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Notes: This was the first paper we found that cited Dr. McDonnell's reference to the U.S.D.A. control program that found where supplemental copper was fed, CWD was stopped completely.

How do deer get CWD? There are some known methods of susceptibility and risk for deer, but first let's clear out the myths and recognize the actual science that is out there. Myth #1 CWD, is spread by nose to nose contact. Many experts believe this is the case, since CWD seems to occur in area where the deer are heavily populated, but the actual science behind the prions dispels this belief. Brain proteins are not found in bodily fluids. This means there is no vehicle to transport the prions to one animal to another. In fact, prions or any other infectious agents have never been identified in the blood, urine, saliva, manure or mucus of a CWD infected animal. This is true after almost forty years of extensive research in the US and three hundred years of incidence and research in Europe. If it could be identified, there would be a live test that could easily be incorporated on commercial farms in the field. There is no such test available. The closest live test is research today is to remove the tonsils (part of the nervous system) of a deer and that procedure is difficult and often not conclusive. The only conclusive test is to take a brain or tonsil sample after the deer is dead and check for the rouge prion. Myth #2, deer get CWD from eating antlers or bones of a contaminated animal. While consumption of massive amounts of infected prions may cause CWD, the likelihood that deer contract CWD from eating bones or antlers alone does not make common sense. Antlers and other bone material are consumed by deer for the mineral value when no other sources are available. Bone and antlers are gnawed in small amounts, stripping the outer calcium-phosphorous layer for supplementation. Prions, as part of the nervous system, may be present in marrow. The amount of marrow in a finished antler or a discarded bone is very small and not likely to be consumed by deer seeking mineral supplementation. Myth #3, deer contracted CWD from supplements that contained animal by-products. There is plenty of evidence to disprove this theory. First of all, ruminant by-products have been banned in the US since 1994 (FDA ruling 21 CFR part 589). Secondly, TSE related diseases have rarely been found in sheep in the US and never in cattle. The likelihood of contaminated ruminants by-products consumed by wild deer is next to none. Finally, ruminant by-products are not very digestible or palatable for the deer. They will simply not eat a product that contains ruminant by-products, much less in the amounts needed for massive contamination.

After dispelling the myths, let's take a look at the possible causes of CWD. In-vitro contamination… In-vitro contamination simply suggests that the does passes the rouge prion to her fawn before the fawn is born. This is the only scientifically confirmed mode of transmission of CWD and it is often dismissed prematurely. The doe may pass prions that have the potential for mutation. If the right environment or stress conditions occur, the prions mutate and become the disease. You can appreciate how an outbreak can occur by doing some simple

math on deer populations. Let's say a doe is carrying the potential rouge prion. According to the USDA veterinary service, CWD has a long incubation period so she may not develop symptoms of CWD for several years, if at all. It is very common for a doe to produce 11 new deer in a lifetime (estimated lifetime at 5 ½ years old). The reproductive rate is normally 50% does and 50% bucks. She passes this prion to her offspring. Her five does will produce another 55 deer in a lifetime. If 50% of those deer are does, they will produce another 300 deer in a lifetime, all with the potential for developing CWD symptoms. In areas where some outbreaks have occurred, there is some evidence of possible in-vitro contamination. In 1967, the disease was first recognized at Fort Collins at Colorado Division Wildlife facility. Deer from this facility were released into the wild. In Wisconsin in the 1970's, a game farm/research facility was operated near Mt. Horeb, near the 'hot zone'.

Copper deficiency and heavy metal contamination. The copper deficiency/heavy metal contamination theory of TSE related diseases is accepted in Europe and New Zealand as the environmental factor to the CWD equation. Studies have been shown that copper deficiency leads to developmental abnormalities in the cerebellum and the demyelination of the spinal cord of ruminants and aids in the binding and formation of rouge prions to predispose those animal to TSE related disease (Rehbinder & Petersson, 1994; McKenzie, 1998). In other words, copper deficiencies may cause prions to form abnormally. Nebraska elk researcher Dr. Michael McDonnel describes how it works: Imagine a prion like a screen-door spring, a long length with a curling structure like DNA. At each end of the prion are hooks to carry copper to various body tissues. The prion then goes to the liver to pick up more copper. When the copper is low or manganese is high, the manganese gets stuck on these hooks. Manganese has a different shape than copper, causing the screen-door spring to bend with both ends coming together. These fish hooked shaped prions stick to normal prions and knock off copper ions. Manganese replaces copper and the cycle starts all over. The fish hooks latch on to each other and form chains. These chains with the fish hooks sticking out tears holes in the brain that are the trademark of CWD. McDonnel also cites that in a **USDA control program** where supplemental copper was fed, **CWD was stopped completely.** When the same copper amount from a less digestible form was fed to the group, 5-7% of the herd still tested positive for CWD. When no copper was fed, 33-55% of the elk tested positive for CWD. An environmental factor such as copper deficiency would explain why no transmission of the disease has ever been found or why it suddenly appears in an area. It could explain why commercially ranched deer seldom have CWD, as they are fed copper supplements in their feed. Copper deficiencies, as a rule, are extremely common in cervids. Copper deficiencies can be caused if the natural vegetation is low in copper or copper is low in the soil or water. Copper deficiencies can also occur through natural tie-ups from other nutrients. Other heavy metals such as manganese, iron, molybdenum, lead or phosphorous available in the water or vegetation will interfere with copper digestion and cause it to become unavailable to the animal. In fact, the TSE outbreak that occurred in cattle in England is now thought to have been caused by mutations of healthy prions initially caused by the exposure to an organophosphate pesticide (Purdey, 2000). A

more common source of copper deficiency, however, occurs when deer are overpopulated. Overpopulation of deer in an area causes social and physical stresses that lower the immune system and cause the animal to ruminate or digest ineffectually. Poor rumination and digestion frequently results in poor trace mineral (such as copper) absorption. Combine overpopulation with any of the other possible environmental factors and the result: Outbreak.

Prion contaminated soil… Prions are extremely hardy proteins and will last in the soil for years. If a diseased animal dies in an area and is buried or left to decay, the prions will exist in the soil long after the carcass is gone. It would be unlikely, however, that the amount of contaminated prions in the soil would be enough for the massive amounts needed for contamination.

Spiroplasma… Spiroplasma is spiral bacterium that is often found side by side with the rouge prions after an animal develops symptoms. The supporters of this hypothesis feel that the spiroplasmosis may be the cause of TSE related diseases and not a secondary brain infection contracted as the animal is already weakened from CWD. The theory has some problems. First of all, even though spiroplasmosis was identified in the 1970's, it never has been identified in the blood, urine, saliva, saliva, mucus or manure of an infected animal. This seems to support the idea that spiroplasmosis can show us as a result of an infected animal. Moreover, the theory also states the spiroplasmosis was consumed by deer through animal by-products. Again, the myth continues. Ruminant by-products are nor palatable nor very digestible to deer and have been banned since the early nineties.

How can CWD be controlled? The approach to the control of CWD has not taken current science into consideration. According to Dr. Harry Jacobson and Dr. James Kroll, the end result of current state deer management or hunter opportunity? Models of deer management is overpopulation and disease. It is those same organizations that are now trying to impose the costly destruction of all the deer in hot zones in a futile attempt to eradicate the disease. In the hot zone In Wisconsin, the state department plans to kill more than this slow moving disease will kill in twenty years (based on the 2-5% of the deer population CWD currently affects in hot zone areas). Besides, anyone who has ever hunted deer knows the difficulty of this plan. Also, once an area is relieved of its deer population (as if this would ever happen), who's going to control the borders to stop other deer from entering the area? Will we have to hire more wildlife cops? Sounds like a plan to hire employ more government people and maybe get more federal money involved. Furthermore, these same agencies have put a ban on supplemental feeding because even though transmission of CWD by nose to nose contact has been disproven over and over again in the last 40 years, there is an assumption that gathering of deer may spread the disease. Unfortunately, banning supplemental feeding will only result in the possible spread of the disease. Deer, in fact all cervids, are herd animals. It is their natural behavior to gather or yard up in the winter near a food source. Whether the food source is a supplement grain and minerals, a corn field or browse makes no difference, the deer still gather. Taking away a major healthy food source to an overpopulated species will result in

deficiencies, other diseases are starvation. Unless, of course, that is the goal: to control population through starvation. If the hunters won't do it, nature will. As one unnamed source has said, "controlling population of the deer herd is the only means of managing CWD". I admit, I'm biased. As a ruminant nutritionist, the last thing I want to see is more diseases and/or starvation. Supplemental feeding, at least to supply copper, as a mean to head of further problems until we can get the deer population under control, makes sense to me. States where destroying the deer and banning supplemental feeding as a means of eradication has been a practiced method for years and it has not worked. These states still experience CWD in 2-5% of the wild deer and elk population.