Evolution of resistance to white pine blister rust in high-elevation pines

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The basic ecology
High-elevation White Pines

- Habitat: dry, exposed, high altitude sites (Rocky Mtns.)
- Occupies habitat other species cannot
- Long-lived species
- Keystone species
Threats to HEWPs

Climate change

Mountain Pine Beetle

White pine blister rust
White Pine Blister Rust

- *Cronartium ribicola*
- Non-native fungal pathogen
- Lethal in HEWP
- Spreading despite extensive control efforts
Effects of WPBR
WPBR Life Cycle

Two Obligate Hosts

- Ribes spp.
- Spring
- Sub-lethal

- HEWP
- Fall
- Lethal

aeciospores

basidiospores
B = Basic model
Consider a stage-structured population model in the form of a nonlinear map

\[
x\{n\}(t + 1, p) = h\{n\}(x\{n\}(t, p), p)
\]

\[
x\{n\}(0, p) = x\{n\},0
\]

where

\[
h\{n\} : \mathbb{R}^n \times \mathbb{R}^m \mapsto \mathbb{R}^n.
\]

**Notation**

- The subscript \(\{n\}\) is used to denote the size of the system.
- \(x\{n\}(t, p)\) is the solution vector containing \(x_i\), the density of individuals (individuals per hectare) in stage \(i = 1, \ldots, n\) at times \(t = 0, 1, \ldots, T\) (years).
- \(p\) is the vector of parameters \(p_j, j = 1, \ldots, M\).

Nonlinear o.d.e. models are analogous.
Assumption: Fecundity occurs after survivorship & transition.

Consequently,

$$x_{\{n\}}(t + 1) = g_{\{n\}}(x_{\{n\}}(t)) + f_{\{n\}}(g_{\{n\}}(x_{\{n\}}(t)))$$

where $f$ denotes fecundity and $g$ denotes survivorship and transition.

*Note:* When considering a genetic model, a splitting between survivorship & transition and fecundity arises naturally since survivorship & transition occurs within genotypes while fecundity occurs across genotypes.

Note: For simplicity, we drop the explicit dependence of solution $x_{\{n\}}(t)$ on parameters $p$. 
We will use

- $\hat{x}$ to denote the outcome of survivorship & transition,
- $\hat{y}$ as the vector on which fecundity operates,
- $\bar{x}$ or $\bar{y}$ to denote intermediate quantities.

**Assumption:** Disease status and genotype affect the *arguments (inputs)* of the functions modeling survivorship & transition and fecundity.
D = Disease
**D: Incorporating disease**

Assumption: There are only susceptible and infected populations.

Let

\[
\mathbf{x}_{2n} = \begin{pmatrix} \mathbf{x}_S^{\{n\}} \\ \mathbf{x}_I^{\{n\}} \end{pmatrix},
\]

and

\[
g_{2n}(\mathbf{x}_{2n}) = \begin{pmatrix} g_{\{n\}}(\mathbf{x}_S^{\{n\}}) \\ g_{\{n\}}(\mathbf{x}_I^{\{n\}}) \end{pmatrix}.
\]

White bark pines do not recover from blister rust infection.
Assumption: The effect of disease can be described in terms of a linear weighting applied to the argument of the function modeling survivorship & transition.

Let the cost of infection to survivorship & transition be

\[
C_\{2n\}^\nu = \begin{pmatrix}
I_{\{n\}} & 0 \\
0 & C_\{n\}^\nu
\end{pmatrix}, \quad C_\{n\}^\nu = \text{diag}(c_i), \ i = 1, \ldots, n,
\]

and define

\[
\hat{x}_{\{2n\}} = g_{\{2n\}} \left( C_{\{2n\}}^\nu \ast x_{\{2n\}} \right) = \begin{pmatrix}
g_{\{n\}}(x_{\{n\}}^S) \\
g_{\{n\}}(C_{\{n\}}^\nu \ast x_{\{n\}}^I)
\end{pmatrix}
\]
Fecundity is modeling through the action of the non-linear vector valued function

\[ f_{2n} : \mathbb{R}^{2n} \mapsto \mathbb{R}^{2n}, \]

but

\[ f_{2n} \neq \begin{pmatrix} f_n(x^S) \\ f_n(x^I) \end{pmatrix}. \]

Think vertical vs horizontal transmission.

Both susceptible and infected white bark pine produce susceptible seeds. (Furthermore, seeds cannot become infected.)
Assumption: The effect of disease on fecundity can be described as a weighting applied to the argument of the nonlinear function used to model fecundity.

Let the cost of fecundity be

\[ C_{2n}^\varphi = \begin{pmatrix} I & 0 \\ 0 & C_n^\varphi \end{pmatrix}. \]

and define fecundity in the SI model to be

\[ f_{2n} \left( C_{2n}^\varphi \ast \tilde{x}_{2n} \right). \]
D: Infectivity

Assumption: Infection occurs after survivorship, transition and fecundity and occurs at a constant rate.

Let

\[
B_{\{2n\}} = \begin{pmatrix}
I_{\{n\}} - B_{\{n\}} & 0 \\
B_{\{n\}} & I_{\{n\}}
\end{pmatrix},
\]

where

\[
B_{\{n\}} = \text{diag}(b_i), \ i = 1, \ldots, n
\]

and \(I_{\{n\}}\) is the \(n \times n\) identity matrix.
Finally, the full SI disease model is the nonlinear map

\[ x_{2n}(t + 1) = B_{2n} \ast (\hat{x}_{2n} + f_{2n}(\hat{y}_{2n})) , \]

where

\[ \hat{x}_{2n} = g_{2n}(C^{\nu}_{2n} \ast x_{2n}) , \]

and

\[ \hat{y}_{2n} = C^{\varphi}_{2n} \ast \hat{x}_{2n} . \]
D: SI disease model

The cost of disease on survival, transition, the cost of disease on fecundity and infectivity could all be nonlinear.

The SI disease model would then become

\[ x_{\{2n\}}(t + 1) = B_{\{2n\}} (\hat{x}_{\{2n\}} + f_{\{2n\}}(\hat{y}_{\{2n\}})) , \]

where

\[ \hat{x}_{\{2n\}} = C^{\nu}_{\{2n\}} (x_{\{2n\}}) , \]
\[ \hat{y}_{\{2n\}} = C^{\phi}_{\{2n\}} (\hat{x}_{\{2n\}}) . \]

Conceptually this is no more complicated.
$P = \text{Pine model}$
The basic pine model has six stages or classes,

\[ x \{6\} = \begin{pmatrix}
    SEEDS \\
    SD1 \\
    SD2 \\
    SA \\
    YA \\
    MA
\end{pmatrix} = \begin{pmatrix}
    x_1 \\
    x_2 \\
    x_3 \\
    x_4 \\
    x_5 \\
    x_6
\end{pmatrix}. \]

Units: Individuals / hectare
Consistent with the general framework, we define the nonlinear map

\[ x_{6}(t + 1) = g_{6}(x_{6}(t)) + f_{6}(g_{6}(x_{6}(t))) \]

where \( f \) and \( g \) represent fecundity and survivorship & transition respectively.

Units: Time is measured in years.

Model structure and coefficient values is the result of extensive field studies.
Assumption: Seedling recruitment occurs before survivorship & transition

The modeling of the germination process involves a number of auxiliary quantities, including properties of the surrounding forest, and the foraging and seed storing behaviors of the Clark’s nutcracker.

- Leaf Area Index (LAI)
- Seed caching
- \( P_{\text{find}} \)
- …
Leaf Area Index (LAI) is used as a proxy for tree density and is defined as a function of tree size as measured by the diameter at breast height.

Let

\[ l_{area_1} = l_{area_2} = 0, \quad l_{area_3} = \alpha_1, \quad l_{area_i} = \alpha_2 (d_i)^{\alpha_3}, \quad i = 4, 5, 6, \]

where

\[ \alpha_1 = 0.456, \alpha_2 = 0.117, \alpha_3 = 1.925, \]

and the diameter at breast height (dbh) measurements for the six classes are

\[ d_1 = 0, \quad d_2 = 0, \quad d_3 = 0, \quad d_4 = 2.05, \quad d_5 = 12.5, \quad d_6 = 37. \]

Secondary seedlings have \( d_3 = 0 \) but do possess leaf area, thus \( \tilde{l}_{area_3} \) is calculated separately. Let

\[ \text{LAI}(x_6) = \frac{(l_{area}, x_6)}{10000}. \]
We define

\[ \text{SpB}(x_{\{6\}}) = \frac{x_1}{\text{nBirds}}, \]
\[ r_{cache}(x_{\{6\}}) = \frac{0.73}{1 + \exp \left( (31000 - \text{SpB}(x_{\{6\}}))/3000 \right)} + 0.27, \]
\[ r_{ALS}(x_{\{6\}}) = \frac{1}{1 + \exp \left( 2 \left( \text{LAI}(x_{\{6\}}) - 3 \right) \right)}, \]

where \( x_1 \) is the number of seeds, and finally,

\[ r_2(x_{\{6\}}) = \left[ \frac{(1 - P_{\text{find}}) (1 - P_{\text{cons}})}{\text{SpC}} \right] r_{cache}(x_{\{6\}}) r_{ALS}(x_{\{6\}}). \]

The number of new seedlings is then given by

\[ \gamma_{\{6\}}(x_{\{6\}}) = r_2(x_{\{6\}}) * x_1 * e_2, \]

where \( e_2 \in \mathbb{R}^6 \) is the unit vector with a single nonzero entry in the second position.
We define the intermediate vector

$$ \overline{x}_{6} = x_{6} + \gamma_{6}(x_{6}), $$

where $\gamma_{6}(x_{6})$ is the result of the nonlinear seedling recruitment process.
P: Linear survivorship & transition

Let

\[ S_{\{6\}} = \begin{pmatrix}
1 & 0 & 0 & 0 & 0 & 0 \\
0 & s_2 & 0 & 0 & 0 & 0 \\
0 & t_2 & s_3 & 0 & 0 & 0 \\
0 & 0 & t_3 & s_4 & 0 & 0 \\
0 & 0 & 0 & t_4 & s_5 & 0 \\
0 & 0 & 0 & 0 & t_5 & s_6
\end{pmatrix}. \]

The coefficients along the diagonal of \( S_{\{6\}} \) are

\[ s_2 = 0.636, \quad s_3 = 0.8391, \quad s_4 = 0.9310, \quad s_5 = 0.9653, \quad s_6 = 0.995. \]

Coefficients along the sub-diagonal of \( S_{\{6\}} \) are

\[ t_2 = 0.212, \quad t_3 = 0.0559, \quad t_4 = 0.0490, \quad t_5 = 0.0197. \]
We define the outcome of both nonlinear and linear survivorship and transition as

\[ \hat{x}_6 = g_6(x_6) = S_6 * \bar{x}_6. \]
We define the nonlinear function

\[ f_{\{6\}} : \mathbb{R}^6 \times \rho \mapsto \mathbb{R}^6 \]

where \( \rho \) is a measure of the effect of class on fecundity.

The modeling of the fecundity process involves a number of auxiliary quantities.

- Maximum number of cones per tree
- Number of seeds per cone
- \ldots
Seed production is determined by the number of seeds per cone, which is determined as

\[
C_{\text{tree}}(\mathbf{x}_6) = \left[ \frac{0.5}{1 + \exp(5 \left( \text{LAI}(\mathbf{x}_6) - 2.25 \right))} + 0.5 \right] C_{\text{max}}
\]

and

\[
r_1(\mathbf{x}_6) = S_{\text{cone}} \times C_{\text{tree}}(\mathbf{x}_6).
\]

The number of seeds produced is then given by

\[
f_6 = r_1(\hat{\mathbf{x}}_6) \times (\rho \hat{x}_5 + \hat{x}_6) \times \mathbf{e}_1
\]

where \( \mathbf{e}_1 \in \mathbb{R}^6 \) is a unit vector with a single nonzero entry in the first position.
Finally

\[ x_6(t + 1) = \hat{x}_6 + f_6(\hat{x}_6). \]
PD = Pine + disease
We double the number of classes and define

\[
\mathbf{x}_{\{12\}} = \begin{pmatrix} \mathbf{x}_S^{\{6\}} \\ \mathbf{x}_I^{\{6\}} \end{pmatrix} = \begin{pmatrix} \text{SEEDS} \\ \text{susceptible SD1} \\ \text{susceptible SD2} \\ \text{susceptible SA} \\ \text{susceptible YA} \\ \text{susceptible MA} \\ 0 \\ \text{infected SD1} \\ \text{infected SD2} \\ \text{infected SA} \\ \text{infected YA} \\ \text{infected MA} \end{pmatrix} = \begin{pmatrix} x_1 \\ x_2 \\ x_3 \\ x_4 \\ x_5 \\ x_6 \\ x_7 \\ x_8 \\ x_9 \\ x_{10} \\ x_{11} \\ x_{12} \end{pmatrix}.
\]
1) Seedling recruitment: $r_z(\tilde{x}_n)$

2) Survival & transition: $\tilde{g}_n$

3) Seed production: $\vec{f}_n$

4) Infection: $B_{(n)}$
Arrows:
- Survival & transition
- Infection
- Fecundity

Nodes:
- Susceptible
- Infected
Assumption: Seedling recruitment occurs before survivorship & transition.

We define

$$\bar{x}_{12} = \begin{pmatrix} x^S_{6} \\ x^I_{6} \end{pmatrix} + \begin{pmatrix} \gamma^D_{6} (x_{12}) \\ 0 \end{pmatrix}$$

where the nonlinear function $\gamma^D_{6}$ is the modification of $\gamma_{6}$.

Both susceptible and diseased individuals provide shading.
Let

\[ S_{\{12\}} = \text{diag}(S_{\{6\}}, S_{\{6\}}). \]

The cost of infection on survival & transition is given by

\[ C_{\{12\}}^\nu = \text{diag}(I_{\{6\}}, C_{\{6\}}^\nu) \]

where

\[ C_{\{6\}}^\nu = \text{diag}(0, c_2, \ldots, c_6), \]

with the cost of infection related to tree size, specifically

\[ c_2 = 0.01, \ c_3 = 0.13, \ c_i = 1 - \exp(-\delta d_i), i = 4, 5, 6. \]
We define

\[
\hat{x}_{12} = g_{12}(x_{12})
\]

\[
= S_{12} \ast \left( C^\nu_{12} \ast \overline{x}_{12} \right)
\]

\[
= \begin{pmatrix} S_{6} & 0 \\ 0 & S_{6} \end{pmatrix} \ast \begin{pmatrix} I_{6} & 0 \\ 0 & C^\nu_{6} \end{pmatrix} \ast \begin{pmatrix} \overline{x}^S_{6} \\ \overline{x}^I_{6} \end{pmatrix}
\]

\[
= \begin{pmatrix} S_{6} & 0 \\ 0 & S_{6} C^\nu_{6} \end{pmatrix} \ast \begin{pmatrix} \overline{x}^S_{6} \\ \overline{x}^I_{6} \end{pmatrix}.
\]
Assumptions:

- Infection is not transmitted vertically. Susceptible and infected adults produce susceptible seeds.

- Fecundity is modeled via cone production only because we assume fertilization occurs via a "pollen cloud", with each individual contributing equally to the cloud. Thus pollen is not limiting and differences in fecundity can be seen as a result of differences in available resources allocated to cone production.

- Pollen production is unaffected by infection because the pollen producing branches primarily compose the lower crown, while the seed cone producing branches dominate the upper crown. Infection disproportionately affects the upper crown (top-kill), thus mitigating the effect of infection on pollen relative to cone production.
We define the nonlinear function

\[ f_{12} : \mathbb{R}^{12} \times (\rho, C^{\varphi})^\top \mapsto \mathbb{R}^{12} \]

where

- \( \rho \) is a measure of the effect of class on fecundity,
- \( C^{\varphi} \) is a measure of the effect of disease on fecundity.
Let $y_{\{12\}} = \hat{x}_{\{12\}}$ and

$$y^S = \rho y_5 + y_6, \quad y^I = \rho y_{11} + y_{12},$$

and

$$z = y^S + C^\varphi y^I,$$

so that

$$f_{\{12\}}(\hat{x}_{\{12\}}; \rho, C^\varphi) = r_1(\hat{x}_{\{12\}}) \ast z \ast e_1,$$

where the function $r_1(x_{\{12\}})$ is an obvious extension of $r_1(x_{\{6\}})$. Think shading.
Assumption: Infectivity occurs at a constant rate which may vary according to age class.

We define

$$B_{\{12\}} = \begin{pmatrix} I_{\{6\}} - B_{\{6\}} & 0 \\ B_{\{6\}} & I_{\{6\}} \end{pmatrix}$$

where

$$B_{\{6\}} = \text{diag}(0, b_2, b_3, b_4, b_5, b_6)$$

and

$$b_2 = b_3 = b_4 = b_5 = b_6 = 0.044.$$
Assumption: Disease transmission occurs after fecundity.

Finally

\[ \mathbf{x}_{12}(t + 1) = B_{12} \ast (\hat{\mathbf{x}}_{12} + \mathbf{f}_{12}(\hat{\mathbf{x}}_{12}; \rho, C^\varphi)), \]

where

\[ \hat{\mathbf{x}}_{12} = g_{12}(\mathbf{x}_{12}). \]
Sensitivity analysis
Sensitivity wrt parameters

Using index notation,

\[ x_i(t + 1, p) = h_i(\mathbf{x}(t, p), p), \]
\[ x_i(0) = z_i \]
\[ \begin{array}{c}
\end{array} \] \( i = 1, \ldots, n \)

Differentiating with respect to parameters, \( p_k \), gives

\[ \begin{aligned}
\frac{\partial x_i(t + 1)}{\partial p_k} &= \frac{\partial h_i}{\partial x_m} \cdot \frac{\partial x_m(t)}{\partial p_k} + \frac{\partial h_i}{\partial p_k} \\
\frac{\partial x_i(0)}{\partial p_k} &= 0
\end{aligned} \]
\[ \begin{array}{c}
\end{array} \] \( i = 1, \ldots, n, \quad k = 1, \ldots, K \)

Elasticities are relative sensitivities,

\[ E_{i,k} = \frac{p_k}{x_i} \frac{\partial x_i}{\partial p_k}. \]
Sensitivity wrt initial conditions

To determine stability with respect to the initial conditions, we differentiate with respect to the initial conditions to give

$$\begin{align*}
\frac{\partial x_i(t+1)}{\partial z_k} &= \frac{\partial h_i}{\partial x_m} \ast \frac{\partial x_m(t)}{\partial z_k} + \frac{\partial h_i}{\partial z_k} \\
\frac{\partial x_i(0)}{\partial z_j} &= \delta_{ij}
\end{align*}$$

$$\{ i = 1, \ldots, n, \\
k = 1, \ldots, K \}$$

To solve this for the population and its stability with respect to parameters and initial conditions we evolve all equations simultaneously.

Perform calculations using the MATLAB / MAPLE package SENSAI*.

Results
Model Results I

Regeneration from 1000 SD$_1$

- Stages 2-6
- Eq$^m = 626$
- No disease ($\beta = 0$)

\[
\bar{x}_{12} = \begin{pmatrix}
62580 \\
38 \\
79 \\
65 \\
91 \\
353 \\
0 \\
0 \\
0 \\
0 \\
0 \\
0
\end{pmatrix}
\]
Model Results II

- Introduce rust into Eq^m population
- Infection scenario (β = 0.044)
The Beta Effect

- $\beta = 0$
- $\beta = 0.016$
- $\beta = 0.044$
- $\beta = 0.2$
Beta vs. Delta

100 years

Total Trees

Mature Adults
Disease free sensitivities and elasticities

Mature adults and total population.
Regenerating population after 100 years and equilibrium population.
Elasticities during transience

Elasticities for $\beta = 0.044$ after 100 years.
Mature adult population and total population.
Stage-specific and overall prevalence of blister rust for low ($\beta = 0.016$), medium ($\beta = 0.044$) and high ($\beta = 0.20$) probability of infection.
Conclusions I

- **Sustainability** of HEWP stands infected with WPBR depends on two dominant effects:
  
  (a) Infection probability
  
  (b) Regeneration mediated by competition (e.g. LAI)

Parameters controlling these effects disproportionately remove smaller stages via infection induced mortality and reduced seedling establishment.

- **Stand structure**
  
  Diseased equilibrium stands are less dominated by mature adults than disease-free equilibrium stands.

- **Disease prevalence**
  
  (a) Low prevalence in SD1 is due to high cost of infection, high natural mortality and low residence time, *not* low susceptibility. This may account for low seedling rust prevalence found in field surveys.
  
  (b) High prevalence in mature adults due to low infection cost and low natural mortality. Consistent with field observations.
Conclusions II

- Transient vs equilibrium sensitivity
  
  (a) 100 years after onset of infection, MA are sensitive to mortality and infection probabilities of all stages
  
  (a) At equilibrium, MA are sensitive to mortality and infection probabilities of the MA stage only
What insights can modeling provide?

Simulation

- Reinterpret existing observations
- Understand change in the structure of the forest
- Understand time scale for loss of forest ( » observation time)
- Understand time scale for control strategies ( » observation time)
- Predict behaviors of planted stands vs mature forests

Transient sensitivities

- Identify important parameters to estimate
- Identify important parameters to seek to control
- Equilibrium sensitivities are somewhat moot
Control strategies

Control efforts

1. Eradicate ribes

2. Prune infected branches

3. Select and breed rare resistant individuals

What insights might a genetic model provide?

- Understand time scale for spread of a resistant allele
- Estimate the effect of potential control strategies on pine stands

† = job creation scheme
SLG = Single locus genetics
Assumption: Consider two distinct alleles at a single locus only.

Let

\[ x_{\{3n\}} = \begin{pmatrix} x^{AA}_{\{n\}} \\ x^{aA}_{\{n\}} \\ x^{aa}_{\{n\}} \end{pmatrix}. \]

Survivorship & transition occur independently *within* each genotype, hence

\[ g_{\{3n\}}(x_{\{3n\}}) = \begin{pmatrix} g_{\{n\}}(x^{AA}_{\{n\}}) \\ g_{\{n\}}(x^{aA}_{\{n\}}) \\ g_{\{n\}}(x^{aa}_{\{n\}}) \end{pmatrix}. \]
Random mating is applied through the action of the nonlinear vector-valued function

\[ f_{3n} : \mathbb{R}^{3n} \mapsto \mathbb{R}^{3n}. \]
Assumption: Genotype affects survivorship & transition through fitness function $V_{3n} : \mathbb{R}^{3n} \rightarrow \mathbb{R}^{3n}$ and affects fecundity through fitness function $W_{3n} : \mathbb{R}^{3n} \rightarrow \mathbb{R}^{3n}$.

Putting it all together,

$$x_{3n}(t + 1) = \hat{x}_{3n} + f_{3n}(\hat{y}_{3n})$$

where

$$\hat{x}_{3n} = g_{3n}(V_{3n}(x_{3n}))$$

$$\hat{y}_{3n} = W_{3n}(\hat{x}_{3n}).$$
DSLG = Disease and single locus genetics
Disease and single-locus genetics

Combining our earlier ideas we define

\[ x \{6n\} = \begin{pmatrix} x^{AA} \{2n\} \\ x^{aA} \{2n\} \\ x^{aa} \{2n\} \end{pmatrix}. \]
We extend $g_{2n}$ to create $g_{6n} : \mathbb{R}^{6n} \mapsto \mathbb{R}^{6n}$ as

\[
g_{6n} = \begin{pmatrix} g_{2n} \\ g_{2n} \\ g_{2n} \end{pmatrix}.
\]

Assumption: Genotype can influence the cost of disease on survivorship & transition.

\[
C_{6n}^\nu = \text{diag} \left( C_{6n}^{\nu,AA}, C_{6n}^{\nu,aA}, C_{6n}^{\nu,aa} \right) = \text{diag} \left( I_n, C_{n}^{\nu,AA}, I_n, C_{n}^{\nu,aA}, I_n, C_{n}^{\nu,aa} \right).
\]
We define

\[ f_{6n} : \mathbb{R}^{6n} \rightarrow \mathbb{R}^{6n} \]

to model random mating.

Assumption: Genotype can influence the effect of disease on fecundity.

Let

\[ C_{\{6n\}} = \text{diag} \left( C_{\{2n\}}^{\varphi,AA}, C_{\{2n\}}^{\varphi,aA}, C_{\{2n\}}^{\varphi,aa} \right) \]

\[ = \text{diag} \left( I_{\{n\}}, C_{\{n\}}^{\varphi,AA}, I_{\{n\}}, C_{\{n\}}^{\varphi,aA}, I_{\{n\}}, C_{\{n\}}^{\varphi,aa} \right). \]
Assumption: Infectivity is independent of genotype.

Let

\[ B_{\{6n\}} = \text{diag} \left( B_{\{2n\}}, B_{\{2n\}}, B_{\{2n\}} \right). \]
Putting it all together,

\[
x_{6n}(t + 1) = B_{6n} \ast \left( \hat{x}_{6n} + f_{6n}(\hat{y}_{6n}) \right),
\]

where

\[
\hat{x}_{6n} = g_{6n}(C'_{6n} \ast x_{6n})
\]

and

\[
\hat{y}_{6n} = C'_{6n} \ast \hat{x}_{6n}.
\]
PDSLG = Pine + disease + single locus genetics
Define $x_{\{36\}}$ as

$$x_{\{36\}} = \begin{pmatrix} x_{AA}^{12} \\ x_{aA}^{12} \\ x_{aa}^{12} \end{pmatrix}.$$ 

Recall that survivorship and transition occurs within each genotype.
Assumption: Nonlinear seedling recruitment is independent of infection status and genotype.

We define

\[ \overline{x}_{12} = x_{12} + \begin{pmatrix} \gamma_{DSLG}^{6}(x_{36}) \\ 0 \end{pmatrix} \]

and

\[ \overline{x}_{36} = \begin{pmatrix} \overline{x}_{AA}^{12} \\ \overline{x}_{aA}^{12} \\ \overline{x}_{aa}^{12} \end{pmatrix}. \]

Think shading.
Let\[ S_{\{36\}} = \text{diag}(S_{\{6\}}, S_{\{6\}}, S_{\{6\}}, S_{\{6\}}, S_{\{6\}}, S_{\{6\}}), \]

and \[ C_{\nu}^{36} = \begin{pmatrix}
I_6 & 0 & \cdots & 0 & 0 & \cdots & 0 & 0 \\
0 & I_6 & \cdots & 0 & 0 & \cdots & 0 & 0 \\
\vdots & \vdots & \ddots & \vdots & \vdots & \ddots & \vdots & \vdots \\
0 & 0 & \cdots & I_6 & 0 & \cdots & 0 & 0 \\
0 & 0 & \cdots & 0 & I_6 - h * C_{\nu}^6 & \cdots & 0 & 0 \\
\vdots & \vdots & \ddots & \vdots & \vdots & \ddots & \vdots & \vdots \\
0 & 0 & \cdots & 0 & 0 & \cdots & I & 0 \\
0 & 0 & \cdots & 0 & 0 & \cdots & 0 & I_6 - C_{\nu}^6
\end{pmatrix}. \]
Then

\[ \hat{x}_{36} = g_{36}(x_{36}) \]

\[ = S_{36} \ast \left( C_\nu \ast \overline{x}_{36} \right) . \]
We define the nonlinear function

\[ f_{\{36\}} : \mathbb{R}^{36} \times (\rho, C^{\varphi}, h)^\top \mapsto \mathbb{R}^{36} \]

where

- \( \rho \) is a measure of the effect of class on fecundity
- \( C^{\varphi} \) is a measure of the effect of disease on fecundity,
- \( h \) is the usual partial dominance parameter.
Assumptions

- Males do not have an infection status.
- The number of males linearly dependent on the number of females.

Let \( y = \text{x}_{\{36\}} \) and

\[
\begin{align*}
y_{\text{male}}^{AA} &= (y_5 + y_6) + (y_{11} + y_{12}), \\
y_{\text{male}}^{aA} &= (y_{17} + y_{18}) + (y_{23} + y_{24}), \\
y_{\text{male}}^{aa} &= (y_{29} + y_{30}) + (y_{35} + y_{36}), \\
\text{male} &= y_{\text{male}}^{AA} + y_{\text{male}}^{aA} + y_{\text{male}}^{aa},
\end{align*}
\]

so that

\[
\begin{align*}
p_{\text{pollen}} &= \frac{y_{\text{male}}^{AA} + 0.5y_{\text{male}}^{aA}}{\text{male}} \quad \text{and} \quad q_{\text{pollen}} = \frac{0.5y_{\text{male}}^{aA} + y_{\text{male}}^{aa}}{\text{male}}.
\end{align*}
\]
PDSLG: Females

Assumption: Stage has an effect on viability of females that is independent of infection status and genotype.

Define

\[ y_{AA}^S = \rho y_5 + y_6, \quad y_{AA}^I = \rho y_11 + y_12, \]
\[ y_{aA}^S = \rho y_{17} + y_{18}, \quad y_{aA}^I = \rho y_{23} + y_{24}, \]
\[ y_{aa}^S = \rho y_{29} + y_{30}, \quad y_{aa}^I = \rho y_{35} + y_{36}, \]

and

\[ \text{female} = y_{SS}^{AA} + y_{II}^{AA} + y_{SS}^{aA} + y_{II}^{aA} + y_{SS}^{aa} + y_{II}^{aa}. \]
**PDSLG: Egg viability**

Assumption: Genotype affects egg viability of infected individuals.

Consider the effect of infection status and genotype on egg viability

\[
\begin{align*}
z^{AA} &= y_S^{AA} + y_I^{AA}, \\
z^{aA} &= y_S^{aA} + (1 - h \times (1 - C^\phi)) \times y_I^{aA}, \\
z^{aa} &= y_S^{aa} + C^\phi \times y_I^{aa}.
\end{align*}
\]
Random mating gives

\[ \hat{f}_1(y_{36}) = p_{\text{pollen}} \ast (z^{AA} + 0.5 \ z^{aA}), \]
\[ \hat{f}_{13}(y_{36}) = q_{\text{pollen}} \ast (z^{AA} + 0.5 \ z^{aA}) + p_{\text{pollen}} \ast (0.5 \ z^{aA} + z^{aa}), \]
\[ \hat{f}_{25}(y_{36}) = q_{\text{pollen}} \ast (0.5 \ z^{aA} + z^{aa}), \]

and

\[ \hat{f}_{\{36\}} = \hat{f}_1(y_{36}) \ast e_1 + \hat{f}_{13}(y_{36}) \ast e_{13} + \hat{f}_{25}(y_{36}) \ast e_{25}. \]

Thus

\[ f_{\{36\}} = r_1(\hat{y}_{36}) \ast \hat{f}_{36} \]

where the function \( r_1(x_{\{36\}}) \) is an obvious extension of \( r_1(x_{\{6\}}) \).
Assumption: Infectivity is independent of genotype.

\[
PDSLG: \text{Infectivity} \\
\]

\[
B_{36} = \begin{pmatrix}
B_{12} & 0 & 0 \\
0 & B_{12} & 0 \\
0 & 0 & B_{12}
\end{pmatrix}.
\]
Putting it all together,

\[ x_{36}(t + 1) = B_{36} \ast (\hat{x}_{36} + f_{36}(\hat{x}_{36}; \rho, C^\varphi, h)) \].
Results
Fixation of advantageous allele with and without background pollen
Number of adults
Moderate competition and low quality site.

Frequency of advantageous allele

Evolution of resistance – p. 72/74
Preliminary studies

Number of adults
Low competition, high quality site.

Frequency of advantageous allele
Future directions

- Comprehensive study of single locus genetics including control strategies
- Multilocus genetics
  1. Linkage disequilibrium for small numbers of loci and alleles
  2. Recombination vs selection for small numbers of loci
  3. Parametrization ???
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There is something fascinating about science. One gets such wholesale returns of conjecture out of such a trifling investment of fact. ... Mark Twain