Wildlife disease elimination and density dependence

Alex Potapov, Evelyn Merrill, and Mark A. Lewis

Supplementary material

Derivation of equations (4)-(6)

The population size is $n = S + I + V$ and therefore $dn/dt = dS/dt + dI/dt + dV/dt$ and equation for *n* is obtained by taking sum of (1), (2), and (3):

$$\frac{dn}{dt} = b(n)n - m(n)n - hn - \mu I .$$

The disease prevalence is $i = I/n$, therefore $I = in$, and after the substitution we obtain Eq. (4).

To derive equation for *i*, we use its definition again,

$$i = I/n \quad \text{or} \quad \ln i = \ln I - \ln n .$$

Differentiating the last equality by *t*, we obtain

$$\frac{1}{i}\frac{di}{dt} = \frac{1}{I}\frac{dI}{dt} - \frac{1}{n}\frac{dn}{dt}.$$

Taking into account that $(dI/dt)/I$ is Eq. (2) divided by *I*, and $(dn/dt)/n$ is (4) divided by *n*, we obtain

$$\frac{1}{i}\frac{di}{dt} = [\beta(n)S - (m(n)+\mu) - h] - [b(n) - m(n) - \mu i - h] =$$
$$= \beta(n)S - \mu - b(n) + \mu i.$$

Taking into account that $S = n - I - V = (1 - i - v)n$, after multiplying by *i*, we obtain (5).

Finally, using relations $v = V/n$, $\ln v = \ln V - \ln n$, and $\frac{1}{v}\frac{dv}{dt} = \frac{1}{V}\frac{dV}{dt} - \frac{1}{n}\frac{dn}{dt}$, similar to the previous case we obtain

$$\frac{1}{v}\frac{dv}{dt} = \left[\gamma\frac{S}{V} - m(n) - h\right] - [b(n) - m(n) - \mu i - h] = \gamma\frac{S}{V} - b(n) + \mu i .$$

Substituting $S/V = (1 - i - v)/v$ and multiplying by *v* we obtain (6).

Estimate of R_0 at the disease-free equilibrium

We can estimate R_0 right from its definition, the average number of secondary infections produced by an infected individual in a non-infected population (survival function method in [1]). Let the system be at the state of equilibrium $S=S_0$, $V=V_0$, $I=0$, $n=n_0=S_0+V_0$, and therefore $b(n_0) = m(n_0) + h$. At $t=0$ introduce small number I_0 of

infected individuals (primary infections). They will die at rate $m(n_0)+\mu+h$ and transmit the infection to susceptibles at rate $\beta(n_0)S_0I(t)$ and generate secondary infections. The number of primary infections depends on time as

$$\frac{dI_1}{dt} = -(m(n_0)+\mu+h)I_1,\ I_1(0)=I_0,\quad I_1(t)=I_0\exp(-(m(n_0)+\mu+h)t).$$

The rate of secondary infections produced by these primary ones is

$$\frac{dI_2}{dt} = \beta(n_0)S_0I_1(t),\quad I_2(0)=0,$$

$$I_2(t)=\int_0^t \beta(n_0)S_0I_1(t)dt = \frac{\beta(n_0)S_0I_0}{m(n_0)+\mu+h}(1-\exp(-(m(n_0)+\mu+h)t))$$

Therefore,

$$R_0 = \frac{I_2(\infty)}{I_1(0)} = \frac{\beta(n_0)S_0}{m(n_0)+\mu+h} = \frac{\beta(n_0)(n_0-V_0)}{m(n_0)+\mu+h} = \frac{\beta(n_0)n_0(1-v_0)}{m(n_0)+\mu+h},\qquad v_0=\frac{V_0}{n_0}.$$

Taking into account the equilibrium condition $b(n_0)=m(n_0)+h$, it can be written also as

$$R_0 = \frac{\beta(n_0)n_0(1-v_0)}{b(n_0)+\mu}.$$

It allows us to estimate the immunity threshold v_0, above which the disease cannot get established:

$$R_0(v_0)=1=(1-v_0)R_0(0),\quad v_0=1-1/R_0(0),\quad R_0(0)=\frac{\beta(n_0)n_0}{b(n_0)+\mu},$$

the well-known relation in the epidemiological literature.

Derivation of the critical value $\hat{\beta}_0$ (Fig. 1)

Substituting the equilibrium vaccinated proportion $v_0=\gamma/(\gamma+m+h)$ for the fixed vaccination rate γ into the expression for R_0, in case of FD transmission we obtain

$$R_0 = \frac{\beta_0}{m+h+\mu}\frac{m+h}{m+h+\gamma}.$$

The minimum harvest and vaccination rates allowing to stop the disease spread correspond to R_0=1, that is

$$m+h+\gamma=\frac{\beta_0(m+h)}{m+h+\mu}=\beta_0\left(1-\frac{\mu}{m+h+\mu}\right).$$

Therefore, the minimum necessary vaccination rate given h is

$$\gamma=\beta_0\left(1-\frac{\mu}{m+h+\mu}\right)-m-h.$$

The expression in the brackets is a growing function of h, saturating to 1 for large h, and the last term is a decreasing function of h. The behaviour of $\gamma(h)$ near h=0 depends on

the value of β_0: for small β_0 $\gamma(h)$ is a decreasing function, for large β_0 it can increase for small h values. The boundary between the two cases ($\beta = \hat{\beta}_0$) corresponds to the case when at h=0 $\gamma(h)$ is neither growing, nor a decreasing function, that is $\gamma'(0) = 0$.

$$\gamma'(h) = \beta_0 \frac{\mu}{(m+h+\mu)^2} - 1,$$

therefore, for $\beta = \hat{\beta}_0$

$$\gamma'(0) = \hat{\beta}_0 \left(\frac{\mu}{(m+\mu)^2} \right) - 1 = 0, \qquad \hat{\beta}_0 = \frac{(m+\mu)^2}{\mu}.$$

Endemic equilibrium $n_*,\ i_*,\ v_*$ for the model (4)-(6) in presence of harvest and vaccination, $\gamma > 0$

We assume $m(n) = m$ (DB regulation).

FD transmission, $\beta(n) = \beta_0$, and At the endemic equilibrium, assuming $i > 0$, n, i, v satisfy the following system of equations:

$$b(n) = m + h + \mu i,$$

$$\beta_0(1 - v - i) - \mu(1 - i) - b(n) = 0,$$

$$\gamma(1 - v - i) - (b(n) - \mu i)v = 0.$$

From the first equation $b(n) - \mu i = m + h$, and substituting this into the last one, we obtain

$$\gamma(1 - i) = (\gamma + m + h)v, \quad v = \frac{\gamma}{\gamma + m + h}(1 - i).$$

Then, substituting b and v into second equation

$$\beta_0(1 - v - i) = \mu(1 - i) + b(n) = \mu + m + h,$$

$$\beta_0 \left(1 - \frac{\gamma}{\gamma + m + h} \right)(1 - i) = \beta_0 \frac{m + h}{\gamma + m + h}(1 - i) = \mu + m + h,$$

$$1 - i = \frac{(\mu + m + h)(\gamma + m + h)}{\beta_0(m + h)}$$

Therefore, the final expressions for the endemic equilibrium are

$$v_* = \frac{\gamma(\mu + m + h)}{\beta_0(m + h)},$$

$$i_* = 1 - \frac{(\mu + m + h)(\gamma + m + h)}{\beta_0(m + h)} = 1 - \frac{1}{\beta_0}\left[\frac{\gamma\mu}{m + h} + \gamma + \mu + m + h \right],$$

and n_* should be determined from the relation $b(n_*) = m + h + \mu i_*$. It can be shown that for $\beta_0 > \mu$ n_* decreases with *h*. However, since n_* does enter into the expressions for i_*, v_*, they are always valid provided the solution $n_* > 0$ exists. Since $b(n)$ attains its maximum value at $n = 0$, the condition for existence of the solution is $m + h + \mu i_* < b(0)$, and we assume that it is satisfied.

Differentiating i_*, v_* by *h* we obtain

$$\frac{dv_*}{dh} = -\frac{\gamma\mu}{\beta_0 (m+h)^2} < 0,$$

$$\frac{di_*}{dh} = \frac{1}{\beta_0}\left[\frac{\gamma\mu}{(m+h)^2} - 1\right], \quad \frac{di_*}{dh} > 0 \text{ if } \gamma > \frac{(m+h)^2}{\mu}.$$

Therefore, proportion of immune individuals at endemic equilibrium is always reduced by harvest. However, the behavior of endemic disease prevalence depends on the immunization rate γ. For low immunization rate $\gamma < (m+h)^2/\mu$ harvest decreases the endemic prevalence: decrease of lifespan of infected individuals, and hence R_0, affects the disease spread stronger than the loss of immune individuals. However for $\gamma > (m+h)^2/\mu$, which corresponds to

$$v_* > v_{*C} = \frac{(\mu + m + h)(m+h)}{\beta_0 \mu} = \frac{m+h}{\mu R_0(0)},$$

the situation becomes opposite, and disease spread is more sensitive to the loss of immune individuals. One can see that the threshold value v_{*C} is inversely proportional to β_0 or R_0 of completely susceptible population, that is, the more contagious the disease is, the more likely is hampering of vaccination by harvest.

DD transmission, $\beta(n) = \beta_{DD} n$. Repeating the above derivations, we come to similar formulas,

$$v_* = \frac{\gamma(\mu + m + h)}{\beta_{DD} n_* (m+h)},$$

$$i_* = 1 - \frac{(\mu + m + h)(\gamma + m + h)}{\beta_{DD} n_* (m+h)},$$

$$b(n_*) = m + h + \mu i_*.$$

Now the expressions for i_*, v_* contain n_*, which makes the analysis much more complicated. For example, the endemic population size now satisfies

$$b(n_*) + \frac{\mu(\mu + m + h)(\gamma + m + h)}{\beta_{DD} n_* (m+h)} = m + h + \mu.$$

It can be shown that, like in FD case, n_* decreases with *h*, and hence creates an additional source of i_* decrease. Now the condition for i_* increase with *h* becomes

$$\gamma > \left[1 + (1 - i_*)\beta_{DD}\left|\frac{dn_*}{dh}\right|\right]\frac{(m+h)^2}{\mu}.$$

It is not clear, whether it can be satisfied or not.

Phase plane analysis of system with FD disease transmission and no vaccination

Here we develop the phase plane analysis for the system with $\beta(n)=\beta_0$, no vaccination and $b(n)$ qualitatively similar to one described by (10). We also assume that the mortality is a constant m and does not depend on density n. This is enough to apply phase plane analysis to the system (4), (5) with v=0:

$$\frac{dn}{dt}=[b(n)-m(n)-\mu i-h]n \tag{4}$$

$$\frac{di}{dt}=iF(n,0,i), \qquad F=\beta(n)(1-i)-\mu(1-i)-b(n)=(\beta_0-\mu)(1-i)-b(n) \tag{5}$$

We consider only nonnegative population numbers and disease prevalence so that $n\geq 0$, $i\geq 0$. There are two n-isoclines along which $dn/dt=0$:

a) $n=0$;

b) $b(n)-m(n)-\mu i-h=0$ or

$$i_n(n,h)=\frac{b(n)-m-h}{\mu}. \tag{A1}$$

When $0<i<i_n(n,h)$ n increases, and when $i>i_n(n,h)$ n decreases. There are two i-isoclines, along which $di/dt=0$:

c) $i=0$;

d) $F=(\beta_0-\mu)(1-i)-b(n)=0$ or

$$i_i(n)=1-\frac{b(n)}{\beta_0-\mu}. \tag{A2}$$

When $0<i<i_i(n)$ i increases, and when $i>i_i(n)$ i decreases.

Equilibria of the system are the points where both $dn/dt=di/dt=0$ and they correspond to the points of intersection of the isoclines. The number, location, and stability of the equilibria change with model parameters. We consider the effect of varying two parameters: harvest intensity h, which is our way of disease control, and the recruitment potential or maximum recruitment rate b_0, which is responsible for disease controllability in case of FD transmission. All other parameters are fixed. Depending on the value of b_0 the isoclines may intersect in three different ways shown in Fig. A1.

The system (4), (5) may have four types of equilibria:

- **Trivial equilibrium** $n=0$, $i=0$, intersection of isoclines (a) and (c).
- **Disease-free equilibrium** $n=n_0>0$, $i=0$ corresponds to the intersection point of isoclines (b) and (c) i.e. $i_n(n_0,h)=0$, or $b(n_0)=m+h$.
- **Extinction equilibrium** $n=0$, $i=i_0>0$, intersection of isoclines (a) and (d). Biologically this is the same situation as trivial equilibrium, no animal population and hence no infected individuals too. However, the two equilibria reflect the existence of two ways to extinction, the trivial equilibrium corresponds to population disappearance in the absence of infection, e.g. due to harvest. The extinction equilibrium corresponds to population disappearance in presence of the disease, where a certain proportion of the population is always infected.

- **Endemic equilibrium** corresponds to intersection of isoclines (b) and (d), that is $i_n(n,h)=i_i(n)$. It corresponds to coexistence of animal population and the disease.

The trivial equilibrium always exists. The other three may exist or disappear depending on the values of h and b_0, and their stability can change too. Below we assume that Eq. (7) holds at disease-free equilibrium for h=0, that is $\beta_0 > \mu + b = m + \mu$, and the disease can get established in the non-harvested population. Then the disease free equilibrium i=0, n=n_0 at h=0 is unstable, and the asymptotic state of the population depends on its maximum recruitment rate b_0 or recruitment potential. Stability of equilibria can be investigated by a standard linearization technique. In particular, it is easy to show that endemic equilibrium is always stable provided it exists, so graphically disease eradication means bringing isoclines (A1), (A2) to such a position that they do not intersect for n>0, i>0, but population remains nonzero. We omit linear analysis for brevity. Instead in Fig. A1 we show several example trajectories of the system which illustrate system behaviour in different parts of the phase plane. There are three possibilities:

Case 1. Low recruitment potential b_0: no endemic equilibrium, unstable disease-free equilibrium (disease can get established), stable extinction equilibrium, and hence population size eventually goes to zero, Fig. A1(a). When $i_n(n,h)$ is always below $i_i(n)$, they never intersect for n>0 and any h. This happens when $i_n(0,0) < i_i(0)$ or

$$b_0 < b_{0\text{coll}} = \frac{\beta_0 - \mu}{\beta_0}(m+\mu), \qquad \text{(A3)}$$

that is the recruitment potential is less than the collapse threshold.

Case 2. Medium recruitment potential b_0: at no harvest the endemic equilibrium is stable, and disease-free and extinction equilibria are unstable, Fig. A1(b). As harvest increases, the endemic equilibrium corresponds to lower and lower density, and eventually it merges with the extinction equilibrium, which becomes stable, and situation turns to one shown in Fig. A1(a). Therefore, there is population loss at too large a harvest. When at small n and h=0 $i_n(n,0) > i_i(n) > 0$, the isoclines must have an intersection point because at n=n_c they change ordering: $b(n_c)$=0 and $i_n(n_c,0) < 0 < i_i(n_c) = 1$. This happens when $i_n(0,0) > i_i(0) > 0$ or

$$\frac{\beta_0 - \mu}{\beta_0}(m+\mu) < b_0 < \beta_0 - \mu .$$

For too large h the intersection point disappears, so there is a threshold in harvest h=$h_{\max}$: $i_n(0,h_{\max}) = i_i(0)$ or

$$h_{\max} = \frac{b_0\beta_0}{\beta_0 - \mu} - m - \mu , \qquad \text{(A4)}$$

above which the population collapses.

Case 3. High recruitment potential b_0: the extinction equilibrium does not exist. At zero harvest there is stable endemic equilibrium and unstable disease-free equilibrium, Fig. A1(c). As harvest increases, the endemic equilibrium corresponds to smaller and smaller disease prevalence, and at strong enough harvest the endemic equilibrium merges with

disease-free one and then disappears and only stable disease-free equilibrium remains, Fig. A1(d). This happens when $i_i(0) < 0$ or recruitment potential exceeds the disease elimination threshold

$$b_0 > b_{0\text{elim}} = \beta_0 - \mu. \tag{A5}$$

There is also a harvest threshold $h=h_e$, and it corresponds to situation where the endemic equilibrium occurs at $i=0$. Then in (A1) and (A2) we have $i_n(n, h_e) = i_i(n) = 0$ or $b(n) = m + h_e$, and $b(n) = \beta_0 - \mu$, and eventually $h_e = \beta_0 - m - \mu$. For $h>h_e$ the disease cannot persist. However harvest should not be too strong to eliminate the population as well. From Fig. 1 it is clear that the condition for this is $h < b_0 - m$. Since $b_0 > \beta_0 - \mu$, $h_e < b_0 - m$ and at this harvest level the population persists.

Analysis of these three cases show that only in case 3 population reduction can eliminate the disease without driving the population to extinction, that is this method of population management requires sufficient recruitment potential, that is large enough b_0.

Now we shall consider the dependence of the endemic disease prevalence on harvest intensity.

When the endemic equilibrium n_*, i_* exists, it is easy to find endemic prevalence solving equation $i_n(n_*, h) = i_i(n_*) = i_*$. After simple transformations of these equalities we have

$$b(n_*) = m + \mu i_* + h, \qquad (\beta_0 - \mu)(1 - i_*) = b(n_*) \tag{A6}$$

or, solving this system for $b(n_*)$ and i_*,

$$b(n_*) = \frac{(\beta_0 - \mu)(m + h + \mu)}{\beta_0}, \tag{A7}$$

$$i_* = 1 - \frac{m + h + \mu}{\beta_0}. \tag{A8}$$

The maximum endemic prevalence corresponds to no harvest, $h=0$,

$$i_{*\max} = 1 - \frac{m + \mu}{\beta_0}. \tag{A9}$$

Eqs. (A7) and (A8) allow us to make two conclusions: a) the maximum endemic disease prevalence depends only on the ratio of mortality rate of infected individuals and transmission coefficient and does not depend on recruitment potential b_0; b) an increase of harvest lowers the prevalence provided the population does not collapse.

The endemic equilibrium exists only in cases 2 and 3, that is when (A7) has a solution, which is possible only if $b(n_*) < b_0$, or $h < h_{\max}$, see (A3). Substituting the expression for $h_{\max}$ into (A8), we obtain minimum achievable endemic equilibrium under harvest control,

$$i_{*\min} = 1 - \frac{b_0}{\beta_0 - \mu} \quad \text{or} \quad i_{*\min} = 0 \text{ provided } b_0 > \beta_0 - \mu. \tag{A10}$$

Therefore, the three cases considered above give the following results for the prevalence ranges:

1) with low recruitment potential, $b_0 < b_{0coll}$ (A3), the population collapses;
2) with medium recruitment potential, $b_{0\text{coll}} < b_0 < b_{0\text{elim}}$ (A3), (A5), the harvest can not make the endemic prevalence lower than $i_{*\min}$, and further harvest increase leads to population collapse;
3) with high recruitment potential, $b_0 > b_{0\text{elim}}$ (A5), the harvest can drive the disease prevalence down to zero.

These three cases are shown in Fig. 3.

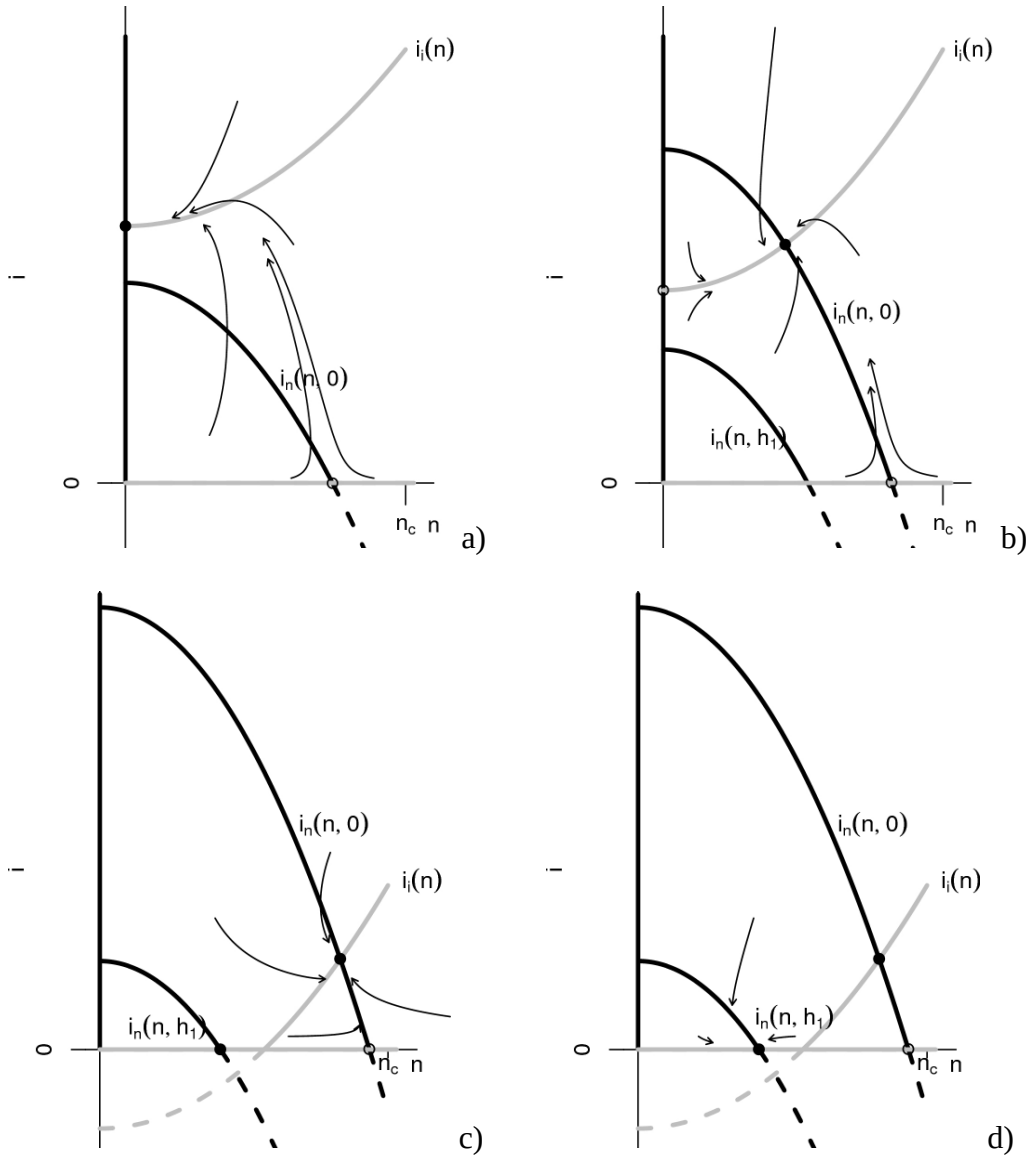

Fig. A1. Scheme of the three types of isocline behaviour. *n*-isoclines are shown as thick black lines, *i*-isoclines as thick gray lines, stable equilibria as black circles, and unstable equilibria as open circles. The behaviour of the system trajectories is shown by thin black lines with the arrows. a) If $i_n(n)$ is always higher than $i_n(n,h)$, these isoclines do not intersect at $n>0$, there is no endemic equilibrium. If the infection can get established (disease-free equilibrium is unstable), the population goes extinct. b) If at no harvest ($h=0$) and small n $i_n(n,h) > i_i(n)$ and $i_n(n) > 0$, the then endemic equilibrium exists (black circle). Harvest decreases endemic prevalence but can destroy the endemic equilibrium if h is too large ($h= h_1$); c and d) if $i_i(0) < 0$ then there is harvest threshold, beyond which only a disease-free equilibrium exists, and disease can be eliminated by population reduction ($h=h_1$).

Using SI model for a disease with environmental transmission: effective exclusion of environment compartment

It is known that prion diseases including CWD can be transmitted indirectly via environment (Miller et al 2004). However, at present there are no data as to how long they may remain accessible to deer, what is the rate of prion loss, how quickly prions decay (see e.g. discussion of prion mobility in soils by Schramm et al. (2006), evidence of prion degradation by Rapp et al. (2006), and review of recent results by Smith et al. (2011)). We base our approach upon the results presented by Miller et al. (2006). In their paper several models were compared against the data, and the best one was the model of environmental transmission. The second best was the SI model. From our point of view, this is not coincidence because, as we show below, under certain conditions SI model can effectively describe disease spread through the environment as well.

The best model in (Miller et al. 2006) is

$$\begin{aligned} \frac{dS}{dt} &= a - S(gE + m), \\ \frac{dI}{dt} &= gSE - I(m + \mu), \\ \frac{dE}{dt} &= \varepsilon I - \tau E. \end{aligned} \qquad \text{(A11)}$$

Here S and I are the number of susceptible and infected deer, E is the amount of prions in the environment, a is rate of deer birth, m is the rate of healthy deer mortality, μ is the mortality increase due to the disease, g is indirect transmission rate, ε is the per capita rate of prion accumulation, and τ is the rate of its loss. The estimated values are τ=2.55 year^{-1}; ε=0.111 mass/year; g=0.787 mass^{-1}year^{-1}, μ=0.57 year^{-1}.

According to these results, if prions are introduced into the environment at t=0, their amount $E(t)$ decreases with time as $E(t) = E_0 \exp(-\tau t)$, see Fig. A2. This means that after one year about 8% of original amount remains, and in 2 years only about 0.6%. The deer density changes slowly compared to this rate of prion washout. 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 than τ reported by Miller et al. (2006).

Therefore, $I(t)$ grows much shower than $E(t)$ decays. In such circumstances we can apply an approach described e.g. by Haken (1983), which is applicable when in the system there are variables with different relaxation rate, fast and slow. Then the slowly changing ones "enslave" the fast, and dynamics of the latter can be approximated by functions of just the slow variables. As a result, the original complex model can be replaced by a simpler one containing slow variables only. The accuracy of this approach depends on the difference between relaxation rates for slow and fast modes: the greater is the difference, the more accurate the method is. In other words, if the infection is washed out of the environment quickly enough, then the current amount of prions is proportional to the

number of infected individuals and a model of direct transmission (SI-type in our case) may be a good approximation of the indirect transmission one.

If we assume that the number of the infected deer in (A11) is changing only slightly in a single year, then the value of E practically converges to its asymptotic value $\varepsilon I/\tau$ before I changes. Hence, we may assume that approximately $E \approx \varepsilon I/\tau$. Substituting this relation into two remaining equations of (A11), we obtain

$$\begin{aligned} \frac{dS}{dt} &= a - S\left(\frac{g\varepsilon}{\tau} I + m\right), \\ \frac{dI}{dt} &= \frac{g\varepsilon}{\tau} SI - I(m+\mu), \end{aligned} \tag{A12}$$

which coincides with the SI model in (Miller et al. 2006) with $\beta=\gamma\varepsilon/\tau$. If we substitute the above values of g, ε, and τ, we obtain $\beta=0.034$ year^{-1}, which is very close to $\beta=0.0326$ year^{-1} obtained by fitting the SI model to data in (Miller et al. 2006). The difference in model performance detected by ΔAIC_c is most probably due to the effect of delay: when an infected deer appears in the area, accumulation of the prion in the environment takes some time, and new infections appear slightly later than required by SI model. Otherwise the disease pattern predicted by both models should be very close.

Therefore, if the hypothesis of a quick prion loss is valid, the model (A12) can be a good approximation to the model of environmental transmission (A11).

Here we apply this approach in deriving equations (1)-(3), where there is the possibility of frequency-dependent infection from environmental contamination. Deer are living in social groups, and direct contacts within a group are much more intensive than between groups. This is the reason why direct transmission should be primarily modeled by FD transmission. However, similar reasons may work for environmental transmission as well: members of the same group are staying close to each other, and have higher probability to pick up the infection after the member of the same group. Then, assuming that the environmental transmission mainly occurs within areas where the groups are, it is possible to show that parameter g in (A11) should be replaced by g/n. The model with within-group environmental transmission has the form

$$\frac{dS}{dt} = b(n)(S+I+V) - m(n)S - (\beta'(n)(I/n) + gE/n)S - \gamma S - hS \tag{A13}$$

$$\frac{dI}{dt} = (\beta'(n)(I/n) + gE/n)S - (m(n)+\mu)I - hI\,, \tag{A14}$$

$$\frac{dV}{dt} = \gamma S - m(n)V - hV\,, \tag{A15}$$

$$\frac{dE}{dt} = \varepsilon I - \tau E\,. \tag{A16}$$

Then, assuming $E \approx \varepsilon I/\tau$, one obtains the model (1)-(3) with $\beta(n) = \beta'(n) + \varepsilon g/\tau$, where the first term corresponds to direct transmission and the last one to environmental transmission. Further details of the approach are given in [11].

Additional references to this section

Haken, H., 1983. Synergetics : an introduction : nonequilibrium phase transitions and self-organization in physics, chemistry, and biology. Berlin; New York: Springer.

Miller, M.W., E. S. Williams, N. T. Hobbs, L. L. Wolfe. (2004) Environmental sources of prion transmission in mule deer. Emerging and Infectious Diseases 10: 1003-1006.

Rapp, D., Potier, P., Jocteur-Monrozier, L., Richaume, A. (2006) Prion Degradation in Soil: Possible Role of Microbial Enzymes Stimulated by the Decomposition of Buried Carcasses. Environ. Sci. Technol., 40, 6324–6329.

Schramm, P.T., Johnson, C.J., Mathews, N.E., McKenzie, D., Aiken, J.M., Pedersen, J.A. (2006) Potential Role of Soil in the Transmission of Prion Disease. Reviews in Mineralogy and Geochemistry 64, 135-152.

Smith, C.B, Booth, C.J., Petersen, J.A. (2011) Fate of prions in soil: A review. Journal of Environmental Quality 40, 449-461.

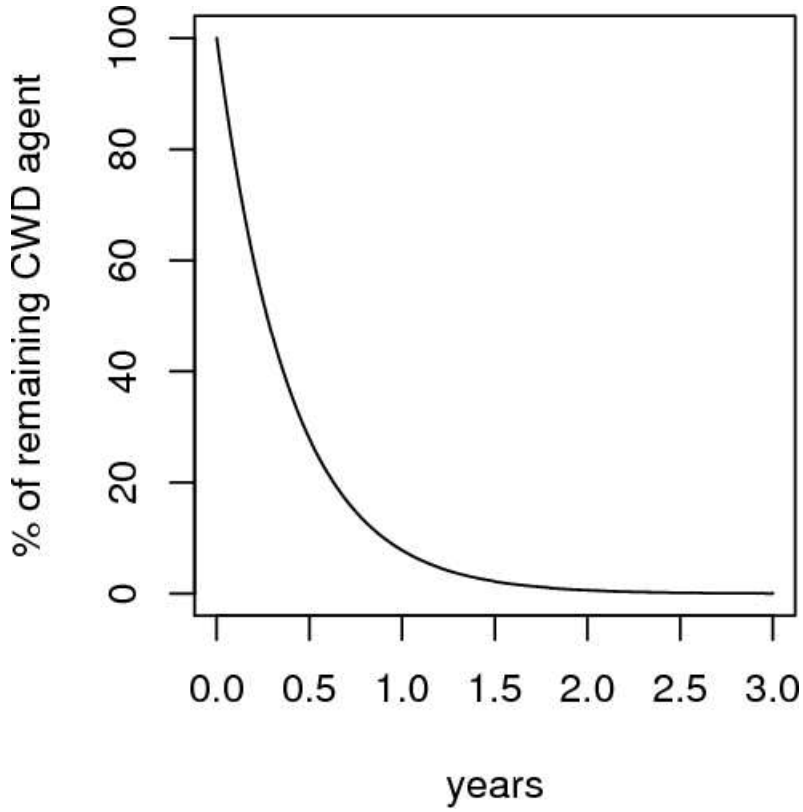

Fig. A2

Estimates of harvest effect for the model of Choisy & Rohani [20]

Here we derive some values important for the comparison of the effects found in [20] with the results of our paper. To simplify the estimates we 1) use the same notation *d* for all parameters equal to it; 2) denote *h=qH*. Then the version of the model system used for the analysis of the steady state has the form

$$S' = (b - dN(t-\tau))N - (dN + h + \lambda)S,$$
$$E' = \lambda S - (dN + h + \sigma)E,$$
$$I' = \sigma E - (dN + h + \gamma + v)I,$$
$$R' = \gamma I - (dN + h)R,$$
$$\lambda = \beta \frac{I}{N}, \qquad N = S + E + I + R.$$

The parameters are

$$\beta = 2000\,\text{year}^{-1},\ \gamma = 73\,\text{year}^{-1},\ \sigma = 45.6\,\text{year}^{-1},$$
$$b = 0.2\,\text{year}^{-1},\ d \sim 10^{-9},\quad v = 0.$$

Since $\beta, \gamma, \sigma >> b, dN, h$, it is possible to do approximate calculations of the equilibrium state:

$$0 = (b - dN(t-\tau))N - (dN + h + \lambda)S,$$
$$0 = \lambda S - (dN + h + \sigma)E,$$
$$0 = \sigma E - (dN + h + \gamma)I,$$
$$0 = \gamma I - (dN + h)R.$$

Summing up the equations gives

$$N = (b - h)/2d, \quad dN + h = (b + h)/2.$$

Then,

$$E = \frac{\lambda S}{dN + h + \sigma},$$
$$I = \frac{\sigma E}{dN + h + \gamma} = \frac{\sigma}{dN + h + \sigma} \times \frac{\beta SI/N}{dN + h + \gamma},$$

or, up to linear terms in *h*,

$$S = N\frac{(dN + h + \sigma)(dN + h + \gamma)}{\beta\sigma} \approx \frac{\gamma}{\beta} N\left(1 + \frac{dN + h}{\gamma} + \frac{dN + h}{\sigma}\right),$$

On the other hand

$$R = \frac{\gamma}{dN + h} I, \qquad E = \frac{dN + h + \gamma}{\sigma} I,$$
$$N - S = E + I + R,$$
$$\left[1 - \frac{\gamma}{\beta}\left(1 + \frac{dN + h}{\gamma} + \frac{dN + h}{\sigma}\right)\right] N = \left[1 + \frac{dN + h + \gamma}{\sigma} + \frac{\gamma}{dN + h}\right] I,$$

Neglecting the term $(dN + h)^2/\sigma << 1$, we have

$$\left[1-\frac{\gamma}{\beta}-\left(1+\frac{\gamma}{\sigma}\right)\frac{dN+h}{\beta}\right](dN+h)N=\left[\gamma+\left(1+\frac{\gamma}{\sigma}\right)(dN+h)\right]I=\gamma\left[1+\left(1+\frac{\gamma}{\sigma}\right)\frac{dN+h}{\gamma}\right]I,$$

Substituting here $dN+h=(b+h)/2$ and taking into account that up to linear terms

$$\left[1+\left(1+\frac{\gamma}{\sigma}\right)\frac{b+h}{2\gamma}\right]^{-1}\approx 1-\left(1+\frac{\gamma}{\sigma}\right)\frac{b+h}{2\gamma},$$

and neglecting quadratic terms in $(b+h)$ and terms ~b/β, after simple but bulky algebra we come to population proportions under harvest:

$$\frac{S}{N}\approx\frac{\gamma}{\beta}+\frac{\gamma+\sigma}{2\beta\sigma}h=s_0+s_1h,\quad s_0=0.037,\ s_1=6.5\times10^{-4},$$

$$\frac{R}{N}\approx 1-\frac{\gamma}{\beta}-\frac{(\gamma+\sigma)}{2\gamma\sigma}b-\left(1-\frac{\gamma}{\beta}\right)\frac{(\gamma+\sigma)}{2\gamma\sigma}h=r_0-r_1h,\quad r_0=0.96,\ r_1=0.018,$$

$$\frac{I}{N}\approx\frac{1}{2\gamma}(b+h)=i_0+i_1h,\quad i_0=0.0013,\ i_1=0.0066,$$

$$\frac{E}{N}\approx\frac{1}{2\sigma}(b+h)=e_0+e_1h,\quad e_0=0.0021,\ e_1=0.011.$$

We can see that the strongest relative effect harvest has on the disease prevalence, however the strongest absolute effect it has on the proportion of recovered individuals. Harvest removes immune individuals, they are replaced by new susceptibles due to increased birth rate, and since the disease is highly contagious, the susceptible class almost does not grow. Almost all additional susceptibles go straight into the exposed and infected classes, which noticeably grow, but cannot become very big because of short disease duration.

Harvest reduces the lifetime of the infected individuals, but since β is very large, they still leave behind too many secondary infections for a noticeable effect on the decrease of the disease spread rate.

Repeating the derivations of R0 for the model (1)-(3) for the case of the model [20], one obtains the following.

The disease-free equilibrium is

$$S=N,\ E=I=R=0.$$

Then primary infectives $I_1(t),\ I_1(0)=I_0<<N$ satisfy the equation

$$I_1'=-(dN+h+\gamma+v)I_1,\qquad I_1(t)=I_0\exp(-(dN+h+\gamma+v)t).$$

Secondary exposed $E_{21}(t),\ E_2(0)=0$ satisfy

$$E_2'=\beta\frac{I_1(t)}{N}S-(dN+h+\sigma)E_2,$$

$$E_2(t)=\frac{\beta I_0}{\gamma+v-\sigma}[\exp(-(dN+h+\sigma)t)-\exp(-(dN+h+\gamma+v)t)].$$

The growth rate for the number of secondary infections $I_2(t)$, $I_2(0)=0$ is $\sigma E_2(t)$, and hence the total number of secondary cases is

$$I_2(\infty)=\int_0^\infty \sigma E_2(t)dt = I_0\,\frac{\beta\sigma}{(\sigma+dN+h)(\gamma+v+dN+h)},$$

and, taking into account that $dN+h=(b+h)/2$, we obtain

$$R_0=\frac{\beta\sigma}{(\sigma+(b+h)/2)(\gamma+v+(b+h)/2)}\approx\frac{\beta}{\gamma}\left(1-\frac{b+h}{2\sigma}-\frac{b+h}{2(\gamma+v)}\right)\approx 27.3-0.5h\,.$$

We can see that harvest can slightly decrease the basic reproduction number, but the relative effect is much weaker than that of the decrease of the number of immune individuals.

Estimate of transmission coefficient from deer population and harvest data for Wisconsin

According to Wisconsin Department of Natural Resources [WI1], deer population is about 1 million of individuals, and about 350,000 deer are harvested per year. Therefore, about 30% of population is removed, and harvest intensity for Wisconsin can be estimated as

$$h\approx\log(1-0.3)/1\text{year}=0.36\,\text{year}^{-1}.$$

The CWD prevalence growth rate in Wisconsin is shown in [WI2], separately for males and females, with the average exponent $\lambda\approx 0.087\,\text{year}^{-1}$ (growth about two times in 2002-2010). Assuming $\mu\approx 0.57\,\text{year}^{-1}$ and $m\approx 0.06\,\text{year}^{-1}$, we obtain from (11)

$$\lambda=\beta_0-\mu-m-h\approx 0.087\,\text{year}^{-1},$$

or

$$\beta_0=\lambda+\mu+m+h\approx 1.08\,\text{year}^{-1}.$$

Assuming that Wisconsin deer population is near equilibrium, we obtain the estimate of the recruitment rate for disease free population,

$$b(n)=m+h\approx 0.42\,\text{year}^{-1}$$

and in case of CWD prevalence near 10%

$$b(n)=m+h+i\mu\approx 0.48\,\text{year}^{-1}.$$

[WI1] Wisconsin's Deer Management Program,
http://dnr.wi.gov/org/land/wildlife/hunt/deer/deerbook.pdf

[WI2] CWD in Wisconsin, Prevalence & Surveillance,
http://dnr.wi.gov/org/land/wildlife/whealth/issues/CWD/prevalence.htm