# The Washington Department of Fish and Wildlife's Chronic Wasting Disease Program 1996-2003



Washington Department of FISH and WILDLIFE

Kristin G. Mansfield, P. Briggs Hall, And Gerald P. Nelson Washington Department of Fish and Wildlife, 600 Capitol Way N., Olympia, WA 98501

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Abstract: From 1996-2000, a total of approximately 80 brainstems from deer and elk throughout Washington were collected by the Department of Fish and Wildlife (WDFW) and tested for evidence of chronic wasting disease (CWD) by immunohistochemistry. From 2001-2003, 2,149 brainstems from deer and 336 brainstems from elk where collected and similarly tested. The majority of the samples were collected during established hunting seasons from hunter-harvested animals and from road kills. Approximately 2-3 samples per year were collected from animals demonstrating clinical signs compatible with CWD. Of the 2,287 usable samples collected, all tested negative for CWD. From 2001-2003, an adequate number of samples was collected from 10/36 (28%) of WDFW deer population management unit (PMUs) to allow us to conclude with 95% confidence that CWD would have been detected if it were present in the population at a prevalence of 5%; and from 3/36 (8%) of the deer PMUs to allow us to conclude that it would have been detected if present at a prevalence of 1%. An adequate number of samples was collected from 2/10 (20%) Washington elk herds to allow us to conclude with >95% confidence that CWD would have been detected if it were present at a prevalence of 5%.

## INTRODUCTION

#### The Disease

Chronic wasting disease (CWD) is a condition seen in mule deer, white-tailed deer, and elk that may be characterized by relatively non-specific clinical signs such as weight loss, abnormal behavior including indifference to human activity, difficulty walking, tremors, hyper-excitability, excessive salivation, teeth grinding, difficulty chewing or swallowing, and excessive drinking and urination. It is always fatal in affected animals (Williams and Young 1980, 1982).

CWD was first observed in captive-held deer and elk at research facilities in Colorado and Wyoming during the 1960's and 1970's. In 1978, researchers classified this disease as a "transmissible spongiform encephalopathy" (TSE) with remarkable similarities to the well-known TSE, scrapie (Williams and Young 1980), which has been known to occur in domestic sheep and goats for over 250 years.

TSEs are believed to be caused by prions, which are abnormally shaped proteins that accumulate in and disrupt the normal cellular structure of the brain. Several species or groups of species, including humans, mink, sheep and goats, deer and elk, and cattle have their own form(s) of TSE. There are at least half a dozen different TSEs that naturally occur in humans. In general, TSEs from one species are not transmissible to another species. However, an acquired human form of TSE, termed variant Cruetzfeldt-Jakob disease (vCJD) is linked to the consumption of meat from cattle afflicted with bovine spongiform encephalopathy (BSE or "mad cow disease"). Variant Creutzfeldt-Jakob Disease is distinct from sporadic CJD, which is the most common TSE of humans and which has no known links to TSEs of other species (Brown and Bradley 1998).

There is no vaccine, treatment, or practical live animal test available for CWD. The incubation period (interval between natural infection and the development of clinical signs) of CWD is generally 12-24 months, although it can range from 17-34 months. Once clinical signs appear, affected animals usually die within a year. Animals with early infections might not be identified by currently available tests (Williams et al. 2002).

CWD is believed to be most commonly spread from animal to animal through direct contact; likely via the saliva, urine, and feces of infected animals. CWD can also be transmitted by exposure to environments that have been contaminated by infected animals or their carcasses (Miller et al. 2004). The risk that carcass parts of infected animals could contaminate the environment has led some states and provinces to impose restrictions on the importation of certain hunter-killed deer and elk parts from outside areas. Regulations that ban the importation into Washington of certain deer and elk carcass parts from states and provinces where CWD is known to occur have been implemented effective September 1, 2004.

Several years after CWD was described in captive deer and elk facilities, the first cases in *wild* deer and elk were seen beginning in 1981 (Spraker et al. 1997). For many years CWD was believed to be limited to wild deer and elk in certain areas of Wyoming and Colorado. It was also diagnosed with some frequency in captive deer and elk in other states, and was most likely spread from Wyoming and Colorado to several states and two Canadian provinces via the transport of these infected captive animals (Williams et al. 2002).

CWD is a rare disease. Where it occurs in wild populations, the prevalence generally ranges from <1-5% in deer and <1% in elk, although prevalences as high as 31% have been reported in deer from certain focal areas (Miller et al. 2000, Joly 2003, T. Kreeger pers. comm.). Recent findings indicate that prime-aged bucks are more likely to be infected than other sex and age classes (Wolfe et al. 2004), and that CWD-positive deer might be over-represented as road kills and disproportionately harvested during muzzle-loader and archery hunting seasons (J. ver Steeg and A. Clark pers. comm.)

Beginning in 2001, many states and provinces began intensively testing wild deer and elk for CWD. This increased surveillance revealed that CWD was present in wild deer and elk in areas outside the previously known "endemic" areas of Wyoming and Colorado. In some instances, CWD-positive wild deer and elk were found concentrated around infected game farms. In other instances, there was no apparent link between CWD-positive wild animals and game farms (Williams et al. 2002).

As of June 2004, CWD has been diagnosed in wild deer and/or elk in the states of Wyoming, Colorado, Utah, New Mexico, Nebraska, South Dakota, Wisconsin, and Illinois and in the Canadian province of Saskatchewan. CWD has been diagnosed in captive deer and elk in the states of Montana, Wyoming, Colorado, South Dakota, Nebraska, Kansas, Oklahoma, Minnesota, Wisconsin, and the Canadian provinces of Alberta and Saskatchewan.

#### **Risks to Humans and Other Animals**

CWD is only known to occur in mule deer, white-tailed deer, and elk. All research to date indicates that CWD is not naturally transmissible to livestock or wildlife species other than deer and elk (Williams et al. 2002).

Although both CWD and BSE are believed to be caused by prions, unlike BSE, there have been no identified links between CWD and human vCJD (World Health Organization 2000). In this respect, CWD appears to be more similar to scrapie of sheep and goats, a disease also believed to be caused by prions, but which has never been associated with human disease despite being present in sheep and goat populations for over two centuries (Brown and Bradley 1998).

In 2003, an alleged cluster of human CJD cases was reported in the state of Washington in hunters who consumed venison (Murionova et al. 2003). Subsequent investigation revealed that all three had died of sporadic CJD (not vCJD), and that there was no evidence to conclude that the development of their disease was related to their consumption of venison (Belay et al. 2004). Several other alleged CJD clusters in humans who consumed venison have been investigated. Investigations revealed that the people had died of either a non-TSE neurological illness or of the more common sporadic CJD (Belay et al. 2001, 2004; Davis et al. 2003).

### Acknowledgments

Tom Owens developed and maintains the WDFW CWD database, conducts CWD data analysis, and generates CWD sampling graphics; dozens of WDFW field staff, tribal biologists, and volunteers collected brainstem samples; and Alan Clark from the Utah Division of Wildlife Resources, Terry Kreeger from the Wyoming Game and Fish Department, and Jeff ver Steeg from the Colorado Division of Wildlife shared unpublished CWD data from their respective agencies. Funding for this project was provided by the WDFW, Federal Aid in Wildlife Restoration, and the United States Department of Agriculture.

## **METHODS**

The Washington Department of Fish and Wildlife began testing deer and elk for CWD in 1996. From 1996-2000, efforts were focused on testing animals that showed clinical signs consistent with CWD ("target animals"), such as emaciation and other characteristics as described above. Healthy appearing animals were tested opportunistically and comprised the majority of animals testing during this period.

Beginning in 2001, WDFW began a more intensive CWD surveillance program, focusing on animals harvested during the fall hunting season. Several training sessions were held throughout the state to instruct WDFW biologists, tribal biologists, and volunteers in brainstem collection techniques. Samples were immediately placed in individual formalin containers and labeled with information on the sex, age, and species of cervid, location of harvest, and hunter information. Only animals >16 months of age were sampled. Information from sample labels was entered into a WDFW database, then transported to the Washington Animal Disease Diagnostic Laboratory (WADDL) at Washington State University, Pullman, WA for testing via immunohistochemistry (Spraker et al. 2002).

This approach relied on the efforts of over 100 WDFW employees and volunteers and the cooperation of hunters and meat processors. Game meat processors and check stations were the main sources of samples. Road-killed animals were sampled opportunistically. In addition to areas managed by the state, several areas under tribal jurisdiction were also sampled by tribal biologists and submitted to WADDL by WDFW.

During the first two years of intensive hunter harvest surveillance, samples were collected based upon the convenience with which they could be obtained. In an effort to improve the distribution of sample collection throughout the state, in 2003 we prioritized collection efforts in areas of the state in proportion to the contribution that area made to statewide harvest. Furthermore, since the prevalence of CWD in wild elk appears to be much lower than it is in wild deer (Miller et al. 2000), biologists were instructed to prioritize collection of samples from deer over collection of samples from elk in 2003.

Thirty-six WDFW deer "population management units" (PMUs) were used as the sampling units for deer. Each deer PMU is comprised of several "game management units" (GMUs), which are geographical areas of the state defined for the purpose of administrating hunting programs. In some instances, two PMUs were combined into one, based on local WDFW biologists' opinion that doing so was biologically justified. The 10 major elk herds in Washington were used as the sampling units for elk.

The confidence in our ability to detect the presence of a disease in any population is a function of the size of the population and the percentage of the population that is affected by the disease (i.e. the prevalence)(Figures 1 and 2). The methods and software (FreeCalc; <u>http://epiweb.massey.ac.nz</u>) of Cameron and Baldock (1998) were used to calculate sample sizes needed to substantiate freedom from disease, and the probability of detecting disease based on the number of samples actually collected. For software calculations, Type I and Type II errors were set at 0.05, and test sensitivity and specificity were set at 90% and 99% respectively (Miller and Williams 2002). Calculations were performed assuming minimum expected disease prevalence of both 1% and 5%. For deer, population sizes of between 1,600 and 30,000 were assumed for each PMU. For elk, published estimated population sizes were used (Washington Department of Fish and Wildlife 2003).

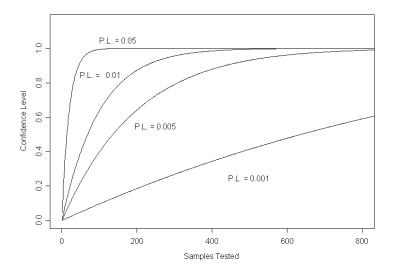


Figure 1. The confidence level in ability to detect presence of a disease in a population according to the number of samples tested and the minimum assumed prevalence level (P.L.) of the disease in a population of 4000 animals.

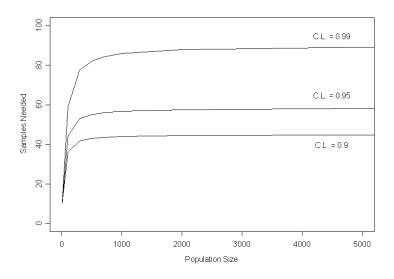


Figure 2. The sample size needed to detect disease at 5% prevalence level according to size of population and desired confidence level (C.L.).

Additional assumptions were that samples were randomly collected; mule deer, blacktailed deer, and white-tailed deer comprise a single uniformly susceptible cervid population within a PMU; and CWD, if present, was randomly distributed throughout the deer and elk populations.

## RESULTS

From 1996 to 2000, a total of approximately 80 deer and elk brainstems were tested for evidence of CWD. None tested positive. From 2001 to 2003, a total of 1,915 usable samples from deer and 292 usable samples from elk were tested throughout the state. None have tested positive for CWD (Table 1). Since emphasis in 2003 was placed on sampling areas of the state that tended to be the ones that were more difficult to obtain samples from, and sampling of elk was de-emphasized, the number of total samples collected dropped from a yearly average of 907 during 2001-2002 to approximately 670 in 2003 (Table 1). During all years of the study period, an average of 2-3 animals fitting the "target" profile (e.g. showing clinical signs compatible with CWD) was tested per year. None tested positive for CWD.

An adequate sample was collected from 10/36 (28%) deer PMUs to allow us to conclude with 95% confidence that we would have detected CWD if it were present in the deer population at a prevalence of 5% (Table 2, Figure 3); and from 3/36 (8%) deer PMUs to allow us to conclude with 95% confidence that we would have detected CWD if it were present in the deer population at a prevalence of 1% (Table 2, Figure 4).

An adequate sample was collected from 2/10 (20%) elk herds to allow us to conclude with 95% confidence that we would have detected CWD if it were present in the elk population at a prevalence of 5% (Table 3, Figure 5). No elk herds were adequately sampled to allow us to conclude with 95% confidence that we would have detected CWD if it were present in the elk population at a prevalence of 1% (Table 3, Figure 6).

Species	Result		Year		
-		2001	2002	2003	Total Samples
					_
Black-tailed deer	Negative	374	293	144	811
	Indeterminate <sup>a</sup>	(72)	(30)	(4)	(106)
	Positive	0	0	0	0
White-tailed deer	Negative	67	189	214	470
	Indeterminate	(21)	(34)	(10)	(65)
	Positive	0	0	0	0
Mule deer	Negative	111	296	195	602
	Indeterminate	(17)	(32)	(9)	(58)
	Positive	0	0	0	0
Elk	Negative	103	119	70	292
	Indeterminate	(17)	(21)	(6)	(44)
	Positive	0	0	0	0
Unrecorded deer sp.	Negative	4	11	17	32
	Indeterminate	(0)	(3)	(2)	(5)
	Positive	0	0	0	0
Totals	Negative	659	908	641	2207
	Indeterminate	(127)	(120)	(31)	(278)
	Positive	0	0	0	0
	Collected	786	1028	672	2485

Table 1. Washington Department of Fish and Wildlife chronic wasting disease surveillance results by species, 2001-2003.

<sup>a</sup> Indeterminate results were usually obtained when the wrong part of the brainstem was collected.

Table 2. Chronic wasting disease (CWD) samples collected from deer by Washington Department of Fish and Wildlife population management unit (PMU) 2001-2003, and confidence level achieved assuming that CWD was present at a prevalence of 5% or 1% (Cameron and Baldock 1998). Assumes population size of 1,600 – 30,000 deer for each PMU.

Tribe or	Usable	Confidence le	evel achieved	Sampling goa	l met to achieve 95%
PMU <sup>a</sup>	samples	assuming that	t CWD would have		vel that CWD would
	collected		l if present at <sup>b</sup> :	have been det	ected if present at:
		5%	1%	5%	1%
		prevalence	prevalence	prevalence	prevalence
Colville	23	73	36		
Quinault	1	5	2		
Yakama	26	77	39		
P1122	49	94	61		
P13	319	>99	>99	Yes	Yes
P14	48	93	60		
P15	45	92	58		
P16	68	98	73	Yes	
P17	36	87	49		
P21	241	>99	99	Yes	Yes
P2324	82	99	79	Yes	
P25	24	74	37		
P26	24	74	37		
P31	6	29	11		
P32	7	32	13		
P3335	23	72	36		
P34	37	87	51		
P36	5	24	9		
P41	25	76	38		
P43	6	29	11		
P44	1	5	2		
P45	8	36	14		
P46	6	29	11		
P47	11	46	19		
P48	2	11	4		
P51	61	97	69	Yes	
P52	54	95	65	Yes	
P53	10	43	17		
P54	28	79	42		
P55	4	20	7		
P56	74	98	76	Yes	
P57	46	93	59		
P61	70	98	74	Yes	
P62	208	>99	98	Yes	Yes
P63	64	97	71	Yes	105
P64	35	86	49		
P65	18	64	29		
P66	6	29	11		
P67	45	92	58		
ppp	98	,2	20		

<sup>a</sup> Population management unit; sample labels lacking sufficient information to allow assignment to a PMU (including most samples from tribal lands), were placed in the PMU category "ppp". <sup>b</sup> Assuming test sensitivity of 90% and specificity of 99% (Miller and Williams 2002).

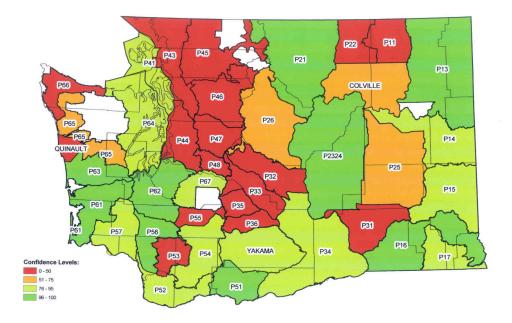


Figure 3. Confidence level that chronic wasting disease would have been detected if present at a prevalence of 5% based on Washington Department of Fish and Wildlife sampling by deer population management unit, 2001-2003.

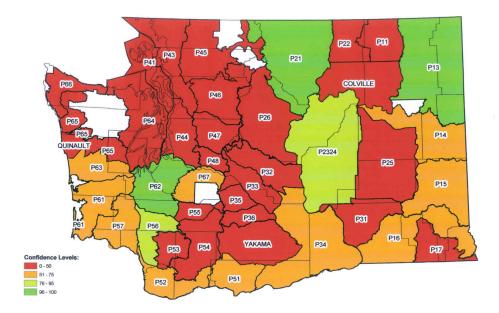


Figure 4. Confidence level that chronic wasting disease would have been detected if present at a prevalence of 1% based on Washington Department of Fish and Wildlife sampling by deer population management unit, 2001-2003.

Table 3. Chronic wasting disease (CWD) samples collected by elk herd 2001-2003, and confidence level achieved assuming that CWD was present at a prevalence of 5% or 1% (Cameron and Baldock 1998).

Elk Herd	Estimated	Usable	Confidence level achieved	achieved	Sampling goal met to achieve 95%	to achieve 95%
	Population	samples	assuming that CWD would have	VD would have	confidence level that CWD would	at CWD would
	Size	collected	been detected if present at <sup>a</sup> :	present at <sup>a</sup> :	have been detected if present at:	l if present at:
			5% prevalence	1% prevalence	5% prevalence	1% prevalence
Yakima	10,460	83	66<	62	Yes	
Olympic	8,620	92	>99	83	Yes	
Colockum	4,500	18	64	29		
North Rainier	1,450	1	5	2		
South Rainier	2,100	2	11	4		
North Cascades <sup>b</sup>	425	0	0	0		
Selkirk	1,450	1	5	2		
Willapa Hills	7,600	47	93	59		
Mount St. Helens	13,350	26	77	39		
Blue Mountains	4,400	5	24	6		
Herd not recorded		17				
TOTAL	54,355	292				

<sup>a</sup> Assuming test sensitivity of 90% and specificity of 99% (Miller and Williams 2002). <sup>b</sup> The North Cascades herd is not hunted.

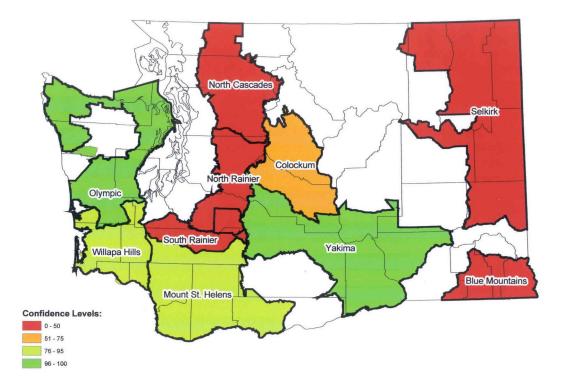


Figure 5. Confidence level that chronic wasting disease would have been detected if present at a prevalence of 5% based on Washington Department of Fish and Wildlife sampling by elk herd 2001-2003.

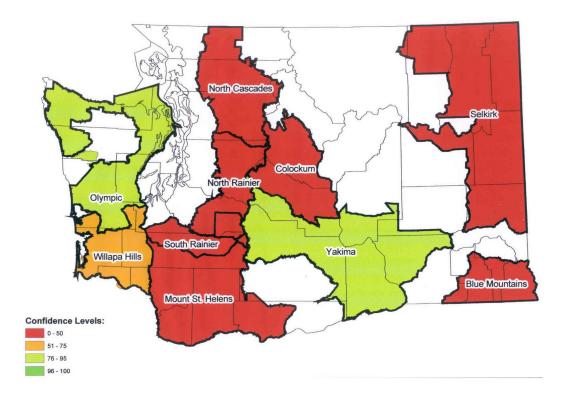


Figure 6. Confidence level that chronic wasting disease would have been detected if present at a prevalence of 1% based on Washington Department of Fish and Wildlife sampling by elk herd, 2001-2003.

## DISCUSSION

Without testing every deer and elk in the state, it is impossible to say with 100% certainty that Washington herds are free of CWD. However, statistical methods allow conclusions to be drawn with a given confidence level relative to the probability of disease being detected if it is present in a given area.

We assumed that each of the 36 deer PMUs and each of the 10 elk herds sampled in Washington represented a discrete population and therefore chose these as our sampling units. Based on this approach, 28% of the deer PMUs and 20% of the elk herds were adequately sampled to allow us to conclude with 95% confidence that we would have detected CWD if 5% of the animals were infected in these areas. However, where CWD occurs in nature, it is often at a prevalence closer to 1% (Miller et al. 2000, Joly 2003). We were only able to sample 8% of the deer PMUs, and none of the elk herds, at a level sufficient for us to conclude that we would have detected CWD if only 1% of the animals were infected.

To be able to conclude with 95% confidence that we could detect CWD if present in our deer and elk populations at a prevalence of 1% would require that a minimum of 156 animals be tested from each of the 36 deer PMUs and each of the 10 elk herds (Cameron and Baldock 1998). Thus far, financial and logistical constraints have prevented WDFW from sampling at this level.

It is important to acknowledge that some of the assumptions underlying the sampling and statistical methods we used were likely not met. This is a common problem of almost any study of free-ranging wildlife populations. For example, we assumed that samples were randomly collected, when in fact hunter selection of deer and elk is likely not random. Also, we assumed that CWD, if present, was randomly distributed throughout the population, when in fact the disease appears to occur in a clustered pattern (Joly et al. 2003).

Despite the shortcomings discussed above, and considering the logistical and financial resources required to overcome them, we feel that the sampling and statistical frameworks we used represent the best options available to us.

#### **Future CWD Activities in Washington**

Future CWD activities in Washington will depend on available funding. At this point, it appears that for the indefinite future, WDFW will be able to continue sampling at a level similar to that conducted over the past 3 years.

While it may not be possible to collect a large enough sample from every deer PMU and elk herd in Washington to allow us to conclude with a high level of confidence that CWD is absent from the state, this may be an achievable goal in select areas, and in fact has already been achieved in some areas (Table 2, Table 3). Sampling efforts in the near future will continue to be focused on areas of the state that remain underrepresented in

terms of the number of samples collected, while maintaining baseline surveillance in areas that have already been well-sampled.

Targeted surveillance, whereby deer and elk exhibiting clinical signs compatible with CWD are targeted for testing, can be a very efficient way to sample for the presence of CWD (Miller et al. 2000, Samuel et al. 2003). WDFW should consider increasing emphasis on targeted surveillance, particularly if adequate funding for intensive harvest-based sampling does not remain available. Implementing a targeted surveillance program will require a fairly extensive outreach and education campaign directed towards WDFW staff (field biologists, customer service agents, enforcement officers), hunters, and other members of the public.

CWD diagnostic technology is advancing rapidly and may dictate the samples that WDFW collects from deer and elk in the future. For example, most diagnostic laboratories that test for CWD are now switching away from routine IHC testing of the brainstem in favor of enzyme-linked immunosorbent assay (ELISA) screening of retropharyngeal lymph nodes, and using IHC of the brainstem or retropharyngeal lymph node only for confirmatory testing of positive ELISA results. The advantages of this approach include easier sample collection, greatly reduced testing costs, more rapid turnaround time for results, and the ability to use previously frozen lymph nodes. The disadvantage is that the ELISA test is slightly less specific (99.6%) (Hibler et al. 2003) than the IHC gold standard, meaning that the probability of false positive ELISA results is 0.4% or about 4 per 1,000 samples tested. In the case of positive ELISA results, formalin-fixed retropharyngeal lymph node (for deer) or brainstem (for elk) tissue would need to be available for confirmatory testing.

## SUMMARY AND CONCLUSIONS

Of the 42 states currently believed to be free of CWD, Washington ranks very near the top in terms of the total number of deer and elk that have been tested (International Association of Fish and Wildlife Agencies 2004), with no positive results to date. Furthermore, because game farming and the importation of deer and elk was banned in this state over a decade ago, and because CWD is not known to be present in any neighboring states or provinces, the risk of CWD entering Washington is believed to be very low. Current understanding of the disease and available information suggests that Washington is free of CWD. However, considering the rarity of this disease, much more testing will be required in order to conclude with a high level of confidence that this is true.

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