March 2, 2026

TO: Oklahoma Department of Wildlife Conservation

FROM: Mark Ruder, Director
Southeastern Cooperative Wildlife Disease Study

RE: Wild white-tailed deer GEBV scores and interest in release of farmed deer

Below are some thoughts on the data file I was able to read titled "20250206_Wild_Deer_OK". The spreadsheet provided GEBV scores and *PRNP* gene polymorphisms for 500 free-ranging WTD from Oklahoma that were sampled in 2024. I will also provide some other supporting information that may help ODWC navigate continued outside interest in the release of farmed deer into wild populations for the purpose of CWD management. As I have stated previously, there is no scientific justification for the release of farmed white-tailed deer into wild populations in Oklahoma for the purpose of CWD management, or otherwise. These data do not change that, nor does any peer-reviewed scientific research support such an action. As you know, I will help ODWC navigate this issue however needed. I am happy to provide information as I am here, or engage more directly. I am also in regular contact with experts in the field from various scientific disciplines, including conservation geneticists, and I can help connect ODWC with these experts. These other voices and perspectives would be helpful to the discussion. I know numerous wildlife professionals and researchers who would engage in this matter. The proposed release of farmed cervids into wild populations, if realized in Oklahoma, would set a dangerous precedent for deer management in other jurisdictions. In that vein, many people around the country are paying attention to what happens in Oklahoma and I am confident many wildlife managers, researchers, and conservation organizations around the country would be ready to assist ODWC with this matter.

Take home points:

- The data provides GEBV scores and *PRNP* gene polymorphisms of 500 wild deer from Oklahoma but does not provide scientific justification for the release of farmed deer into the wild. Simply having GEBV data on a small number of wild deer from Oklahoma does not help justify the release of deer, it honestly only causes us to ask more questions. These are simply numbers in a spreadsheet and dots on a map. In short, the data changes nothing. All the arguments and points offered previously still apply.
- The entire concept of utilizing a GEBV score to manage selective breeding choices within the farmed cervid industry is based on two publications: Seabury et al 2020 and 2022. The genetic tool being explored in these papers was based on genomic data from farmed white-tailed deer. This is an important limitation because there is genetic variation among not only different captive populations of deer, but also different wild populations of deer. The correlation of GEBV scores with CWD susceptibility based on intensive study of captive herds should not be directly applied to wild populations of deer. There have been no published research studies to advance the science beyond the two proof of concept studies mentioned above (Seabury et al 2020, 2022) and these publications have **not** been sufficient to provide consensus within the scientific community. There needs to be more published research to build on these two studies. Importantly, because even deer with favorable GEBV scores are ultimately susceptible to CWD, such research should clearly define the onset and duration of prion shedding, the onset of disease, and the ability of diagnostic assays to successfully detect infected deer. The underlying mechanisms driving the potential relationship should be explored. Finally, research into how such a program designed for farmed deer could be

implemented in the wild remains completely unexplored. Until more research provides clarity and confidence, this is just a theory that puts wild deer at risk.

- Releasing farmed deer into a healthy and thriving wild population is a radical idea. Such an aggressive wildlife management intervention would typically be reserved for a species in crisis and there would be significant goal-oriented planning, coordination and monitoring. This idea of releasing farmed deer carries many risks to the deer population in Oklahoma and none of these risks have been defined, considered, or mitigated. I am not aware of any science-based management approaches being discussed as it relates to preventing pathogen introduction, monitoring genetic profiles, monitoring disease status, or monitoring vital rates in wild or released deer, etc.
- As outlined below, the release of farmed deer into the wild for the purposes of CWD prevention and management in Oklahoma would be against the scientific recommendation of all relevant and respected regulatory authorities and experts who manage or study disease in wild deer. The state of Oklahoma would be taking an action that is not supported by science and would go against the scientific opinion of experts who work in the best interest of natural resources.

Scientific opinion by scientific experts and regulatory authorities

CWD Research Consortium:

Excerpts from: Scientific Comment on use of Selective Breeding and release of Captive White-tailed Deer for CWD Prevention and Management, https://www.cwd-research.com/home/selective-breeding-and-release-of-captive-white

"*There is **no feasible approach to releasing captive-bred white-tailed deer that would meaningfully change a free-ranging deer population's genetic structure**, neither locally nor at scales relevant to management, particularly in hunted populations*."

"***All white-tailed deer are susceptible to CWD**. No alleles or combinations of alleles have been identified that prevent CWD infection, thus there are no known truly 'resistant' genotypes*."

"*Despite ongoing research in this area, there are critical knowledge gaps and scientific uncertainties about the relationship between CWD and deer genetics. **Further research is needed to better identify and understand potential unintended consequences prior to the application of genetic tools to CWD management in free-ranging cervids***."

Notably, Seabury et al. 2020 and 2022 were both published at the time this document was written. No additional scientific research in support of this approach has been published since that time. The science has not advanced.

National Academies of Sciences, Engineering, and Medicine:

Excerpt from: National Academies of Sciences, Engineering, and Medicine. 2025. State of Knowledge Regarding Transmission, Spread, and Management of Chronic Wasting Disease in U.S. Captive and Free-Ranging Cervid Populations. Washington, DC: The National Academies Press. doi: 10.17226/27449.

"*Conclusion 9: **Genetic selection**, vaccines, environmental decontamination, and therapeutic options **are being investigated as tools for CWD control but need further inquiry and review**. Although none of these approaches can, at present, replace existing forms of management and control, in the future they may, in combination with current methods, reduce CWD on the landscape."*

Notably, Seabury et al. 2020 and 2022 were both published at the time this statement was made. No additional scientific research in support of this approach has been published since that time. The science has not advanced.

Association of Fish and Wildlife Agencies

Excerpt: AFWA Resolution 2024-09-10 in opposition to the release of captive-bred cervids into free-ranging white-tailed deer populations

> "…***the Association opposes the release of any captive cervids into the wild to influence free-ranging cervid population genetics for the purpose of controlling or managing CWD****, based on the current best scientific information, and encourages its members in their own jurisdictions to promote and implement the best scientific management practices for CWD*."

Notably, regarding the comment above "…current best scientific information…", again both Seabury et al 2020 and 2022 were published at the time of this statement and no new scientific research in support of this action (releasing farmed cervids) that would support the action has been published.

USDA-APHIS Veterinary Services

Excerpt: Letter to the Association of Fish and Wildlife Agencies from the Deputy Administrator of APHIS-Veterinary Services, dated November 24, 2025 in response to AFWA Resolution 2024-09-10

> "***APHIS does not support the release of livestock, including farmed cervids, into wild populations.*** *The one allowance APHIS recognizes as a legitimate reason for releasing captive cervids is outlined in title 9, Code of Federal Regulations, section 81.3(b), which provides for the release of captive deer, elk, or moose that were captured from a wild population for interstate movement and subsequent release*."

USDA-APHIS Wildlife Services

Excerpt: Letter to the Association of Fish and Wildlife Agencies from the Deputy Administrator of APHIS-Wildlife Services, dated January 28, 2025 in response to AFWA Resolution 2024-09-10

> "***APHIS does not support the release of captive-bred cervids into free-ranging populations****. The only allowance APHIS recognizes as a legitimate reason for releasing captive cervids is outlined in Title 9, Code of Federal Regulations, section 81.3 (b), which provides for the release of captive deer, elk, or moose that were captured from a wild population for interstate movement and subsequent release*."

Summary of data provided by ODAFF/Oklahoma Whitetails

My assumption is the spreadsheet was generated by the North American Deer Registry (NADR) and that Dr. Seabury is the one who compiled the data and reported these results. Dr. Seabury is the one who created the algorithm used to calculate GEBV scores, and to my knowledge he has not shared it with anyone. Therefore, any GEBV score to date has been generated by Dr. Seabury. The spreadsheet provides the GEBV score along with variation at multiple different codons of the *PRNP* gene (codons 37, 95, 96, 116, 226) for all 500 deer. Numerous studies across multiple investigators have demonstrated that the *PRNP* gene underlies the genetic basis of CWD susceptibility. And notably, studies have shown that all deer are susceptible to CWD, regardless of genetics. However, some *PRNP* gene variations (e.g., 96 S/S) have been associated with a longer incubation period (i.e., delayed disease onset). This importance of the *PRNP* gene is also supported by our understanding of other prion diseases in other species. However, Dr. Seabury's two publications (2020 and 2022) offer new information - that CWD susceptibility is more nuanced and involves more than the *PRNP* gene. Dr. Seabury presents evidence that CWD susceptibility is polygenic – meaning it is driven by thousands of loci (locations) across the genome. Thus, this polygenic approach to CWD susceptibility is a new concept and departs from prior scientific understanding of CWD susceptibility. The two scientific studies by Seabury should be considered first steps in exploring a new understanding of the mechanisms underlying this polygenic theory and should be explored further. It is important to understand that these two publications (that are 4-6 years only now) do not demonstrate or explain the hypothesized mechanisms for the influence these other areas in the

genome (outside of the *PRNP* gene) have on CWD susceptibility. So, **nothing has changed** with these data from Oklahoma wild deer - there is still no scientific justification for releasing farmed deer. While **Dr. Seabury's original study results are interesting and novel, these two studies should encourage more research, not initiate premature management action**. This is especially true for free-ranging deer.

Below is a basic summary of the spreadsheet data to help visualize the data. I have pulled together the summary data for codon 96 polymorphisms for the *PRNP* gene because the literature clearly shows that codon 96 is important to CWD susceptibility, with the least susceptible being 96 S/S and most susceptible being 96 G/G. Importantly, 96 S/S deer are generally not common in most wild deer populations. However, the predictive genomics approach for farmed cervids emphasizes that you can see favorable GEBV scores (i.e., deer you want to keep) that are 96 G/G, G/S or S/S. Again, you can see how this line of thought (without mechanistic explanation or further exploration) is a departure from previous scientific understanding. More and diversified research is needed.

Table: polymorphisms at codon 96 of *PRNP* gene for 500 deer sampled in Oklahoma

Total	G/G	%	G/S	%	S/S	%
500	272	*54*	189	*38*	39	*8*

Tables: breakdown of polymorphisms at codon 96 of *PRNP* gene for deer with favorable GEBV scores (n=75, < -0.056), compared with those deer that have unfavorable GEBV scores (n= 424; > -0.056)

< -0.056	%	G/G	%	G/S	%	S/S	%
76	*15*	44	*58*	26	*34*	6	*8*

>-0.056	%	G/G	%	G/S	%	S/S	%
424	*85*	228	*54*	163	*38*	33	*8*

So, of the 500 wild deer sampled, 76 (15%) had favorable GEBV scores. The percentage of deer with various codon 96 polymorphisms (i.e., G/G, G/S, S/S) are basically the same between deer with favorable GEBV scores and unfavorable GEBV scores.

Figure: Counties (gray) where wild deer (n=500) were sampled in Oklahoma in 2024. Black number = no. sampled for each county. Red number = no. of deer with favorable GEBV scores (< -0.056).

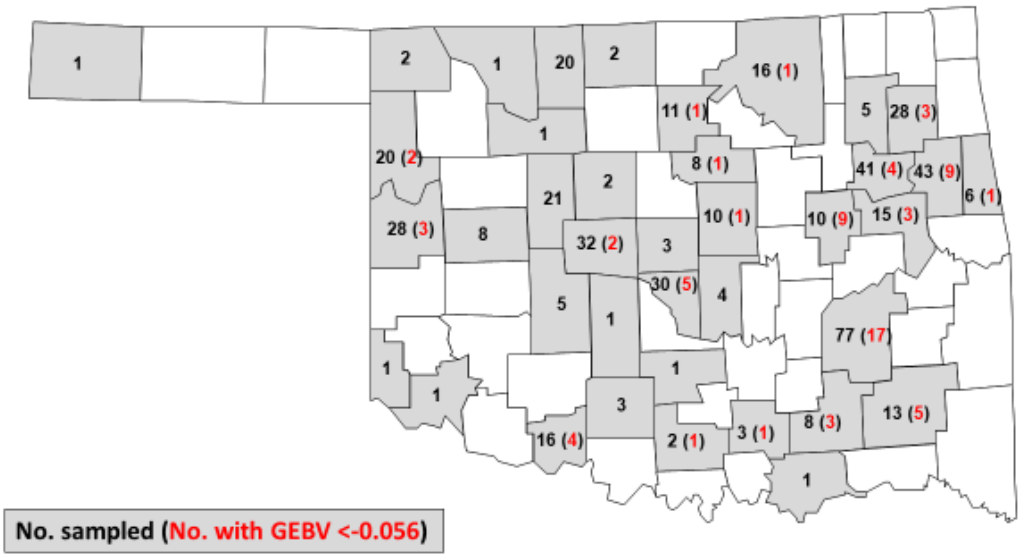
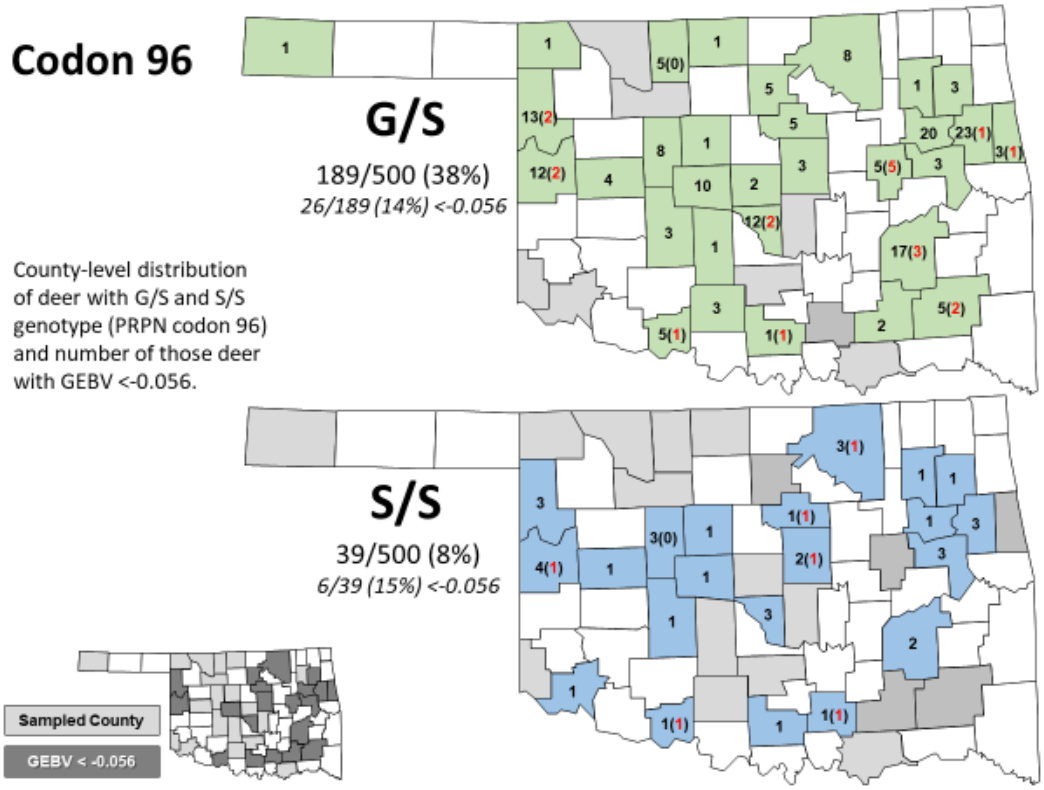


Figure: County level locations of deer sampled in Oklahoma that are G/S (top map) and S/S (bottom map) at codon 96 of the *PRNP* gene. Black number = no. of G/S or S/S in each sampled county. Red number = no. of deer that also have a favorable GEBV score (< -0.056).



What is GEBV and how is it used?

The paragraph and bullet points below are a summary of information provided in an APHIS document titled, *Genomically Estimated Breeding Value Predictive Genetics and Chronic Wasting Disease*. The document promotes GEBV for farmed cervids. This is effectively a document promoting the GEBV method to the captive industry and it clearly outlines their approach and I summarize some of it here for your understanding so you can better wrap your head around the GEBV score and their approach.

GEBV is a statistically generated score that estimates total genetic potential of an animal with respect to a heritable trait. [This is used by agricultural geneticists to drive breeding practices and programs that typically focus on maximizing production and economic efficiency. Wildlife population management should seek guidance from conservation geneticists – not geneticists focused on agricultural production]. The traits can be influenced by many different regions across the genome. Dr. Seabury's studies found that >120,000 different regions may have a role in CWD susceptibility. However, the document states that some regions will have greater contributions than others and these are the regions that Dr. Seabury has based his GEBV score on. Based on this USDA document, deer with GEBV scores that are **-0.056 or lower** (meaning more negative) are those animals that should be used as breeders to generate a herd with reportedly reduced CWD susceptibility. ***Below are the USDA outlined "steps to using GEBV scores for herd improvement". Keep in mind, these are written for farmed cervids***. No such document exists for wild cervids.

- Identify all animals with scores above the minimum cutoff (-0.056)
- Keep meticulous herd and breeding records that includes GEBV scores and codon 96 data

- Determine if the herd has a sufficient number of deer with scores <-0.056 to sustain viable breeding population without breeding too tightly.
- Develop a culling plan to remove as many deer above the cutoff (>-0.056) as possible. With careful breeding over several generations, the overall GEBV scores can be improved.

None of these critical steps outlined by USDA are possible in a free-ranging herd. At best, altering herd genetics through these steps is very challenging and expensive in a farmed deer herd. In an abundant free-ranging population of deer, it is simply not financially feasible or biologically possible to meaningfully influence population genetics. From what I have seen, the advocates for this action have provided zero scientific evidence that this approach is effective outside the fence. However, I have also not heard what the goal of such a program would be, what metrics would be used to monitor post-release, and what the metrics of success or failure would be. All of these are fundamentally important questions to make financially sound science-based management decisions. The USDA document I mention above (nor the two publications that it is based on, Seabury et al. 2020, 2022) have relevance to wild deer. The entire concept was developed using farmed deer samples and was intended to be applied farmed deer. It cannot simply be applied to wild deer and expected to work. If the polygenic sites identified by Dr. Seabury are actually important to CWD susceptibility in deer, this only applies to the farmed deer populations that he developed this theory in (the study population). We do NOT know if these same loci are important in other farmed deer herds, or across the genetic diversity of numerous free-ranging populations of deer. We cannot simply apply results of one study to the entire population (farmed and wild) of white-tailed deer in America and assume such an approach will work. Much more science is needed.

I continue to stress that the specific method Dr. Seabury used to calculate GEBV scores has not been made available to other scientists to confirm its validity. This is a major limitation. A fundamental part of the scientific method is independent reproducibility. Meaning, other scientists should be able to have the information needed to independently replicate and verify the result. However, this is not possible because Dr. Seabury has not made the method available. This lack of transparency is not how science works; other geneticists should be able to reproduce his work. In a study by Navarro et al (2026), the team investigated 55 locations in the WTD genome outside of the *PRNP* gene that Dr. Seabury identified as important for CWD status in his 2020 paper. However, 15 loci (37%) showed a significant allele frequency difference, implying potential differences in CWD risk between the two study populations (farmed deer vs free-ranging deer). However, the authors highlighted that more research is needed, and CWD+ wild deer should be included in future studies. This paper, while limited in scope, highlighted the point above that what applies to the captive study population Dr. Seabury based his work on, may not apply to populations of wild deer. Indeed, numerous other studies by multiple researchers are ongoing that may shed light on this area of research in the near future.