Model Forecasting of the Impacts of Chronic Wasting Disease on the Jackson Elk Herd

Nathan L. Galloway[*], Ryan J. Monello[*†], Doug Brimeyer[‡], Eric Cole[§], and N. Thompson Hobbs[¶]

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[*]National Park Service, Biological Research Division
[†]Present Address: National Park Service, Inventory and Monitoring Program, Pacific Island Network
[‡]Wyoming Game and Fish Department
[§]United States Fish and Wildlife Service, National Elk Refuge
[¶]Colorado State University, Natural Resource Ecology Laboratory; Corresponding author.

Executive Summary

Chronic wasting disease (CWD) is a contagious transmissible encephalopathy that infects members of the deer family (Cervidae) in North America. Although CWD does not currently occur in the Jackson elk herd, it is present in mule deer (*Odocoileus hemionus)* 35 miles away and in elk within 160 miles. Observations from captive and free-ranging herds suggest CWD may have greater impacts on elk in relatively high-density settings or where environmental contamination may occur in a smaller area, such as those found on the National Elk Refuge (NER).

We developed a Bayesian model for the Jackson elk herd to achieve three goals; 1) Examine the potential impacts of the CWD on population dynamics of the Jackson elk herd, 2) Aid in designing future sampling for CWD in the population, and 3) Provide a proactive framework for assimilating new data on herd health to guide adaptive management of the population.

We fit the model using three sources of data on the Jackson population: total census, demographic classifications, and CWD test results and included information from a CWD-infected elk population in Rocky Mountain National Park (RMNP). The model enables predictions of the future state of the population accompanied by rigorous, statistically defensible estimates of uncertainty. These forecasts can be used to understand the short-term dynamics of the population infected with CWD before the disease actually arrives.

Analysis revealed that CWD is likely to drive the population growth rate (λ) below one, indicating a decline in elk numbers over time. A declining population becomes more probable with increasing disease prevalence. Given the predictions from this study and no hunting, the most probable threshold between an increasing and a declining population ($\lambda = 1$) occurred when prevalence of CWD in yearling and adult females reached 7%; however, we cannot rule out a threshold as high as 23% prevalence before λ falls below one. The addition of CWD into the model with current hunting levels leaves little possibility that the population is growing. Note, however, that recent harvest levels are designed to decrease the population size. Although there is significant overlap in the forecasts for an infected and uninfected population, it is clear that CWD has the potential to decrease the population size.

The model framework presented here is a first step towards informing managers of the Jackson elk herd of the possible effects of CWD on the population. Our results depend in part on results

of research conducted on elk populations infected with CWD in RMNP. Given that the modeled effects were based on a smaller and presumably less concentrated elk population, CWD may lead to higher disease prevalence and larger impacts of CWD on the population growth rate due to crowded herds returning annually to the same location, as occurs on some feedgrounds.

Problem Statement

Chronic wasting disease (CWD) is a contagious transmissible encephalopathy that infects members of the deer family (Cervidae) in North America. Although CWD does not currently occur in the Jackson elk herd, it is present in mule deer (*Odocoileus hemionus)* 35 miles away and in elk within 160 miles. Previous research suggests that the disease will continue to slowly spread in a fashion that has been described as an "epizootic with a protracted time-scale" (Miller et al., 2000). It is impossible to predict when or even if CWD will occur in the Jackson valley. However, there is no evidence to suggest the performance of the Jackson elk herd wouldn't be affected by the disease in a way that resembles other elk populations through reduced adult survival and consequent reductions in population growth rate (Sargeant et al., 2011; Monello et al., 2014). Observations from captive and free-ranging herds suggest CWD may have greater impacts on elk in relatively high-density settings or where environmental contamination may occur in a smaller area (e.g., Peters et al., 2000; Williams and Miller, 2002; Monello et al., 2014), such as those found on the National Elk Refuge (NER). Given the profound importance of the Jackson elk herd to ecosystem processes and to human livelihoods and recreational opportunity in this region, it is prudent to use existing data to help understand potential implications of CWD on this elk population and to plan for managing the impacts of the disease.

Supplemental winter feeding for elk has occurred on the NER and U.S. Forest Service lands in Wyoming for over 100 years. This practice typically results in a highly concentrated elk herd from January to April. An interagency Bison and Elk Management Plan (2007) developed for the elk that winter on or near the NER identified an overarching strategy of reducing reliance on supplemental feeding to achieve management goals and objectives. To date, successful reduction of total population size and improvements to available natural standing forage have been unsuccessful in reducing elk density on the NER winter range. A spatial redistribution of animals onto the NER has maintained high winter densities(see Cole et al., 2015), which will likely be conducive to disease transmission.

Past modeling attempts make long-term projections of the arrival and subsequent transmission of CWD in the Jackson Hole area highly uncertain, predicting an enormous range of outcomes (e.g., Osnas, 2011), which limits their value for guiding management and policy. However, extensive de-

mographic data are available for elk in this region and can be combined with data from studies of CWD in elk elsewhere in the Rocky Mountains to reduce uncertainty about how CWD may shape the dynamics of the Jackson Hole elk population. This approach is enabled by the advent of new Bayesian state-space models and recent CWD studies on free-ranging elk (LaDeau et al. 2011; Monello et al. 2013, 2014, and unpublished/ongoing NPS project data). This approach allows managers to forecast how differing levels of CWD will interact with elk recruitment, survival, and migration to determine the Jackson elk herd's population trajectory. Additionally, these forecasts can be properly tempered by rigorous estimates of uncertainty and allow managers to fully understand the range and accuracy of predicted outcomes.

Here we develop these analyses and associated products for the entire Jackson elk herd, recognizing that it is shared and not exclusive to the NER. This work will benefit the long-term goals and understanding of all pertinent agencies in the region. To that end, Wyoming Game and Fish Department is a key partner in our work. National Park Service collaborators have a strong record of research on CWD, producing results relevant to elk management across agencies.

We developed a Bayesian state-space model to achieve three goals:

1. Examine the potential impacts of the CWD on dynamics of the Jackson elk herd.

2. Aid in designing future sampling for CWD in the population.

3. Provide a proactive framework for assimilating new data on herd health to guide adaptive management of the population (see Walters, 1986).

Modeling Approach

Here, we provide a general overview of our inferential approach, leaving detailed, technical treatment of the mathematics and statistics to Appendix 1. All inference is built from a model that uses a projection matrix to annually update a state vector (Caswell, 1988). The state vector consists of the number of individuals in four age and sex classes (juveniles, yearling and adult females, yearling males, and adult males) and two disease states (infected and not infected with CWD). The projection matrix is composed of parameters representing survival, recruitment and disease transmission. The structure of our model closely resembles the matrix models used to inform decisions on harvest of ungulates throughout the Western United States (e.g., White and Lubow, 2002)

We fit the model using three sources of data on the Jackson population: total census, demographic classifications, and CWD test results. We use Markov chain Monte Carlo (MCMC) methods to approximate the statistical distributions of all parameters and unobserved states conditional on the data. These distributions provide honest estimates of uncertainty arising from the failure of the model to represent population and disease dynamics (process variance) and from sampling and calibration error in the data (observation variance).

Estimating process variance separately from observation variance enables forecasting. We are able to make predictions of the future state of the population accompanied by rigorous, statistically defensible estimates of uncertainty. These forecasts can be used to understand the short-term dynamics of the population infected with CWD before the disease actually arrives.

We make inference on the effect of CWD on population growth rate by calculating the dominant eigenvalue of the projection matrix using the values of survival, recruitment, and probability of transmission of CWD at each MCMC iteration. We repeat this sampling for different hypothetical levels of prevalence of CWD in the population, allowing us to see how population growth rate would change with different levels of prevalence.

We further examined the potential influence that CWD may have on population growth across varying levels of harvest. When included, harvest was set equal to the average hunting levels from 2011-2015 .

We use posterior predictive checks to assure that the model meets the fundamental assumption

of statistical inference, that the inferential model is capable of giving rise to the data. Lack of fit is indicated if test statistics (mean, coefficient of variation, and discrepancy) calculated on datasets simulated at each MCMC iteration are consistently smaller or consistently larger than the same test statistics calculated from the observed data. We calculate these test statistics for all datasets used to fit the model.

Results

The model passed all posterior predictive checks. There was no evidence of lack of fit (Table 1).

Table 1: Posterior predictive checks fail to show lack of fit, which is indicated by Bayesian P values close to 0 or 1.

Observation	Bayesian P value
Total counts	0.24
Proportion juveniles	0.46
Proportion yearling and adult females	0.52
Proportion yearling males	0.49
Proportion adult males	0.45

Data modified posterior distributions of parameters relative to priors for uninfected juvenile survival, uninfected yearling and adult male survival, proportion of juveniles surviving to the yearling stage that are male, recruitment, and probability of transmission (Figure 1, Tables 2 and 6). The posterior distribution of probability of transmission shifted away from the prior distribution and toward zero because of extensive testing data showing no positive cases in the Jackson elk herd. Data caused small shrinkage in posterior distributions of yearling and adult female survival, confirming prior knowledge.

The population showed a weak downward trend during 1998 - 2015 (Figure 2)[1]. Model predictions were strongly influenced by sex and age classification data (As evidenced by the extremely small credible interval around each data point in Figure 3).

Eigen analysis revealed that infection of the population with CWD is likely to drive the population growth rate (λ) below one (Figure 4), indicating a decline in elk numbers over time. A declining population becomes more probable with increasing disease prevalence, particularly in the presence of recent harvest levels.

[1] 2016 is treated as a forecast due to pending CWD test results.

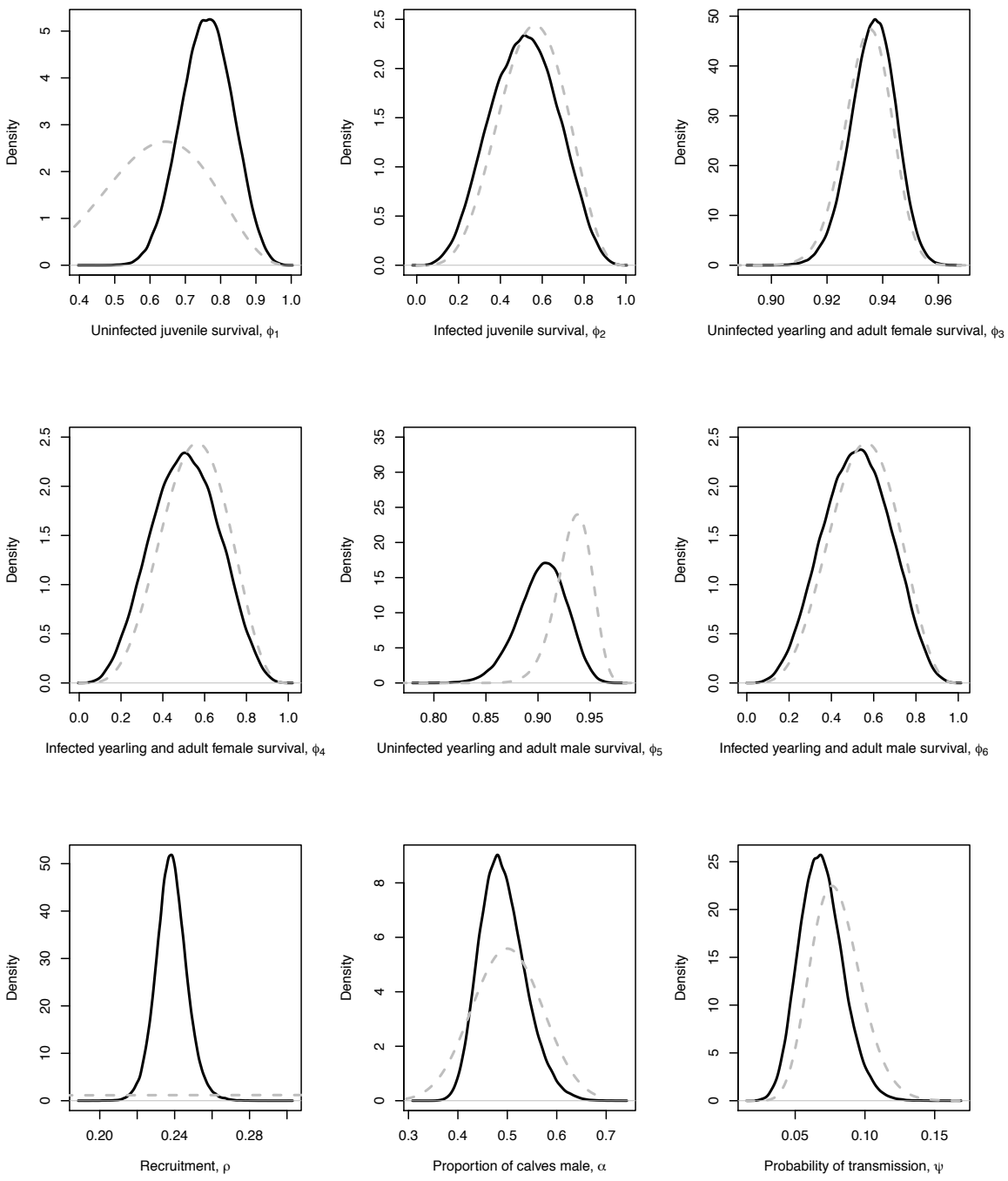

Figure 1: Solid and dashed lines show marginal posterior and prior distributions, respectively, of model parameters.

Table 2: Marginal posterior distributions of model parameters are summarized with means, standard deviations (SD), medians and upper (97.5%) and lower (2.5%) highest posterior density intervals.

	Mean	SD	Median	2.5%	97.5%
Recruitment α	0.49	0.046	0.49	0.41	0.58
Uninfected juvenile survival ϕ_1	0.76	0.072	0.76	0.62	0.9
Ininfected juvenile survival ϕ_2	0.52	0.16	0.52	0.22	0.82
Uninfected yearling and adult female survival ϕ_3	0.94	0.008	0.94	0.92	0.95
Infected yearling and adult female survival ϕ_4	0.51	0.16	0.51	0.21	0.82
Uninfected yearling and adult male survival ϕ_5	0.9	0.023	0.91	0.86	0.95
Infected yearling and adult male survival ϕ_6	0.52	0.16	0.52	0.22	0.81
Probability of transmission ψ	0.069	0.016	0.068	0.039	0.099
Recruitment ρ	0.24	0.0083	0.24	0.22	0.25
Process standard deviation for adult and yearling females σ_{pf}	0.11	0.031	0.11	0.057	0.17
Process standard deviation for juveniles σ_{pj}	0.14	0.033	0.13	0.078	0.2
Process standard deviation for yearling and adult males σ_{pm}	0.21	0.035	0.2	0.14	0.27

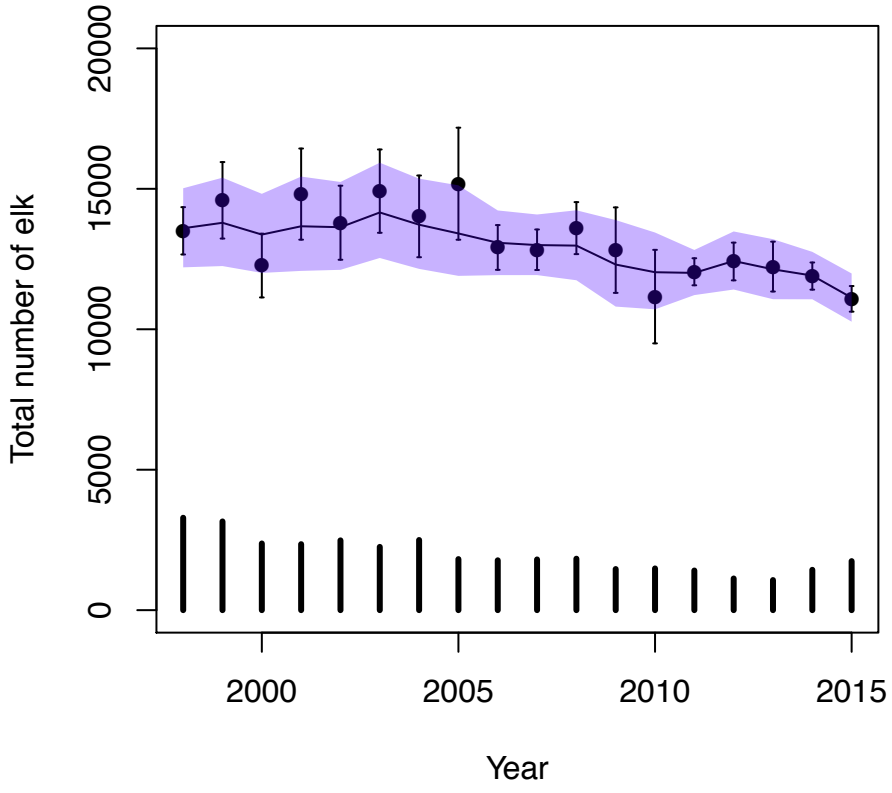

Figure 2: Number of elk in the Jackson Elk Herd is shown on the y-axis during 1998 to 2015. The population showed a weak downward trend during this time, coinciding with management efforts to reduce abundance. Filled circles are mean counts and vertical bars are ± 2 standard deviations. Solid line is the median estimate of the true, unobserved population size and shaded areas give 95% equal-tailed credible intervals. Vertical bars show total harvest preceding census.

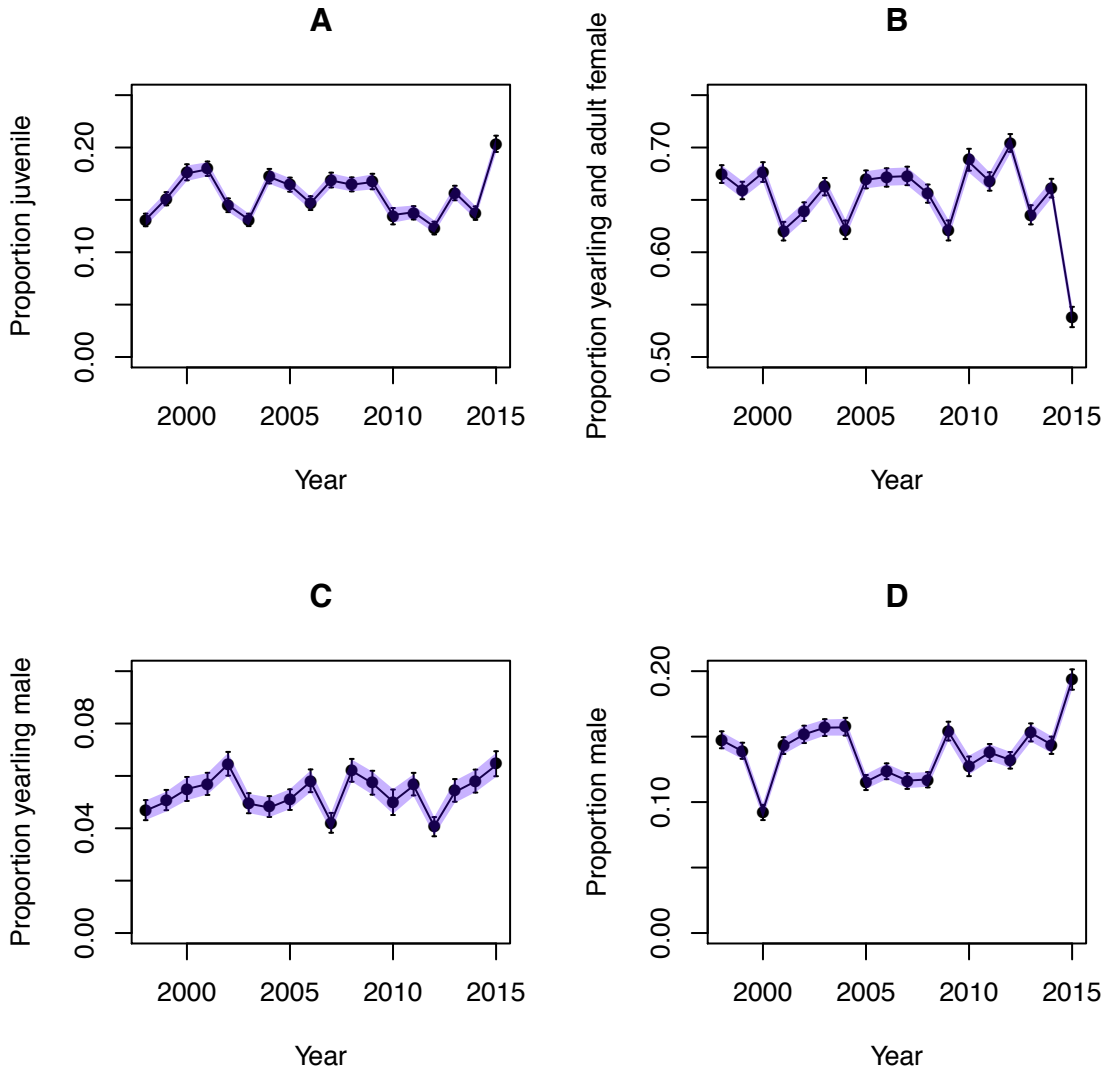

Figure 3: Model fit to data on population composition: **A**. Juveniles **B**. Yearling and adult females **C**. Yearling males **D**. Adult males. Filled circles are the proportion observed in annual classification counts and vertical bars are ± 2 standard deviations of the proportion. Solid line is the median of the unobserved, true population composition. The shaded area gives 95% highest posterior density interval of the true proportion.

Given the predictions from this study and no hunting, the most probable threshold between an increasing and a declining population ($\lambda = 1$) occurred when prevalence of CWD in yearling and adult females reached 7%; however, we cannot rule out a threshold as high as 23% prevalence before λ falls below one. The probability that the current population growth rate, in the absence of both hunting and CWD, is less than one is 0.055. Recent (2011-2015) levels of hunting reduce the population growth rate significantly, but this is by design, as the state and federal agencies are seeking to reduce abundance of the Jackson elk herd. The addition of CWD into the model leaves little possibility that the population is growing (See Figure 4, Panels A and B).

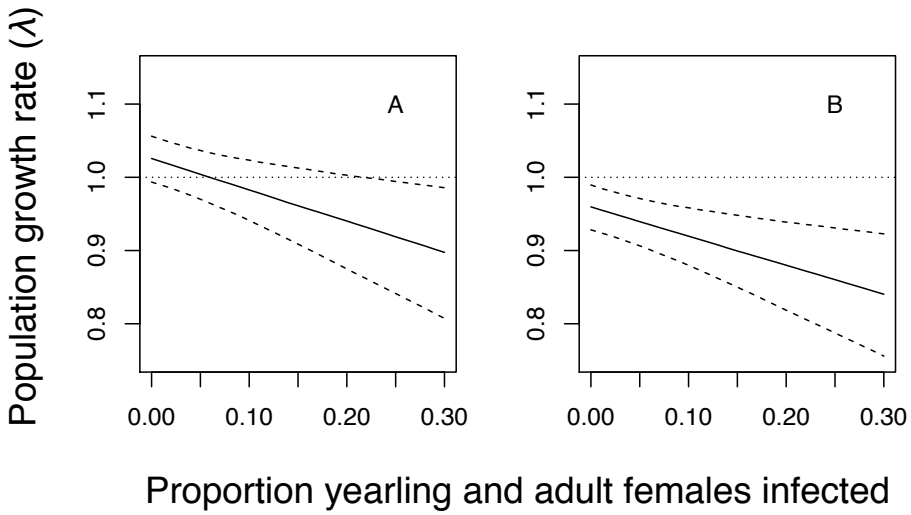

Figure 4: Estimated potential effects of chronic wasting disease on the growth rate of the elk population in the Jackson elk herd differ with assumptions about hunting. Solid lines are the median of the marginal posterior distribution of lambda. Dashed lines give 95% equal-tailed credible intervals. The horizontal dotted line is the threshold between population increase and decline. **A**. Population growth rate assuming no hunting and recruitment estimated in this study. **B**. Effect of hunting on adult females survival is estimated as a random variable from hunting removals observed during the last five years, 2011-2015. This effect is included in survival of healthy and infected animals. Recruitment is again estimated in this study, just as in (A).

Model forecasts showed the potential for CWD to reduce the abundance of the Jackson elk herd (Figure 5) if transmission is similar to the level (0.08; BCI=0.05, 0.12) observed by Monello et al. (2014). Although there is significant overlap in the forecasts for an infected and uninfected population, it is clear that CWD has the potential to decrease the population size.

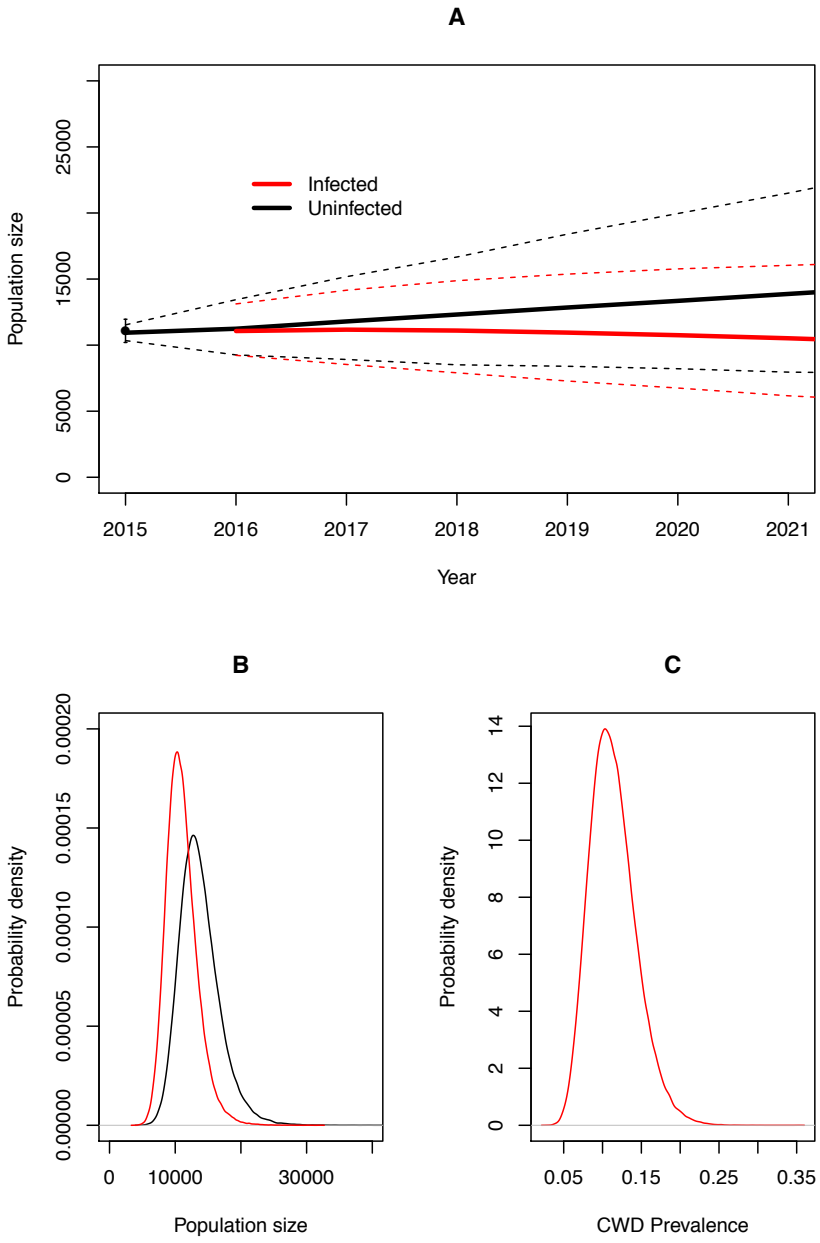

Figure 5: Forecast of the population size of the Jackson elk herd during 2016 - 2021 with and without CWD infection (upper panel) assuming time-invariant disease transmission similar to that observed in Rocky Mountain National Park (Monello et al., 2014). Solid lines are the median of the posterior predictive distribution. Dashed lines are 95% equal-tailed credible intervals. **B**. Posterior predictive distributions of healthy and infected populations in 2021 are shown. **C**. Posterior predictive distributions of CWD prevalence in the infected population in 2021 is shown.

Discussion

Forecasts suggest that effects on population abundance are likely to be slow to develop and difficult to detect; there is strong overlap between predictive process distributions for populations with and without CWD (Figure 5). The assumption of a constant transmission probability over time drives the observed forecasts. Time-invariant transmission is almost certainly not the case but represents a starting point until parameters in a dynamic model can be estimated from data. The NER may offer an unusual opportunity to do that given a long history of monitoring for CWD, with the arrival of CWD presenting a unique opportunity to understand the epidemic behavior of the disease over time. The Bayesian model in place would facilitate the development of the understanding of dynamic transmission probability after disease invasion by providing a way to assimilate data from multiple sources, including annual census data and data from prior studies in other locations. It can also be used annually to update the role of harvest and disease and provide managers with the probability of attaining population goals in the ensuing 1-3 years (Ketz et al., In review). With some modifications, it will allow for the comparision of multiple theoretical models of dynamic disease transmission (See Miller et al., 2006). The model makes clear that if CWD becomes established and increases, conditions that are more likely in higher density areas, hunting and the disease will compete and result in higher mortality than would be present in the absence of one or the other.

Although model forecasts showed prevalence of the disease increasing to an asymptote, we emphasize that this result depends on the assumed constant transmission probability. It is reasonable to expect the modeled transmission probability to be excessively high in the years immediately after the arrival of CWD, before both the environmental prion pool and infected animal contacts on the landscape increase, and excessively low in later years. Data from Rocky Mountain National Park (RMNP) which were used to estimate transmission probability were collected well after the initial CWD outbreak, giving time for disease establishment. The density of elk in RMNP in winter is substantially lower than that on the NER, and on feedgrounds specifically. We hypothesize that this higher density and the resulting concentrated environmental contamination would lead to elevated rates of disease transmission on the NER after the disease fully invades, increasing impacts on abundance.

Selection for genotypes with a longer incubation period for CWD that allows infected elk to

persist longer and continue reproducing could decrease the impact of the disease (Williams et al., 2014). The potential for selection of genotypes with a longer CWD incubation time were not incorporated into our model. However, we used survival data from infected cow elk that lived in a relatively high-density population that has been exposed to CWD for 30-50 years (Monello et al., 2014) and the effects of such selection would be incorporated in survival outcomes described here. This means that the modeling results in this paper are unlikely to be largely influenced by prion genotype selection in the next several decades, suggesting that CWD-related declines in population growth rate may be prolonged. This is consistent with work to date that suggested the effects of selection on prion genotypes will only take root after 75-100 years based on a "worst-case" scenario that used presumably higher transmission rates from a captive population confined to contaminated pens (Williams et al., 2014). Interestingly, this "worst-case" scenario leads to faster evolution and associated population recovery, whereas lower disease transmission delays potential beneficial evolution, suggesting that declines in population growth rate may be drawn out over a longer time-frame than described in Williams et al. (2014).

The model also does not currently consider the potential recruitment decrease for CWD-infected females. This effect would further exacerbate the decline in growth rate with increasing prevalence, (See Dulberger et al., 2010), which would in turn increase disease impacts on abundance. This is one area where observation after CWD invasion would provide valuable insight to clarify the impact of disease. Continued use of this model along with annual census and disease data will provide valuable information on the potential effects of CWD on evolutionary selection and recruitment.

The potential outcomes of CWD invasion into the Jackson elk herd are also complicated by the different migratory segments of the population and their differential recruitment (Cole et al., 2015). Disease may also act differentially in these groups, with the long-distance migrants perhaps having a slight advantage by spending more time over a larger area and thereby decreasing exposure to the most concentrated environmental prion reservoir and infected animal contacts. However, both segments of the population intermix on the winter grounds, which may negate such benefits. There also exists the possibility that the higher observed recruitment for short-distance migrants (Cole et al., 2015) may mitigate the disease impacts on population growth rate as long-distance migrants are lost. There are foreseen drawbacks to an increase in short-distance migrants in the population, however, including more vehicle collisions, more commingling with livestock and property damage,

and decreased hunting opportunity (See Cole et al., 2015).

It is important to note that the recruitment ratio reported by Cole et al. (2015) is from summer months, and is not directly comparable with that in our work. Data collected in the summer excludes calf mortality occurring between summer and the winter census (the ratio represented by our classification data and recruitment parameter).

The model framework presented here is a first step towards informing managers of the Jackson elk herd of the possible effects of CWD on the population. However, some caveats are needed. Our work depends fundamentally on prior research on CWD in elk in RMNP (Monello et al., 2014). CWD may have a larger impact over the long term than our results suggest because crowding of elk annually at feedgrounds will plausibly accelerate disease transmission, leading to a higher prevalence and reduced population growth rate than observed in RMNP. As disease impacts become more clear, newly available data can be incorporated into the existing model framework, which will enable adaptive management as it was originally conceived by Walters (1986).

Acknowledgements

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Appendix 1: Model Details

Overview

Here we describe in detail the mathematics and statistics used to model the emergence of chronic wasting disease (CWD) in the greater National Elk Refuge elk population. The overall structure follows Ketz et al. (In review); Hobbs et al. (2015) and Raiho et al. (2015). The purpose of the model is to examine the potential impacts of the disease on the population's dynamics, to aid in designing future sampling for CWD in the population, and to provide a proactive framework for assimilating new data on herd health. This framework can be used to guide adaptive management of the population (Walters, 1986).

Notation

Matrices will be notated in uppercase bold font, vectors in lowercase bold, and scalars in plain font. Greek letters will be used to represent parameters. We will notate data using the convention

$y^{\text{data type}}$. All other quantities are unobserved.

Deterministic model

State vector

The state of the population at year t is represented in a seven element vector ($\mathbf{n}_t$) representing four sex and age classes and two disease states (Table 3).

Table 3: Elements of column vector ($\mathbf{n}_t$) representing the state of the National Elk Refuge elk population.

State	Definition
$n_{1,t}$	Uninfected juveniles of both sexes aged six months on their first census
$n_{2,t}$	Uninfected yearling and adult females aged 18 months and older at census
$n_{3,t}$	Uninfected yearling males aged 18 months at census
$n_{4,t}$	Uninfected adult males aged 30 months and older at census
$n_{5,t}$	CWD infected yearling and adult females aged 18 months and older at census
$n_{6,t}$	CWD infected yearling males aged 18 months at census
$n_{7,t}$	CWD infected adult males aged 30 months and older at census

Note that there are no infected juveniles, which represents the assumption animals do not become infected with CWD before eight months of age. This assumption is consistent with studies of epidemiology of CWD in mule deer (Miller and Williams, 2003; Miller and Conner, 2005). We pool yearling and adult females into the same class because preliminary analyses revealed that the data do not allow us to separately identify these states.

Summary of model parameters

Model parameters are defined in Table 4.

Note the important assumption; the probability that an individual animal aged older than six months becomes infected (ψ) is represented as a time-invariant constant.

Representing CWD transmission

The probability that an individual animal aged older than six months becomes infected (ψ) is represented as a time-invariant constant based on capture-mark-recapture studies of elk infected with CWD in Rocky Mountain National Park (Monello et al., 2014). We explored the possibility of representing transmission using a density dependent formulation and obtained unreasonable results based on the available data. We recognize that initially transmission is likely to be lower while in the long-term it may be higher as more animals become infected (Miller et al., 2006). Our model will

Parameter	Definition
$\boldsymbol{\phi}$	Vector of natural survival probabilities indexed by $i = 1$ for juveniles surviving to become uninfected yearlings, $i = 2$ for juveniles surviving to become CWD infected yearlings, $i = 3$ for uninfected yearling and adult females, $i = 4$ for CWD infected yearling and adult females, $i = 5$ for uninfected yearling and adult males, and $i = 6$ for CWD infected yearling and adult males.
ρ	Number of calves produced per yearling and adult female alive at the birth pulse $(t - 1 + \frac{1}{4}\Delta t)$ that survive to their first census at year t.
α	Proportion of juveniles surviving to the yearling stage that are male
ψ	Probability that an uninfected animal becomes infected with CWD during $t - 1$ to t.

Table 4: Definitions of parameters in deterministic model of elk population dynamics on the National Elk Refuge.

provide a basis for selecting among alternative, dynamic transmission models if and when infection occurs in the population and monitoring data become available.

Probability of transmission controls the state change of animals from healthy to infected. We illustrate how state change occurs using adult males as an example. Recall that

$$\begin{aligned}
\phi_5 &= \text{survival probability of an uninfected yearling or adult male during } t-1 \text{ to } t;\\
\phi_6 &= \text{survival probability of a CWD infected yearling or adult male during } t-1 \text{ to } t;\\
\psi &= \text{probability that an uninfected yearling or adult male becomes infected;}\\
n_{3,t} &= \text{number of yearling males at time } t;\\
n_{4,t} &= \text{number of uninfected adult males at time } t;\\
n_{6,t} &= \text{number of CWD infected yearling males at time } t;\\
n_{7,t} &= \text{number of CWD infected adult males at time } t.
\end{aligned}$$

The parameter ϕ_6 reflects mortality from CWD; ϕ_5 does not. Assume for expository purposes that all CWD transmission occurs in a pulse at time point $t + q_{tr}\Delta t$, such that q_{tr} represents the proportion of the time step that has elapsed before CWD is transmitted. This means that

$$n_{4,t} = (1-\psi)\phi_5(n_{3,t-1} + n_{4,t-1}), \tag{1}$$

$$n_{7,t} = \phi_5^{q_{tr}}\psi\phi_6^{1-q_{tr}}(n_{3,t-1} + n_{4,t-1}) + \phi_6(n_{6,t} + n_{7,t-1}). \tag{2}$$

Thus, all transmission occurs immediately before census if $q_{tr} = 1$ and all transmission occurs immediately after census if $q_{tr} = 0$. We set $q_{tr} = \frac{1}{2}$ to represent the average time of transmission for a process that occurs continuously throughout the year.

Recruitment and survival to the yearling stage

We assume that females breed for the first time as yearlings, such that the juveniles at time $t-1$ do not contribute to recruitment at time t. We define ρ as the number of offspring produced per adult yearling and female alive at the birth pulse that survive to their first census in the absence of harvest. Thus, the parameter ρ is the product of the proportion of females surviving to the birth pulse that successfully carry pregnancies to term and the probability of natural survival of neonates to their first census. Thus, the number of juveniles at time t is

$$n_{1,t} = \rho \left(n_{2,t-1}\phi_3^{\frac{1}{4}} + n_{5,t-1}\phi_4^{\frac{1}{4}} \right). \tag{3}$$

We include terms for adult female survival uninfluenced by harvest ($\phi_3^{\frac{1}{4}},\ \phi_4^{\frac{1}{4}}$) because females must survive the first one quarter of the year (the interval between census and the birth pulse) before they give birth (Noon and Sauer, 1992). This formulation assumes that CWD has no influence on recruitment other than its effects on adult survival (equation 3). We realize that yearling elk females rarely breed and that it would be desirable to differentiate between yearlings and adult females in the model to allow ρ to apply solely to the breeding age class. However, preliminary analyses demonstrated that the harvest and classification data do allow these states to be separately identifiable. Thus, it is important to recognize that our estimate of ρ is somewhat reduced than it would be if applied to breeding females alone.

We define ϕ_1 as the probability that a juvenile survives from its first to second census (i.e., age 6 months to 18 months) and α as the proportion of males in the surviving yearlings. Thus, yearling and adult females in uninfected and CWD infected states are calculated as

$$n_{2,t} = (1-\alpha)\phi_1(1-\psi)n_{1,t-1} + \phi_3 n_{2,t-1}, \tag{4}$$

$$n_{5,t} = (1-\alpha)\phi_1^{\frac{1}{2}}\psi\phi_2^{\frac{1}{2}}n_{1,t-1} + \phi_4 n_{5,t-1}, \tag{5}$$

and yearling males as

$$n_{3,t} = \alpha\phi_1(1-\psi)n_{1,t-1}, \quad (6)$$

$$n_{6,t} = \alpha\phi_1^{\frac{1}{2}}\psi\phi_2^{\frac{1}{2}}n_{1,t-1}. \quad (7)$$

Projection matrix

The state of the population in the absence of harvest can be updated using the deterministic model

$$\mathbf{n}_t = \mathbf{A}\mathbf{n}_{t-1} \quad (8)$$

where $\mathbf{A}$ is a nine $\times$ nine projection matrix (Table 5)(Caswell, 1988). If all harvest occurs immediately before census, then

$$\mathbf{n}_t = \mathbf{A}\mathbf{n}_{t-1} - \mathbf{g}_t, \quad (9)$$

where $\mathbf{g}_t$ is a seven element column vector specifying the number of animals harvested from each state during $t-1$ to t. If all harvest occurs immediately after census, then the state of the population in year t is given by

$$\mathbf{n}_t = \mathbf{A}(\mathbf{n}_{t-1} - \mathbf{g}_t). \quad (10)$$

However, assume that harvest occurs at $t + q_h\Delta t$ where q_h is the proportion of the year elapsed before harvest occurs ($0 < q_h < 1$). In this case, survival terms in the matrix $\mathbf{A}$ must reflect annually varying harvest if q_h is not relatively close to 0 or 1 (e.g., Hobbs et al., 2015). This modification would require that we express recruitment as the product of parameters for the number of offspring produced per female at the birth pulse and the survival probability of offspring from the birth pulse to the first census. These parameters can trade-off to produce the same value for recruitment, which virtually assures that they would not be identifiable given the data we have available for this study. The relatively brief interval between harvest (which occurs during November - December) and census (which occurs in February) does not justify a more complex formulation for recruitment. Thus, we use equation 9 to represent effects of harvest on population dynamics.

Table 5: Non-zero elements of the projection matrix **A**.

Transition during $t-1$ to t	Element	Value
Offspring produced per uninfected yearling and adult female	$a_{1,2}$	$\phi_3^{\frac{1}{4}}\rho$
Offspring produced per CWD infected yearling adult female	$a_{1,5}$	$\phi_4^{\frac{1}{4}}\rho$
Uninfected juveniles to uninfected yearling females	$a_{2,1}$	$(1-\alpha)\phi_1(1-\psi)$
Uninfected juveniles to uninfected yearling males	$a_{3,1}$	$\alpha\phi_1(1-\psi)$
Uninfected juveniles to CWD infected yearling females	$a_{5,1}$	$(1-\alpha)\phi_1^{\frac{1}{2}}\psi\phi_2^{\frac{1}{2}}$
Uninfected juveniles to CWD infected yearling males	$a_{6,1}$	$\alpha\phi_1^{\frac{1}{2}}\psi\phi_2^{\frac{1}{2}}$
Uninfected yearling and adult females to uninfected adult females	$a_{2,2}$	$\phi_3(1-\psi)$
Uninfected yearling and adult females to CWD infected adult females	$a_{5,2}$	$\phi_3^{\frac{1}{2}}\psi\phi_4^{\frac{1}{2}}$
Uninfected yearling males to uninfected adult males	$a_{4,3}$	$\phi_5(1-\psi)$
Uninfected yearling males to CWD infected adult males	$a_{7,3}$	$\phi_5^{\frac{1}{2}}\psi\phi_6^{\frac{1}{2}}$
Uninfected adult males to uninfected adult males	$a_{4,4}$	$\phi_5(1-\psi)$
Uninfected adult males to CWD infected adult males	$a_{7,4}$	$\phi_5^{\frac{1}{2}}\psi\phi_6^{\frac{1}{2}}$
CWD infected yearling and adult females to CWD infected adult females	$a_{5,5}$	ϕ_4
CWD infected yearling males to CWD infected adult males	$a_{7,6}$	ϕ_6
CWD infected adult males to CWD infected adult males	$a_{7,7}$	ϕ_6

Stochastic model

Process model

The deterministic model described thus far assumes that equation 9 is able to represent changes in the state of the population perfectly. This is to say that the model can account for all of the dynamics of the population based on annually varying harvest and time-invariant survival, recruitment, and time-invariant CWD transmission. However, there are many influences on the population's dynamics that are not included in equation 9, notable among them predation, other diseases, weather, poaching, density dependence, and non-linearities of CWD transmission producing a time-varying probability of transmission.

We represent these stochastically by including influences not represented in the deterministic model in a seven $\times$ seven variance-covariance matrix $\mathbf{\Sigma}_p$. This matrix contains four terms for process variance on the diagonal and zeros elsewhere. We estimate separate process variances for healthy juveniles, yearling and adult females, and yearling and adult males as well as for infected adults. We assume that these process variances are uncorrelated to reduce the number of estimated

parameters.

We can now represent the unobserved, true state of the population as a probability distribution reflecting the uncertainty that arises from influences on the true state that are not represented in the deterministic model using

$$\log(\mathbf{n}_t) \sim \text{multivariate normal}(\log(\mathbf{A}\mathbf{n}_{t-1} - \mathbf{g}_t), \mathbf{\Sigma}_p). \tag{11}$$

Equation 11 represents the true state of the population at time t as a nine element vector of continuous and non-negative, lognormally distributed random variables with medians $\mathbf{A}\mathbf{n}_{t-1} - \mathbf{g}_t$ and variances $\boldsymbol{\sigma}_p^2 = (\sigma^2_{\text{p.juvenile}}, \sigma^2_{\text{p.female}}, \sigma^2_{\text{p.male}}, \sigma^2_{\text{p.CWD}})$.

Prior distributions

Prior distributions, also known as parameter models, express current knowledge about parameters uninformed by new data. Informative parameter models improve estimation of unobserved quantities, aid in identifiability, and speed convergence of computational algorithms (Hobbs and Hooten, 2015). Prior distributions of parameters are summarized in Table 6.

We derived prior distributions for survival probabilities for uninfected animals as follows. Meta-analysis results of Raithel et al. (2007) parameterize priors for natural survival of juveniles. The age categories in Raithel et al. (2007) did not correspond directly to the age categories in the state vector because juveniles in Raithel et al. (2007) were assumed to be born immediately before census, where juveniles in our model are born six months after census. Therefore we use a mixture model of juvenile and yearling survival from Raithel et al. (2007) for a prior on survival appropriate for our juvenile age class.

We used the meta-analysis of Brodie et al. (2013) to form priors for natural survival of yearling and adult females. The prior for yearling and adult females includes effects of predation by wolves and puma. There are far fewer data on natural survival of yearling and adult males. We used the mean for corresponding female age classes and doubled the standard deviation to reflect greater uncertainty about males and to allow a greater influence of the data in estimating male survival.

Recruitment was informed by the mean estimate from Raithel et al. (2007) with standard deviation set equal to 0.2 to make this prior weakly informative and allow the data to drive the posterior.

A prior distribution for survival of CWD infected yearling and adult females was based on the mark-capture-recapture studies of females by Monello et al. (2014) in Rocky Mountain National Park, Colorado. We assumed that survival of CWD infected yearling and adult males did not differ substantially from the values for females. Priors for survival of infected juveniles were derived as follows. We estimated an offset in survival resulting from CWD infection by sampling from the distributions of infected and uninfected survival probabilities of adult females and calculating a ratio of infected survival probability relative to uninfected survival probability. We then used this ratio to adjust the prior natural survival for juveniles obtained from Raithel et al. (2007).

Probability of transmission ψ is based on Monello et al. (2014) for females in Rocky Mountain National Park.

We increased the dispersion of beta prior distributions for survival and recruitment by dividing parameters by two to allow the data to have greater influence on their posterior distributions.

Data and initial conditions

Harvest data

We use data from the WGFD Jackson Herd Unit to estimate animals harvested from each demographic state. We define a vector $\mathbf{y}_{t-1}^{\text{harvest}}$ as a vector of mean number of animals harvested[2] during the interval $t-1$ to t in four categories: juveniles ($y_{1,t-1}^{\text{harvest}}$, calves), yearling + adult females ($y_{2,t-1}^{\text{harvest}}$), yearling males ($y_{3,t-1}^{\text{harvest}}$, spikes), and adult males ($y_{4,t-1}^{\text{harvest}}$, bulls). We assume that animals harvested in November are classified in the same way they would be classified during the following February. Model fitting treated harvest data as measured without error.

Seven stages are modeled (Table 3), which means that modeled stages are subsets of the four categories of harvest observations. For example, there is a single observation for harvest of yearling and adult females (one category), but we model uninfected and CWD infected yearling and adult females (two states). This means we must decompose the harvest data to assign the number of animals removed to appropriate states. To do so, we assume that animals within a larger category, for example adult + yearling females in both disease states, are harvested in proportion to their numbers in the population at the time of harvest.

This assumption is represented by defining a seven element vector $\mathbf{g}_t$ containing the number of

[2]Estimated from phone surveys of hunters.

Table 6: Prior distributions of model parameters.

Parameter	Definition	Distribution[1]	Mean	Std. dev.	Source / notes
ϕ_1	Natural survival probability of juveniles surviving to become uninfected yearlings	beta(13.1, 8.12)	.616	10.3	Raithel et al. (2007)
ϕ_2	Natural survival probability of juveniles surviving to become CWD infected yearlings	beta(2.00, 7.76)	.363	.095	Derived from Raithel et al. (2007)and Monello et al. (2014)
ϕ_3	Natural survival probability of uninfected yearling and adult females	beta(1598, 112)	.934	.006	Brodie et al. (2013)
ϕ_4	Natural survival probability of CWD infected yearling and adult females	beta(10.7, 8.7)	.55	.11	Monello et al. (2014)
ϕ_5	Natural survival probability of uninfected yearling and adult males	beta(398, 28.2)	.934	.012	Brodie et al. (2013)(doubled standard deviation)
ϕ_6	Natural survival probability of CWD infected juvenile and adult males	beta(10.7, 8.7)	.55	.11	Monello et al. (2014)
ρ	Proportion of adult females producing a calf that survives to its first census	beta(2.42, 2.80)	.464	.2	Raithel et al. (2007) with standard deviation = .2 to make weakly informative
α	Proportion of juveniles surviving to be yearling males	beta(49.5,49.5)	.5	.05	Parameters provide weakly informative distribution centered on .5.
ψ	Probability of transmission	beta(18.1,208)	.08	.018	Monello et al. (2014)

[1]Parameters of beta distribution were derived by moment matching (Hobbs and Hooten, 2015) means and standard deviations reported in the literature. The tabulated values for all parameters except ψ were divided by two to allow greater influence of data on posterior distributions.

animals harvested from all states at time $t-1+q_h\Delta t$. We calculate the elements of $\mathbf{g}_t$ as follows. We first approximate the number of animals in each age class that would be alive at the time of harvest, $\boldsymbol{\mu}_t$ using

$$\boldsymbol{\mu}_t = \mathbf{A}\mathbf{n}_{t-1}. \tag{12}$$

We notate the total number of females (adults and yearlings) alive at harvest as

$$f_t = \mu_{2,t} + \mu_{5,t}. \tag{13}$$

Similarly, we notate the number of yearling males (spikes) alive at harvest as

$$s_t = \mu_{3,t} + \mu_{6,t}, \tag{14}$$

and the number of adult males (bulls) as

$$b_t = \mu_{4,t} + \mu_{7,t}. \tag{15}$$

We approximate the elements of $\mathbf{g}_t$ using

$$\begin{aligned}
\text{juveniles, } g_{1,t} &= h_{1,t-1}, && (16)\\
\text{uninfected yearling and adult females, } g_{2,t} &= h_{2,t-1}\frac{\mu_{2,t}}{f_t}, && (17)\\
\text{uninfected yearling males, } g_{3,t} &= h_{3,t-1}\frac{\mu_{3,t}}{s_t}, && (18)\\
\text{uninfected adult males, } g_{4,t} &= h_{4,t-1}\frac{\mu_{4,t}}{b_t}, && (19)\\
\text{CWD infected yearling and females, } g_{5,t} &= h_{2,t-1}\frac{\mu_{5,t}}{f_t}, && (20)\\
\text{CWD infected yearling males, } g_{6,t} &= h_{3,t-1}\frac{\mu_{6,t}}{s_t}, && (21)\\
\text{CWD infected adult males, } g_{7,t} &= h_{4,t-1}\frac{\mu_{7,t}}{b_t}. && (22)
\end{aligned}$$

The numerator in the fraction on the right hand side of these equations is the number of individuals in state i alive at the time of harvest and the denominator is the total number of individuals in harvest class j containing state i that are alive at harvest. Multiplying this proportion times the number of animals harvested in class j approximates the number of animals harvested from state

i. Approximating the vector $\mathbf{g}_t$ allows us to update the state of the population reflecting effects of natural mortality and harvest (Equation 11).

We assume that vulnerability to harvest is the same for CWD infected and uninfected animals, which means that we may underestimate the effect of harvest on CWD infected animals. CWD infected mule deer are more vulnerable to vehicle collisions (Krumm et al., 2005) and to predation by mountain lions (Krumm et al., 2010) than uninfected mule deer, suggesting that infected animals would plausibly be more vulnerable to hunters (also see Conner et al., 2000).

Census data

We approximate the posterior distributions of unknown quantities conditional on three sources of data, annual census (y_t^{count}), sex and age classification ($\mathbf{y}_t^{\text{class}}$) and testing for CWD ($\mathbf{y}_t^{\text{CWD}}$). For notational convenience, we define $\eta_t = \sum_{i=1}^{7} n_{i,t}$. Thus, η_t is the true, unobserved total population size[3].

We estimate observation uncertainty in the total count by dividing the annual census into two parts, animals counted off the Refuge (y_t^{off}) and animals counted on the Refuge (y_t^{on}). We assume that the counts of animals on the refuge were made without error. We include observation uncertainty in the off refuge counts using results of the sightability model of Lubow and Smith (2004) who found that 67.3% (se = 14%) of animals present in an area sampled off the Refuge were counted. For each year, the number of true number of animals on and off the refuge (y_t) is modeled as

$$p^{\text{sight}} \sim \text{beta}(m(0.673, 0.14)) \tag{23}$$

$$y_t^{\text{count}} = y_t^{\text{on}} + \frac{y_t^{\text{off}}}{p^{\text{sight}}} \tag{24}$$

where $m()$ is the function that matches the moments of the beta distribution to its parameters. The standard deviation of the annual, total count ($y_t^{\text{count.sd}}$) is estimated by applying an MCMC algorithm to equations 23 and estimating the standard deviation of the converged output for y_t^{count}.

The probability of observing the annual count y_t^{count} conditional on η_t is modeled as

$$y_t^{\text{count}} \sim \text{normal}\left(\eta_t, y_t^{\text{count.sd}}\right). \tag{25}$$

[3]Strictly speaking, η_t is the mean of the data distribution, equation 25.

We justify using a normal distribution for the likelihood because corrected counts are a continuous random variable and because they are sums of several many samples, which by the central limit theorem, distribute normally as the number of samples becomes large.

Classification data

The classification data include counts of individuals in four categories, juveniles (y_1^{class}, calves), yearling males (y_2^{class}, spikes), yearling and adult females (y_3^{class}) and adult males (y_4^{class}, bulls). The probability of observing the annual classification data is modeled using

$$\mathbf{y}_t^{\text{class}} \sim \text{multinomial}\left(\sum_{i=1}^{4} y_{i,t}^{\text{class}}, \left(\frac{n_{1,t}}{\eta_t}, \frac{n_{3,t}+n_{6,t}}{\eta_t}, \frac{n_{2,t}+n_{5,t}}{\eta_t}, \frac{n_{4,t}+n_{7,t}}{\eta_t}\right)'\right) \tag{26}$$

The first term in the multinomial distribution (equation 26) is the number of individuals classified during year t. The second term (within the parentheses followed by ') is a four element vector of the true, unobserved proportions of juveniles, yearling males, yearling and adult females, and adult males in the population. We will explore using hunt areas as replicates to estimate sampling variance in estimates of population sex and age structure.

Prevalence data

We use data on test results for CWD as a likelihood for the model's prediction of prevalence

$$y_t^{\text{CWD}} \sim \text{binomial}\left(J_t^{\text{CWD}}, \frac{n_{5,t}+n_{6,t}+n_{7,t}}{\eta_t}\right), \tag{27}$$

where y_t^{CWD} is the number of positive tests, J_t^{CWD} is the total number of tests during year t.

Initial values for states

We treat initial conditions as priors informed by census and classification data. The initial, total population size is treated as a Poisson random variable with mean y_1^{count}, the elk count during 2000:

$$\eta_1 \sim \text{Poisson}(y_1^{count}). \tag{28}$$

We assume all infected states (n_5, n_6, n_7) are zero in year 2000. Therefore, we need initial conditions for the non-infected states (n_1, n_2, n_3, n_4). We use the sex and age classifications ($\mathbf{y}_1^{\text{class}}$)

observed during 2000 to partition the total population into the appropriate states. For notational convenience, define $\mathbf{w}_1 = \mathbf{y}_1^{\text{class}}$. Let

$$\text{juveniles, } z_1 = w_{1,1} + 1, \tag{29}$$

$$\text{yearling and adult females, } z_2 = w_{2,1} + 1, \tag{30}$$

$$\text{yearling males, } z_3 = w_{3,1} + 1, \tag{31}$$

$$\text{adult males, } z_4 = w_{4,1} + 1. \tag{32}$$

We then define the random variable $\mathbf{k}$, a vector specifying the initial composition of the population

$$\mathbf{k} \sim \text{Dirichlet}(\mathbf{z}), \tag{33}$$

$$n_{\forall i \in \{1...4\}} = k_i \eta_1, \tag{34}$$

$$n_{\forall i \in \{5...7\}} = 0. \tag{35}$$

Posterior and joint distributions

Marginal posterior distributions of unobserved states and parameters conditional on the observed annual census, classification and harvest data are approximated using

$$\begin{aligned}
\left[\mathbf{N}, \boldsymbol{\phi}, \rho, \alpha, \beta, \boldsymbol{\sigma}_p^2 | \mathbf{y}^{\text{count}}, \mathbf{y}^{\text{harvest}}, Y^{\text{class}}\right] \quad \propto \quad & \prod_{t=2}^{T} \Bigg(\left[y_t^{\text{count}} \,\middle|\, \eta_t, y_t^{\text{count.sd}} \right] && (36) \\
\times \quad & \left[y_t^{\text{class}} \,\middle|\, \sum_{i=1}^{4} y_{i,t}^{\text{class}}, \right. \\
& \left. \left(\frac{n_{1,t}}{\eta_t}, \frac{n_{3,t} + n_{6,t}}{\eta_t}, \frac{n_{2,t} + n_{5,t}}{\eta_t}, \frac{n_{4,t} + n_{7,t}}{\eta_t} \right)' \right] \Bigg) \\
\times \quad & \left[y_t^{\text{CWD}} \,\middle|\, J_t^{\text{CWD}}, \frac{n_{5,t} + n_{6,t} + n_{7,t}}{\eta_t} \right] && (37) \\
\times \quad & \prod_{t=2,}^{T+f} \left[\log\left(\mathbf{n}_t\right) | \log(\mathbf{A}\mathbf{n}_{t-1}), \boldsymbol{\Sigma}_p \right] \\
\times \quad & [\boldsymbol{\phi}][\rho][\alpha][\beta][\boldsymbol{\sigma}_p^2][\mathbf{n}_1]
\end{aligned}$$

The notation $[a|b, c]$ reads the probability distribution of the random variable a conditional on b and c. Specific probability distributions needed for equation 36 are given in the text above (equations 11, 25, 26, 27 and Table 6). The quantity T is the total number of sequential years with data and

f gives the number of years of forecasts[4]. Note that the quantity $\mathbf{N}$ is a $7 \times (T + f)$ matrix of random variables where rows contain values for the true states at each of the $T + f$ years.

Model checking

We test for lack of fit using posterior predictive checks (Hooten and Hobbs, 2015; Gelman and Hill, 2009). We simulate a new dataset for total counts and population composition at each iteration in the MCMC chain. We then calculate test statistics τ based on the simulated and the real data,

$$\tau_{\text{data}} = \sum_{i=1}^{n} \left(y_i^{\text{predicted}} - y_i^{\text{real data}} \right)^2 \tag{38}$$

$$\tau_{\text{simulated}} = \sum_{i=1}^{n} \left(y_i^{\text{predicted}} - y_i^{\text{simulated data}} \right)^2. \tag{39}$$

Lack of fit was evaluated using a Bayesian P value P_B computed as

$$P_B = \Pr(\tau_{\text{simulated}} \geq \tau_{\text{data}}) \tag{40}$$

for each of the datasets used to fit the model. Values of P_B close to 0 or to 1 indicate lack of fit.

Analysis

Effects of CWD on population growth rate

We use the approach of Monello et al. (2014) to approximate the potential impact of CWD on the growth rate of the greater National Elk Refuge elk population. We created a females-only projection matrix, $\mathbf{F}$:

$$\mathbf{F} = \begin{pmatrix} 0 & (\phi_3^{\frac{1}{2}}(1-p) + \phi_4^{\frac{1}{2}}p)(1-\alpha)\rho \\ \phi_1(1-p) + \phi_2 p & \phi_3(1-p) + \phi_4 p \end{pmatrix} \tag{41}$$

where p is the proportion of the population that is infected with CWD and other parameters are as defined previously (Table 4). We vary levels of prevalence from 0 to .3 in steps of .05 and approximated the marginal posterior distribution of the ergodic population growth rate λ for each step. We calculated the dominant eigenvalue λ^k for each iteration k of the MCMC output (combined over chains) using the four survival probabilities $\phi^k_{\forall i \in 1..4}$, α^k, and ρ^k as input to the

[4]The quantity f is often called the forecast horizon.

`lambda()`function of the `popbio` package in R. Each resulting value of λ^k was stored in a vector for each level of prevalence. We use those vectors for inference (Hobbs and Hooten, 2015).

We estimated effects of CWD on population growth rate (λ) including and excluding effects of hunting. We included effects of hunting by estimating the proportion of yearling adult females alive at time t that were harvested during t to $t+1$ and adjusting annual yearling and female survival to reflect this proportional source of mortality in the projection matrix used to approximate population growth rate.

Forecasting

We examine the effects of CWD by comparing predictive process distributions, i.e. forecasts, (Hobbs et al., 2015; Raiho et al., 2015; Ketz et al., In review), ten years into the future (2016-2025) using the process model (equation 8), illustrated here for the total population size. Given T years with data, the predictive process distribution of the total population size for time $T+1$ (η_{T+1}) is defined as

$$\int_{\theta_1 \ldots} \int_{\theta_P} \int_{\eta_1 \ldots} \int_{\eta_T} [\eta_{T+1} | \eta_T, \boldsymbol{\theta}][\eta_1 .... \eta_T, \theta_1 ... \theta_p | \mathbf{Y}_1^{\text{all}} ... \mathbf{Y}_t^{\text{all}}] d\theta_1 ... d\theta_p, d\eta_1 ... d\eta_t \tag{42}$$

where $\boldsymbol{\theta}$ is a vector containing the P parameters in the process model (equation 11) and $\mathbf{Y}^{\text{all}}$ is the full monitoring dataset including classification, census, harvest, and CWD testing. Predictive process distributions reflect the inherent uncertainty in the model (process variance) as well as uncertainty in all model parameters, initial conditions, and future, latent states. This equation can be expanded for any time point beyond T, $T+1 .... T_f$ where f is the forecast horizon, the number of years beyond the data for which we compute posterior process distributions. We approximate this integral using Monte Carlo integration.

We compare predictive process distributions for CWD-free and CWD-infected populations five years into the future. We first assume initial conditions in 2016 of 0 infected animals and a transmission probability equal to 0 to represent CWD-free dynamics. We compare the resulting posterior process distributions with those obtained by assuming a probability of transmission as defined in the model description. This results in a posterior distribution which considers both the prior (parameter model) equivalent to the transmission probability observed in the elk population of Rocky Mountain National Park (Monello et al., 2014) as well as the CWD test results included in the data, y_t^{CWD} and J_t^{CWD} . This transmission probability informs the dynamics of the population infected

with CWD.

We treat future harvest as a normally distributed random variable with mean equal to the average harvest for the last five years in each age class and corresponding standard deviation. This treatment reflects uncertainty in the ability to implement future harvest objectives.

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