Prions are a symptom of the (metabolic) „chronic wasting disease“ and do not cause the disease **(as a part of my eco-detective study)**

Contents

Introduction

Cattle ranchers in areas with infected deer and elk express concern that the disease could spread to their herds. But Beth Williams, a University of Wyoming veterinarian leading efforts to track the disease, says there is no evidence to support such fears. In continuing experiments run by her lab, **cows kept in pens with infected elk, and cows fed diseased brain tissue didn't get sick. Only when infected tissue was directly injected into their brains did cattle catch the disease**. Scientists say it's still not clear how CWD is passed between animals, although experiments on penned deer and elk show that it is contagious. Some experts say there also is no evidence that CWD poses a threat to people. "I would think there would be virtually no way history could repeat itself," says Dr. Miller, the Colorado wildlife veterinarian, referring to the way mad cow disease jumped the species barrier between cattle and human**(http://www.wapiti.net/news/default2.cfm?articleID=62)**. After 25 years and hundreds of millions of research dollars, no one has actually proved that prions cause disease! A small group of scientists, myself included, believe **prions are a symptom of the disease and do not cause the disease**. Prions are to TSE diseases as cancer cells are to cancer: a result, not a cause!… Here is a non-technical explanation of my CWD-TSE theory. The Spiroplasma theory I have presented in my paper is sufficiently different from current CWD causative theory to create a bit of a stir... (http://www.deer-library.com/artman/publish/article_142.shtml). Wildlife division veterinarian Mike Miller, who has done extensive study on CWD, doubts Schoonveld's theory. "In all the literature we have searched there never has been a mention of scrapie in sheep in those pens during that period. And even if there was, there is nothing to prove CWD is the result of a transfer of scrapie from infected sheep to deer or elk," Miller said (http://www.deer-digest.com/html/nov01.html#1).

Recent research (October, 2006) shows for the first time that chronic wasting disease may **spread through saliva and blood of infected deer, which poses new possibilities** that the disease may spread by blood-sucking insects or social contact between animals. The study also reinforces that no tissue from an infected animal can be considered free of prions, the disease-causing agent… The research, released in the Oct. 6 edition of the journal Science, tested the blood, saliva, feces and urine of deer infected with CWD to determine ways the disease may be transmitted from animal to animal, which has remained a mystery to scientists... **(http://www.cwd-info.org/index.php/fuseaction/news.detail/ID/19ad730128d756fd0a309e67a9ec92d2).**

However, according to the recent article „Should we still be worried?“ (January 10, 2007) (http://www.guardian.co.uk/g2/story/0,,1986657,00.html), there is a different view about the BSE infectiosity - see following text from this article; „ But despite billions spent on efforts to save Britain's beef industry and protect its citizens, all the major questions remain unanswered. The origin of the disease? **A mystery**. The number of people infected with vCJD? **A mystery**. The risk that those harbouring the disease will infect others? **Again, a mystery**... At the end of 1986, pathologists at the Central Veterinary Laboratory were analysing slivers of brain tissue sliced from cattle that appeared to have contracted a new disease. It left the cattle uncoordinated and jerky, and ultimately proved fatal. Under a microscope, the brain damage resembled scrapie, a disease caused by rogue proteins known as "prions" that had been endemic in the national sheep flock for nearly 200 years. **Without publicity, an investigation was launched to find the cause** of the outbreak. It revealed an alarmingly widespread disease. One year later, 95 cases of BSE had been confirmed on 80 farms. By February 1988, 264 cases had been tracked back to 223 farms. The number of cases began to grow exponentially**. It was turning into a major crisis** and there was all sorts of wild guesswork going on because no one understood it, says Chris Higgins, who now chairs the government's advisory committee on spongiform encephalopathy diseases. **The politicians didn't know what to do and the scientists didn't know what to do.** We didn't know where it came from, what caused it, how bad it might be. **We didn't know anything….** „The **danger now is not from cattle**, it's from other human beings," says another expert in vCJD ….“.

„The story of BSE in Britain is a case study in the ruthless efficiency of intensive farming, the self-serving behaviour of government departments and the patronising caution extended to the public when explaining risk. It reveals **the impotence of the scientists involved - at least at the outset**, when they were being called upon to give meaningful advice while still battling to understand a disease they had never encountered before…“- say britain scientists, who **had been summoned to an emergency meeting in London** (http://www.guardian.co.uk/g2/story/0,,1986657,00.html).

According to my opinion BSE (CWD…) can be a "not infectious disease"- see also recent article in “Neuron”. Authors of this article wrote; “Early functional impairments precede neuronal loss in prion disease…they occur before extensive PrP^{Sc} deposits accumulate… supporting the concept that they are caused by a transient neurotoxic species, distinct from aggregatedPrP^{Sc}“ (http://www.neuron.org/content/article/abstract?uid=PIIS0896627307000086). This can be in connection that the story of BSE in Britain is a consequence of „intensive farming“ (metabolic disease and „neurotoxicity“) and belongs in the „Organic Research“ (http://organicresearcher.wordpress.com/2007/01/06/bse-an-alternative-theory/). This theory also explains the link between BSE and Alzheimer´s disease

(http://www.medicalnewstoday.com/youropinions.php?opinionid=11677) and is based on the chronic Mg-deficiency- potentiated by hyperammonemia (high protein intake…). These mechanisms have a strong influence on CNS, especially in ruminants and carnivora animals. My alternative "BSE ecological view" can be well documented on the following "Chronic Wasting Disease" (CWD) example.

1. The „great mistake“ about the infectosity of the CWD?

The known natural hosts of „chronic waste disease“ (CWD) are mule deer, white-tailed deer, elk, and moose. CWD was first identified as a fatal wasting syndrome **in captive mule deer in Colorado in the late 1960s and in the wild in 1981**. It was recognized as a **spongiform encephalopathy in 1978**. To date (January 4, 2007), no strong evidence of CWD transmission to humans has been reported. By the mid-1990s, CWD had been diagnosed among free-ranging deer and elk in a **contiguous area in northeastern Colorado and southeastern Wyoming,** where the disease is now endemic. The geographic range of diseased animals currently includes 11 U.S. states and two Canadian provinces and is **likely to continue to grow**. Surveillance studies of hunter-harvested animals indicate the overall prevalence of the disease in northeastern Colorado and southeastern Wyoming from 1996 to 1999 was estimated to be **approximately 5% in mule deer**, 2% in white-tailed deer, and <1% in elk **(http://www.cdc.gov/ncidod/dvrd/cwd/).**

(September, 2002);
Mark Purdey says; „Despite the long term tradition of CWD haunting the Front Range foothills, a surge of near hysteria has bestruck the official US wildlife departments whose job it is to preside over CWD. Following in the footsteps of the official furore over mad cow disease in Europe, **the US government has sadly adopted the same unproven hypothetical mindset on the origins of these diseases**; that TSEs stem from exposure to **hyper infectious 'prions'** that are readily transmitted via body to body contact (saliva, etc), or via 'prion' contaminated feed. In this respect, blame has been conveniently offloaded onto the deer themselves – for sharing the same feed troughs, etc, – or onto the hunters for transporting the 'infectious' agent around with them from shooting region to region…“.
(http://www.markpurdey.com/articles_thewastingland.htm)

September, 2006;
***Ross Reinhold*, editor of Wisconsin CWD Watch says**; After over four years of experimentation with CWD Eradication in and around the Mount Horeb, Arena and Blue Mounds killing zone triangle, it is clear to those of us in the center of the "zone" that eradication is an impossible mission. Deer remain numerous despite massive amounts of money and firepower being employed to eliminate them. And evidence is mounting that CWD is much more broadly distributed than alleged by the Department of Natural Resources (DNR).

To date the clear results from the DNR CWD program are: miilions of dollars diverted from other wildlife & environmental programs, widespread fear of eating venison, and significant damage to the venerable sport of deer hunting by corrupting it into a panic slaughter of white-tailed deer in Southwest Wisconsin.

The DNR response to this disease has created what no one thought possible - putting animal rights activists and advid deer hunters on the same side! In both camps there is repulsion at slaughtering wild deer for the dumpster - like rats - and starving them to death through unreasonable restrictions on feeding. This has led some pundits to suggest there is an outbreak of another form of the cognitive disease whose locus is the GEF II State Office

Building in Madison, Wisconsin. A newly discovered disease called **CPD* (Chronic Panic Disorder)** appears to be spreading at the agency headquarters!

The DNR accounted for $26.8 million of the $32.3 million spent on CWD through FY 2005-06. To date, DNR's efforts to eradicate CWD have not been effective. Hunters must wait longer to receive CWD testing results for their deer. Wisconsin's approach to CWD should be reevaluated.

It is becoming an axiom: the more we test for CWD, the more we find. And with further testing, the original zone continues to expand (it has more than quadrupled in geographic area) and new zones are defined….

In order to mitigate this dramatic expansion of the area of known CWD, the DNR has developed the "sparks theory" of CWD spread. This allows single new cases of CWD found several miles outside of the current disease zone boundaries to be characterized as isolated "sparks" from the original zone . . . and not an indication that the geographic extent of the disease is larger than the current designation. Only through such Orwellian concepts as their "sparks" program, is the DNR is able to claim they are containing CWD (http://www.caids-wi.org/).

March, 2006; State stops culling herds of deer, elk

The Colorado Division of Wildlife is giving up on the controversial practice of killing deer and elk to help contain the spread of chronic wasting disease. The agency's lead scientist on CWD and an early proponent of culling, Mike Miller, said data collected over the years don't show that thinning herds has had any significant effect on the rate of the disease in the wild. Since 2001, Division of Wildlife officials culled roughly 2,300 animals on both sides of the Continental Divide in hopes of containing initial outbreaks or reducing populations in "hot spots" where prevalence of the disease was highest. That's a small fraction of the roughly 100,000 deer and elk killed by hunters annually in recent years. Typical rate of infection of deer in the state's "hot spots" for CWD (10%), typical rate of infection for elk in infected areas (1%).(http://www.cwd.cc/state_stops_culling.htm).

2. Why CWD can be a naturally occurring disease?

The CWD was first **discovered in 1967 in a state-owned research facility at Fort Collins, Colorado**. After 35 years of research, no one can say for sure how it started and how it spreads. Even Beth Williams (who discovered CWD) states, **"It's equally as plausible that CWD is a naturally occurring disease."**

Also Mark Purdey says (September, 2002); „CWD disease lies in the particular ENVIRONMENT where these animals were pastured. The answer must lie with some specific idiosyncratic **factors commonly shared by all of the ecosystems** where these spongiform hotspots erupt. An analytical field study of these regions provided a golden opportunity to pinpoint the aetiological needle in the causal haystack…".

a/ Elk digestive anatomy (small stomach disagned for more rapidly digestible feed);

To properly understand the nutrient requirements of elk, and how various crops and storage methods may work with elk, a brief examination of their digestive anatomy is useful. Elk have been described as intermediate or mixed feeders that will **naturally select a mixture of food from grasses to browse material such as leaves.** This is different from cattle or bison, which are classified as roughage/grass eaters and will select almost exclusively grass.

The implications this has when considering feeds for **elk is that they have a smaller stomach size relative to body weight and their digestive systems are designed for more rapidly digestible feed.** Conversely, cattle and bison hold feed in their rumens for a longer time to allow for digestion of more fibrous plant material. These **anatomical differences** should be considered when making feed decisions for elk based on information from cattle research. **Cattle are easier to feed since they have been bred and adapted over hundreds of generations** to farm production. However, deer have a digestive system that remains more **closely linked to their environment**. Feeding them on a farm requires a careful balance of rations (http://www.deer-library.com/artman/publish/article_133.shtml).

b/ Elk digestive anatomy; and a high nitrogen intake and fertilization recommendations

White-tailed deer in most southern habitats have access to adequate amounts of forage most of the year. However, **white-tailed deer need a minimum of 17% crude protein in their forage** year-round for maximum body and antler-growth and other natural deer forages do not contain this minimal amount of protein. **Thus food quality**, not quantity, may be the limiting factor in **producing high quality white-tails on a sustainable basis.**

There probably is no better off-season forage that one can plant for deer than soybeans. **Soybean foliage is high in protein, the soybean seed is extremely high in protein** and other nutrients. Soybean plants are utilized heavily from sprouting through seed production. **Cowpeas are annual legumes like soybeans and produce high protein forage during the off-season**. It has been our experience that cowpeas often are not browsing by deer until they reach a certain stage of maturation. However, when this stage is reached, deer may direct their attention to these plantings and eliminate them in a matter of a week or so. This may defeat the intended purpose of supplying deer high-quality forage throughout the summer season.

Generally speaking, areas selected for natural forage enhancement on most Alabama soils would benefit from an application of the equivalent of 1-3 tons of lime per acre before fertilization. These areas, whether fallow fields, roadsides, honeysuckle patches, etc. should then receive a broadcast treatment of 13-13-13 at a rate of 4-500 pounds per acre (an acre is about the size of a football field). **Fertilization should coincide with spring green-up** or about mid-March. Around the first to the middle of May **apply ammonium nitrate at a rate of 100 pounds per acre.** Natural forage **production can be doubled and crude protein content of many plants can be pushed** well beyond the basic requirements of white-tailed deer(http://www.pfmt.org/wildlife/deer_nutrition.htm). **Reindeer are selective feeders and, if conditions permit, eat only the top (younger) portion of plants (higher protein content).**

The nutritive value of forages is based on the nutritional level (energy, protein, etc.) and its availability to the digestive system of the animal. As percent **digestible dry matter increases**, animal performance in terms of weight gain, milk production, weaning weight, and conception rate increases. Legumes are more digestible and higher in protein, calcium and phosphorus than grasses. In general, cool-season grasses are more digestible **than warm-season grasses** and annuals are more digestible than perennials. **Deer and elk prefer legumes over grasses** because they are higher quality (higher protein content…) and resemble forbs because of their broad leaves (http://www.deer-library.com/artman/publish/article_57.shtml).

3. Nutrition requirements for deer and elk (recommendations for the captive deer herds)

Fallow and whitetail deer are very selective eaters, **choosing to consume only the most succulent and digestible plants** and plant parts. Red deer and elk will eat and digest feeds higher in fiber content, but will leave plant parts that bison, sheep and cattle will readily consume. All deer are highly seasonal in their eating habits. Voluntary intake decreases by as much as 40 - 60% during the winter, as compared to spring and summer peaks.

Crudeprotein(CP)

Protein is needed for maintenance, muscle and bone growth, and tissue repair. Elk that are rapidly growing or lactating and bulls recovering from the rut **have higher protein needs**. To some extent, **greater protein requirements** can be met by increased intake. However, the percentage of protein in diets designed for these animals is usually increased as well. Maintenance rations should be at 10 - 12% crude protein, whereas rations for lactation or antler growth should provide **14 - 18% crude protein.** Growing rations should contain **16 - 20% crude protein**. If a single diet is fed to all gender and age groups, a **16 - 17% crude protein level is optimal**. Total dietary protein content must be determined to follow these general guidelines.

Calcium and phosphorus

The actual amount of calcium and phosphorus required by elk increases greatly during growth, lactation and antler growth. **A minimum of 0.7% calcium and 0.4% phosphorus is suggested for elk rations**. Of equal importance is ensuring a calcium to phosphorus ratio of at least 1.5 to 1. How wide a ratio is tolerated by elk has not been researched although other ruminant species can tolerate calcium to phosphorus ratios as high as 5:1. Calcium levels are relatively high in hay, especially legumes, but phosphorus content is quite low. Access to browse (twigs and leaves) improves natural mineral intake on pastures. Check trace element levels for copper, zinc and manganese. Elk, unlike sheep, require supplemental copper. Next check the levels of vitamins A, D, E, and selenium…(http://www.deer-library.com/artman/publish/article_117.shtml).

About **high protein recommendations into the practice** – see one exaple as the „ protein feeders offer“ (http://www.sweeneyfeeders.com/home.php?cat=5); „Nutrition is the key to successful wildlife and game management. And nothing is more important than protein. Protein is absolutely essential to the health of your entire herd and to the potential for trophy bucks. Whitetail deer need protein all year round... **Automatic Protein Feeders** are totally dependable, providing **protein on time, every time, all year round…**are ideal **for free choice protein feeding**“.

4. Conclusions

A/ High dietary crude protein intake;

The high dietary crude protein (CP) for „adult deer“ (16- 18%), recommended by „deer nutrition scientists“ is significantly higher- compared with „high yielding dairy cows“. So for example dairy cow; **milk yield 55 kg/ day**, according to the NRC (1989 and 2001) – requires **only 17,5 and 16,7% of CP, respectively**. In addition, recent research (**during 1990s) resulted to the decrease of CP** content in dairy cow ration – compared NRC (1989) and NRC (2001)- (http://www.nap.edu/catalog/9825.html)- and **significant or „dramatic“ decrease in „early lactation**“(19 and 15.9%, respectively).

Dairy cow:600-680 kg body weight							
	Lactation				Early lactation		Dry pregnant
Milk yield (kg/day)	25	35	45	55	25	35	
Degradable protein – „DP" (%):							
NRC,1989	8,8	9,7	10,4	10,4		9,7	-
NRC,2001	9,5	9,7	9,8	9,8	10,5	10,3	9,9
Undegradable protein- „UDP" (%):							
NRC,1989	5,4	5,7	6,0	6,3		7,2	-
NRC,2001	4,6	5,5	6,2	6,9	5,4	5,6	3,2
Crude protein – „CP"- (%):							
NRC,1989	15,0	16,0	17,0	**17,5**		**19,0**	12,0
NRC,2001	14,1	15,2	16,0	**16,7**	15,9	**15,9**	13,1

Lush grass innately has an increased level of crude protein. This factor, **combined with increased use of nitrogenous fertilizers** in the soil, causes an **increase in ammonia in ruminal fluid**, leading to a decrease in the availability and **absorption of magnesium**. (MARTENS et SCHWEIGEL, 2000; URDAZ et al., 2003; FONTENOT et al., 1989).

So in high crude protein (CP) intake; deer rumen NH_3 levels are excessively high, the NH_3 is absorbed into the blood and either recycled or excreted in the urine as urea. Excess NH_3 in the portal system can readily pass through the liver and enter the arterial system. Brain tissue rapidly extracts NH_3 from the arterial blood. Ammonia then builds up in bloodstream (hyperammonemia) and more and more NH_3 accumulates in tissue cells. The **CNS is first to malfunction** because it has a large requirement for energy. Behavioral and nervous signs do seem to appear first, cellular energy and respiration deficits probably cause ultrastructural **damage and the degenerative changes in the CNS** (BOOTH and McDONALD, 1988).

B/ High dietary calcium intake (magnesium deficit in „intracellular fluid"**);**
The above recommended high dietary calcium level for the „adult deer" (minim. 0.7 %)– it is the same example- compared with the requirements for high yielding dairy cow (55 kg milk yield/ day) (see; NRC, 2001; http://www.nap.edu/catalog/9825.html).

A high content of calcium in the ration **increases the magnesium requirements** of the animal. The lower the magnesium level in the animal ration (and in the tissue cells); the **more marked is „calcium effect excitotoxicity" in the CNS.** It can be also accentuated; a low temperature raises magnesium requirements. Impairment of neuronal energy metabolism may sensitize neurons to excitotoxic cell death. However, **calcium in cells is tightly regulated** and mostly unrelated to necessary dietary calcium.

C/ Higher dietary potassium intake (magnesium deficit in „extracellular fluid"**);**

In addition**, potassium (K) concentrations were found with a high positive correlation with crude protein content** (REID and JUNG, 1988) in tropical grasses. The same high

positive correlation between K and crude protein were found in temperate forages with highest coefficients in ryegrass forage (HLÁSNÝ, 1990). So if the mule deer;

(a) naturally select a mixture of food from grasses to browse material such as leaves (higher CP content)

(b) have a smaller stomach size relative to body weight and their digestive systems are designed for more rapidly digestible feed, **choosing to consume only the most succulent and digestible plants** and plant parts (young plant- higher CP content).

© prefer legumes (higher protein and potassium content) over grasses because they are higher quality,

there is not only higher protein but also higher potassium intake.

While non-ruminants absorb Mg primarily from the small intestine, ruminants are able to absorb much of their Mg requirement from the rumen. In fact, the reticulum and **rumen can account for up to 80% of the Mg absorption** along the entire digestive tract (REMOND, et al, 1996). . Probably, the nutrient having the **greatest adverse effect on Mg absorption is an excess of K in the ration**, as shown by at least four sheep experiments (GRACE, et al, 1988; YANO, et al, 1990; DALLEY, et al, 1997; WACIRAPAKORN, et al, 1996). In addition, experiments demonstrated (Van MOSEL, et al, 1990) that younger cows are better able to mobilize Mg from the body reserves than older cows (see the **BSE occurrence only in older cattle**…).

5. Summary

Hyperammonemia plus hypomagnesaemia „simultaneous“ action (www.bse-expert.cz)

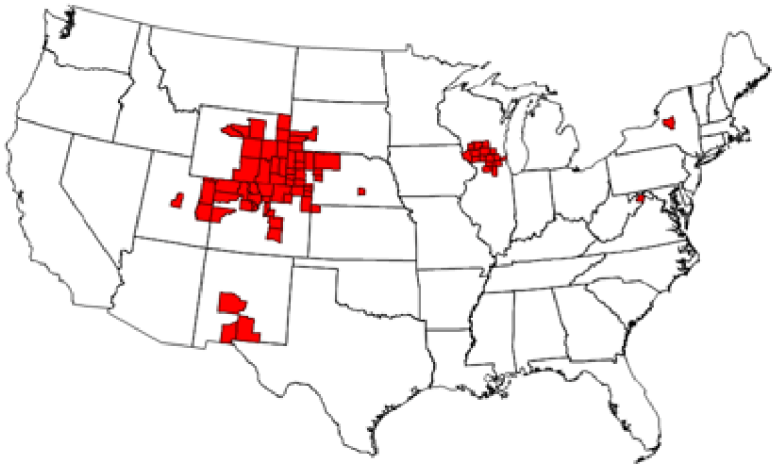
There is the possibility that these mechanisms have a strong influence on CNS, and that the **CWD** has its roots in a more common **nutritional problem- and not an „infectious disease“**. This **alternative „ ammonia- magnesium“ theory** is based on the chronic Mg-deficiency potentiated by hyperammonemia 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 „CWD“ disease.** If the CWD is involved; a longer- chronic action of corresponding biochemical changes in the blood (CSF) is necessary, to rise irreversible neurodegenerative changes. **So, prions are a symptom of the metabolic „chronic wasting disease“ and do not cause the disease…** The sad twist to this tale is that straightforward **magnesium supplementation of deer in CWD risk areas** may be all that is required to prevent CWD. However, also **dietary protein should be lowered**…

6. Two supporting views about CWD and the „ammonia-magnesium theory“

A/ Why the CWD has not been found in Florida or any other southeastern state?

At this time (5/5/2006), CWD has been found **in free-ranging cervids** in Colorado, Illinois, Wyoming, Nebraska, South Dakota, Wisconsin, **New Mexico**, Saskatchewan, and Alberta. It has been found **in captive herds** in Colorado, Wyoming, Nebraska, South Dakota, Oklahoma, Kansas, Montana, Wisconsin, Minnesota and South Korea. The core endemic area is contiguous portions of Wyoming, Colorado, and Nebraska. The prevalence of CWD in this area is approximately <1% - 15% in mule deer and <1% in elk, although this varies greatly by location. **CWD has not been found in Florida or any other southeastern state (http://www.myfwc.com/cwd/).**

Chronic Wasting Disease Among Free-Ranging Cervids by County, United States, January 2007



Click on the image above to enlarge

***However, why CWD has been found in free-ranging cervids- in southweastern New Mexico?* (the same has been found about the BSE high incidence in „southweastern Europe"- in Galicia).**

Explanation according to the „ammonia-magnesium theory"- see my website (www.bse-expert.cz);

Response; „Galicia is green with mad cows due to the more luxurious ryegrass pastures"; which has a climate and greenery like Ireland 's, and the uplands of the Catalan province of Girona , also relatively lush... (see this „Galicia Chapter" in my website).

However, **Galicia is one exception in the world**, because the BSE in „free- ranging" cattle; „mostly" is not observed in the „world south- located" countries. Because, there grasses are grown under a low NPK-fertilizers application (hot weather- water stress is obvious), so **grasses are low in crude protein and potassium, and higher in magnesium content** (warm season grasses)…

In Spain, more than one third of the BSE cases was found in Galicia, and Spain is the **second country in the world** about the **highest BSE incidence**;

2006; United Kingdom- 124 cases, Spain- 64 cases, Ireland- 41cases**… 2005**; United Kingdom- 224 cases, Spain- 98 cases, Ireland- 69 cases… **2004**; United Kingdom- 398 cases, Spain- 137 cases, Ireland- 126 cases…(**http://home.hetnet.nl/~mad.cow/**).

In contrast to „Dry Spain", Galicia is green due to the more luxurious pastures, which account for 11.1 percent of the total area**.** This province covers only 5.8 percent of Spain´s total surface and is located in Northwest Spain, with the 7.5 percent of the Spanish population. Annual mean temperatures vary from 8.3oC in the highest altitudes inland to 16oC in the southwestern coast. **Annual rainfall** varies between **700 to more than 2000 mm (see my website).**

However, rainfall and the **available water capacity of the soil are major yield determinants**, with output in the U.K., for example ranging from 6000- 14000 kg DM/ha **under intensive fertilization**. However, there are notable exceptions such as Benelux , wchich although characterized by the highest pasture yields in Europe, has a comparatively low share of grassland in total ruminant feed composition (50- 55%) – compared with Ireland (97%), U.K. (83%), France (71%). The permanent grazings of the Mediterranean zone are subject to severe moisture stress with annual production being limited to about 1000 kg DM/ ha. However, in this climatic zone, **irrigated legume and legume/ grass swards are capable of outputs of 20000 kg DM/ ha** (LEE, 1988).

Why hypomagnesemia (and BSE) is not observed in ruminants on warm season grasses?
(see Chapter „Publikace týkající se šílených krav, zveřejněné v časopisech ČR"; p. 24-43)

There is the explanation; these grasses are **low in crude protein and potassium**, and **higher in magnesium content;** grown under a low NPK-fertilizers application (hot weather-water stress is obvious). The main advantages of the grasses are their summer growth habit, providing when temperate (cool) grasses (perennial ryegrass, orchardgrass…) have become semi- dormant , and their ability to grow to use soil moisture efficiently. By the same token, they share the disadvantage of all tropical (warm) or C_4 grasses in that their nutritive quality for livestock is lower than that of temperate (C_3) species. This appears to be related to higher fiber and lower crude protein, and potassium concentrations in the warm season grasses (REID and JUNG, 1982).

It should be noted that **grazing cattle preferentially select leaf material and protein consumption** would therefore be expected to be markedly higher than indicated by whole plant analysis. It is well established that **tropical grasses** contain relatively high concentration of fiber and low levels of protein (PAYNE, 1966; BUTTERWORTH, 1967). The fundamental differences in leaf structure (Kranz anatomy) and metabolism of C_4 grasses result in marked differences in composition and nutritional quality of tropical and temperate forages (NORTON, 1982). Environmental conditions exert a strong effect on composition of C_4 grasses result in slower rates of degradation of fiber components in rumen (AKIN, 1986), and lower digestibility by ruminants (MINSON, 1981).

Under tropical or subtropical conditions, pastures based on C_4 grasses are generally considered to provide no more than a maintenance level of nutrition for grazing animals. The effects of temperature on cell wall development were examined by FORD et al. (1979); with increasing temperature , leaf neutral detergent fiber (NDF) concentrations in temperate species perennial ryegrass(Lolium perenne) increased, **while in tropical species (Panicum maximum) NDF levels decreased**. The decrease was due to a decline in cellulose concentration, while hemicellulose and lignin contents increased. There are clear differences in the concentration of minerals; lower levels of Ca and P, and higher concentrations of Mg, Cu in tropical than in temperate grasses (NORTON, 1982); and **potassium (K) concentrations quite low (mean 1.23% for 378 samples) with a high positive correlation with crude protein**(REID and JUNG, 1988) in tropical grasses. The same high positive correlation between K and crude protein were found in temperate forages with

highest coefficients in ryegrass forage- Lolium multiflorum (HLÁSNÝ, 1990). Tropical grasses appear to contain higher concentrations of Mg (0.36%) than temperate grasses (0.18%)- according to Norton (1982). However, REID and JUNG (1988) found a mean concentration (in 414 samples) **only of 0.16 percent Mg in warm season grasses of Northeast of the USA**. It is interesting to note that grass tetany at this area has not been observed in animals on warm season grasses. In balance trials with cattle and sheep fed a range of switchgrass (Panicum virgatum) and big bluestem hays in West Virginia , VONA et al (1984) found that both animal species remained in positive Mg balance at all stages of maturity of the hays.

B/ Capture myopathy as a „clinical syndrome“ of the subclinical hypomagnesemia?

The study found that mortality due to **capture myopathy was 6% among fawns, 12.5% among yearlings and 20.6% among adults.** Capture myopathy (or **white muscle disease**) is a response by the **deer to stressors in its environment**. The type of response to stress is affected by several factors - species, age, previous experiences, general health, genetics and learned/innate behaviour. The immediate reaction to stress is the "fight or flight" syndrome. The adrenals secrete adrenaline. Persistent stress raises reaction to a dangerous level. The mid-term effects of stress are: a) release of ACTH from the pituitary gland, b) the animal is on high alert, c) the animal becomes worn out, and d) the deer becomes susceptible to disease. Severe stress over days or weeks can cause **chronic corticosteroid production and adrenal exhaustion.** The secondary effects include metabolic upset, loss of body condition, loss of reproduction, and increased susceptibility to stress and death. Stress causes anaerobic metabolism, which results in chemically stored energy, lactic acid and cramping and muscle damage. Lactic acid damage contributes to capture myopathy. **Capture myopathy is a syndrome of acute or chronic degradation resulting from stressful activity** such as a pursuit of the susceptible animal. It can occur without exercise (animal does not have to be chased). Fear and anxiety plus **excessive body heat plus too much adrenaline** will result in capture myopathy **(http://www.deer-library.com/artman/publish/article_98.shtml).**

The similar „malignant hyperthemia“ is well known in pigs as **Porcine stress syndrome (PSS)** – see in Chapter „Spongiform encephalopaties- as a loss of parasympathetic function?- Controversy surrounds the efficasy of enteral Mg in growing pigs (p. 5-8). **Dietary magnesium asparate (MgAsp) supplementation to pigs** can be used to reduce the effects of preslaughter "stress", possibly through **reducing catecholamine secretion** at slaughter, and can reduce the incidence of pale, soft, exudative meat. The data demonstrated the efficasy of dietary MgAsp supplementation as a method for improving meat quality and reducing the incidence of PSS (D´SOUZA et al. 1998). Magnesium supplementation is thought to reduce pre-slaughter stress through a reduction in the release of stress hormones, including **cortisol and the catecholamines,** however a **definitive mechanism has not been established**. Further work in this area is needed to evaluate the potential benefits of increased dietary magnesium levels in pig diets on reducing the incidence of PSS, including an examination of the effects of diet composition, sex of the animal, pig genetics and stress susceptibility, pre-slaughter handling protocols, and the optimal level (Mg-dose) and **duration of magnesium supplementation** (HOUSE, 2001).