Happy spring! Here in Colorado winter is making way for spring with wild weather fluctuations. One day it’s blue skies and warm, the next is a foot of heavy snow. It’s also a time of transition for our working group. Our WDWG Chair for the past two years, Rich Chipman, has moved from serving as our leader to Past Chair and supporting me as the new Chair as well as our other new officers: Chair-Elect Julie Blanchong and Secretary-Treasurer Anne Ballmann. We also welcome Steve Sweeney, Barb Bodenstein, and Jordona Kirby as Board Members. We sincerely thank Rich, as well as Tim Algeo, our past Secretary-Treasurer, and past Board Members for their dedication and leadership the last couple years.

I am excited and humbled to be serving as the working group Chair. There is lots of work to be done and, like the Colorado weather, our working group activity tends to fluctuate with the seasons. We are always busy in fall preparing for the annual conference and early winter brings developing proposals for the next year’s conference. There has been a bit of a lull the last couple months as we’ve transitioned, but we’re looking forward to a busy year. To keep the work going throughout the year, Board members are stepping up to serve as leads for our standing committees. For example, Julie Blanchong is coordinating responses to wildlife health legislation such as the recent Bsal rule to contribute to TWS’s response (see following article). Our web editors are working to transition to the new TWS web format. And soon we’ll be soliciting more involvement from our membership to contribute to these important tasks. So be thinking about how you might like to contribute and check back in our next issue of The Vector for more information.

The editors of The Vector welcome your contributions. If you wish to submit an article, but suspect you will not quite make the deadline, please contact Samuel M. Goldstein.

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Your membership in the WDWG can only be obtained by joining and renewing your annual TWS Membership each year. At the time that you join simply indicate that you want to be a member of this working group on the TWS application. Membership dues are $5.

With your membership you will receive our quarterly newsletter to keep up-to-date with our group business and the most current disease issues. Your membership also provides an opportunity to work closely with other wildlife disease professionals.

The Vector
The Newsletter of The Wildlife Society Wildlife Diseases Working Group

**From the Chair:**

Each quarter, the WDWG distributes The Vector showcasing the wonderful work of our students, ongoing research, and current topics related to wildlife disease. We need your help!! With writings contributed from our large and diverse membership, we can supplement the content of The Vector and augment the number of informative articles disseminated through the newsletter. This is an opportunity for you to share information on a topic you find important and valuable to our members.

Please consider providing a short article about your profession or path to becoming a wildlife disease expert, major projects, research findings, or a hot topic in the wildlife disease field. Senior-level professionals may feel free to share lessons learned in their career to benefit students and early career professionals. Please encourage your students or technicians to do the same. Articles are not only a great way to share your current work, but they can also open doors for future collaboration. Articles need not be long or formal, and will go through an editing process by the editors and/or Student Affairs Committee upon submission. We encourage you to submit a few photos to accompany your writing.

Please jump at this opportunity to get involved, give back to your profession, share a little bit about what you do, and help shape your working group for the future. Inquiries and articles can be submitted at any time to Sam Goldstein (Samuel.M.Goldstein@aphis.usda.gov) or Michelle Clayson (rosenmi1@gmail.com).
Chronic wasting disease (CWD), a transmissible spongiform encephalopathy of cervids, reached record prevalence in southeastern Wyoming in 2011 with 57% of harvested mule deer (Odocoileus hemionus) infected with the fatal disease. The southern Converse County mule deer herd, located in the northern reaches of the Laramie Mountains, provided a special opportunity to study the impacts of high CWD prevalence (>30%) on the long-term viability of this population. The significance of CWD and its impact on free-ranging cervid populations has only recently been documented, specifically in free-ranging white-tailed deer (Odocoileus virginianus) and elk (Cervus elaphus) (Edmunds, 2013; Monello et al., 2014). CWD surveillance conducted by the Wyoming Game and Fish Department (WGFD) showed a presumptive correlation between CWD prevalence and a decade-long decline in mule deer numbers, prompting an epizootiological study of CWD in free-ranging mule deer. I began this research in 2010 as a Ph.D. student in the Department of Veterinary Sciences at the University of Wyoming and in collaboration with WGFD and U.S. Geological Survey National Wildlife Health Center, Madison, WI.

The main objective of my study was to investigate how CWD affects vital rates, mule deer behavior, and consequently population sustainability. We performed antemortem CWD tests using tonsil biopsies collected during capture of 143 radio-collared adult mule deer in southern Converse County, Wyoming (Herd Unit (HU) 756) from 2010 to 2014. Disease status of radio-collared deer was re-evaluated annually during capture events. Additionally, we collected blood to determine pregnancy status and prion protein genotype (Prnp). Radio-collared deer were monitored throughout the study to determine survival, number of fawns recruited annually, and causes of mortality.

Most surprising during our investigation was the discovery of potential disease-driven evolution of Prnp at codon 225. In the early 2000s, 152 mule deer from HU 756 were genotyped and were predominantly homozygous serine (225SS; 98%), followed by heterozygous serine/phenylalanine (225SF; 2%), and no homozygous phenylalanine (225FF) deer (Jewell et al., 2005). In contrast, ten years later we found a statistically significant (p < 0.01) increase of the F allele (12% compared to <0.9% in 2000) in mule deer from the same HU. Research using captive and free-ranging mule deer demonstrated greater CWD prevalence and suggested shorter incubation periods in 225SS deer compared to 225SF deer (Jewell et al., 2005). In our study, 99% of CWD-positive deer were the 225SS genotype. Only one 225SF deer and none of the 225FF deer tested positive for CWD during our longitudinal study. We observed considerably higher CWD incidence and reduced annual survival in 225SS deer compared to deer that possessed at least one F allele (225*F). We believe the increase in F alleles in the population was the result of strong selection pressure driven by CWD favoring less susceptible genotypes (225SF and 225FF).

While we found reduced CWD incidence and protracted survival of 225*F deer, we also considered alternative forces shaping currently observed Prnp frequencies. Survival analysis of CWD-negative deer showed similar annual survival rates for 225SS deer and 225*F deer. These results suggested higher infection rates and lower survival of 225SS deer resulted in reduced fitness. Additionally, fitness characteristics such as annual fawn recruitment and body condition were similar between CWD-negative 225SS and 225*F deer. Thus, differential survival among genotypes presumably resulted in greater lifetime reproduction in 225SF and 225FF deer and consequently the observed increase of F alleles in the population. Our findings suggest CWD can drive Pnp selection and result in a significant increase of the F allele.

While our work is ongoing, our preliminary analyses propose significant future declines in mule deer numbers. Our study also demonstrated that one way free-ranging mule deer populations respond to CWD is through natural selection of the prion protein gene. However, this process may not adequately buffer the negative impacts of CWD on long-term viability of populations with high CWD incidence and prevalence. For example, changes in CWD susceptibility due to prion adaptation and differential immigration/emigration of deer to and from neighboring populations may diminish the influence of natural selection (Robinson et al., 2012). Furthermore, little is
In the News: First Detections of CWD and WNS

Bat with white-nose syndrome confirmed in Washington state

OLYMPIA – White-nose syndrome (WNS) has been confirmed in a little brown bat (Myotis lucifugus) found near North Bend – the first recorded occurrence of this devastating bat disease in western North America. The presence of this disease was verified by the U.S. Geological Survey’s National Wildlife Health Center.

WNS has spread quickly among bats in other affected areas, killing more than six million beneficial insect-eating bats in North America since it was first documented nearly a decade ago.

WNS is not known to pose a threat to humans, pets, livestock or other wildlife.

On March 11, hikers found the sick bat about 30 miles east of Seattle near North Bend, and took it to Progressive Animal Welfare Society (PAWS) for care. The bat died two days later, and had visible symptoms of a skin infection common in bats with WNS.

PAWS then submitted the bat for testing to the USGS National Wildlife Health Center, which confirmed through fungal culture, molecular and pathology analyses that it had WNS.

“We are extremely concerned about the confirmation of WNS in Washington state, about 1,300 miles from the previous westernmost detection of the fungus that causes the disease,” said U.S. Fish and Wildlife Service Director Dan Ashe. “Bats are a crucial part of our ecology and provide essential pest control for our farmers, foresters and city residents, so it is important that we stay focused on stopping the spread of this fungus.

People can help by following decontamination guidance to reduce the risk of accidentally transporting the fungus.”

First seen in North America in the winter of 2006/2007 in eastern New York, WNS has now spread to 28 states and five Canadian provinces. USGS microbiologist David Blehert first identified the unknown fungus, Pseudogymnoascus destructans, which causes the disease. WNS is named for the fuzzy white fungal growth that is sometimes observed on the muzzles of infected bats. The fungus invades hibernating bats’ skin and causes damage, especially to delicate wing tissue, and physiologic imbalances that can lead to disturbed hibernation, depleted fat reserves, dehydration and death.

“This finding in a far-western location is unfortunately indicative of the challenges we face with the unpredictability of WNS,” said Suzette Kimball, director of the USGS. “This underscores the critical importance of our work to develop tools for early detection and rapid response to potentially devastating wildlife diseases.”

The U.S. Fish and Wildlife Service leads the national WNS response effort, working with state and federal partners to respond to the disease. The Service’s National White-nose Syndrome Coordinator Jeremy Coleman said the first step will be to conduct surveillance near where the bat was found to determine the extent of WNS in the area. The Washington Department of Fish and Wildlife (WDFW) is responsible for bat management and conservation in Washington and will coordinate surveillance and response efforts.

WDFW veterinarian Katie Haman said the disease is transmitted primarily from bat to bat, although people can carry fungal spores on their clothing, shoes or caving gear.

“The bat found near North Bend most likely had been roused from hibernation and was attempting to feed at a time of very low insect availability,” Haman said. “At this point we don’t know where the infected bat may have spent the winter, but it seems likely that it was somewhere in the central Cascades.”

Haman said Washington state has 15 species of bats that benefit humans by consuming large quantities of insects that can impact forest health and commercial crops.

WDFW advises against handling animals that appear sick or are found dead. If you find dead bats or notice bats exhibiting unusual behavior such as flying outside during the day or during freezing weather, please report your observation online at http://wdfw.wa.gov/conservation/health/wns or contact the WDFW Wildlife Health Hotline at (800) 606-8768.

To learn more about WNS and access the most updated decontamination protocols and cave access advisories, visit www.whitenosesyndrome.org.

Persons with disabilities who need to receive this information in an alternative format or who need reasonable accommodations to participate in WDFW-sponsored public meetings or other activities may contact Dolores Noyes by phone (360) 902-2349, TTY (360-902-2207), or email (dolores.noyes@dfw.wa.gov). For more information, see http://wdfw.ca.gov/accessibility/reasonable_request.html.

CWD Found In Arkansas

In late February, the Arkansas Game and Fish Commission (AGFC) reported that an elk harvested on the Buffalo National River in October 2015 tested positive for chronic wasting disease (CWD). This was the first detection of CWD in the state. To begin to determine the extent of the disease, AGFC planned to collect 300 white-tailed deer and elk in a 5 mile radius of the index case. This focal area expanded quickly when a clinically ill deer was diagnosed with CWD about 12 miles from the elk.

A month later on March 31, AGFC reported that they had detected 50 positive deer and elk in the focal area and just beyond in adjacent lands. Results from 110 samples are still pending. Many of these positive animals were collected based on targeted surveillance, i.e., collection of sick-appearing animals. Others were hit by car or apparently healthy deer that were randomly culled in the focus area. Targeted surveillance biases prevalence estimates, but clearly prevalence and establishment of the disease in the area is higher than anyone anticipated.

As the next step, AGFC plans to focus on targeted surveillance and testing road-killed deer and elk on a much broader scale through May 20. This information will inform additional actions moving forward.
known regarding the evolutionary fitness of the Prnp 225FF polymorphism. While we observed similar survival and reproduction among CWD-negative Prnp groups, small sample size of 225FF mule deer (N=2) in our study precluded us from making strong conclusions regarding their fitness. Thus, additional research regarding the fitness and effects of genetic-specific CWD susceptibility in free-ranging cervid populations is warranted.

Lastly, the mechanisms by which CWD disease dynamics are affected by Prnp frequency are largely unknown. Longer survival of 225*F deer is promising for the species; however, it is yet to be determined if these deer also contribute more to direct transmission and environmental contamination with infectious prions due to prolonged incubation periods. Further research regarding Prnp-specific CWD transmission and prion shedding is necessary in future studies.

This research was supported by funding from the USGS National Wildlife Health Center, WGFD, and the Mule Deer Foundation. A graduate stipend was also provided by the University of Wyoming, Department of Veterinary Sciences. I thank all those that helped with deer captures, field work, and genotyping. I also thank the many private landowners in southern Converse County, WY for granting us access to their land to collect data.

Citations

Melia DeVivo completed a M.Sc. degree in Biology at Indiana University of Pennsylvania in 2010 and is expected to complete her Ph.D. at the University of Wyoming in 2015. She has conducted research on cervid biology for over a decade and is especially interested in wildlife disease epidemiology. Melia can be contacted at mdevivo@uwyo.edu
Current Research in Wildlife Disease

Nuno Santos, Virgilio Almeida, Christian Gortazar, and Margarida Correia-Neves. PATTERNS OF MYCOBACTERIUM TUBERCULOSIS COMPLEX EXCRETION AND CHARACTERIZATION OF SUPER-SHEDDERS IN NATURALLY-INFECTED WILD BOAR AND RED DEER. Veterinary Research 2015 46:129. doi:10.1186/s13567-015-0270-4. Wild boar (Sus scrofa) and red deer (Cervus elaphus) are the main maintenance hosts for bovine tuberculosis (bTB) in continental Europe. Understanding Mycobacterium tuberculosis complex (MTC) excretion routes is crucial to define strategies to control bTB in free-ranging populations, nevertheless available information is scarce. Aiming at filling this gap, four different MTC excretion routes (oronasal, bronchial-alveolar, fecal and urinary) were investigated by molecular methods in naturally infected hunter-harvested wild boar and red deer. In addition MTC concentrations were estimated by the Most Probable Number method. MTC DNA was amplified in all types of excretion routes. MTC DNA was amplified in at least one excretion route from 83.0% (CI95 70.8–90.8) of wild ungulates with bTB-like lesions. Oronasal or bronchial-alveolar shedding were detected with higher frequency than fecal shedding (p < 0.001). The majority of shedders yielded MTC concentrations <10⁴ CFU/g or mL. However, from those ungulates from which oronasal, bronchial-alveolar and fecal samples were available, 28.2% of wild boar (CI95 16.6–43.8) and 35.7% of red deer (CI95 16.3–61.2) yielded MTC concentrations >10⁴ CFU/g or mL (referred here as super-shedders). Red deer have a significantly higher risk of being super-shedders compared to wild boar (OR = 11.8, CI95 2.3–60.2). The existence of super-shedders among the naturally infected population of wild boar and red deer is thus reported here for the first time and MTC DNA concentrations greater than the minimum infective doses were estimated in excretion samples from both species.

Chandy C. John, Helene Carabin, Silvia M. Montano, Paul Bangitana, Joseph R. Zunt, and Phillip K. Peterson. GLOBAL RESEARCH PRIORITIES FOR INFECTIONS THAT AFFECT THE NERVOUS SYSTEM. Nature 527, S178-2015. Doi: 10.1038/nature16033. Infections that cause significant nervous system morbidity globally include viral (for example, HIV, rabies, Japanese encephalitis virus, herpes simplex virus, varicella zoster virus, cytomegalovirus, dengue virus and chikungunya virus), bacterial (for example, tuberculosis, syphilis, bacterial meningitis and sepsis), fungal (for example, cryptococcal meningitis) and parasitic (for example, malaria, neurocysticercosis, neuroschistosomiasis and soil-transmitted helminths) infections. The neurological, cognitive, behavioural or mental health problems caused by the infections probably affect millions of children and adults in low- and middle-income countries. However, precise estimates of morbidity are lacking for most infections, and there is limited information on the pathogenesis of nervous system injury in these infections. Key research priorities for infection-related nervous system morbidity include accurate estimates of disease burden; point-of-care assays for infection diagnosis; improved tools for the assessment of neurological, cognitive and mental health impairment; vaccines and other interventions for preventing infections; improved understanding of the pathogenesis of nervous system disease in these infections; more effective methods to treat and prevent nervous system sequelae; operations research to implement known effective interventions; and improved methods of rehabilitation. Research in these areas, accompanied by efforts to implement promising technologies and therapies, could substantially decrease the morbidity and mortality of infections affecting the nervous system in low- and middle-income countries.

Hot Topics: The Dilution Effect by LDCR Danielle Buttke

A recent publication in PNAS and highlighted in Science News conducted a broad-scale meta-analysis of over 200 assessments and found ‘overwhelming evidence’ supporting the dilution effect. The dilution effect is the hypothesis that disease systems with variations in host competence, host resilience, and non-density-dependent transmission, biodiversity can decrease infectious disease risk. The dilution effect has been hotly debated and studied for its promise for conservation: if biodiversity does indeed protect humans and animals from infectious disease, it provides another argument for protecting natural spaces and resources. The meta-analysis is available electronically here: http://www.pnas.org/content/112/28/8667.abstract.
Mission Statement

The mission of the Wildlife Diseases Working Group is to promote better scientific understanding of the causes and consequences of disease in ecosystems and wildlife populations; to apply the principles of wildlife science, ecology, and epidemiology to the prevention and management of diseases in wildlife; to foster education and transfer of information on diseases to wildlife management professionals and the public; and to apply this knowledge to enhance the health and conservation of wildlife populations and their interactions with humans and domestic animals.

Have you worked on a wildlife disease research project? Have you engaged in a wildlife-related veterinary externship?

We want to hear about it!

‘The Vector’ is the quarterly newsletter distributed by the Wildlife Disease Working Group.

We feature a student article in every newsletter, which highlights how students across the country are involved in wildlife disease projects.

As an appreciation for preparing the article, the WDWG is happy to sponsor a 1-year membership to both TWS and WDWG for student and postdoctoral authors!

Interested in learning more?

Contact a member of the WDWG Student Affairs Committee:

- Katrina Alger: kealger@syr.edu
- Sarah A. Hamer, Texas A&M University: shamer@cvm.tamu.edu
- Michelle Clayson: rosenmi1@gmail.com
- Graham Hickling, University of Tennessee: ghicklin@utk.edu
- Mike Samuel, University of Wisconsin: mdsamuel@wisc.edu

Need financial help to attend the annual meeting in Raleigh? Look for student travel grant details in the next issue of The Vector!