is so, as their findings indicate, it might also be so for ostensibly infectious nature of prions in vCJD. That, however, leaves open the issue of what the disease's infectious agent might be (Jeffrey et al., 2006). Roger Highfield as science journalist in his report- article "Can this really kill you?"(The Telegraph; May 30, 2006) wrote; “The Nobel prize-winning hypothesis that infectious proteins can cause CJD and 'mad cow disease' is still being challenged…But even today, and almost a decade after Prusiner's Nobel prize, findings still challenge his hypothesis so that, at best, it seems incomplete and, at worst, it may even be wrong”.

Six years later, Prusiner´s team (Stohr et al., 2012) found the right recipe to show that the amyloid-beta (Abeta) protein involved in Alzheimer´s disease (AD) are prions. So really, abnormal proteins are a consequence of the disease process, rather than a cause? In the AD, the most prevalent cerebral proteopathy, the two principal aggregating proteins are β-amyloid (Aβ) and tau. Pusiner´s team investigators inoculated transgenic mice with purified brain-derived Aβ fibrils or aggregates made of synthetic Aβ peptides. Their results provide incontrovertible evidence that Aβ aggregates are prions and that the formation of Aβ prions does not require additional proteins or co-factors. This was the first to definitively show that Aβ deposition can be seeded in the brain by synthetic Abeta alone, solidifying the conclusion that a prion-like process of corruptive protein templating is involved. Knowing that amyloid-β and similar proteins act like prions, researchers are left wondering why no one has recorded a case of the proteins passing from person to person, when on the basis of laboratory results, all neurodegenerative diseases should be infectious… Taken together, these results point to amyloid-β and other neurodegenerative proteins as behaving like prions, says Neil R. Cashman, a neurologist at the University of British Columbia, in Vancouver. “It’s becoming a widely accepted idea,” he adds. “But it’s also opening a Pandora’s box” (Wolf, 2012).

However to date, Pandora’s box is still closed and the BSE still raises among people fears, that humans can infect from cows by tainted meat, infectious medical equipment, by infectious blood... As far as the economic consequences are concerned, these are enormous finances losses in the European Union, where all MBM produced since 2001 is disposed of (mostly incinerated) and american soya is being compensated for this loss of protein. It has been calculated that in Europe is annually destroyed 16 million tonnes of MBM, who is replaced by 23 million tonnes

of soybeans. In 2010, it was considered to lift the ban on the use of MBM in animal feed in EU countries, when there were enough reasons to lift this ban (Ziggers, 2010). Unfortunately, the European Commission finally did not give its approval. However, in the United States, Canada , where BSE has also been detected, MBM continues to be used in pig and poultry. In addition, from 1989 the UK was exporting about 25,000 tonnes of MBM to EU countries and about 7,000 tonnes to nations outside Europe mostly in the Middle East and Africa. By 1991, sales of MBM to Europe dropped to zero. At the same time exports of MBM to the Third World had soared to 30,000 tonnes (Barnett, 2000). However, not to Japan (see after two decades later); in Japan 36 cases and in Third World, no case of BSE.

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