Curriculum vitae (professional)

HLASNY Josef, born on 30 August 1946 in Velke Losiny (Sumperk district, Czech Republic), retired from December, 2007. Last occupation; private veterinary surgeon in Bludov and external animal nutrition scientist in Research Institute for Cattle Breeding in Rapotin (both; Sumperk district, North Moravia, Czech Republic). After the retirement, I attended as a presenter, at three World Veterinary Congresses (2008, 2013, 2015).

1. Education

a) 1960- 1963 High School (Gymnasium) in Sumperk
b) 1963- 1969 University of Veterinary Medicine in Brno (Czech Republic).
   There I obtained the diploma „Doctor of Veterinary Medicine“ (MVDr.) on 30 March 1969 and the „CSc.“ diploma (Candidate of Veterinary Sciences) on 23 March 1991.

2. Activity

a) 1969- 1972 Veterinary surgeon in district of Znojmo
b) 1973- 1975 Veterinary surgeon in district of Strakonice
   There also as veterinary hygiene inspector in two abattoirs; large animals and poultry in Vodnany
c) 1975- 1990 Animal nutrition specialist and veterinary surgeon in district of Pisek
d) 1990- 1995 https://www.vfu.cz/index_en.html Assistant professor – Department of Animal Nutrition, University of Veterinary Medicine and Pharmacy, Brno
e) 1995- 2000; http://www.vuchs.cz/agrovyzkum-rapotin/index.php Animal nutrition scientist in Research Institute for Cattle Breeding in Rapotin
f) 2000- 2007; http://www.bludovska.cz/ Private veterinary surgeon in a large agricultural enterprises (joint-stock company) „Bludovska,a.s.“ in Bludov (80% cattle, 10% pigs, 10% poultry), in the area between the town of Zabreh and Sumperk (about 15 villages)
g) 2004- 2007; External animal nutrition scientist (Research Institute for Cattle Breeding in Rapotin)
h) 1999- 2015; Veterinary surgeon, private veterinary ambulance (small animals) in Bludov

To date I published 150 articles (see my „curriculum vitae“ in Czech), mostly in Czech journals (1986- 2015), including of three Czechoslovak patents (1990- 1992) and also my website www.bse-expert.cz.

I finished this website to the end of August 2006. From September 2006 to date I sent hundreds of comments to the world media. It concerns (my opinion, supported by the world literature) that the BSE (bovine spongiform encephalopathy) and other neurodegenerative diseases; are not infectious, so it is a spontaneous (metabolic) disease in some individuals. Concerning this subject, I attended as a „poster presenter“, at three last World Veterinary Congresses (2008, 2013, 2015).

Some details about my professional activity (from 1980s)

Ad 2c. From July 1, 1975 to September 30, 1990 (District Veterinary Administration in Pisek, South Bohemia)

Continuously „specialist- inspector for animal nutrition“ (Pisek district), including input and output veterinary control activities, in two feed mills (Zahori and Kvechov).

Sampling of biological material (cattle and pigs) for biochemical examination, and interpretation of the findings following a comprehensive assessment of the animal nutrition (an average; about 40 farms per year; period 1975-1990).

In addition to these activities, veterinary jobs at these positions- sites;

a) from January 1, 1977 to December 31,1977; veterinary surgeon; „Dairy cows- factory farm“ in Cizova
b) from April 1 to December 31,1978; poultry slaughterhouse in Mirovice; Head of Veterinary hygiene Centre
c) from June 1 to December 31, 1978; veterinary surgeon in large „Pigs- factory farm“ in Lety
d) from January 1,1984 to 1990; as an external specialized worker for;
   - Regional Agricultural Laboratory in Pisek; interpretation of laboratory results into practice (chemical feed analysis)
   - Central management for cattle feeding in Pisek; Optimization of cattle feed rations, with the use of computer technology in practice

To the end of the 1980s I published laboratory results in two Czechoslovak scientific journals (Veterinary Medicine and Biology- chemicals- veterinary);

- HLASNY,J.: Content of macrominerals, nitrates, and soluble sugars in feedstuffs. Veterinary Medicine (Prague), 1989; 34: 567-576
- HLASNY,J.:_Mineral feed additives and macro mineral composition of preserved roughage. Veterinary Medicine (Prague), 1989: 34; 689-698
- HLASNY,J.:Providement for an optimum supply of sodium and magnesium to the feed rations of dairy cows and high pregnant heifers. Biol.Chem. Vet. (Prague), 1989; 25: 157-169
- HLASNY,J.: Evaluation of a new mineral supplement in young cattle feeding during winter season. Veterinary Medicine (Prague), 1989; 34: 717-725 (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=2631375&dopt=Abstract).

In the last two publications, there is the recommendation that much more magnesium (by 180%) is necessary in dairy mineral supplements. Really; it was commonly realized in

Europe, at the beginning of 1990s. Therefore if we will put this „phenomenon“ into European practice; significantly higher additional dietary Mg-supplementation - can be a cause about the BSE incidence decrease, especially in the UK, after 1993/94 period. These similar results, as a field experiences were also patented by the“Czechoslovak Patent Office“, see; HLASNY,J.; Mineral supplement for breeding cattle. Patent No. 274 171. Prague 1991.

This phenomenon about the „European great Mg interest“ at the beginning of 1990s, it was also presented at the „3rd European Congress on Magnesium“ (March, 1990) in Geneve. During 1980s I collaborated with well known Czech neurologist Ladislav STEIDL (Palacky University, Olomouc), a member of the editorial board (1975- 2000) of the International scientific journal „Magnesium research“ (London- Paris).

I had a lecture as oral presentation (in French) in Geneve at „3rd European Congress on Magnesium“ (March, 1990).

See our publication;

- HLASNY,J.- STEIDL,L.: Mechanism of magnesium deficiency in feedstuffs and in nutrients. Magnesium Research. 1990, 3: p.48

This was my first travel to „western countries“ (Austria, Switzerland), because I had my passport as far as from January 1990. Our family in Bludov was persecuted (about the passport), because my uncle Karel Hlasny (brother of my father; born 1911) escaped from Czechoslovakia in 1948- as chief of Staff of the 1st Armored Division in Prague with the rank of Gen Staff Lt/Col. In California, there he was as instructor (Defense Language Institute, Presidio of Monterey) from 1952 to 1979. See;
http://www.rimbachvets.org/index.php/main/content/links-to-related-web-sites/

Ad 2d. From October 1, 1990 to February 28, 1990 (University of Veterinary Medicine and Pharmacy in Brno)

After the „1989 revolution“ in former Czechoslovakia, there were changes about the staff of the University of Veterinary Medicine and Pharmacy. I was selected (from October 1, 1990 for animal nutrition teaching, because I had more of experiences as veterinarian- animal nutritionist in the field conditions (see above- bellow mentioned scientific journal- articles). During my teaching in Brno, see these publications;

a) Laboratory results (as a field experiences from South Bohemia) have been patented, see three Czechoslovak patents;

- HLASNY,J.: Mineral supplement for breeding cattle. Prague 1991. Patent No. 274 171

It has been concluded from the results (see; total of 260 tons of a new mineral feed supplement was used on farms in the Písek district) of the experimental and farm-scale testing

that the new mineral supplement, containing much more magnesium (by 180%) and less phosphorus (by 46%); than the commercially produced supplements produced in Czechoslovakia; is able to meet all the phosphorus requirement, together with a rational management of magnesium, in young breeding cattle.

Similarly (higher magnesium content), we patented with my brother- chemical engineer , two drugs for newborn calves (especially);

- HLASNY, Josef.- HLASNY,Karel.: Electrolyte replacement for oral application I. Prague 1990. Patent No 268881
- HLASNY.Josef.- HLASNY,Karel.: Electrolyte replacement for oral application II. Prague 1992. Patent No 277602

b) Other laboratory results (as a field experiences from South Bohemia) were published in these two scientific Czechoslovak journals;

- HLASNY,J.: Evaluation of some relationships between macrominerals and nitrogen compounds in forages. Agrochémia (Bratislava), 1990 (1); 30: 28-32
- HLASNY,J.: Influence of nutrition on acid base balance and serum magnesium in dairy cows. Biopharm (Prague), 1991;1: 51-60

During animal nutrition teaching in Brno I continued about the „mechanism of Mg-deficiency“ concluded as the disertation work („PhD thesis“)“Causes and compensation of magnesium deficiency in ruminants“ (March 23, 1991). In Geneve (3rd European Congress on Magnesium, March, 1990) I obtained some connections with Mg- researchers and after few months later (during my teaching in Brno) I received the invitation (December 1990) from West Virginia University as a „Visiting Research Fellow“. There I collaborated (April 1-December 31, 1991) with an international team headed by professor R.L.Reid . We participated at the grazing trial; it has been as a cooperative grant between West Virginia University and the USDA-ARS-NAA Appalachian Soil and Water Conservation Research Laboratory, Beckley, WV (CRIS No.l932-23330 - 00l- 00D) on a project entitled „Improved Forage and Sheep Production Efficiency“.

The metabolism of ewes has been tested at three different pastures during the five- weeks period (April- May 1991). Spring pasture growth and its feeding, it was an example of the nutrition changes after the winter ruminants feeding when nitrogen- potassium level in feeds is low compared with their level in young pasture. The results with almost of 400 nutritive values of forages and nearly 5000 biochemical values were obtained.

See our work published in Journal of Animal Science;

- COX-GANSER,J.M.- PUOLI,J.R.- HLASNY,J.- REID,R.L.: Mineral status of ewes and quality of different grass and legume pastures for spring grazing. J. Anim.Sci. /Suppl./, 70, 1992: 182.

During my cooperation with professor R.L.REID at WVU (Morgantown), there my work- schedule has been following (about these activities, see the „photo gallery“ in my website);

a) WVU- experimental station; taking forage- pasture, blood, urine (catethrization) samples (April- May, 1991)
b) WVU; collection of obtained results (June)
c) WVU; study of the etiology and treatment of „switchgrass toxicity“ in sheep (based on the Ca- Mg antagonism, and vegetative nervous system function), according to the findings of Czech professor PhDr., MUDr. et MVDr.h.c. Jan BEČKA; in lambs from experimental ewes (July)
d) WVU; nervous diseases of ruminants study and interpretations of experimental results (August)
e) lectures and consultations (State Universities): about the K (Ca) and Mg antagonism concerning following subjects (September, 1991);
   Colorado- Fort Collins (Dr.Mortimer); neonatal diarrhea in calves
   Utah- Logan (Dr. Ralphs); kochia scoparia toxicosis in animals
   Oregon- Corvallis (Dr. Blythe); degenerative myeloencephalopathy in horses with access to pasture grasses
   Oklahoma- Stillwater (Dr.Tucker); tall fescue toxicosis in cattle
f) literature collection about obtained results interpretation (October- December, 1991).

Ad 2e. From March 1, 1995 to December 31, 2000 (Research Institute for Cattle Breeding in Rapotin)

I finished my teaching-work in Brno to the end of February 1995 and I obtained a new post as animal nutrition scientist in Research Institute for Cattle Breeding in Rapotin (from March 1, 1995 to December 31, 200O). There I continued about my field experiences on the grant (as leader of the working team) „The causes of milk production stagnancy in the dairy herds- Moravia and East Bohemia regions“ (January 1995- December 1997) as the grant NAZV No 5149. Simultaneously I continued with the collaboration in two selected dairy farms about the work „The efficiency of the rapeseed products feeding in dairy cows“ (January 1997- December 2000) as the grant NAZV No EP7150.

There I had these scientific presentations;

a) 47th Annual Meeting of the European Association for Animal Production (1996);

   - HLASNY,J.- PINDAK,J.; Thymus development in calves kept under normal feeding regimen. In: Book of Abstracts of the 47th Annual Meeting of the European Association for Animal Production. Lillehammer 1996; 145.

The full text of our paper, it was „reprinted“ in the scientific Croatian journal „Stočarstvo“ (Zagreb, 51, 1997(1); 15-21

b) 48th Annual Meeting of the European Association for Animal Production (1997);

- HLASNY,J.- PINDAK,J.; Ketonuria as an indikator of hyperketonaemia in stanchion housed dairy cows especially. In: Book of Abstracts of the 48th Annual Meeting of the European Association for Animal Production. Vienna 1997; 112

c) 31st International Congress on the History of Veterinary Medicine (University of Veterinary Medicine and Pharmacy in Brno) Czech Republic, 2000);

- HLASNY,J.: Professor Jan Bečka and his still useful magnesium research of the thirties. In: Book of abstracts of „31st International Congress on the History of Veterinary Medicine“, 2000: 44

During my research in Rapotin I studied the literature sources obtained in West Virginia University- Evansdale library (1991), especially about the dietary protein surplus and the Mg-deficiency in ruminants.

This work or the „BSE study“ I finished and published in „Výzkum v chovu skotu“;

- HLASNY,J.: Nervous diseases and connections with nutrition in ruminants. Výzkum v chovu skotu (Rapotín), 43, March 2001(1): 13-29

This article it was „mostly reprinted“ in the International Journal „Feed Mix“;

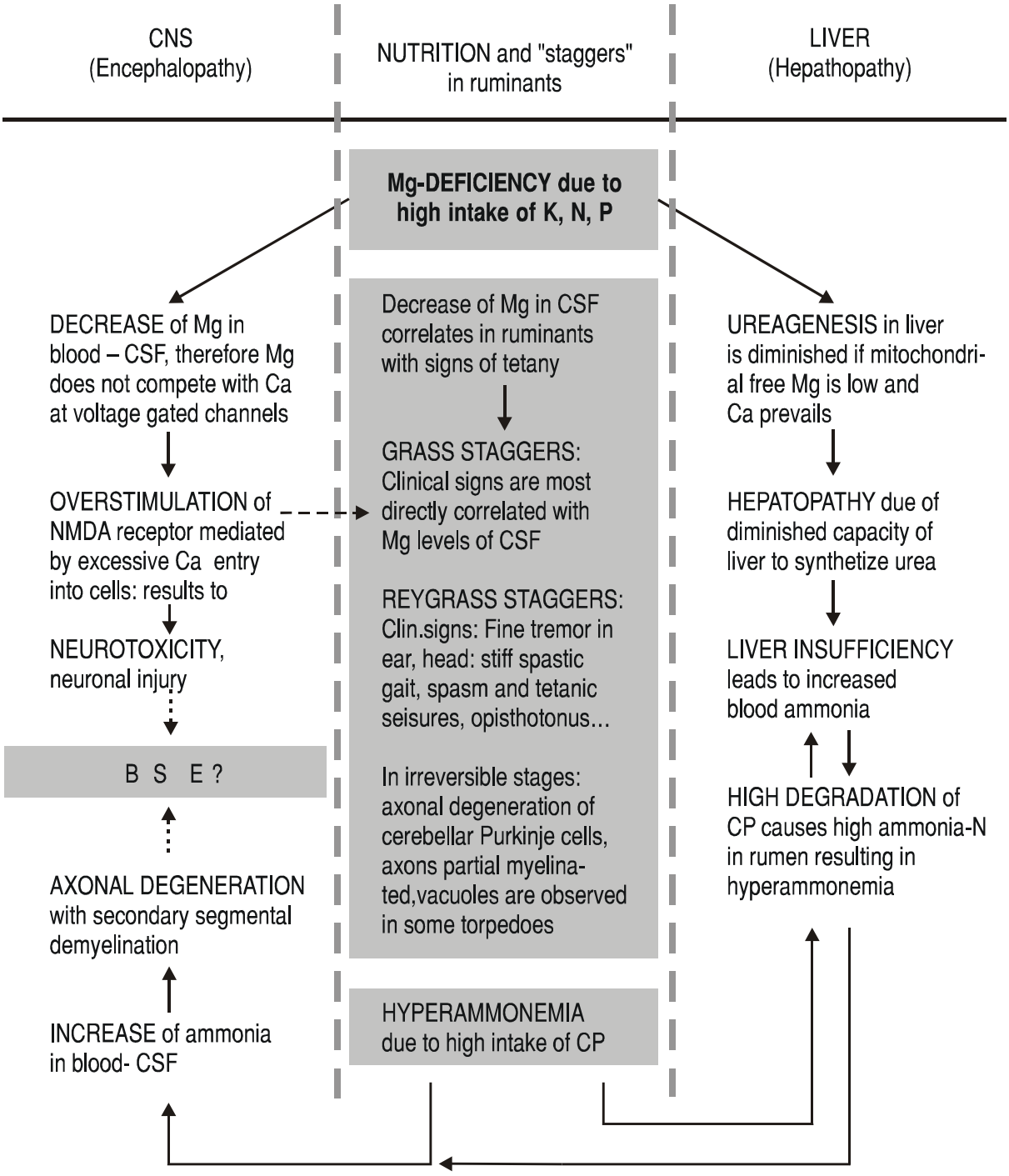
- Lone voices in the BSE debate. Feed Mix (International Journal on Feed, Nutrition and Technology- Elsevier Business Informations), May 2002 (2), 10: 12-15 http://www.warmwell.com/lone_voices_in_the_bse_debate%5b1%5d.pdf

There I wrote;

Hyperammonemia plus hypomagnesaemia „simultaneous“ action have a strong influence on the CNS, especially in ruminants (Mg absorption in the rumen, especially), so that the BSE has its roots in a more common nutritional problem. This alternative „ ammonia- magnesium theory“ is based on the chronic Mg-deficiency potentiated by hyperammonemia in ruminants. As a typical example; the ryegrass staggers is showed in ruminants. So, various clinical symptoms can be observed because the nervous system controlling both voluntary and unvoluntary muscles is affected (Mg and Ca disturbances). It seems, that during the chronic hypomagnesemic disease, the heavy weather changes (cold- rainy, windy...) or nutrition (high intake of crude protein...) stress - these episodes of acute abruptions, may accelerate the nervous, like to „BSE“ disease. If the BSE is involved; a longer- chronic action of corresponding biochemical changes in the blood (CSF) is necessary, to rise the irreversible neurodegenerative changes...

See the Figure 1, from; Nervous diseases and connections with nutrition in ruminants.Výzkum v chovu skotu (Rapotín), 43, March 2001(1): 13-29

Nervous diseases and connections with nutrition in ruminants



CNS: Central nervous system • CSF: Cerebrospinal fluid • CP: Crude protein
NMDA: "N-Methyl-D-Aspartate" receptor.

This figure (from 2001) I presented at three World Veterinary Congresses (2008, 2013, 2015).

However, it was a different „non infectious BSE view“, compared with the scientific „BSE infectious theory“. So I become as a „BSE disident“ in Czech Republic, without following projects-grant working, therefore I finished my research work in Rapotin.

Ad 2f. From January 1, 2001 - to the end of 2007 Private veterinary surgeon in a large agricultural enterprises (joint-stock company) „Bludovska,a.s.“ in Bludov

At this time (from July 1, 2004 to May 30, 2007) I also obtained the post as the „external animal nutrition scientist“ in Research Institute for Cattle Breeding in Rapotin.

So I returned to my former profession- veterinary surgeon (after 25 years!). This job I obtained at a large agriculture enterprise in „Bludovska a.s.“ with about 1,000 cows (mostly dairy farms) and about the same number of pigs and about 80,000 poultry (hens and chickens-broilers). And concerning the small animal veterinary practice; I continued in my private Veterinary ambulance, established at my home in Bludov (1999).

Thanks this private veterinary job, I continued about my „BSE - detective study“ – see following publications;

- HLASNY,J.: Nervous diseases and connections with nutrition in ruminants, or only the story of BSE ? Výzkum v chovu skotu (Rapotín), 44, December 2002(4): 10-33
- HLASNY,J.: Nervous diseases in connection with nutrition in ruminants, and the BSE. In; Book of Abstracts of the International Meeting- professor Jaroslav Kabrt, 100 years birthday. Brno, January 2003; 42-50
- HLÁSNÝ,J.: Některé poznatky z historie výzkumu transmisibilních spongiformních encefalopatií. 2003 (1): str.15-22
- HLASNY,J.: Some informations from United Kingdom concerning the history about Mg- research in ruminants; and the BSE. Výzkum v chovu skotu (Rapotín), 45, March 2003 (1): 22-34
- HLASNY.J.: Two views about the origin and spread of the BSE. Výzkum v chovu skotu (Rapotín), 46, March 2004(1); 28-40
- HLASNY,J.: BSE incidence with the dietary protein surplus in dairy cows – see one forgotten experiment . Výzkum v chovu skotu (Rapotín), 46, June 2004(11); 25-35
- HLÁSNÝ,J.: BSE differently? Zverokruh, The journal of the Chamber of Czech veterinary surgeons. 2004 (3); 11-13
- HLÁSNÝ,J.: It is indeed meat and bone meal originator of „ BSE infection“? Zverokruh, The journal of the Chamber of Czech veterinary surgeons; 2004 (5); 13-16
- HLASNY,J.: Long-term excess dieteary protein in the cows ration and the BSE (part 1). Zverokruh, The journal of the Chamber of Czech veterinary surgeons. 2004 (7); 9-10
- HLASNY,J.: Long-term excess dieteary protein in the cows ration and the BSE (part 2). Zverokruh, The journal of the Chamber of Czech veterinary surgeons. 2004 (8);11-13.
- HLASNY.J.: The retreat of the BSE infectious positions, crazy hypothesis; prion infection not exists. Zverokruh, The journal of the Chamber of Czech veterinary surgeons 2006(8);13-16

- HLASNY,J. : What it was, or still is, BSE? . Zverokruh, The journal of the Chamber of Czech veterinary surgeons. 2015 (12); 28- 30

From November 1, 2007 I prepared my papers concerning my website (www.bse-expert.cz) established in August 2006. These conclusions I presented as „Josef Hlasny, Veterinary ambulance, Bludov, Czech Republic“ at the last three World Veterinary Congresses;

1. 29th World Veterinary Congress, Vancouver, July 2008;

Neurodegenerative diseases and schizophrenia as a hyper or hypofunction of NMDA receptors

See the abstract;

Neurodegenerative diseases, including BSE, Alzheimer's disease etc. are caused by different mechanisms but may share a final common pathway to neuronal injury due to the overstimulation of glutamate receptors, especially of the N-methyl-D -aspartate (NMDA) receptor subtype. It is generally accepted that the influx of Ca2+ as a result of excessive activation of the NMDA receptor underlies the toxic actions of glutamate in many systems. Also, ammonia intoxication leads to excessive activation of NMDA receptors in brain. On the other hand, Mg2+ competes with Ca2+ at voltage- gated calcium channels both intracellularly and on the cell surface membrane. So, Mg2+ can protect against NMDA- induced neurodegeneration and Ca2+ deficiency can be important about „NMDA hypofunction“ in schizophrenia. In addition there can be another example about hypoglutamatergic condition; cannabinoids are known to inhibit Ca2+ channels- glutamate release in schizophrenia, and to inhibit progression of certain neurodegenerative diseases.

There are no scientific references to date in which high intake of crude protein (and potassium) high enough to lead to a state of hyperammonemia (and hypomagnesemia) during the incubation period of the BSE. Therefore there is the first idea of this review; to show the hyperammonemia plus hypomagnesemia“simultaneous“ action on the ruminant tissues. So the various clinical symptoms can be observed because the nervous system controlling both voluntary and unvoluntary muscles is affected (Mg and Ca disturbances). If the BSE is involved; a longer- chronic action of corresponding biochemical changes in the blood (CSF) is necessary, to rise irreversible neurodegenerative changes.

Recently was found that elevated manganese in blood was associated with „prion infection“ in ruminants. These findings about „manganese theory“ act in concert with this „BSE ammonia- magnesium theory“. So I will perform some interpretations about this connection and some details will be presented to the Congress, and also second idea of this review; to show that cannabis use can be a proof about the link between the NMDA receptor hyperfunction (neurodegeneration) and hypofunction (schizophrenia).
(http://www.vin.com/Members/proceedings/Proceedings.plx?CID=WRLDVC2008&Category=3840&PID=23744&O=Generic)

2. 31st World Veterinary Congress, Prague, September 2013.

a) Hypotheses and facts about BSE/ vCJD in the UK, so meat and bone meal as infectious agens should be revised. ID 422

See the abstract;

In Britain, perennial ryegrass mostly fed (1980s); protein content reached to over 300 g/kg dry matter in young, heavily with nitrogen fertilized grasses (high protein, potassium content); conditions for chronic hypomagnesemia (see; England- Wales; highest BSE incidence; rainy, cool weather and ryegrass - low magnesium content- feeding in high producing dairy cows, exception in the world).

In 1987/88; there 156 confirmed BSE cases in 145 cattle herds (with at least one confirmed case) and hypothetically 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- not classical infectious epidemy; without experimental confirmation- MBM in calves feeding).

In 1991/92; there 513 dairy and 1266 suckler cow herds were sampled, and serum blood Mg below 0.8 mmol/l was found in 28% of cows. Later, 1993/94, there significantly higher dietary Mg- supplementation was realized and BSE incidence significantly decreased (Note; Czechoslovak patent -1991; recommendation to increase the magnesium content by 180%, in feed supplements for cows).

Eurostat data (1990-2000) indicate that UK exports of MBM was in total; 229,000 tonnes, mostly to the Africa- Asia; however, not to Japan (see; after two decades later; in Japan 36 cases and Third World- no case of BSE, to date).

Dietary experiment (England, 2000) confirmed BSE (13% of 47 dairy cows), after long-term protein surplus, with ryegrass feeding (without MBM).

Some other important details and relationships will be presented at the Congress.

b) The origins of the neurodegenerative diseases may lie in chronic magnesium deficiency coupled with a high protein intake. ID 430

See the abstract;

Epidemiological incidence of neurodegenerative diseases in a certain period, was only detected in cattle in the UK (BSE). This happened at a time, after significant increase of crude protein, in dairy rations (ARC, 1980) in the mid of 1980s, without equality of dietary Mg-supplementation. Feeding readily fermentable young grass (there especially most common ryegrass), leads to intraruminal ammonia concentrations (up to 30-70 mmol/l), and to decrease of magnesium absorption.

Neurodegenerative diseases, are caused by different mechanisms, but the common denominator of neuronal injury, is overstimulation of glutamate receptors. In excess, glutamate triggers a process called excitotoxicity, causing neuronal damage, particularly when NMDA receptors are activated. An important consequence of NMDA receptor activation is the influx of Ca2+ into neurons, Mg2+ can protect against NMDA- induced neurodegeneration.

So the lower the Mg2+ level in the animal tissue cells, the more marked is „Ca-effect excitotoxicity“. It should be noted that NMDA receptor channel in Purkinje cells, has a more

extreme sensitivity to Mg2+, than that in other brain regions. Excitotoxicity can be found even with normal levels of glutamate, if NMDA receptor activity is increased, e.g., when neurons are injured-depolarized (more positively charged); this condition relieves the normal block of the ion channel by Mg2+, and thus abnormally increases NMDA receptor activity.

Well known is ammonia induced depolarization in cortical astrocytes, what results in removal of Mg2+, that normally blocks the NMDA receptor channel. Prolonged activation of NMDA receptors, was recently reported (Alzheimer´s research), to increase the neuronal production of amyloid β.
http://worldvet.org/uploads/docs/wvc-2013-03-bovine-medicine-posters-abst.pdf

3. 32nd World Veterinary Congress, Istanbul, September 2015;

Alzeimer´s disease and bovine spongiform encephalopathy (BSE) connections

See the abstract;

Prion diseases, also called transmissible spongiform encephalopathies (TSEs), are a group of fatal neurodegenerative disorders affecting animals (BSE, scrapie…) and humans (CJD…). Until recently, TSEs encapsulated a distinct category of neurodegenerative disorder, exclusive in their defining characteristic of infectivity (prion diseases). It now appears sclerosis (ALS).that similar mechanisms of self-propagation may underlie other proteinopathies (prion-like diseases) such as Alzheimer's disease (AD), Parkinson's disease (PD), and Amyotrophic lateral sclerosir (ALS).

However, only prion disease has been established as the sole „bona fide infectious" disease among these protein misfolding disorders (neurodegenerative diseases). The misfolding and aggregation of endogenous proteins in the central nervous system is a neuropathological hallmark of above mentioned neurodegenerative diseases. Prions („infectious") are produced by recruiting the normal cellular prion protein (PrPc) and stimulating its conversion into the disease causing isoform PrPsc (derived from scrapie). The new prion diseases that have emerged in the last 25 years are BSE and variant Creutzfeldt-Jakob disease (vCJD). The accepted cause of vCJD is that BSE spread from cattle to humans by the consumption of infected beef. However, the evidence that supports this is very thin. Despite probable widespread exposure of the UK population to BSE-contaminated food in the 1980s, there have been only fewer cases of vCJD, than researchers anticipated. The reasons for this are to date uncertain. The temporal relationship between BSE and vCJD (1990s) only coincidentally supported the notion that BSE caused vCJD, and as such is not evidence. The evidence other than this comes from research using mouse models and analysis of subtypes of abnormal prion protein. This supporting evidence was related to four papers published in high-ranking journals (1996- 1999).

These experimental mouse models were later supported by the mathematical BSE/vCJD models in about 13 scientific articles (1996- 2006). So these „infectious conclusions" were finished in 2006, when it was found that „Alzheimer's may 'seed' itself like BSE", if proteins taken from the brains of Alzheimer's patients and injected into the brains of genetically engineered mice trigger Alzheimer's-like lesions in the mouse brains, Later, many other similar studies showed that the pathology of AD, PD and ALS can be

transmitted to animals in a way similar to that by which a prion disease was transmitted with PrP inoculation. These neurological disorders can be produced by either peripheral (extracerebral) or direct brain (intracerebral) inoculation. Those findings provide evidence of cell-to-cell spread of pathologic proteins of neurological disorders in experimental animals, suggesting those pathological proteins may have seeding abilities, like prion diseases, to transmit pathology. Experimental studies have shown that the aggregation of the AD-associated proteins amyloid-β (Aβ) and tau, and of the PD-associated protein α-synuclein, can be stimulated in laboratory animal models by intracerebral injection of inocula containing aggregated species of the respective proteins.

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. However, to date, there is no direct evidence in humans indicating that the diseases caused by misfolded Aβ, tau, α-synuclein are infectious. Again, as in the case of BSE / CJD infection, reasons for this are uncertain.

Taken together, these results are consistent with the fact that BSE and scrapie (prion diseases) are not infectious (it has never been scientifically proven), as has been presented at the last World Veterinary Congresses (2008, 2013). Similarly, also other neurodegenerative diseases are not infectious, relevant connections mentioned above will be interpreted at the Congress.

Book of Congress Abstracts, p.225
http://www.wvcistanbul2015.com/files/Poster_Presentations.pdf

At all three World Veterinary Congresses, I presented as a „poster presenter“, because I do not speak English properly, required for oral presentation. In 1963 I graduated (high school in Sumperk) only from Russian and French...
Recently (October 2015), I summed up some important facts from all three World Veterinary Congresses, in the discussion at the British newspaper The Independent, see article; http://www.independent.co.uk/news/uk/home-news/mad-cow-disease-in-the-uk-what-is-bse-and-what-are-the-symptoms-a6675351.html

Even more details, it was in August 2012 for the American newspaper „USA Today“ (see my discussion; approx. 60 of typewritten pages), in article;
www.usatoday.com/money/industries/health/story/2012-08-03/Mad-cow-disease/56748052/1.

This happened after the discovery of the team of American neurologist Prusiner (Nobel Prize 1997), where he writes; „We report compelling evidence that Aβ aggregates are prions by demonstrating widespread cerebral β-amyloidosis induced by inoculation of either purified Aβ aggregates derived from brain or aggregates composed of synthetic Aβ“. http://www.ncbi.nlm.nih.gov/pubmed/22711819.

See other discussion (Aβ Sufficient for Seeding - But Is It a Prion?) about this publication; http://www.alzforum.org/news/research-news/av-sufficient-seeding-it-prion.

Also, from this time, see article „Alzheimer's Prion Connection“ (including my discussion); http://cen.acs.org/articles/90/i27/Alzheimers-Prion-Connection.html. There researchers concluded; knowing that amyloid-β and similar proteins act like prions, these

results point to amyloid-β and other neurodegenerative proteins behaving like prions, therefore have the potential to be infectious...

To date, I published 150 professional articles, the vast majority; (a) as my experiences from the „veterinary field conditions“ (b) in Czech - as a „separate author“ (see my professional biography in Czech version).

Mostly, thanks of my staying in West Virginia University (1991) as a Visiting Research Fellow.

Bludov, January 2016