Laying Deer to Waste

Project Module Associated with
2nd Edition, Introduction to Computational Science:
Modeling and Simulation **by**

Angela B. Shiflet and George W. Shiflet
Wofford College

Agent-Based Modeling Techniques, such as in Chapter 11

Introduction

A disease similar to Mad Cow Disease (*bovine spongiform encephalopathy*) is decimating some wild populations of deer and other related animals, like elk and caribou. Chronic Wasting Disease (CWD) is a slowly advancing disease, where the signs and symptoms may not appear for years. In advanced stages of CWD, infected brains have deteriorated so that the animals begin to drool and stagger (Figure 1). Listless and emaciated, they will soon die. What is the cause of this horrific disease?

Figure 1 Deer showing signs of chronic wasting disease (Kreeger 2000)

We usually worry about diseases caused by bacteria, viruses, fungi, etc., but now there is an even more insidious health threat to concern us. Mad Cow Disease and Chronic Wasting Disease are both caused by proteinaceous agents called **prions** (proteinaceous infectious particles). These proteins (PrP^{cwd}) are characterized as abnormally shaped versions of a normal neuronal protein (PrP^{c}), which can induce the normal protein into assuming an abnormal shape. The abnormal proteins then form clumps, which disrupt normal functioning of neurons and promote neuronal loss, particularly in the brain. Animals acquire such a disease primarily by ingestion of the particles, which may be found in body fluids or remains of dead animals; and the particles may persist for years in soils. Because cooking does not destroy the prions, hunters that kill and eat the meat from such animals are at risk of contracting the disease.

Without a vaccine or other preventative tool, scientists are searching for some effective countermeasure to fight the disease's spread. Infected herds of deer around Yellowstone National Park could potentially spread the disease to the park's herds of elk and deer. Furthermore, there is an unknown risk of its spread to other wildlife, as well as human populations (Osterholm et al. 2019). Concerned wildlife scientists have desperately sought possible solutions to the advancing problem (Gillin and Mawdsley 2018).

From an extensive survey of literature that report results from field and experimental studies, often associated with mathematical and computational analysis, four intervention/control strategies were examined (Uehlinger et al. 2016):

1. selective/preferential removal of infected deer, through consumption by large carnivores, or through increased harvest of males (higher prevalence), or through testing and culling
2. nonselective population reduction (hunting, intensive agency culling, harvesting permits)
3. change of season harvest (summer hunting)
4. vaccination

Each of these or a combination of methods may prove to be useful, but one of the approaches has intrigued some researchers–predators. The idea is to use predators to remove those animals that are infected and weakened, which could reduce prevalence and improve the overall health of the herd. So far, the results are mixed for this practice, and introduction of predators may meet with opposition from local human populations. Still, predation may be one tool that can help decrease the spread of a horrible disease (Krumm et al. 2010, Team 2020, Wild et al. 2011).

Overview of Model

Gross and Miller (2001) developed an individual-based (agent-based) model for the spread of chronic wasting disease (CWD) in mule deer, employing data from northeastern Colorado and elsewhere, and used the model to examine strategies for limiting the spread of the disease. The projects below are based on their research and approach.

The user should be able to specify the length of the simulation. The simulation usually advances for 100 simulated years, using a one-year time step. A year is divided into two periods, summer-fall (about May to October) and winter-spring (about November to April). The simulation starts at the beginning of the winter, when animals are about 0.5, 1.5, 2.5, … years old.

An agent (i.e., mule deer) can be susceptible to the disease; latent, in which the animal has been infected but cannot spread the disease yet; or infectious, in which the disease can spread from the sick animal. The state of an agent includes the following parameters: sex; disease-state (susceptible, latent, or infectious); latent-period, or the number of six-month periods an animal has been in the latent stage of the disease; and infectious-period, or the number of six-month periods a deer has been infectious with CWD. The disease progresses by 6-month periods with probabilities as indicated in Table 1.

Table 1. Transition probabilities for 6-month disease periods

From	To	Probability
Latent 1	Latent 2	1.0
Latent 2	Latent 3	0.9
Latent 2	Infectious 1	0.1
Latent 3	Latent 4	0.1
Latent 3	Infectious 1	0.9
Latent 4	Infectious 1	1.0
Infectious 1	Infectious 2	0.9
Infectious 1	Dead	0.1
Infectious 2	Infectious 3	0.05
Infectious 2 (winter)	Dead	0.95
Infectious 2 (summer)	Dead	0.47
Infectious 3 (winter)	Dead	1.0
Infectious 3 (summer)	Dead	0.5

A female becomes sexually mature at about 1.5 years-of-age. For simplification, assume all sexually mature females become pregnant. Also, at the end of first spring of sexual maturity, a female gives birth to one fawn; whereas, afterwards, she has two offspring. About half the offspring are male and half female. If a mother is infectious, there is a 5% chance a fawn will be born latent infected.

The probability of a fawn surviving the first year of life is 0.312. For a female of age 1 yr to 11 yr, the chance of survival from one year to the next is 0.85. However, from 11 yr to 16 yr, the yearly survival probability decreases linearly from 0.85 to 0. For males 1-10 years of age, this chance of survival for deer is 0.85; but for ages 10-12 yr, the probability decreases linearly from 0.85 to 0.

The model should allow the user to specify whether winter harvesting (sacrificing) of specified fractions of all animals is to occur or not when the population is greater than 500 animals. With harvesting, the population density is reduced, so that the population is not allowed to grow exponentially. Table 2 presents the harvest levels (fractions) when the population densities are normal (501-1000 deer) or high (over 1000 animals).

Table 2. Harvest levels when the population is greater than 500 deer at the beginning of winter

		501-1000 Deer	**> 1000 Deer**
Sex	**Age Category**	**Harvest Level**	**Harvest Level**
Female	Fawn	0.01	0.03
	Yearling	0.02	0.05

	Adult	0.07	0.10
Male	Fawn	0.02	0.03
	Yearling	0.06	0.09
	Adult	0.20	0.27

Gross and Miller (2001) considered two CWD-management strategies, early and infectious culling. In both cases, testing for the disease and culling occurs after CWD prevalence reaches some designated threshold. With early culling, rangers destroy tested animals that have been latent for one or more periods; while with infectious culling, rangers sacrifice infectious deer. Because of the difficulty in testing every animal and in test accuracy, the model also specifies a program-efficacy fraction, which is the probability that an appropriate diseased animal will be detected and eliminated. At the time of the paper, scientists were still experimenting with non-destructive means for early detection, so the authors envisioned that their model could be used as a predictor of the effectiveness of such approaches when appropriate testing was available.

After initialization of the agents and the parameters in the agent-based simulation, the following steps are repeated for 100 years (time steps):

For winter-spring:
- census count of deer
- infect
- move
- half-year disease progress
- become half-year older

For summer-fall:
- reproduce
- update CWD prevalence
- if harvesting and census-count > 500, then harvest
- infect
- move
- half-year disease progress
- test and slaughter
- natural mortality
- become half-year older
- advance clock by one year
- update population rate

Other Parameters and Initialization

The user should specify the initial numbers of female and male deer. Gross and Miller (2001) initialized their simulations with 592 females and 408 males, for a total of 1000 deer, to reflect post-harvest proportions, with the greater harvesting of males than females. These agents should be placed in random locations and move at random to adjacent cells throughout the simulation.

Determined by simulation, Table 3 lists cumulative probabilities that a female will be at the indicated age or younger when the simulation begins; while Table 4 displays the initial cumulative probabilities for males. For example, initially about 21.0% (fraction 0.21022558) of the females will be fawns of age 0.5 years, and about 37.8% (fraction 0.37805661) of the females will be fawns of age 0.5 years or yearlings of age 1.5 years. Thus, about 16.8% (0.37805661 - 0.21022558 = 0.16783103) of the females will be 1.5-years old.

Table 3. Cumulative probabilities that a female will be at the indicated age or younger when the simulation begins

Age	Cumulative Probability
0.5	0.21022558
1.5	0.37805661
2.5	0.51300385
3.5	0.62057494
4.5	0.70705341
5.5	0.7764097
6.5	0.83180883
7.5	0.87607919
8.5	0.91178266
9.5	0.93968734
10.5	0.96239207
11.5	0.98059691
12.5	0.99227922
13.5	0.99795252
14.5	0.99967141
15.5	1

Table 4. Cumulative probabilities that a male will be at the indicated age or younger when the simulation begins

Age	Cumulative Probability
0.5	0.21669247
1.5	0.38978931
2.5	0.52852135
3.5	0.63977397
4.5	0.72835782
5.5	0.80011554
6.5	0.85682727
7.5	0.90226187
8.5	0.93859574

9.5	0.96751988
10.5	0.99055054
11.5	1

Users should be able to specify the initial numbers of latent females and infectious females, each of age 2.5 years. Typical values employed by Gross and Miller (2001) were 4 each. In the simulation projects, disease spreads from an infectious agent to a susceptible one in the same or an adjacent cell with a designated probability and with a default value of 0.57.

For early or infectious culling, the user should be able to specify a CWD threshold (typically 0.01, 0.05, or 0.10), after which culling is to occur.

Output

Simulation graphs typically should include the following:

- A plot of the number of mule deer in each major disease category (susceptible, latent, infectious) as well as the total number of animals
- A plot of the annual population rate and mean of all annual population rates
- Optionally, histograms of female ages and of male ages

Displayed current results typically should include the following:

- Year
- CWD prevalence
- Total number of susceptible deer
- Total number of latent deer
- Total number of infectious deer
- Total number of deer
- Total number of dead
- Total number of dead due to disease
- Total number harvested
- Total number culled
- Annual population rate and mean of annual population rates
- Annual harvest level and mean of annual harvest levels
- For simulations with culling, the year CWD was eliminated from the population

Projects

1. **a.** Develop an agent-based population model of mule deer, including harvesting, using the information and data above. For this version, do not include items related to CWD.

The following parts of this project involve experimenting with your model. Run each simulation at least 10 times to a specific time, such as 80 or 100 years, averaging the results.

b. With no harvesting, determine the average of mean population growth levels. Gross and Miller's paper states, "Populations without simulated disease or harvest grew at an average rate of 6.4% per year." Do your results agree with those in the paper? (Other simulations may have obtained different results than those of Gross and Miller.)

c. With harvesting, determine the mean population growth level, number harvested, and mean harvest level. Is the population size stable? Gross and Miller's paper states, "For harvested populations, a mean harvest level of 12.8% of the population per year resulted in a stable population size." Do your results agree with those in the paper? (Other simulations have obtained different results than those of Gross and Miller.)

d. In unharvested populations, determine the sensitivity of productivity (i.e., growth rate) to changes in fecundity.

e. In unharvested populations, determine the sensitivity of growth rate to survival rate of female fawns.

f. In unharvested populations, determine the sensitivity of growth rate to survival rate of male fawns.

g. In unharvested populations, determine the sensitivity of growth rate to survival rate of adult females.

2. a. Refine the model in Project 1 to include CWD but no culling, using the information and data above.

The following parts of this project involve experimenting with your model. Run each simulation at least 10 times to a specific time, such as 80 or 100 years, averaging the results. In all cases, include harvesting but no culling.

b. Start with 4 latent and 4 infectious 2.5-year-old females and a probability of 0.57 that an infectious agent will infect a susceptible one in the same or an adjacent cell. Determine the following: average population rate, final CWD prevalence, total population, latent number, infectious number, total disease dead. Compare these results to those for uninfected populations.

c. Use the same scenario as for Part b. For infected populations, describe the graphs of the different sub-populations (susceptible, latent, sick, total) and variations in these graphs for the simulation runs. For simulations in which CWD prevalence was at least 2%, describe graphs of the total population and the proportion of infectious animals.

d. Indicate how similar the results of your simulations to the data of Miller et al. (2000) from certain CWD-endemic areas for mule deer of Colorado and Wyoming: Approximate overall prevalence of 4.9%; about 1% CWD prevalence in 15-20 yr and about 15% in 37-50 yr.

e. Miller et al. (2000) determined the age- and sex-specific data for CWD prevalence in CWD-endemic areas for mule deer of Colorado and Wyoming (Table 5). Adjust your model to display such information at simulation year 31. How do your results compare with the field data?

Table 5. CWD prevalence for mule deer by age and sex in certain CWD-endemic areas of Colorado and Wyoming (Results estimated to within 0.005, Figure 4, Miller et al. 2000)

	Age	**Females**	**Males**
	1	0.025	0.015
	2-3	0.075	0.060
	4-6	0.060	0.130
	7-9	0.050	0.005
	10+	0.060	0.005

f. In your model, how sensitive are CWD prevalence and CWD persistence to the transmission rate, or the probability that disease spreads from an infectious agent to a susceptible one in the same or an adjacent cell? That is, if the transmission rate is decreased by 10% (from a default value of 0.57), determine the resulting mean CWD prevalence and the likelihood that CWD is eliminated from the population within 50 years. Also, determine the impact on the population if the transmission rate is increased by 10%,

3. **a.** Refine the model in Project 2 to include the possibility of test and slaughter programs (early- and infectious-culling), using the information and data above.

The following parts of this project involve experimenting with your model. Run each simulation at least 10 times to a simulation time of 80 years, averaging the results. In all cases, start with 4 latent and 4 infectious 2.5-year-old females and a probability of 0.57 that an infectious agent will infect a susceptible one in the same or an adjacent cell and include harvesting.

b. With a CWD threshold of 0.01 and early culling, determine the year CWD is eliminated from the population for program efficacies of 0.1, 0.2, …, 0.7. Discuss the results.

c. Repeat Part b for a threshold of 0.05.

d. Repeat Part b for a threshold of 0.10.

e. Comparing the results of Parts b-d, discuss the results and its implications for early-culling programs.

f. With a CWD threshold of 0.01 and infectious culling, determine the year CWD is eliminated from the population for program efficacies of 0.1, 0.2, …, 0.7. Discuss the results.

g. Repeat Part b for a threshold of 0.05.

h. Repeat Part b for a threshold of 0.10.

i. Comparing the results of Parts f-h, discuss the results and its implications for infectious-culling programs.

j. Use your results to make recommendations for efforts to eliminate/reduce CWD.

4. a. Develop a program to run the simulation from Project 2 (CWD with harvesting but no culling) a user-designated number of times and to display the fraction of simulation runs that result in CWD persisting for 50 time steps (i.e., simulation years). Thus, stop each individual simulation at 50 time steps or when no animal has the disease (latent or infectious).

b. Consider starting each simulation with one infectious 2.5-year-old female. Adjust the infection probability (the probability that disease spreads from an infectious agent to a susceptible one in the same or an adjacent cell) so that a little less than 0.3 fraction (i.e., less than 30%) of 100 simulation runs results in CWD persisting for 50 "years."

c. Using the infection probability from Part b, run the simulation 100 times each for an initial number of infectious 2.5-year-old females being 1, 2, …, 7, and record the fractions of runs that result in CWD persisting for 50 "years." Plot the fractions persisting versus the initial numbers of infectious 2.5-year-old females. Discuss the results and their implications related to preventing the spread of CWD from infected populations to uninfected populations.

References

Gillin, Colin M., and J. R. Mawdsley. "AFWA technical report on best management practices for surveillance, management and control of chronic wasting disease." *Association of Fish and Wildlife Agencies, Washington, DC, USA,* 2018).

Gross, John E. and Michael W. Miller. "Chronic Wasting Disease in Mule Deer: Disease Dynamics and Control." J. of Wildlife Management 65(2):205-215, 2001.

Kreeger, Terry, Wyoming Game and Fish and Chronic Wasting Disease Alliance, 2000, accessed July 1, 2023. https://commons.wikimedia.org/wiki/File:Deer1_tkreeger.jpg.

Krumm, Caroline E., Mary M. Conner, N. Thompson Hobbs, Don O. Hunter, and Michael W. Miller. "Mountain lions prey selectively on prion-infected mule deer." *Biology Letters* 6, no. 2 (2010): 209-211.

Miller, M. W., E. S. Williams, C. W. McCarty, T. R. Spraker, T. J. Kreeger, C. T. Larsen, E. T. Thorne "Epizootiology of chronic wasting disease in free-ranging cervids in Colorado and Wyoming." J Wildl Dis, 2000 Oct;36(4):676-90.

Osterholm, Michael T., Cory J. Anderson, Mark D. Zabel, Joni M. Scheftel, Kristine A. Moore, and Brian S. Appleby. "Chronic wasting disease in cervids: implications for prion transmission to humans and other animal species." *MBio* 10, no. 4 (2019): e01091-19.

Team, CWD Action. "Montana Chronic Wasting Disease Management Plan." 2020.

Wild, Margaret A., N. Thompson Hobbs, Mark S. Graham, and Michael W. Miller. "The role of predation in disease control: a comparison of selective and nonselective removal on prion disease dynamics in deer." *Journal of Wildlife Diseases* 47, no. 1 (2011): 78-93.

Uehlinger, F. D., A. C. Johnston, T. K. Bollinger, and C. L. Waldner. "Systematic review of management strategies to control chronic wasting disease in wild deer populations in North America." 2016.

General Information on Human Prion Disease

Holman, Robert C., Ermias D. Belay, Krista Y. Christensen, Ryan A. Maddox, Arialdi M. Minino, Arianne M. Folkema, Dana L. Haberling et al. "Human prion diseases in the United States." *PloS one* 5, no. 1 (2010): e8521.