Spatio-temporal model for the genetic resistance to ash-dieback

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Ash Dieback

- Invasive **fungal disease** of european ash trees causing large numbers of deaths and **threatening the survival** of the species
- 2 symptoms: *Crown Dieback* (CD) and *Collar Lesion* (CL)

Scientific goals

- Estimate genetic parameters for the resistance
- Assess genetic covariation across symptoms
- Account for differences in exposure to pathogen across space and time
- Quantify the effect of potential covariates

Data

- $N = 777$ trees from 23 OP progenies.
- CD assesed visually in a 6-level scale. CL measured as a girdling index.
- Other variables: bud flush precocity (BF) provenance (3-levels), and basal circumference

Crown Dieback progress and model

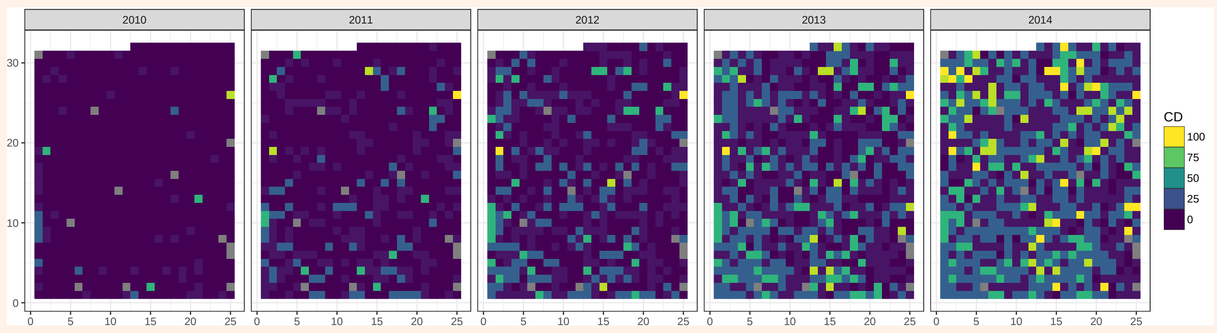


Figure: Spatio-temporal progression of CD. Each pixel is a tree.

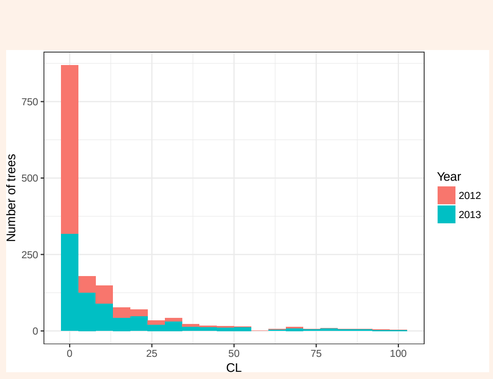
After a normalizing transformation and a variable selection procedure, we model the response in year i, location j for the individual k as:

$$\begin{aligned} y_{ijk} =& \text{Year}_i + \text{BF}_k + \eta_{ij} + a_k + \varepsilon_{ijk} \\ \boldsymbol{\eta} \sim& \mathcal{N}\left(\mathbf{0}, \tau_\eta^2 \mathbf{Q}(\rho_t, \rho_s)\right) \\ \boldsymbol{a} \sim& \mathcal{N}\left(\mathbf{0}, \tau_a^2 \mathbf{A}^{-1}\right) \\ \boldsymbol{\varepsilon} \sim& \mathcal{N}\left(\mathbf{0}, \mathbf{I}\right), \end{aligned} \tag{1}$$

which accounts for the year-wise progression of the disease, its spatio-temporal variability, the effect of the individual bud flush precocity, the individual additive genetic effect (*a.k.a. Breeding Value*) and the residual effects.

- The parametric structure matrix $\mathbf{Q}(\rho_t, \rho_s)$ is the tensor product of a Matérn spatial process and a exchangeable temporal structure
- The relationship matrix $\mathbf{A}$ is determined from the family kinkship
- Prior spatial range constrained within the field dimensions. Other priors are *reasonably* vague. See paper for details.

Collar Lesion progress and model



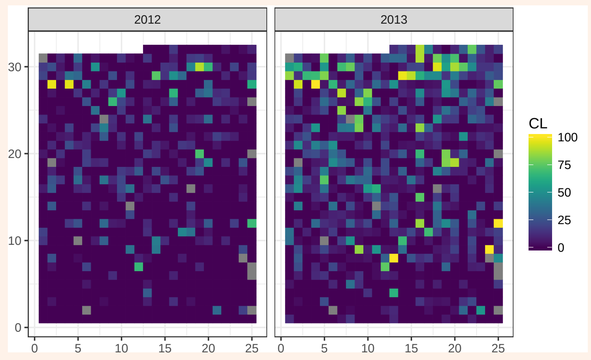


Figure: Marginal and spatio-temporal progression of CL.

- No normalizing transformation possible here: too many zeros we model the response in year i, location j for the individual k as:

For a measurement of CL y_{ijk} taken in year i, at location j for individual k, we assume that

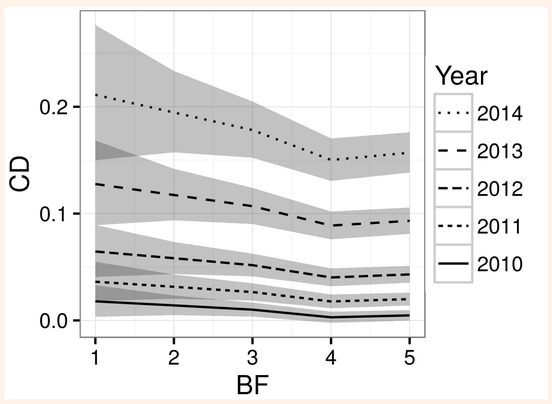
$$\begin{aligned} \Pr[y_{ijk} = 0] &= p_{ijk}, & 0 < p_{ijk} < 1 \\ \pi(y_{ijk} | y_{ijk} > 0) &= \text{Ga}(a_{ijk}, b_{ijk}), & a_{ijk}, b_{ijk} > 0. \end{aligned} \tag{2}$$

We define a hierarchical model for the parameters p, a and b using appropriate link functions of the expected values of the respective distributions. Specifically, calling $\mu = E(y|y > 0) = \frac{a}{b}$, we define two linear predictors

$$\begin{aligned} \text{logit}(p_{ijk}) &= \text{Year}_i^{(1)} + \eta_{ij}^{(1)} + a_k^{(1)} \\ \log(\mu_{ijk}) &= \text{Year}_i^{(2)} + \eta_{ij}^{(2)} + a_k^{(2)} \end{aligned} \tag{3}$$

for the binary and continuous components, respectively. The elements in the latent linear predictors are defined as for Eq. 1

Results for CD



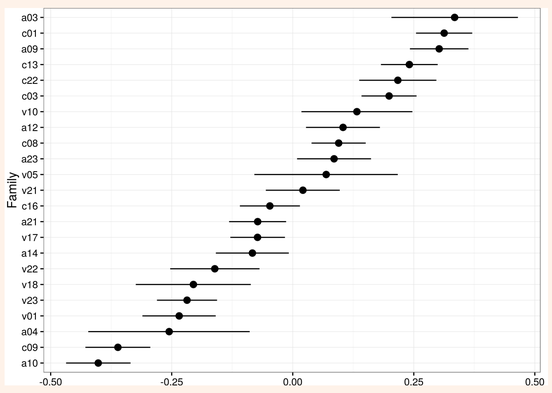


Figure: Posterior means and 95% credible intervals of BF and family effects.

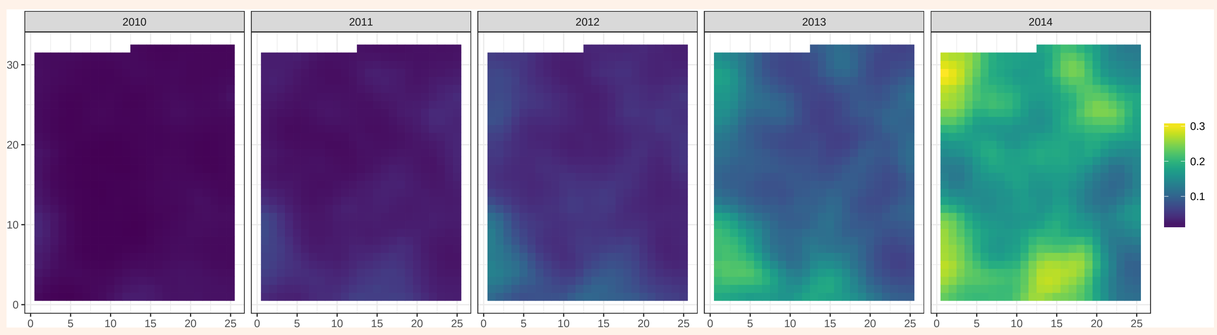
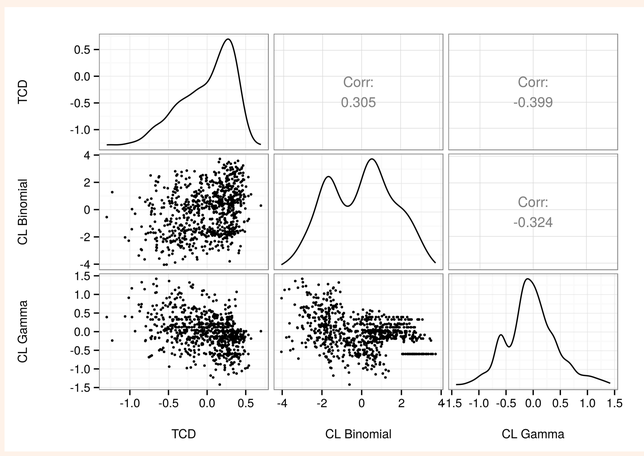


Figure: Posterior mean spatio-temporal effect for $BF = 3$

Results for CL



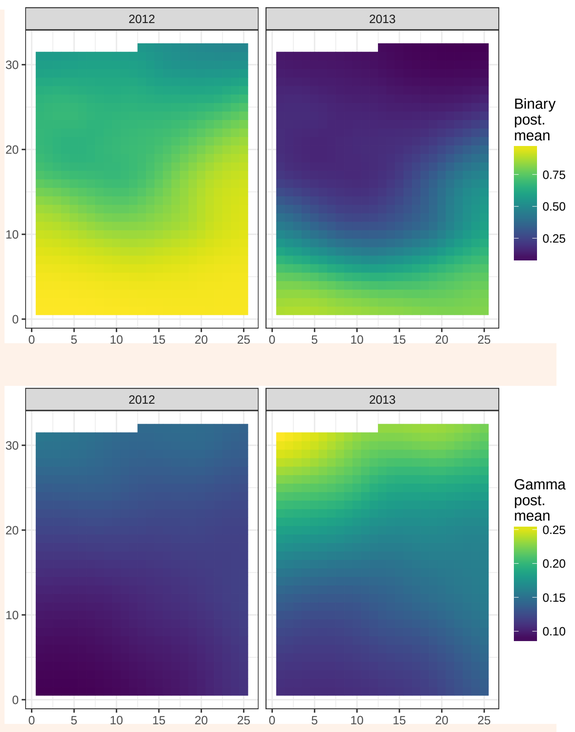


Figure: Posterior mean spatio-temporal effect and empirical genetic correlations

Conclusions

- Narrow-sense heritabilities estimated at .42, .49 and .42 for CD, CL prevalence (Binomial component) and CL severity (Gamma component) respectively.
- Genetic correlations of up to .4 between the severities of crown and collar symptoms
- No evidence for differences between provenances. More genetic variability within than between families.
- Early flushing correlates with healthier crown.

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