



CWD 102 for Managers

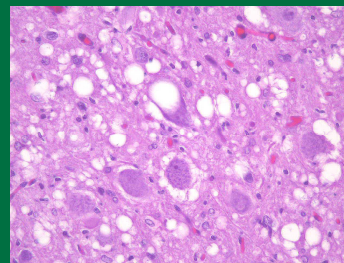
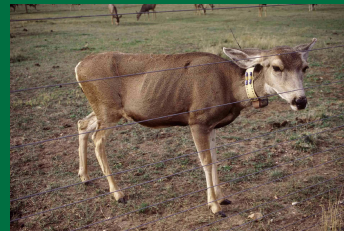
Bryan Richards
CWD Project Leader
USGS National Wildlife Health Center

U.S. Department of the Interior
U.S. Geological Survey

Chronic Wasting Disease - the beginnings

- 1967 – Colorado
 - Mule deer research facility
 - Loss of condition
 - Neurological symptoms
 - Uniformly fatal

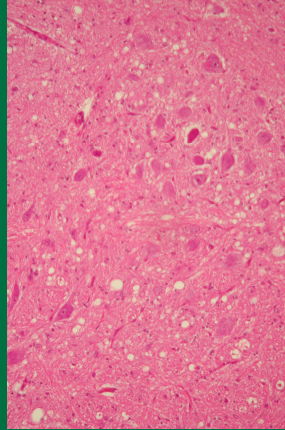
- 1978 – Univ. of Wyoming
 - Dr. Elizabeth Williams
 - Examined brain sections
 - Vacuolization
 - Similar to sheep scrapie
 - Transmissible Spongiform Encephalopathy (TSE)



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Transmissible Spongiform Encephalopathy (TSE) - definition

- **Transmissible**
 - Capable of being transmitted from one to another
- **Spongiform**
 - Resembling a sponge, porous
- **Encephalopathy**
 - Disorder or disease of the brain



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TSEs - examples

- **Humans**
 - Creutzfeldt-Jakob Disease (CJD)
 - Kuru
 - Fatal Familial Insomnia (FFI)
 - Gerstmann-Straussler-Scheinker (GSS)
 - Variant CJD (vCJD or nvCJD)
- **Domestic animals**
 - Scrapie – sheep
 - Transmissible Mink Encephalopathy (TME) – mink
 - Bovine Spongiform Encephalopathy (BSE) – cattle
 - Feline Spongiform Encephalopathy (FSE) – cats
 - BSE in other ungulates
- **Wildlife**
 - Chronic Wasting Disease – N.A. deer, elk and moose



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TSE Management Examples

- Kuru



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TSE Management Examples

- Bovine Spongiform Encephalopathy
- Kuru



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TSE Management Examples

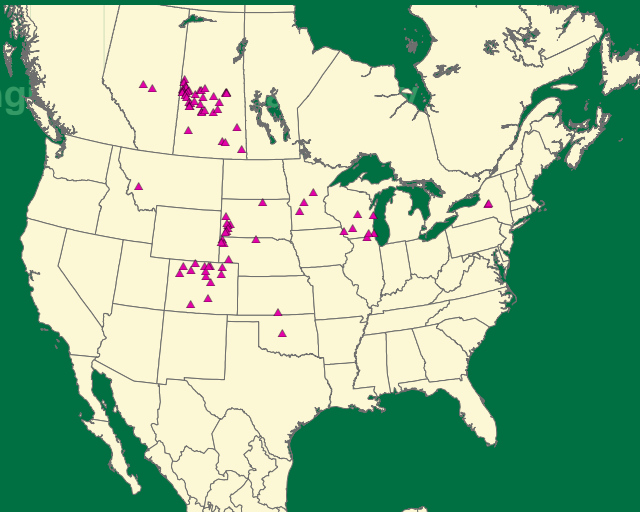
- **Scrapie**
- Bovine Spongiform Encephalopathy
- Kuru



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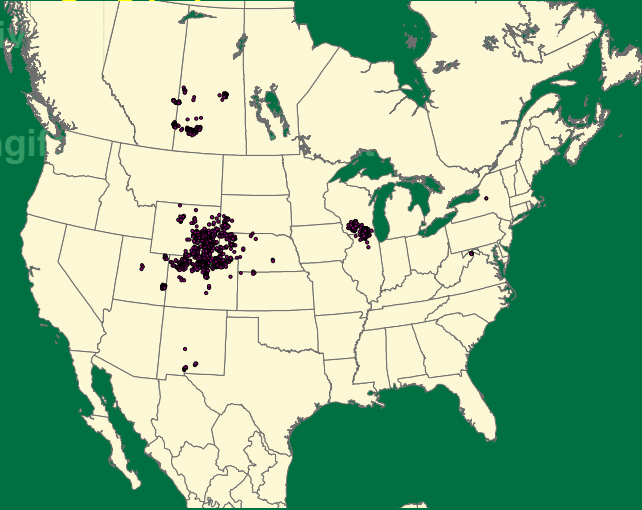
TSE Management Examples

- **CWD in captive facilities**
- Scrapie
- Bovine Spongiform Encephalopathy
- Kuru



TSE Management Examples

- CWD in free-ranging populations
- CWD in captivity
- Scrapie
- Bovine Spongiform Encephalitis
- Kuru



USGS

Chronic Wasting Disease

- what we know

- TSE (prion disease) of North American deer, elk, & moose
- Rare, geographically dispersed, local foci with higher prevalence
- Progressive neurological degeneration
- Uniformly fatal
- Contagious (only CWD & Scrapie)
 - Transmissible via contact (direct transmission)
 - Transmission via contaminated environment (indirect transmission)
- Long incubation period (months to years)
- Animals are infectious (shed prions) before onset of clinical signs
- Tests are post-mortem *
- No vaccination or treatment
- No strong evidence of widespread genetic resistance
 - Possible genetic resistance in elk (Hamir et al. 2006)
 - Possible genetic resistance in deer (Johnson et al. 2006)
 - Resistance does not confer immunity in elk (O'Rourke et al. 2007)
- **Effective management in free-ranging herds - extremely difficult**

Environmental Sources of Prion Transmission in Mule Deer

Michael W. Miller,* Elizabeth S. Williams,† N. Thompson Hobbs,‡ and Lisa L. Wolfe*

Whether transmission of the chronic wasting disease (CWD) prion among cervids requires direct interaction with infected animals has been unclear. We report that CWD can be transmitted to susceptible animals indirectly, from environments contaminated by excreta or decomposed carcasses. Under experimental conditions, mule deer (*Odocoileus hemionus*) became infected in two of three paddocks containing naturally infected deer, in two of three paddocks where infected deer carcasses had decomposed in situ ~1.8 years earlier, and in one of three paddocks where infected deer had last resided 2.2 years earlier. Indirect transmission and environmental persistence of infectious prions will complicate efforts to control CWD and perhaps other animal prion diseases.

(9.11.14). CWD epidemics do not appear to have been perpetuated by exposure to contaminated feed, but because ingestion of brain tissue can transmit CWD experimentally to deer (11.17), decomposed carcasses could serve as sources of infection in the environment.

Environmental sources of CWD infection represent potential obstacles to control in natural and captive settings. To investigate their role in transmission of this disease, we compared three potential sources of infection: infected live deer, decomposed infected deer carcasses, and an environment contaminated with residual excreta from infected deer.

Emerging Infectious Diseases • www.cdc.gov/eid • Vol. 10, No. 6, June 2004



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Infectious Prions in the Saliva and Blood of Deer with Chronic Wasting Disease

Candace K. Mathiason,¹ Jenny G. Powers,³ Sallie J. Dahmes,⁴ David A. Osborn,⁵ Karl V. Miller,⁵ Robert J. Warren,⁵ Gary L. Mason,¹ Sheila A. Hays,¹ Jeanette Hayes-Klug,² Davis M. Seelig,¹ Margaret A. Wild,³ Lisa L. Wolfe,⁶ Terry R. Spraker,^{1,2} Michael W. Miller,⁶ Christina J. Sigurdson,¹ Glenn C. Telling,⁷ Edward A. Hoover^{1*}

A critical concern in the transmission of prion diseases, including chronic wasting disease (CWD) of cervids, is the potential presence of prions in body fluids. To address this issue directly, we exposed cohorts of CWD-naïve deer to saliva, blood, or urine and feces from CWD-positive deer. We found infectious prions capable of transmitting CWD in saliva (by the oral route) and in blood (by transfusion). The results help to explain the facile transmission of CWD among cervids and prompt caution concerning contact with body fluids in prion infections.

www.sciencemag.org SCIENCE VOL 314 6 OCTOBER 2006



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Findings Relevant to Aim 1: Describe Mechanisms of Transmission

Investigations of Shedding of PrP^{Sc} and Vertical Transmission

Data from our experimental study support the likely transmission of CWD from infected to susceptible deer via dried fecal matter. Deer in each of four previously unexposed "recipient" groups that consumed air-dried feces from orally inoculated "donors" became infected within 209–297 days after initial exposure to infectious feces. Donor deer apparently shed infectious agent within the first 399 days after inoculation, and infectivity in feces persisted for at least 383 days.

Unfortunately, we subsequently learned that one control deer being maintained at a separate location ("Red Buttes") had somehow become infected with CWD. Although epidemiological investigation suggested that infection most likely occurred at the Red Buttes facility after deer were moved into a new pen, we cannot be certain that the original transmission study was not compromised. Consequently, this experiment will need to be repeated under much more stringent control conditions before fecal-oral CWD transmission can be reported with absolute certainty.

Colorado State University CWD Annual Report - 2005



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PLoS PATHOGENS

Prions Adhere to Soil Minerals and Remain Infectious

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An unidentified environmental reservoir of infectivity contributes to the natural transmission of prion diseases (transmissible spongiform encephalopathies [TSEs]) in sheep, deer, and elk. Prion infectivity may enter soil environments via shedding from diseased animals and decomposition of infected carcasses. Burial of TSE-infected cattle, sheep, and deer as a means of disposal has resulted in unintentional introduction of prions into subsurface environments. We examined the potential for soil to serve as a TSE reservoir by studying the interaction of the disease-associated prion protein (PrP^{Sc}) with common soil minerals. In this study, we demonstrated substantial PrP^{Sc} adsorption to two clay minerals, quartz, and four whole soil samples. We quantified the PrP^{Sc}-binding capacities of each mineral. Furthermore, we observed that PrP^{Sc} desorbed from montmorillonite clay was cleaved at an N-terminal site and the interaction between PrP^{Sc} and Mte was strong, making desorption of the protein difficult. Despite cleavage and avid binding, PrP^{Sc} bound to Mte remained infectious. Results from our study suggest that PrP^{Sc} released into soil environments may be preserved in a bioavailable form, perpetuating prion disease epizootics and exposing other species to the infectious agent.

Citation: Johnson CJ, Phillips KE, Schramm PT, McKenzie D, Aiken JM, et al. (2006) Prions adhere to soil minerals and remain infectious. *PLoS Pathog* 2(4): e32. DOI: 10.1371/journal.ppat.0020032



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Short
Communication

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Infectious agent of sheep scrapie may persist in the environment for at least 16 years

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In 1978, a rigorous programme was implemented to stop the spread of, and subsequently eradicate, sheep scrapie in Iceland. Affected flocks were culled, premises were disinfected and, after 2–3 years, restocked with lambs from scrapie-free areas. Between 1978 and 2004, scrapie recurred on 33 farms. Nine of these recurrences occurred 14–21 years after culling, apparently as the result of environmental contamination, but outside entry could not always be absolutely excluded. Of special interest was one farm with a small, completely self-contained flock where scrapie recurred 18 years after culling, 2 years after some lambs had been housed in an old sheep-house that had never been disinfected. Epidemiological investigation established with near certainty that the disease had not been introduced from the outside and it is concluded that the agent may have persisted in the old sheep-house for at least 16 years.



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Oral Transmissibility of Prion Disease Is Enhanced by Binding to Soil Particles

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Soil may serve as an environmental reservoir for prion infectivity and contribute to the horizontal transmission of prion diseases (transmissible spongiform encephalopathies [TSEs]) of sheep, deer, and elk. TSE infectivity can persist in soil for years, and we previously demonstrated that the disease-associated form of the prion protein binds to soil particles and prions adsorbed to the common soil mineral montmorillonite (Mte) retain infectivity following intracerebral inoculation. Here, we assess the oral infectivity of Mte- and soil-bound prions. We establish that prions bound to Mte are orally bioavailable, and that, unexpectedly, binding to Mte significantly enhances disease penetrance and reduces the incubation period relative to unbound agent. Cox proportional hazards modelling revealed that across the doses of TSE agent tested, Mte increased the effective infectious titer by a factor of 680 relative to unbound agent. Oral exposure to Mte-associated prions led to TSE development in experimental animals even at doses too low to produce clinical symptoms in the absence of the mineral. We tested the oral infectivity of prions bound to three whole soils differing in texture, mineralogy, and organic carbon content and found soil-bound prions to be orally infectious. Two of the three soils increased oral transmission of disease, and the infectivity of agent bound to the third organic carbon-rich soil was equivalent to that of unbound agent. Enhanced transmissibility of soil-bound prions may explain the environmental spread of some TSEs despite the presumably low levels shed into the environment. Association of prions with inorganic microparticles represents a novel means by which their oral transmission is enhanced relative to unbound agent.

Citation: Johnson CJ, Pedersen JA, Chappell RJ, McKenzie D, Aiken JM (2007) Oral transmissibility of prion disease is enhanced by binding to soil particles. PLoS Pathog 3(7): e93. doi:10.1371/journal.ppat.0030093



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COMMUNICATIONS

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DYNAMICS OF PRION DISEASE TRANSMISSION IN MULE DEER

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Abstract. Chronic wasting disease (CWD), a contagious prion disease of the deer family, has the potential to severely harm deer populations and disrupt ecosystems where deer occur in abundance. Consequently, understanding the dynamics of this emerging infectious disease, and particularly the dynamics of its transmission, has emerged as an important challenge for contemporary ecologists and wildlife managers. Although CWD is contagious among deer, the relative importance of pathways for its transmission remains unclear. We developed seven competing models, and then used data from two CWD outbreaks in captive mule deer and model selection to compare them. We found that models portraying indirect transmission through the environment had 3.8 times more support in the data than models representing transmission by direct contact between infected and susceptible deer. Model-averaged estimates of the basic reproductive number (R_0) were 1.3 or greater, indicating likely local persistence of CWD in natural populations under conditions resembling those we studied. Our findings demonstrate the apparent importance of indirect, environmental transmission in CWD and the challenges this presents for controlling the disease.

Key words: basic reproductive number (R_0); chronic wasting disease (CWD); epidemic model; mule deer; *Odocoileus hemionus*; prion disease; transmissible spongiform encephalopathy.



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Chronic Wasting Disease - is it a problem?

- **Common Allegations:**
 - It doesn't cross into cows
 - People don't get sick from it
 - It hasn't hurt deer populations
- **So why should we worry?**
- **Why should we try to manage CWD?**
- **It's just another deer disease**
- **We need to learn to "live with it"**



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What are the risks?

- Domestic livestock
 - Low?
- Human health
 - Low?
- Other wildlife
 - Largely unknown
 - Exposure occurs
- Cervids
 - Presumably high
 - Long-term issue
 - Not yet realized
- Other?

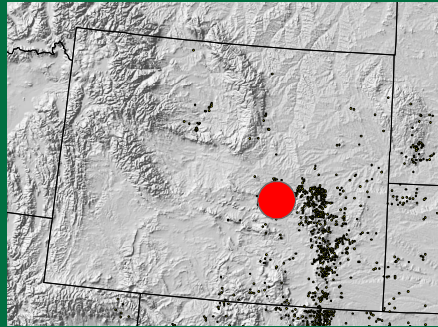


Chronic Wasting Disease - risk to cervids?

- In free-ranging populations:
 - Higher prevalence in males (2-4x females)
 - Higher prevalence in adults
 - Geographic spread is apparent
 - Prevalence is increasing locally
 - >30% in localized populations
 - No evidence of “equilibrium”
 - No strong evidence of management success
- In captive populations:
 - Prevalence can near 100%
- Population-level impacts?
- Caribou?



Examples of high CWD prevalence



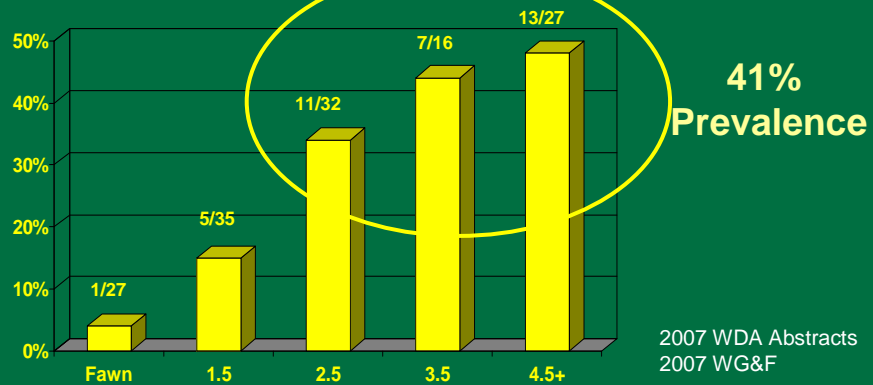
Wyoming



Wisconsin

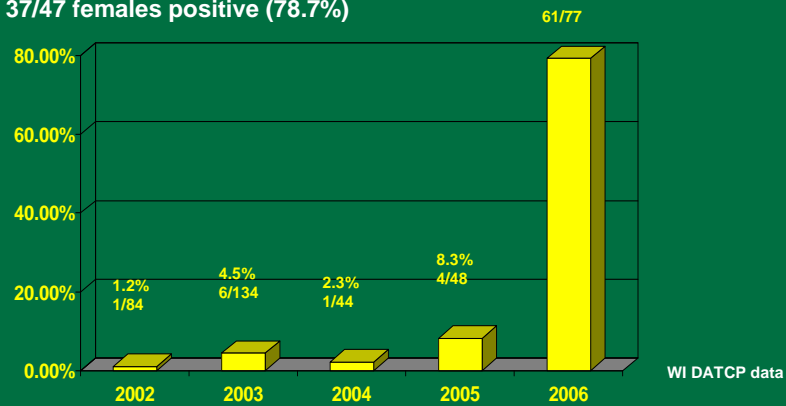
High prevalence in Wyoming white-tailed deer (WTD)

- Overall CWD prevalence in study area is 27%
- CWD prevalence is higher in female WTD (32%) than in male WTD (22%)



High prevalence in a Wisconsin game farm

- CWD first detected in 2002
- 79.2% prevalence at depopulation (2006)
 - 23/29 males positive (79.3%)
 - 37/47 females positive (78.7%)



CWD management

- If you don't have it:
 - Implement preventative measures to minimize risk of introduction
- If you do have it:
 - Establish management goals
 - Implement management actions
 - Monitor and adapt
- Conduct and support research



Preventative measures

- reduce risk of disease introduction/amplification

- Introduction risks
 - Game farm movements/imports
 - Game farm/wild contacts, escapes
 - Hunter carcass movements/imports
- Establishment/Amplification risks
 - Feeding and baiting
 - High deer densities
- Mitigating risks
 - Regulations
 - Education & Information
 - Conduct surveillance



CWD management goals

- No action
- Monitor distribution
- Monitor prevalence
- Slow the spread
- Contain spread
- Eliminate "sparks"
- Control prevalence
- Eradicate CWD



CWD management goals – “passive”

- No action
- **Monitor distribution**
- **Monitor prevalence**
- Slow the spread
- Contain spread
- Eliminate “sparks”
- Control prevalence
- Eradicate CWD



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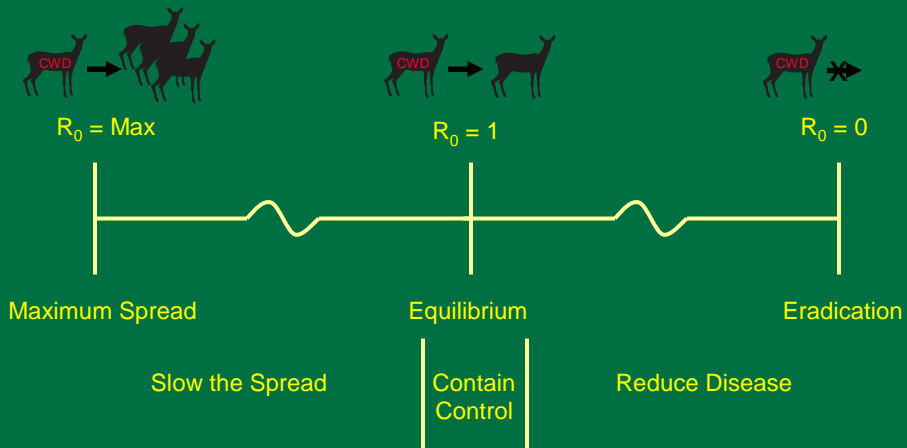
CWD management goals – “active”

- No action
- Monitor distribution
- Monitor prevalence
- **Slow the spread**
- **Contain spread**
- **Eliminate “sparks”**
- **Control prevalence**
- Eradicate CWD



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Disease control - theoretical



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Active CWD management (I)

- Live cervid restrictions
- Carcass movement restrictions
- Selective removal of CWD+ animals
 - Tonsil biopsies
 - RAMALT biopsies
 - Predators?



Active CWD management (II)

- Dramatic, sustained deer population reduction
 - Hunting
 - Landowner culling
 - Alternative (supplemental) techniques
 - Agency personnel (sharpshooting)
 - Other techniques?
- Goals:
 - Reduce # of CWD infected deer
 - Reduce environmental contamination
 - Reduce # of potential carriers
 - Reduce disease transmission
- **Prevalence cannot be “shot down” in the short-term**



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Hunters and disease control?

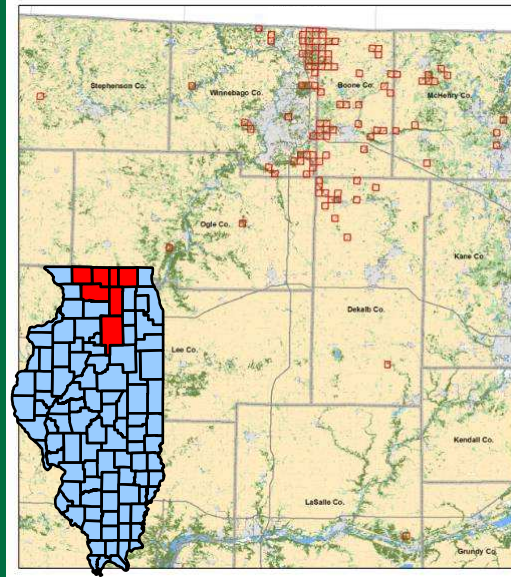
- Hunter numbers
- Access issues
- Time issues
- Per capita maximum harvest
 - Consumption limit
- Desire to “not harm the resource”
- **Hunters do not view themselves as disease control agents**
- **Human dimensions research suggests hunters will avoid high prevalence herds**



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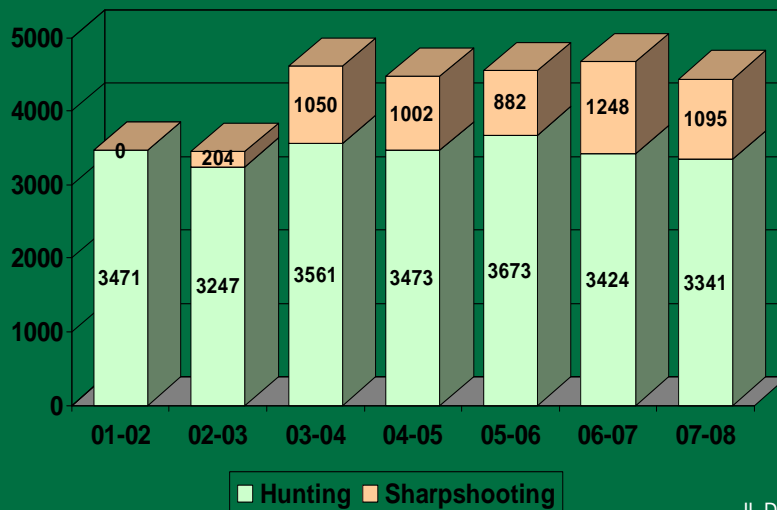
CWD Management in Illinois

- Extended hunting opportunities
- Intensive agency sharpshooting
 - Aerial surveillance
 - Land access



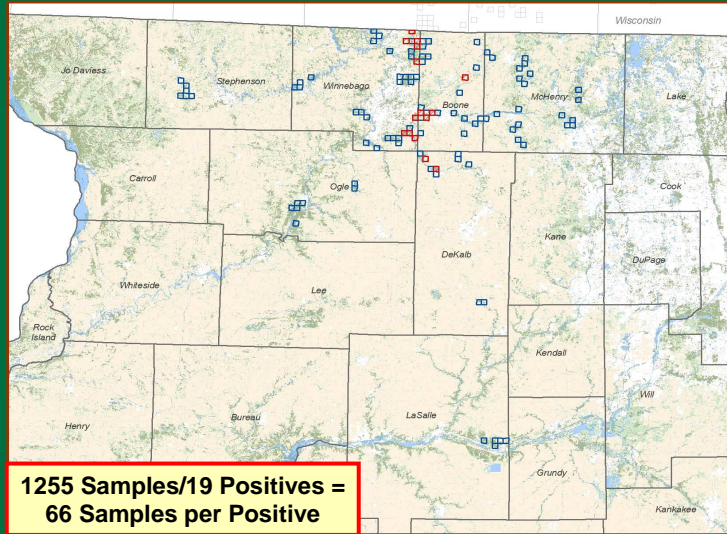
IL DNR data

Deer Removals in Illinois CWD Area - Boone, Winnebago, McHenry, DeKalb counties

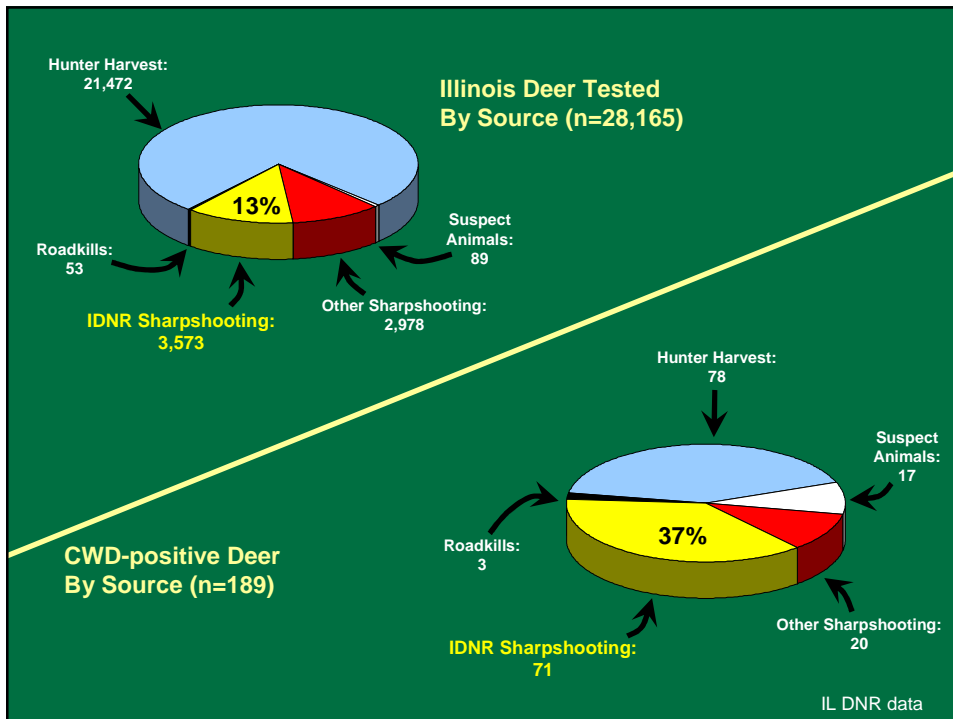


IL DNR data

2007-2008 Sharpshooting

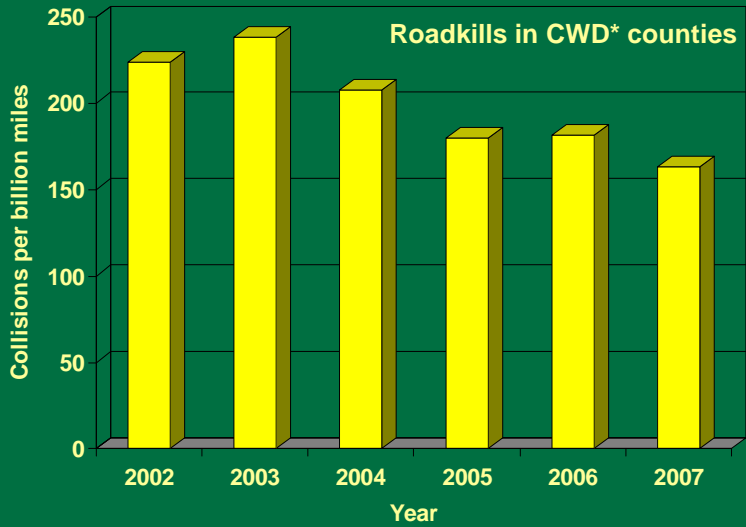


IL DNR data



IL DNR data

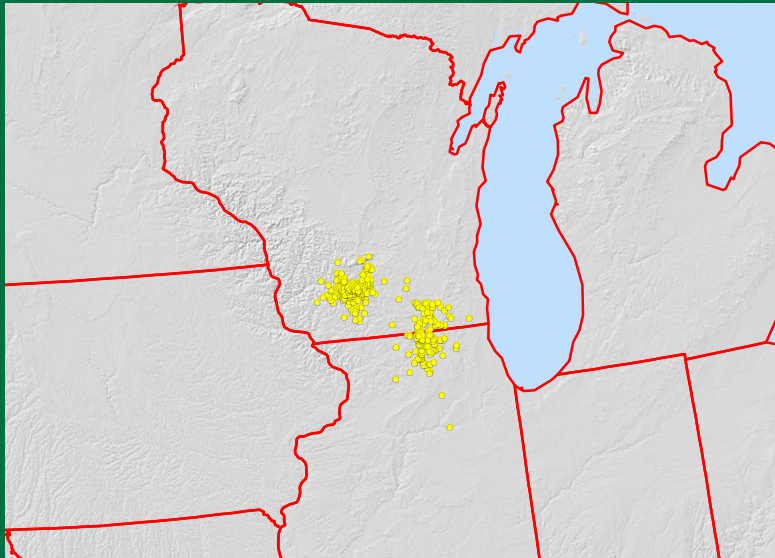
Sharpshooting in Illinois - population impact?

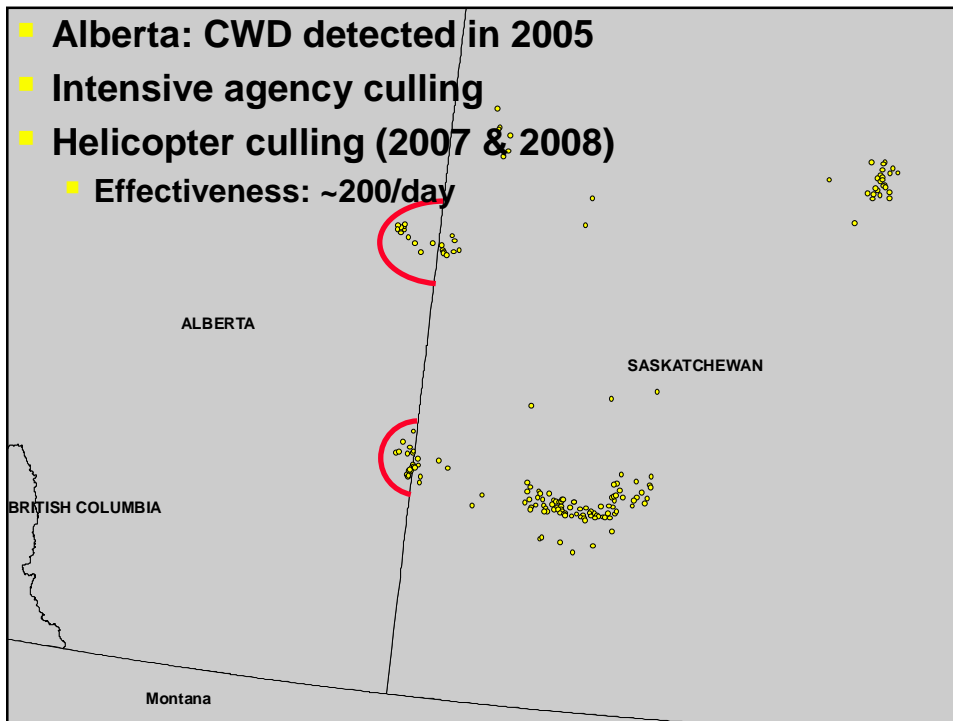


* Boone, Winnebago, McHenry, DeKalb counties

IL DNR data

Can Illinois succeed?





CWD management in Alberta

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Top News

Residents upset with Alberta deer cull

Published: Feb. 22, 2008 at 10:58 AM

EDMONTON, Alberta, Feb. 22 (UPI) -- Residents of southeast Alberta are upset with a provincially ordered deer cull to prevent the spread of Chronic Wasting Disease, the Edmonton Sun reported.

The affected area is a 6.5-mile radius around Chauvin, where provincial game officials have been ordered to shoot every deer on sight to prevent the spread of CWD from neighboring Saskatchewan, the report said.

Locals, backed by the Alberta Fish and Game Association, have complained the cull targets more deer than necessary, upsets the natural balance, threatens tourism, and spooks livestock, the Sun said.

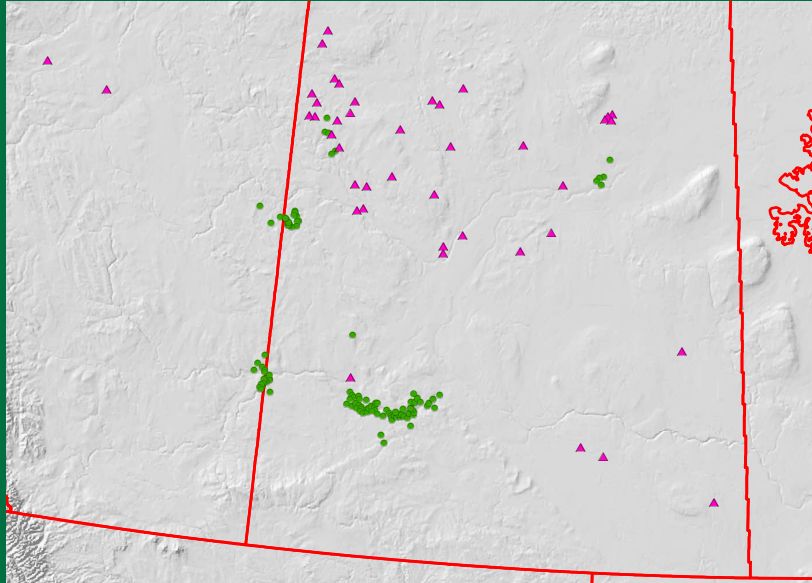
Dave Ealey, a spokesman for Sustainable Resource Development, said the province's efforts are based in science.

"This is not a hunt, let me make that clear. This is a cull," he said. "This is disease management."

Because of several unseasonably warm winters, Alberta's deer population has exploded, the Calgary Herald said. A similar cull last winter reduced the numbers by about 1,850, the report said.

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Can Alberta succeed?



Considerations for this workshop

- Realistic timeline for management
- True management experiments
- Monitoring issues
- Appropriate disease metrics
- Effective communications
- Regional aspects of disease management
- Explore alternative means for population reduction
- Explore alternative means for disease control