Appendix A. Details of population model and its parameterization

Density dependent mortality

We incorporated density-dependent fawn survival but not fecundity rate (FR) because density-fecundity relationships for mule deer are not well developed in the literature. For example, from the only direct estimate of density-dependence FR for white-tailed deer (Swihart et al. 1998, Fig 2: FR= (1.86±0.5)–0.0058*D*), density dependence provides population stabilization at densities *D*~300 deer/km^2, which is not realistic for the CWD area in Alberta (Merrill et al. 2011).

To estimate F_R in (7) we use mean daily food requirements for each kind of deer, $F_{v,x}$, where $v=\{S,I\}$ and $x=\{m,f,j\}$. If we denote winter duration by T_0, then the food density needed for perfect deer existence is

$$F_R = T_0 \times \left(F_{S,f} S_f + F_{S,m} S_m + F_{S,j} S_j + F_{I,f} I_f + F_{I,m} I_m + F_{I,j} I_j\right). \quad \text{(A01)}$$

Estimates of F_A from the existing vegetation types are very difficult and require too detailed information, which is hardly available. It is easier to use the mean equilibrium densities of healthy population S_{0f}, S_{0m}, S_{0j}, which are available from deer observations, and to determine "typical" value V_0 from average deer mortality. Then we obtain the equality

$$V_0 = 1 - \frac{F_A}{T_0 \times \left(F_{S,f} S_{0f} + F_{S,m} S_{0m} + F_{S,j} S_{0j}\right)}, \quad \text{(A02)}$$

and

$$\frac{F_A}{T_0} = \left(1 - V_0\right) \times \left(F_{S,f} S_{0f} + F_{S,m} S_{0m} + F_{S,j} S_{0j}\right). \quad \text{(A03)}$$

This immediately allows us to obtain the expression for the starvation index for current population densities,

$$V = \max\left\{0,\ 1 - \frac{\left(1 - V_0\right)\left(F_{S,f} S_{0f} + F_{S,m} S_{0m} + F_{S,j} S_{0j}\right)}{F_{S,f} S_f + F_{S,m} S_m + F_{S,j} S_j + F_{I,f} I_f + F_{I,m} I_m + F_{I,j} I_j}\right\}, \quad \text{(A04)}$$

which contains only values assumed to be known. It is convenient to introduce the relative food consumption coefficients

$$C_{v,x} = \frac{F_{v,x}}{\left(1 - V_0\right)\left(F_{S,f} S_{0f} + F_{S,m} S_{0m} + F_{S,j} S_{0j}\right)}, \quad \text{(A05)}$$

then

$$V = \max\left\{0,\ 1 - \left(C_{S,f} S_f + C_{S,m} S_m + C_{S,j} S_j + C_{I,f} I_f + C_{I,m} I_m + C_{I,j} I_j\right)^{-1}\right\} \quad \text{(A06)}$$

F_A is calculated from observed mean deer density of 1.58 deer/km^2 (Merrill et al.) and F_R from mean daily food intake of all deer categories. We used extreme winter fawn survival values of (White and Lubow 2002), which we associate with V close to 0 and 1, and also equilibrium fawn mortality, which allows obtaining the value of V_0.

The effect of winter food availability on juvenile mule deer mortality has been studied in (Baccante and Woods 2008). Winter food availability depends not only on population

density, but on winter severity and snow depth in particular. (Baccante and Woods 2008) have shown strong linear correlation between winter severity index and juvenile survival. In (White and Lubow 2002) there are data on both juvenile and adult survival. If we plot them in one graph, we can see very little correlation between juvenile and adult female survival (Fig. A1).

For healthy juveniles White and Lubow (2002) give the estimates of per year survival probability of mule deer fawns and adult females. Fawn survival varies between $s_{j\min}$=0.05 and $s_{j\max}$=0.77. If we assume that the lower estimate corresponds to V close to 1, and the upper one to V=0, then, using (8) and converting per year survival s_j into mortality coefficients m_{xj} corresponding to each year conditions (see below), we obtain the values of V for each year. This in turn allows us to estimate the average starvation index V_0=0.17.

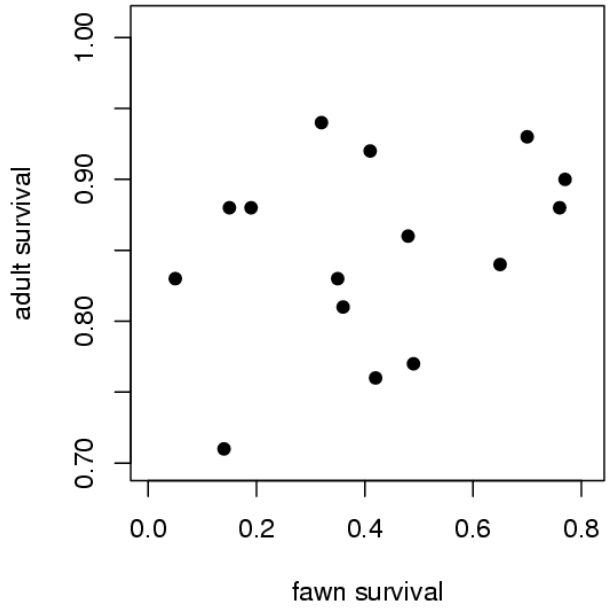

Fig. A1. Adult survival vs. juvenile survival in Colorado mule deer, data from (White and Lubow 2002).

Deer harvesting

For harvest modeling it is convenient to use population proportions regardless of the infection status,

$$\pi_m = \frac{S_m + I_m}{D}, \quad \pi_f = \frac{S_f + I_f}{D}, \quad \pi_j = \frac{S_j + I_j}{D}. \tag{A07}$$

We assume that the rate of harvesting of each deer category is proportional to deer density and hunting effort: the number of hunters or the number of licences purchased. If hunters were choosing the game at random, then proportions of killed animals of different kind

$$\pi_{Kx} = \frac{\text{the number of killed animals of category } x}{\text{total number of killed animals}}, \quad x = \{m, f, j\}, \tag{A08}$$

would coincide with proportions π_x in deer population. However, published data show that there may be strong difference between π_x and π_{Kx}, see e.g. data in (Medin and Anderson 1979): the proportion of killed adult males (antlered) is significantly greater their proportion in the studied Colorado population. There may be two reasons for this effect: 1) hunters may prefer to get antlered deer and 2) hunting regulation may be directed towards preferred killing of a certain type of deer category.

We may consider the following model of hunters' success. To kill an animal, a hunter must encounter it, then decide whether to shoot or not, then, if decided, try to kill. Assuming all steps being random, we obtain that the number of killed animals of category *s* is proportional to

$$\text{Kill}_x \sim \Pr(\text{encounter}_x) \times \Pr(\text{shoot} \mid \text{encounter}_x) \times \Pr(\text{kill} \mid \text{shoot}). \tag{A09}$$

Probability to encounter an animals of category *s* is proportional to its density, probability to kill we assume equal for all deer, then

$$\text{Kill}_x = a(S_x + I_x)h_{Px} = ap\pi_x h_{Px}, \quad h_{Px} = \Pr(\text{shoot} \mid \text{encounter}_x), \tag{A10}$$

where *a* is a constant proportional to the total number of hunters and the area, and h_{Px} reflects hunting preferences to kill different types of animals. Then

$$\pi_{Kx} = \frac{\text{Kill}_x}{\text{Total kill}} = \frac{\pi_x h_{Px}}{T}, \quad T = \pi_f h_{Pf} + \pi_m h_{Pm} + \pi_j h_{Pj}, \tag{A11}$$

or

$$h_{Px} = T\frac{\pi_{Kx}}{\pi_x}. \tag{A12}$$

The proportions of killed and living animals can be measured. The value *T* is unknown, but we assume that at least for one category of animals hunters always attempt to shoot, that is the probability to shoot is one. This immediately gives that

$$T \times \max\left\{\frac{\pi_{Kf}}{\pi_f}, \frac{\pi_{Km}}{\pi_m}, \frac{\pi_{Kj}}{\pi_j}\right\} = 1. \tag{A13}$$

According to data (e.g. Medin and Anderson 1979), most hunters want to kill antlered males, and so we set h_{Pm}=1, h_{Pf}<1, h_{Pj}<1.

Using the introduced hunters' preference h_{Pj}, the harvest rate of the category *x* is $h \times h_{Px} \times x_x$, where *h* is the total harvest effort. The total number of harvested animals from the area *A* during time interval Δt is $\Delta N = hp(\pi_f h_{Pf} + \pi_m h_{Pm} + \pi_j h_{Pj})A\Delta t$. This relation allows estimating the hunting effort *h* from the data.

We denote for brevity

$$h_x = h \times h_{Px}, \tag{A14}$$

the effective harvest rate for each deer category. We assume that hunters are unable to distinguish between healthy and sick animal, and for infected deer the harvest rate is estimated in the same way.

Model equations for healthy population

For healthy population all I_x=0, λ_x=0 and only Eq. (1)-(3) remain:

$$\frac{dS_j}{dt} = BS_f - \left(m_{0S,j} + Vm_{1S,j}\right)S_j - h_j S_j - \tau^{-1} S_j, \quad (1a)$$

$$\frac{dS_f}{dt} = 0.5\tau^{-1} S_j - m_{0S,f} S_f - h_f S_f, \quad (2a)$$

$$\frac{dS_m}{dt} = 0.5\tau^{-1} S_j - m_{0S,m} S_m - h_m S_m, \quad (3a)$$

These equations always have zero solution S_j=S_f=S_m=0. Linearizing the system near it (that is, just setting V=0) we find that the zero solution becomes unstable when

$$B > 2\tau\left(m_{0S,j} + h_j + \tau^{-1}\right)\left(m_{0S,f} + h_f\right). \quad (A15)$$

The meaning of this condition is transparent: population grows when the birth rate exceeds death rate. Another interpretation of (A15) is related with harvest intensities. If they are so great that (A15) does not hold, then population collapses.

When zero solution looses stability, population tends to a nonzero steady state. For the latter, all time derivatives vanish, and we have a system of algebraic equations

$$0 = BS_f - \left(m_{0S,j} + Vm_{1S,j} + h_f + \tau^{-1}\right)S_j, \quad (A16)$$

$$0 = 0.5\tau^{-1} S_j - \left(m_{0S,f} + h_f\right)S_f, \quad (A17)$$

$$0 = 0.5\tau^{-1} S_j - \left(m_{0S,m} + h_m\right)S_m. \quad (A18)$$

According to Fredholm's alternative, this system possesses nonzero solution only if determinant of its matrix is zero, that is

$$\left(m_{0S,m} + h_m\right)\left(\left(m_{0S,f} + h_f\right)\left(m_{0S,j} + Vm_{1S,j} + h_f + \tau^{-1}\right) - 0.5\tau^{-1}B\right) = 0.$$

This equation gives the equilibrium value of V^*,

$$m_{0S,j} + V^* m_{1S,j} + h_f + \tau^{-1} - \frac{0.5\tau^{-1}B}{m_{0S,f} + h_f} = 0 \quad (A19)$$

or

$$V^* = \frac{1}{m_{1S,j}}\left(\frac{0.5\tau^{-1}B}{m_{0S,f} + h_f} - m_{0S,j} - h_f - \tau^{-1}\right). \quad (A20)$$

We assume that V^*<1, otherwise it would mean that population may stay at nonzero equilibrium without winter food at all, which means incorrect model parameterization. From (A15) it now follows that V^*>0, which means that (A15) defines the condition of existence of the nonzero equilibrium as well.

Knowing V^*, we can express S_m and S_f through S_j from (A17) and (A18), and then determine S_j from (A06):

$$C_{S,f} S_f + C_{S,m} S_m + C_{S,j} S_j = S_j\left(C_{S,f}\frac{0.5\tau^{-1}}{m_{0S,f} + h_f} + C_{S,m}\frac{0.5\tau^{-1}}{m_{0S,m} + h_m} + C_{S,j}\right) = \left(1 - V^*\right)^{-1} \quad (A21)$$

By means of standard but bulky calculations it can be shown that the matrix of system (1a)– (3a) linearized near the positive equilibrium does not have positive eigenvalues if (A15) holds, and hence the nonzero solution is stable. To save space, we shall not present these calculations here.

Below we use this equilibrium solution for model parameterization.

Model parameterization at equilibrium

Mortality coefficients and per year survival. At equilibrium, population size *p*, starvation index *V*, and mortality do not depend on time. For constant mortality coefficient we can analytically obtain relations for per year survival.

Let us consider a fixed group of N_{f0} adult females at t=0 (in experiment this is the number of collared deer). Their number $N_f(t)$ will diminish with time due to all types of mortality as

$$\frac{dN_f}{dt} = -m_{0f}N_f - h_f N_f, \qquad N_f(t) = N_0 \exp\left(-\left(m_{0f} + h_f\right)t\right). \tag{A22}$$

After one year the number of survivors is $N_f(1) = N_0 \exp\left(-\left(m_{0f} + h_f\right)\right)$, and hence the female per year survival is

$$s_f = \frac{N_f(1)}{N_0} = \exp\left(-\left(m_{0f} + h_f\right)\right),$$

Or equivalently

$$m_{0f} + h_f = -\ln\left(s_f\right) \tag{A23}$$

Similarly for males we have

$$m_{0m} + h_m = -\ln\left(s_m\right). \tag{A24}$$

For juveniles there is a more complicated relation because they not only die, but maturate as well. Similarly, let at t=0 there are N_{j0} juveniles. Their dynamics with time satisfy

$$\frac{dN_j}{dt} = -m_j N_j - h_j N_j - \tau^{-1} N_j, \qquad N_j(t) = N_0 \exp\left(-\left(m_j + h_j + \tau^{-1}\right)t\right), \quad . \tag{A25}$$

$$m_j = m_{0j} + V m_{1j}$$

The number $M_j(t)$ of those who has died satisfy

$$\frac{dM_j}{dt} = m_j N_j + h_j N_j, \quad M_j(0) = 0, \tag{A26}$$

$$M_j(t) = \left(m_j + h_j\right)\int_0^t N_j(t)dt = \frac{m_j + h_j}{m_j + h_j + \tau^{-1}} N_0\left(1 - \exp\left(-\left(m_j + h_j + \tau^{-1}\right)t\right)\right), \tag{A27}$$

and per year survival is

$$s_j = \frac{N_{0j} - M_j(1)}{N_{0j}} = 1 - \frac{m_j + h_j}{m_j + h_j + \tau^{-1}}\left(1 - \exp\left(-\left(m_j + h_j + \tau^{-1}\right)\right)\right). \tag{A28}$$

or

$$\frac{m_j + h_j}{m_j + h_j + \tau^{-1}}\left(1 - \exp\left(-\left(m_j + h_j + \tau^{-1}\right)\right)\right) = 1 - s_j\,. \tag{A29}$$

This equation cannot be solved analytically, and for the sake of brevity we shall denote its solution as

$$m_j + h_j = \phi\left(s_j\right). \tag{A30}$$

Plots of $\phi\left(s_j\right)$ are shown in Fig. A2 for $\tau = 1.0$ (WTD) and $\tau = 1.5$ (MD).

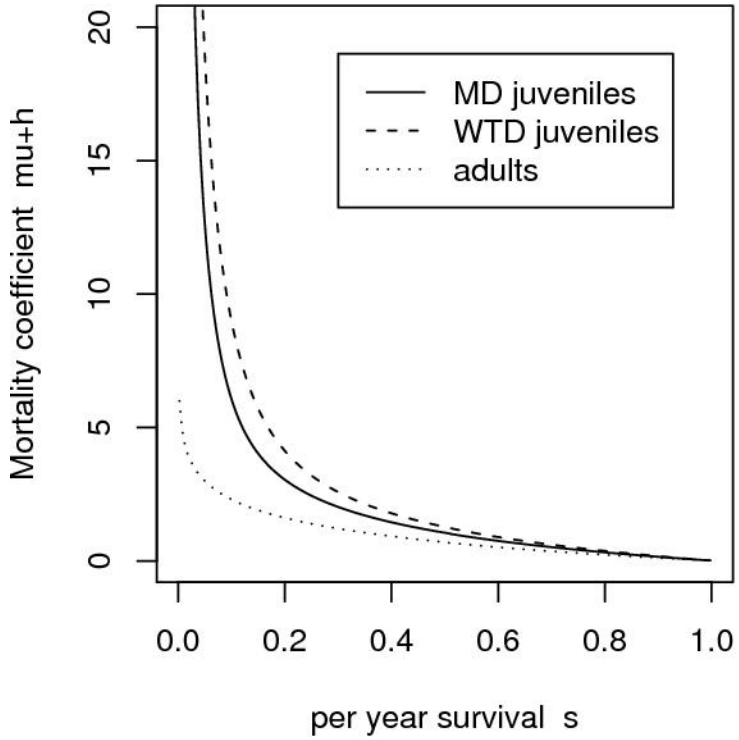

Fig. A2. Mortality estimates from equilibrium per year survival estimates

Equilibrium proportions and per year survival. From equilibrium conditions (A16)–(A18) we obtain the following ratios, relating equilibrium population proportions and per year survival:

$$\frac{S_j}{S_f} = \frac{\pi_j}{\pi_f} = \frac{B}{m_j + h_j + \tau^{-1}} = \frac{B}{\phi\left(s_j\right) + \tau^{-1}}\,, \tag{A31}$$

$$\frac{S_j}{S_f} = \frac{\pi_j}{\pi_f} = 2\tau\left(m_{0f} + h_f\right) = -2\tau\ln\left(s_f\right), \tag{A32}$$

$$\frac{S_j}{S_m} = \frac{\pi_j}{\pi_m} = 2\tau\left(m_{0m} + h_m\right) = -2\tau\ln\left(s_m\right). \tag{A33}$$

Dividing the last two equations one onto another, we obtain

$$\frac{\pi_m}{\pi_f} = \frac{\ln\left(s_f\right)}{\ln\left(s_m\right)}. \tag{A34}$$

If both proportions and per year survival are known, these relations allow one to verify, whether current population state is close to equilibrium or far from it. If only a part of them are known, then the assumption that the population is at or near the equilibrium allows one to obtain the others. For example, knowing the proportions to estimate survival or vice versa.

Survival rates from population proportions based upon aerial survey data at WMU 728 and 730 (CFB Wainwright). Population proportions estimated from survey data are shown in Fig. A3. We used only years when population structure data were available. When estimating population proportions, we ignored the unclassified part of animals, assuming that there is no bias in counting and hence proportions in observed, unobserved, and unclassified parts are equal. There are no visible trends in the data, and we assume that average proportions can characterize the state of equilibrium.

From (A31)-(A33) we have

$$s_f = \exp\left(-\frac{\pi_j}{2\tau\pi_f}\right), \quad s_m = \exp\left(-\frac{\pi_j}{2\tau\pi_m}\right), . \tag{A35}$$

$$m_j + h_j = B\frac{\pi_f}{\pi_j} - \tau^{-1}, \quad s_j = 1 - \frac{m_j + h_j}{m_j + h_j + \tau^{-1}}\left(1 - \exp\left(-m_j - h_j - \tau^{-1}\right)\right) \tag{A36}$$

The results are shown in Table A1, estimates from data are shown in bold, estimates from (A35), (A36) in normal text.

Estimating hunting preferences and intensity from WMU 728 & 730 hunting data. Hunting registration data contain information about the number of killed males, females, and juveniles, which allows us to estimate the proportions of each category in harvested animals π_{Ks}. To estimate the hunting intensity we need to know the removed proportion of the population, and hence we need the estimate of the population size at the beginning of the hunting season. However, there are two problems:

1) Aerial surveys contain the estimates of the total population in the end of December or beginning of January, that is after the hunting season. Hence to obtain population for which hunters have made their decisions, we must add killed animals back to the population.
2) Aerial surveys observe only a part of deer population. By default, SRD assumes that surveys observe 50% of deer. We make estimates for the cases when observed are 50%, 75%, and 100% of deer.

Let us denote the number of deer observed in survey as N_S, the number of killed animals as N_K, the proportion of observed animals as δ. Then the true number of animals during survey is $\delta^{-1}N_S$, the proportion of killed animals in the population is

$$b = \frac{N_K}{\delta^{-1}N_S + N_K}. \tag{A37}$$

We also assume that during a hunting season we can neglect all other mortality reasons besides hunting, as well as deer migration inside and outside the hunting region. Then the proportion of females in the population before the hunting is

$$\pi_{bf} = \frac{\pi_f \delta^{-1} N_S + \pi_{Kf} N_K}{\delta^{-1} N_S + N_K} = \pi_f \frac{\delta^{-1} N_S}{\delta^{-1} N_S + N_K} + \pi_{Kf} \frac{N_K}{\delta^{-1} N_S + N_K} = , \quad \text{(A38)}$$
$$= (1-b)\pi_f + b\pi_{Kf}$$

where π_f is proportion from the survey. The hunters' preference for females is

$$h_{Pf} = T \frac{\pi_{Kf}}{\pi_{bf}} = T \frac{\pi_{Kf}}{(1-b)\pi_f + b\pi_{Kf}} . \quad \text{(A39)}$$

Similarly we estimate preference for males and juveniles, and *T* is estimated from analog of (A13). The estimate gives that h_{Pm}=1, hence the proportion of males removed from population shows the hunting intensity *h*:

$$h = \frac{\pi_{Km} N_K}{\pi_{bf} \left(\delta^{-1} N_S + N_K\right)} = b \frac{\pi_{Km}}{\pi_{bf}} . \quad \text{(A40)}$$

The estimates of proportions before hunting, *h*, h_{Ps}, and mortality coefficients for three values of δ are given in Tables A2 and A3.

In simulations the values for mule deer and δ=0.75 were selected.

Appendix B. Derivations of transmission functions in Table 3

B.0 Implicit accounting for seasonality in contacts

We incorporated seasonal contact rates implicitly by assuming simplified dynamics in groupings of deer as: 1) *Summer* (May to October): males and females stay in separate groups and there are practically no direct contacts between them, fewer interactions among both female and male groups in this season due to spatial dispersion after migration, but high intra-group contacts. 2) *Rut*: (November to-December): male and female groups remain separated but new types of contacts appear: mating contacts between males and females and fights between some males. 3) *Winter* (January to April): deer form larger, mixed-sex groups. Migration to winter locations where food is more accessible under winter snows results in groups staying near each other, and between-group contacts are more frequent but within group contact remain similar to other seasons.

Seasonal changes in direct and environmental contacts by deer require different expressions for the force of infection terms during different seasons when groups are separate (summer and rut) and mixed (winter) together on winter ranges. To avoid introduction of too many parameters, we accounted for seasonality implicitly: we combine terms corresponding to different seasons into a weighted sum, and use it for the whole year.

We incorporated group dynamics by weighting the effect of groups by the length of the season when deer spent time in the group. We assumed seasons had durations Δt_1, Δt_2 and Δt_3 respectively, and across the year they summed as $\Delta t = \Delta t_1 + \Delta t_2 + \Delta t_3$. We modelled the change in population size over time, $dy/dt = f(y,t)$, by assuming that y changes slowly during a year, but $f(y,t)$ varies quickly, such that during winter time $f(y,t) = f_1(y)$, during summer $f(y,t) = f_2(y)$, and during rut $f(y,t) = f_3(y)$. That is, we have a model

$$\frac{dy}{dt} = \begin{cases} f_1(y), & 0 < t < \Delta t_1, \\ f_2(y), & \Delta t_1 < t < \Delta t_1 + \Delta t_2, \\ f_3(y), & \Delta t_1 + \Delta t_2 < t < \Delta t. \end{cases} \qquad \text{(B1)}$$

Neglecting terms proportional to Δt^2 (due to assumption of slow y change) one obtains

$$\Delta y_1 = y(\Delta t_1) - y_0 \approx f_1(y_0)\Delta t_1, \qquad y_0 = y(0),$$
$$\Delta y_2 = y(\Delta t_1 + \Delta t_2) - y(\Delta t_1) \approx f_2(y_0 + \Delta y_1)\Delta t_2 \approx f_2(y_0)\Delta t_2,$$
$$\Delta y_3 = y(\Delta t) - y(\Delta t_1 + \Delta t_2) \approx f_3(y_0 + \Delta y_1 + \Delta y_2)\Delta t_3 \approx f_3(y_0)\Delta t_3.$$

We would like to find a single model $dy_Y / dt = f_Y(y_Y)$ such that $y_Y(t + \Delta t)$ approximately equals $y(t + \Delta t)$ provided $y_Y(t) = y(t)$. This means that $\Delta y_1 + \Delta y_2 + \Delta y_3 \approx \Delta y_Y$, or

$$f_1(y_0)\Delta t_1 + f_2(y_0)\Delta t_2 + f_3(y_0)\Delta t_3 \approx f_Y(y)\Delta t.$$

This gives the averaging rule

$$f_Y(y) \approx w_1 f_1(y) + w_2 f_2(y) + w_3 f_3(y), \qquad \text{(B2)}$$

$$w_1 = \frac{\Delta t_1}{\Delta t}, \quad w_2 = \frac{\Delta t_2}{\Delta t}, \quad w_3 = \frac{\Delta t_3}{\Delta t}, \quad w_1 + w_2 + w_3 = 1,$$

that is the resulting function is a weighted average of the three functions with the weights proportional to the duration of these periods. This result can easily be generalized for other numbers of seasons.

Therefore, if we have several seasons with the relative durations of w_1, w_2, w_3, and different expressions for force of the infection during each season, then the effective term in our model is

$$\lambda(I) \approx w_1\lambda_1(I) + w_2\lambda_2(I) + w_3\lambda_3(I) + ..., \qquad w_1 + w_2 + w_3 + ... = 1. \qquad \text{(B3)}$$

This allows us to account for seasonality without increasing the model complexity. More accurate description of within-year population variations would require using explicit seasonality like in (B1).

B.1 Direct vertical transmission.

Vertical or maternal transmission is implemented through two different birth rates for infected females: they produce healthy juveniles at the rate B_{IS} and infected juveniles at the rate B_{II}. Although some fawns born at late stages in the disease may be not viable (Mathiason et al. 2010), reducing fertility (Dulberger et al. 2010), we assume that fertility for infected and healthy females coincide, $B_{IS} + B_{II} = B$ (see Discussion below). If we denote the probability of vertical transmission by p_V, then $B_{IS} = (1 - p_V)B$, $B_{II} = p_V B$. According to studies on penned mule deer (Miller et al. 2000), for CWD p_V does not exceed 0.05; when vertical transmission occurs in our models, we use this value.

B.2 Direct horizontal transmission

Direct horizontal transmission assumes that with direct contact, such as grooming and mating, the host infects a healthy individual with some probability. At present there are no measures of the frequency of direct transmission, although several studies provide metrics of pair-wise proximity based on GPS-telemetry as surrogates for contact rates (Kjaer et al. 2008, Schauber et al. 2007, Habib et al. 2011). To keep our approach general, we assumed three types of deer social groups (matrilinear family group of females + juveniles, males only groups, and mixed groups) whose proportion in the population varied by season, and the efficiency of transmission for pairs of deer within these groups varied as described below.

B.2.1 Direct contacts within a group. We assume that the population consists of the groups of size k, and the number of groups in the population is $N_G=AD/k$, where A is the area occupied by the population and D is deer density, therefore AD is the total number of deer in the area (Table 1). We assume that the groups are representative such that the proportions of healthy and infected animals in the groups are similar to with the proportions within whole population. We derive the formula for the force of infection corresponding to transmission from deer of type u to type x, e.g., females to females ($u=x=f$) or males to juveniles ($u=m$, $x=j$), by assuming the number of contacts between healthy and infected deer is proportional to the product of the number of healthy and infected deer in the group. The number of infected deer of type u in a group therefore is $k(I_u / D)$ and the number of healthy deer of type x is $k(S_x / D)$. Then for the total number of new infections in the population due to within-group contacts of u and x during a small time interval Δt is:

$$\left(\Delta N_{I,x}\right)_u = A\left(\Delta I_x\right)_u = b_{xu}\left(\frac{kI_u}{D}\right)\left(\frac{kS_x}{D}\right)\left(\frac{AD}{k}\right)\Delta t = \frac{kb_{xu}I_u}{D}S_x A\Delta t\,. \qquad \text{(B4)}$$

where b_{xu} is the rate of disease transmission (contact rate times probability of transmission). Summing up for $u=\{m,f,j\}$ and dividing by A and Δt, we obtain the rate of new infections per unit area in category x as

$$\frac{\Delta I_x}{\Delta t} = \frac{k\left(b_{xm}I_m + b_{xf}I_f + b_{xj}I_j\right)}{D}S_x = \lambda_x S_x, \qquad x = m,\, f,\, j\,. \qquad \text{(B5)}$$

If we denote $\beta_{xu}=kb_{xu}$, then we obtain the expression for respective force of infection λ_x in (1)–(6) as

$$\lambda_x = \frac{\beta_{xm}I_m + \beta_{xf}I_f + \beta_{xj}I_j}{D} \qquad \text{(B6)}$$

In case of male groups in summer and rut, we consider only the male subpopulation instead of the whole population and use S_m+I_m instead of D when calculating the number of infected and healthy individuals within a group and the total number of male groups:

$$\lambda_m = \frac{\beta_{mm}I_m}{S_m + I_m}\,. \qquad \text{(B7)}$$

Similarly, for female family groups (females and juveniles) in summer and rut we use only female and juvenile population $S_f + I_f + S_j + I_j$ instead of D and obtain as a result

$$\lambda_x = \frac{\beta_{xf}I_f + \beta_{xj}I_j}{S_f + I_f + S_j + I_j}, \qquad x = f,\, j\,. \qquad \text{(B8)}$$

Coefficients β_{xu} may change independently across seasons, e.g. due to change in mean group size k. As a simplification, we assume that disease transmission coefficients do not change independently, and hence the relative values of transmission coefficients within the matrix

$$\psi_{xu} = \beta_{xu} / \max_{st}\{\beta_{st}\}, \qquad \text{(B9)}$$

do not change with season. Therefore, we use the factor $\max_{st}\{\beta_{st}\}$ to standardize the transmission coefficient during summer, rut and winter as β_{1S}, β_{1R}, and β_{1W}. Now the expressions for force of infection in males take the form for males of

$$\lambda_{m1} = \left(w_S\beta_{1W} + w_R\beta_{1R}\right)\frac{\psi_{mm}I_m}{S_m + I_m} + w_W\beta_{1W}\,\frac{\psi_{mm}I_m + \psi_{mf}I_f + \psi_{mj}I_j}{D}, \qquad \text{(B10)}$$

and for a family group

$$\lambda_{x1} = \left(w_S\beta_{1S} + w_R\beta_{1R}\right)\frac{\psi_{xf}I_f + \psi_{xj}I_j}{S_f + I_f + S_j + I_j} + w_W\beta_{1W}\,\frac{\psi_{xm}I_m + \psi_{xf}I_f + \psi_{xj}I_j}{D}, \quad x = f,\, j\,. \qquad \text{(B11)}$$

To simplify these force of infection expressions, we introduce the amplitude factor, $\beta_1 = \left(w_S\beta_{1W} + w_R\beta_{1R} + w_W\beta_{1W}\right)/2$, and weights for separate and mixed groups, $w_{S1} = \left(w_S\beta_{1W} + w_R\beta_{1R}\right)/\beta_1$, $w_{M1} = w_W\beta_{1W}/\beta_1$, $w_{S1}+w_{M1}=2$. Now β_1 describes the mean cross-season transmission coefficient, and the weights incorporate seasonal differences in both seasonal duration and magnitude of transmission. Then the final expression for the disease transmission function for direct contacts within groups of males across seasons is

$$\lambda_{m1} = \beta_1 \left[w_{S1} \frac{\psi_{mm} I_m}{S_m + I_m} + w_{M1} \frac{\psi_{mm} I_m + \psi_{mf} I_f + \psi_{mj} I_j}{D} \right], \quad \text{(B12)}$$

and for family groups is

$$\lambda_{x1} = \beta_1 \left[w_{S1} \frac{\psi_{xf} I_f + \psi_{xj} I_j}{S_f + I_f + S_j + I_j} + w_{M1} \frac{\psi_{xm} I_m + \psi_{xf} I_f + \psi_{xj} I_j}{D} \right], \quad x = f, j. \quad \text{(B13)}$$

Weighting factors for seasonal groupings of deer, ψ_{xu} may be important for accurate description of disease transmission through direct contact, and thus we present the general form above (B12)-(B13) because it may be useful in the future work. However, because there are no data on direct disease transmission among age/sex groups, for the simulations presented here we assume all relative transmission coefficients are equal, or all ψ_{xu}=1, and only β_1 and the ratio w_{S1}/w_{M1} are varied in our initial simulations. This results in the simplified expression for the force of infection for males of

$$\lambda_{m1} = \beta_1 \left[w_{S1} \frac{I_m}{S_m + I_m} + w_{M1} \frac{I_m + I_f + I_j}{D} \right], \quad w_{M1} + w_{S1} = 2, \quad \text{(B14)}$$

and for family groups of

$$\lambda_{x1} = \beta_1 \left[w_{S1} \frac{I_f + I_j}{S_f + I_f + S_j + I_j} + w_{M1} \frac{I_m + I_f + I_j}{D} \right], \quad x = f, j. \quad \text{(B15)}$$

After our initial simulations, we also explore an alternative choice for ψ_{xu} based upon sex-related susceptibility (see below).

B.2.2 Direct contacts between groups. The frequency of between group contacts varies strongly by season because of observed shifts in the distribution of deer that alters home range overlap (Habib et al. 2010). We follow the same derivation as for within group contacts, but assume the whole population is one group of the size $k=AD$, which is always mixed at all seasons. Denoting $\beta_{uv} = Ab_{uv}$ and assuming that the relative values of transmission coefficients (B9) do not change with season, we come to the expression, which has a common form for males, females and juveniles,

$$\lambda_x = \left(w_S \beta_{2S} + w_R \beta_{2R} + w_W \beta_{2W}\right)\left(\psi_{xm} I_m + \psi_{xf} I_f + \psi_{xj} I_j\right), \quad \text{(B16)}$$

where x may be m, f, or j. Introducing the amplitude factor β_2, we come to the general expression for between group transmission,

$$\lambda_{x2} = \beta_2 \left(\psi_{xm} I_m + \psi_{xf} I_f + \psi_{xj} I_j\right), \qquad \beta_2 = w_S \beta_{2S} + w_R \beta_{2R} + w_W \beta_{2W}. \quad \text{(B17)}$$

As before, we assume all ψ_{xu}=1 and we obtain the simplified expression

$$\lambda_{m2} = \lambda_{f2} = \lambda_{j2} = \beta_2 \left(I_m + I_f + I_j\right). \quad \text{(B18)}$$

B.2.3 Mating contacts, female to male transfer. Let n_X be the mean number of males that contact one female during mating. The total number of infected females in the population is AI_f, and the total number of mating contacts involving them is $AI_f n_X$, the proportion of susceptible males is $S_m/(S_m+I_m)$. Denoting the probability of disease transfer from a female to male during mating ($f \to m$) by b_{FM}, the number of new male infections during small time interval Δt is

$$\left(\Delta N_{I,m}\right)_X = A\left(\Delta I_m\right)_X = b_{FM} A I_f n_X \frac{S_m}{S_m + I_m} \Delta t. \quad \text{(B19)}$$

These contacts occur only during rut, hence we use only one seasonal weight. Denoting $\beta_3 = w_R b_{FM} k_X$, we obtain

$$\lambda_{m3} = \beta_3 \frac{I_f}{S_m + I_m}. \quad \text{(B20)}$$

B.2.4 Mating contacts, male to female transfer. Repeating the arguments for obtaining λ_{m3} and using the number of susceptible females in the population, AS_f and proportion of infected males $I_m/(S_m+I_m)$, we come to

$$\lambda_{f4} = \beta_4 \frac{I_m}{S_m + I_m}. \quad \text{(B21)}$$

B.2.5 Male fights. Fights during rut typically occur between males from different social groups, and they should be distinguished from sparring matches that contribute to contacts contacts within male groups. We model transmission during fights similarly to between-group disease transmission by assuming that the number of contacts where the disease can be transmitted is proportional to the product of the densities of susceptible and infected males, assuming random mixing. This gives disease transmission due to fighting as

$$\lambda_{m5} = \beta_5 I_m. \quad \text{(B22)}$$

B.3. Environmental transmission

For environmental transmission, we model both the accumulation of prions in the environment and transmission from the environment to deer at both the level of the social group and between groups. We do not explicitly model the environmental compartment *E* for disease transmission, but follow an approach described by Haken (1983) where slowly changing variables "enslave" ones with "fast relaxation", and the latter can be approximated by functions of just the slow variables. As a result, the complex model including both slow and fast variables can be replaced by a simpler model containing slow variables only. Accuracy of the approach depends on the difference between characteristic times for slow and fast modes: the greater is the difference, the more accurate the method is.

The equation for the prion content *E* in the environment is a generalization of the Miller et al. (2006) model:

$$\frac{dE}{dt} = \varepsilon_m I_m + \varepsilon_f I_f + \varepsilon_j I_j - \tau E. \quad \text{(B22)}$$

where ε_x denote rates of environment contamination for the 3 deer age-sex classes, and τ is the rate that prions become inaccessible to deer due to decay or degradation (Rapp et al. 2006) or movement in soils or water (Smith et al. 2011). Miller et al. (2006) reported $\tau = 2.55$ year^{-1} for CWD transmission in penned deer. This rapid rate of removal means that a portion of prions left in the environment decreases with time as $\exp(-\tau t)$ and reduces to 0.078 of its original amount in one year and to 0.006 in two years. Note that these calculations account for the amount of prions actively participating in the disease transmission rather than the total amount of prions in the environment.

The deer density and hence deer infection, $I_x(t)$, changes slowly compared to this rate of prion decline. For example, in Alberta detected prevalence of CWD increased about 6 fold in 5 years after it was once detected (Alberta SRD 2006-2011), which corresponds to growth exponent about 10 times less in magnitude than τ reported by Miller et al. (2006). In such a situation a "fast" variable (prion content in the environment) is determined by current density of CWD infected deer $I_x(t)$, while the influence of the number of infected deer in the past is waning. The solution to (B22), assuming $E(0)=0$, can be written as

$E(t)=\int_0^t \left(\varepsilon_m I_m(t')+\varepsilon_f I_f(t')+\varepsilon_j I_j(t')\right)\exp\left(-\tau(t-t')\right)dt'$ Due to the exponential factor, essential contribution to $E(t)$ comes only from time interval $t-\Delta t<t'<t$ where $\Delta t\sim 1/\tau$ and in our case is close to 1 year. If $I_x(t')$ does not change significantly at this interval, than $I_x(t')\approx I_x(t)$, then the integral easily evaluates, and, neglecting term $\exp(-\tau t)<<1$, one obtains

$$E(t)\approx\frac{\varepsilon_m I_m(t)+\varepsilon_f I_f(t)+\varepsilon_j I_j(t)}{\tau}. \qquad \text{(B23)}$$

The assumption that *E*~*I* makes the force of infection terms for the environmental and direct transmission look similar, though the transmission coefficients have different meaning. Terms in a model describing rate of environmental transmission, e.g. term γES in (Miller et al. 2006), take the form $(\gamma\varepsilon_x/\tau)I_xS=\beta' I_xS$, and formally the model with environmental transmission becomes an SI model with the effective transmission coefficients $\beta'_x=\gamma\varepsilon_x/\tau$.

B 3.1 Discussion of exclusion of environment compartment

The-appropriateness of this approach can be assessed by comparing parameter values presented in (Miller et al 2006) for direct (SI) and environmental transmission (SEI) models. If we use an analog of (B23) for their SIE model and substitute the resulting expression for *E* into two remaining equations for *S* and *I*, formally we obtain a model similar to their SI model for direct contacts with the effective value of transmission coefficient β=0.034 year^{-1}, while direct fitting of the SI model to data gives β=0.0326 year^{-1}. We can conclude that models of quasi-direct contact may quite well describe environmental transmission as well. In (Miller et al 2006) the AIC_c difference between SI and SEI models was only 2.4. Taking into account the number of model parameters, we can find that BIC criterion gives $\Delta BIC\approx 0.4$ for this pair of models, and both values show that the two models can be considered as comparable (Ghosh and Samanta 2001; Burnham and Anderson 2004). The difference in model performance detected by ΔAIC_c is related most probably with the fact that in SIE model arising of an infected deer does not immediately lead to the infection spread: build-up of prions in the environment takes some time, after which the disease transmission effectively starts, while in SI model there is no transmission delay. Otherwise the disease pattern predicted by both models should be very close.

In applying his model of environmental transmission, Miller et al. (2006) ignored social structure because they the study involved a small captive population. In contrast, we

distinguish two types of environmental transmission: within a small social group and across a big population between social groups. For deer within a social group, the chances to contact the environment contaminated by the member of the same group must be higher than that contaminated by a member of other groups (Schauber et al. 2007).

One more assumption made in our model is that we can study CWD transmission without explicit latent stage, when deer already have infection, but do not spread it further. This choice follows from the results in (Miller et al. 2006), where the best model did not have the latent compartment. More detailed comparison of SI and SLI models is given in Appendix D, where we show that, under certain conditions, SI models well describe dynamics of a disease with short enough latent stage. However, the transmission coefficients in such SI model should be smaller than that in SLI model describing the same disease: this is effective accounting for mortality in the latent stage, due to which some individuals getting the infection do not enter infected compartment and do not spread the infection further.

B.3.2 Environmental transmission within a group.

We consider the indirect rate of disease transmission within a mixed group of size k from infected deer of category u to healthy deer of category x. The consideration of separate groups is done similarly. The average number of infected individuals of category u in a group is kI_u/D, and the number of healthy ones is kS_x/D. In general, the rate of contamination of the environment ε_u is proportional to food consumption rate of infected individuals $F_{I,u}$. The rate of transmission from the environment to healthy individuals is proportional to (i) their food consumption rate, $F_{S,x}$, (ii) the coefficient b_x, which incorporates the probability of developing the disease given intake of contaminated food, and (iii) parameters used to reflect prion degradation and increased inaccessibility. The increase in the number of sick deer of age/sex x due to consumption of food contaminated by deer of type u in a mixed group during small time interval Δt is

$$\left(\Delta N_{I,x}\right)_u = A\left(\Delta I_x\right)_u = b_x \frac{kI_u}{D} F_{I,u} \frac{kS_x}{D} F_{S,x} \frac{AD}{k} \Delta t = kb_x F_{I,u} F_{S,x} \frac{I_u}{D} S_x A \Delta t \,. \qquad \text{(B24)}$$

where AD/k is the total number of groups in the population, N_G. Summing up infected individuals in a group $u=\{m,f,j\}$ and dividing by Δt, we obtain change in the number of infected individuals over time as

$$\frac{\Delta I_x}{\Delta t} = kb_x \frac{F_{S,x}\left(F_{I,m} y_m + F_{I,f} y_f + F_{I,j} y_j\right)}{D} S_x \,. \qquad \text{(B25)}$$

We denote the coefficient for transmission of the disease from infected individuals of specific age/sex class (x) to age/sex class (u) via the environment due to food intake as $\beta_{xu}=kb_x F_{S,x} F_{I,u}$, and obtain the force of infection for within-group environmental transmission for the age/sex class x, λ_x as the sum of the rates across all age/sex classes:

$$\lambda_x = \frac{\beta_{xm} I_m + \beta_{xf} I_f + \beta_{xj} I_j}{D} \,. \qquad \text{(B26)}$$

For separate groups such as summer male groups and family groups, instead of the total population density D in the denominator we use the male population ($S_m + I_m$) and female + juvenile population ($S_f + I_f + S_j + I_j$) for the expressions

$$\lambda_m = \frac{\beta_{mm} I_m}{S_m + I_m}, \qquad \lambda_x = \frac{\beta_{xf} I_f + \beta_{xj} I_j}{S_f + I_f + S_j + I_j}, \qquad x = f, j. \tag{B27}$$

Similar to the case of within group direct transmission, we assume that a set of weighting factors showing relative intensity of environmental contacts $\phi_{xu} = \beta_{xu} / \max_{st}\{\beta_{st}\}$ does not depend on season. As above, we assume that males, females, and juveniles have the same probability of getting CWD given a contact, that is $b_m=b_f=b_j$, then

$$\phi_{xu} = \frac{F_{I,u} F_{S,x}}{\max_{v,w}\{F_{I,v} F_{S,w}\}}, \tag{B28}$$

and hence this matrix ϕ can be estimated from deer food consumption. Similar to the case of direct transmission, we denote the amplitude factor $\max_{st}\{\beta_{st}\}$ during summer, rut and winter as β_{6S}, β_{6R}, and β_{6W} so that the force of infection terms with accounting for seasonality (B3) take the form

$$\lambda_m = \left(w_S \beta_{6W} + w_R \beta_{6R}\right) \frac{\phi_{mm} I_m}{S_m + I_m} + w_W \beta_{6W} \frac{\phi_{mm} I_m + \phi_{mf} I_f + \phi_{mj} I_j}{D}, \tag{B29}$$

$$\lambda_{x1} = \left(w_S \beta_{6S} + w_R \beta_{6R}\right) \frac{\phi_{xf} I_f + \phi_{xj} I_j}{S_f + I_f + S_j + I_j} + w_W \beta_{6W} \frac{\phi_{xm} I_m + \phi_{xf} I_f + \phi_{xj} I_j}{D}, \quad x = f, j \tag{B30}$$

where the amplitude factor, $\beta_6 = \left(w_S \beta_{6W} + w_R \beta_{6R} + w_W \beta_{6W}\right)/2$, and weights for separate and mixed groups, $w_{S6} = \left(w_S \beta_{5W} + w_R \beta_{6R}\right)/\beta_6$, and mixed groups, $w_{M6} = w_W \beta_{6W} / \beta_6$, $w_{S6}+w_{M6}=2$. The final expression for the force of infection for environmental transmission within groups is

$$\lambda_{m6} = \beta_6 \left[w_{S6} \frac{\phi_{mm} I_m}{S_m + I_m} + w_{M6} \frac{\phi_{mm} I_m + \phi_{mf} I_f + \phi_{mj} I_j}{D} \right], \tag{B31}$$

$$\lambda_{x6} = \beta_6 \left[w_{S6} \frac{\phi_{xf} I_f + \phi_{xj} I_j}{S_f + I_f + S_j + I_j} + w_{M6} \frac{\phi_{xm} I_m + \phi_{xf} I_f + \phi_{xj} I_j}{D} \right], \quad x = f, j. \tag{B32}$$

As in direct transmission within groups (Section B.2.1), only β_6, and one of w_S/w_M will be varied when we simulate disease spread to explain the observed disease patterns.

B.3.3 Environmental transmission between groups.

Between group transmission arises when home ranges of deer from other groups intersect and deer from one group are exposed to areas infected by the second group. This is less frequent in summer because deer are relatively more dispersed across the landscape than in winter (Habib et al. 2011). However, there is no accounting for social structure in between group transmission, and seasonal weights just add up as in (B16), such that all these differences can be aggregated into single effective transmission coefficient. Repeating the above derivation for the case $k=AD$ and integrating all seasonal-dependent factors into one coefficient β_7 as in (B17), we obtain a general expression for environmental between group contacts:

$$\lambda_{x7} = \beta_7 \left(\phi_{xm} I_m + \phi_{xf} I_f + \phi_{xj} I_j\right). \tag{B33}$$

B 4 Combined force of infection and classification of disease transmission mechanisms

Combining all possible mechanisms of transmission described above , the most general relation for the transmission of CWD for an age/sex class rate is:

$$\lambda_x = \lambda_{x1} + \lambda_{x2} + \lambda_{x3} + \lambda_{x4} + \lambda_{x5} + \lambda_{x6} + \lambda_{x7} \quad (29)$$

This expression denotes the cumulative force of infection for each age/sex class (x) and each hypothesized mechanisms. Corresponding formulas of hypothesized transmission mechanisms 1-7 described above are listed in Table 3. The mechanisms 1, 2, 6, and 7 are in effect year round and affect all deer categories, and below we call them "basic". The rut mechanisms 3, 4, and 5 involve only one or two deer categories, and cannot explain the observed pattern, and by themselves can support the disease only in males. Therefore the minimum combination of transmission mechanisms must include at least one basic mechanism.

In addition the seven mechanisms can be classified as frequency dependent (FD) and density dependent (DD). This classification is often used in disease modeling. It is related with the dependence of force of infection on population density: for FD transmission the force of infection is proportional to disease prevalence and remains constant as density increases, for DD transmission the force of infection is proportional to the number of infected individuals or their density and scales with density (McCallum et al. 2001, Begon et al. 2002). Thus, transmission coefficients β for FD and DD mechanisms have different dimensionality: year^{-1} for FD and year^{-1}km^2 for DD.

When the population density changes, e.g. due to population control measures, but proportions of the infected individuals remain the same, force of infection corresponding to FD mechanisms does not change, but that of DD mechanisms increases or decreases proportionally to the density. For the expressions λ_1 to λ_7 in Table 3, we see that λ_1, λ_3, λ_4 and λ_6 are invariant to density change because both numerator and denominator are proportional to the density, while λ_2, λ_5, and λ_7 scale proportionally to population density. For this reason we refer to the former group as frequency-dependent (FD) transmission mechanisms, and the latter as density-dependent (DD) mechanisms.

The fact that (29) includes both FD and DD mechanisms means that their relative importance depends on population density. We test the mechanisms for the density D close to the disease-free equilibrium one, D_0. In simulations it is then convenient to rescale DD coefficients with respect to the fixed density. For FD transmission force of infection has the form $\lambda_{FD} = \beta_{FD} I / D$, and for DD one $\lambda_{DD} = \beta_{DD} I$. If in the expression for DD force of infection we multiply and divide by the equilibrium density D_0, $\lambda_{DD} = (\beta_{DD} D_0)(I / D_0)$ and introduce $\beta'_{DD} = \beta_{DD} D_0$, then $\lambda_{DD} = \beta'_{DD} I / D_0$. Thus, we note that β'_{DD} has the same dimensionality as β_{FD}, which is convenient for comparison. If at the disease-free equilibrium an infection is introduced and $\beta_{FD} = \beta'_{DD}$, then initially both mechanisms equally contribute to the disease spread.

Appendix C. Forage intake data

Alldredge et al. (1974) present data for Colorado mule deer forage intake in grams of air dry food per kilogram of carcass weight per day. For fawns there are 4 figures for males and females of the ages 6-11 months and 12-17 months: 33.4, 31.7, 32.9, 27.8 with the average 31.5. For adult males the consumption rate is estimated as 16.7, and for adult females 19.2, though statistically their difference was insignificant. We also have to take into account that adult males eat very little during rut, so in the other periods the difference must be smaller, and in spring males consume more food per kg than females (perhaps, because of growing antlers). For this reason, we assume the rate per kg per day equals to 19.2 for both males and females. Therefore, the difference in food consumption between males and females is mainly due to the difference in their body masses.

Alldredge et al. (1974) gives the average weights for males, females, and fawns of the age 6-11 are 72.9kg, 59.4kg, and 32.7kg. Medin and Anderson (1979) in Table 18 give slightly different data with varying female/male weight ratios between the years: average male masses are 52.4, 54.0, 57.1 and female masses 42.7, 44.5, 39.1. This shows big variety in body masses across years, sexes, and locations. However, the ratio of female to male mass stays in a narrow range between 0.82 and 0.68 with the average 0.78. Eventually we assumed male body weight 72.9kg, female weight 56.7kg, and fawn weight 32.7 kg. Multiplying this by per kg per day consumption rates, we obtain the daily intake rates for these categories:

$$F_{Sm} = 1.40 kg/day, \quad F_{Sf} = 1.09 kg/day, \quad F_{Sj} = 1.03 kg/day$$

of air dry forage. For juveniles these data seem to reflect late autumn consumption, when they have already grown and there is a lot of food available. From our point of view, this figure characterizes maximum rather than year average food consumption and in the model we used half of it:

$$F_{Sj} = 0.51 kg/day$$

Appendix D. Relationship between SI and SLI (with latency) models

Among CWD models tested by Miller et al. (2006) were those with disease latency stage. Since CWD does have a latent period during which infection develops internally, but is not transmitted further, it may be necessary to pay attention to two questions that may arise after reading of Miller et al. (2006) and our paper:

a) Why the model with explicit latent stage did not appear the best in Miller et al. (2006) results?

b) Is it appropriate to model a disease with latent stage with SI-type model, and if yes, under what conditions?

To answer these questions, we consider generic SI and SLI models. In the literature these models are typically named SEI for Exposed stage, but not to confuse Exposed and Environment compartments, we follow (Miller et al. 2006) and call it Latency.

Let us consider a generic SLI model without sex/age structure

$$\frac{dS}{dt} = b(S+L+I) - (m+h)S - \beta SI, \tag{D1}$$

$$\frac{dL}{dt} = \beta SI - \rho L - (m+h)L. \tag{D2}$$

$$\frac{dI}{dt} = \rho L - (m+h+\mu)I. \tag{D3}$$

Here ρ is the rate of leaving the latent stage, approximately the inverse of the stage duration. For CWD deer starts to spread the disease after ~9 months after getting the infection, so we can assume that $\rho \approx 1.3\,\text{year}^{-1}$. $m+h$ represents total healthy adult deer mortality, and at disease-free equilibrium it is close to recruitment of new adults, so $m+h \approx 0.6$ year^{-1}. For increased disease-related mortality we take $\mu = 0.57$ year^{-1}.

Equation for L can be written as

$$\frac{dL}{dt} + \rho L + (m+h)L = \beta SI \tag{D4}$$

with formal solution (assuming $L(0)=0$)

$$L = \beta \int_0^t S(\tau) I(\tau) \exp\left(-(\rho+m+h)(t-\tau)\right) d\tau\,. \tag{D5}$$

Now, if the product SI changes with time significantly slower than the exponent, then approximately it can be taken as a constant, and, after integrating and neglecting quickly decreasing exponent $\exp\left(-(\rho+m+h)t\right)$ responsible for initial transient period, we obtain

$$L \approx \frac{\beta SI}{\rho+m+h}\,. \tag{D6}$$

The same result can be obtained slightly differently, as it is done e.g. in (Haken 1983): if the decay rate for L is sufficiently faster than changes in SI, then approximately we can consider L as almost converged to its "asymptotic" state, where $dL/dt \approx 0$. Then the equation for L becomes algebraic rather than differential, and we can express L through SI with the above result (D6).

Substituting the expression for L into the equation for I we come to

$$\frac{dI}{dt} = \frac{\rho}{\rho + m + h}\beta SI - (m + h + \mu)I. \qquad \text{(D6)}$$

This equation looks like one in SI model with a different transmission coefficient,

$$\beta_{SI} \approx \frac{\rho}{\rho + m + h}\beta_{SLI}, \qquad \text{(D7)}$$

however, there one difference: here S is the size of the old susceptible population, and now we need to integrate the excluded latent compartment into it,

$$S' = S + L = \left(1 + \frac{\beta I}{\rho + m + h}\right)S = (1 + \alpha I)S, \qquad \alpha = \frac{\beta}{\rho + m + h}. \qquad \text{(D8)}$$

Then

$$\frac{dI}{dt} = \frac{\rho}{\rho + m + h}\frac{1}{1 + \alpha I}\beta S'I - (m + h + \mu)I. \qquad \text{(D9)}$$

Therefore, formally we can write an effective SI model

$$\frac{dS'}{dt} = b(S' + I) - (m + h)S' - \beta' S'I, \qquad \text{(D10)}$$

$$\frac{dI}{dt} = \beta' S'I - (m + h + \mu)I, \qquad \text{(D11)}$$

but, strictly speaking, the new transmission coefficient

$$\beta' = \frac{\rho}{\rho + m + h} \times \frac{1}{1 + \alpha I}\beta$$

now depends on the infection compartment and decreases as infection progresses. In the beginning of the epidemics $\beta' \approx \beta_{SI}$, see (D7), and then it diminishes until reaching of endemic equilibrium.

Therefore, if we assume that approximation (D6) is valid and associate with the SLI model (D1)-(D3) the following SI model:

$$\frac{dS}{dt} = b(S + I) - (m + h)S - \beta_{SI}SI, \qquad \text{(D10)}$$

$$\frac{dI}{dt} = \beta_{SI}SI - (m + h + \mu)I, \qquad \text{(D11)}$$

where β_{SI} is defined in (D7), then both models provide the same description of the initial stage of the epidemic. As the disease progresses, (D10), (D11) effectively has greater effective transmission coefficient, and hence has equal or worse disease development. Therefore, if population survives and the disease can be controlled in SI model, then the same should be true for SLI model as well.

The above derivations have been made for the case of density-dependent transmission, however similar arguments will work in case of frequency-dependent transmission as well.

We can summarize this section as follows. Depending on the duration of the endemic stage or the rate ρ of leaving it, there are three cases:

1) $\rho >> m+h$, that is the majority of individuals that get the infection leave the latent stage and end up as infected. Then $\beta_{SI} \approx \beta_{SLI}$ and the latent stage can be just ignored.

2) $\rho >\sim 2(m+h)$, then non-negligible part of individuals who get the infection do not make it to infected stage and die being at latent stage. In this case the dynamics of SLI model can be well approximated by SI model, but **with a smaller value of transmission coefficient** given by (D7). This decrease of the transmission coefficient is due to noticeable mortality in latent stage and reflects the fact that these individuals do not spread the infection further.

3) $\rho \leq\sim (m+h)$, then approximation (D6) does not work, and SI model may be not appropriate, though it still may give reasonable qualitative picture.

From the values cited in the beginning of this section, we can obtain that in case of CWD $\rho/(m+h) \approx 2.2$ and $\beta_{SI} \approx 0.7\beta_{SLI}$, so we fit into case 2: SI model well describes the disease dynamics, but must have different transmission coefficient compared to SLI model.

Why different models can have different values of transmission coefficients? Because these coefficients have <u>different meaning</u>. In DD SI model β_{SI} is per capita susceptible per capita infective rate of <u>new infected cases</u>. In DD SLI model β_{SLI} is per capita susceptible per capita infective rate of <u>new latent cases</u>. Since some latent animals die before becoming infected, these values must be different. Only when mortality in latent state is negligible (case 1), these coefficients have to be equal.

We repeated model calculations in the paper for a model with susceptible, latent and infective disease stages. The presence of the latent stage caused decrease of the prevalence ration $r_{\max}$. This decrease depends on the mean duration of the latent stage and for 6-9 months it is about 6%. However, all conclusions concerning transmission mechanisms remain unchanged.

Table A1 Aerial surveys in WMU 728, 730 give average estimates of population proportions. Application of (A35), (A36) give the estimates of per year survival.

Species	B	τ	Estimates from aerial survey data in WMU 728, 730			Estimates from (A35), (A36)		
			π_f	π_m	π_j	s_f	s_m	s_j
MD	1.63	1.5	**0.44±0.02**	**0.18±0.03**	**0.38±0.02**	0.75	0.49	0.45
WTD	1.83	1.0	**0.47±0.02**	**0.16±0.04**	**0.37±0.02**	0.67	0.31	0.49

Table A2. Averaged proportions in aerial survey data, in hunter kill data, and estimated population proportions before hunting season, WMU 728, 730

% deer observed	Average proportions								
	In aerial surveys, all years			Before hunting, estimate			Killed, since 1998		
	m	*f*	*j*	*m*	*f*	*j*	*m*	*f*	*j*
MD, 50	0.19	0.44	0.37	0.23	0.42	0.35	0.53	0.29	0.17
MD, 75	0.19	0.44	0.37	0.25	0.41	0.34	0.53	0.29	0.17
MD, 100	0.19	0.44	0.37	0.26	0.41	0.33	0.53	0.29	0.17
WT, 50	0.16	0.47	0.37	0.19	0.46	0.35	0.53	0.33	0.14
WT, 75	0.16	0.47	0.37	0.21	0.45	0.34	0.53	0.33	0.14
WT, 100	0.16	0.47	0.37	0.22	0.45	0.33	0.53	0.33	0.14

Table A3. Hunters' preferences and mortality coefficients from WMU 728, 730 data.

% deer observed	Hunting intensity h	Hunters' preferences			Mortality without hunting		
		h_{Pm}	h_{Pf}	h_{Pj}	μ_{xm}	μ_{xf}	μ_{xj}
MD, 50	0.30	1	0.30	0.21	0.38	0.19	1.19
MD, 75	0.38	1	0.33	0.23	0.29	0.15	1.16
MD, 100	0.45	1	0.36	0.26	0.22	0.12	1.14
WT, 50	0.24	1	0.27	0.15	0.89	0.32	1.34
WT, 75	0.31	1	0.29	0.17	0.81	0.29	1.32
WT, 100	0.38	1	0.31	0.18	0.74	0.27	1.31

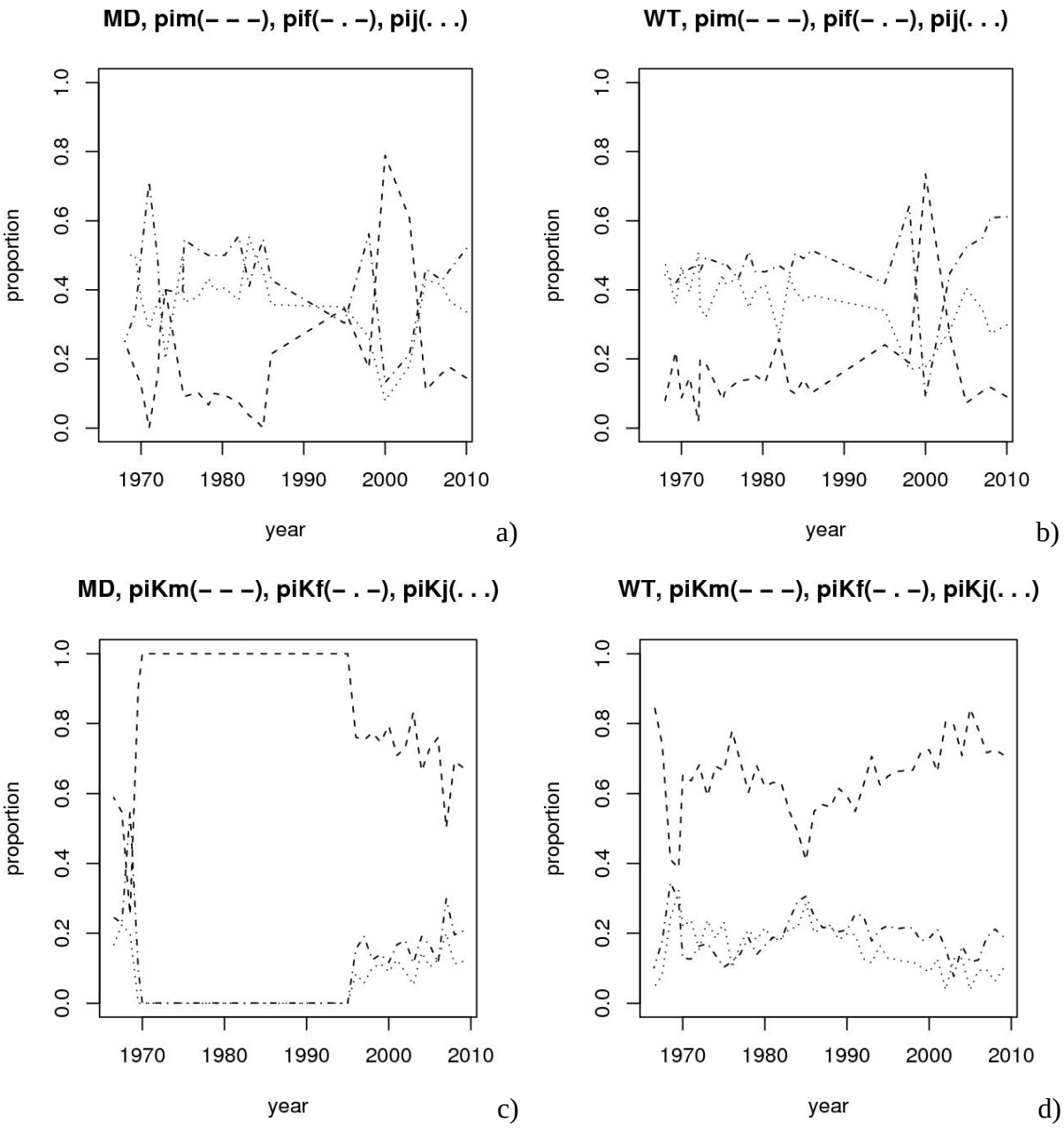

Fig. A3. Population proportions from aerial survey data (a,b) and proportions in killed animals (c,d) for mule deer (a,c) and white-tailed deer (b,d) in WMU 728, 730. Lines: males (dashed), females (dot-dashed) and juveniles (dotted). In 1970-1995 regulations allowed hunting only male mule deer, and for estimating hunters' preferences only the data for 1996-2000 were used.

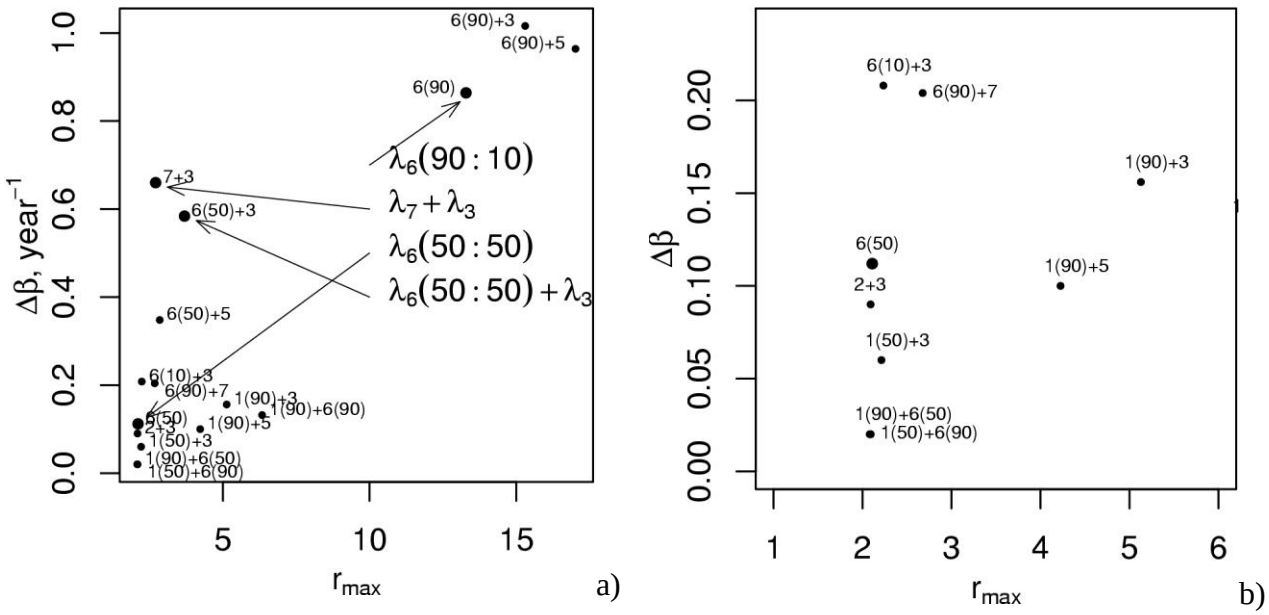

Fig. A4. (a) The plot shows the relationship between maximum male to female prevalence ratio at r_{max} and the range of transmission coefficient Δβ values where the ratio of male to female prevalence r_{mf} >2. Each dot represents either a single transmission mechanism or a combination of two mechanisms from Table 3. Numbers without brackets are the numbers of transmission mechanisms; numbers in brackets show the weight of separate groups in percents. For example, 6(50)+3 means combination of environmental within group transmission λ_6 with 50:50 weights and mating female to male transmission λ_3. We assume that the larger Δβ is, the more likely that given mechanisms are contributing to the observed prevalence difference in males and females. Both Δβ and r_{max} increase when the contribution of separate groups increases or when the rut female to male transmission (λ_3) is added. (b) Enlarged bottom left corner of the panel (a).

Table A4. Similar to Table 4, but with intensive harvest h=0.38.

Mechanism and w_S:w_M,	Basic only	Basic + mating f to m ($+\lambda_3$)	Basic + mating m to f ($+\lambda_4$)	Basic + mating m fights ($+\lambda_5$)
Single basic mechanisms				
Environmental within groups, 90:10	**12.4**	**14.7**	0.90	**15.1**
Environmental within groups, 50:50	1.83	**3.71**	0.75	**2.31**
Environmental within groups, 10:90	1.03	**2.27**	0.66	1.17
Direct within groups, 90:10	1.03	**3.45**	0.73	1.33
Direct within groups, 50:50	0.94	**2.00**	0.72	1.00
Direct within groups, 10:90	0.91	1.60	0.71	0.95
Environmental between groups	0.97	**2.87**	0.52	1.21
Direct between groups	0.90	**2.20**	0.63	0.98
Combinations of 2 basic mechanisms, r_{max}>2				
Direct within groups(90:10)+environmental within groups(90:10)	**3.45**	**5.53**	0.96	**4.34**
Environ within group(90:10)+environmental between groups	**2.43**	**3.94**	0.91	**2.87**
Direct within groups(90:10)+environmetnal within groups(50:50)	1.44	**2.77**	0.89	1.67
Direct within groups(50:50)+environmental within groups(90:10)	1.52	**2.68**	0.92	1.72
Direct between groups+environmental within groups(90:10)	1.58	**2.83**	0.90	1.80
Environ within groups(50:50)+environmental between groups	1.32	**2.48**	0.81	1.50
Direct within groups(90:10)+environmetnal between groups	1.02	**2.25**	0.78	1.15
Direct within groups(90:10)+environmental within groups(10:90)	1.06	1.98	0.83	1.15
Direct within groups(50:50)+environmental within groups(50:50)	1.13	1.99	0.86	1.22
Direct within groups(10:90)+environmental within groups(90:10)	1.19	1.99	0.89	1.29
Direct between groups+ environmental within groups(50:50)	1.28	**2.04**	0.81	1.29

Table A5. Values of $\Delta\beta$ for the combinations of transmission mechanisms given in Table 4, which are plotted in Fig. 5 and Fig. A6. **Bold font** shows the cases shown in Fig. 5 and Fig. A4 as larger circles.

Mechanism and w_S:w_M,	Basic only	Basic + mating f to m ($+\lambda_3$)	Basic + mating m to f ($+\lambda_4$)	Basic + mating m fights ($+\lambda_5$)
Single basic mechanisms				
Environmental within groups, 90:10	**0.86**	1.02	—	0.96
Environmental within groups, 50:50	**0.11**	**0.58**	—	0.35
Environmental within groups, 10:90	—	0.21	—	—
Direct within groups, 90:10	—	0.16	—	0.10
Direct within groups, 50:50	—	0.06	—	—
Direct within groups, 10:90	—	—	—	—
Environmental between groups	—	**0.66**	—	—
Direct between groups	—	0.09	—	—
Combinations of 2 basic mechanisms, $r_{max}>2$				
Direct within groups(90:10)+environmental within groups(90:10)	0.13	0.16	—	0.15
Environ within group(90:10)+environmental between groups	0.20	0.02	—	0.08
Direct within groups(90:10)+environmental within groups(50:50)	0.02	0.11	—	0.06
Direct within groups(50:50)+environmental within groups(90:10)	0.02	0.11	—	0.06
Direct between groups+environmental within groups(90:10)	—	0.17	—	0.08
Environ within groups(50:50)+environmental between groups	—	0.01	—	—
Direct within groups(90:10)+environmental between groups	—	0.09	—	—
Direct within groups(90:10)+environmental within groups(10:90)	—	0.05	—	—
Direct within groups(50:50)+environmental within groups(50:50)	—	0.04	—	—
Direct within groups(10:90)+environmental within groups(90:10)	—	0.04	—	—
Direct between groups+ environmental within groups(50:50)	—	0.06	—	—

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