LANDIS-II EPIDEMIOLOGICAL AGENT EXTENSION

Case study: Development and parameterization of a model of the transmission of *Phytophthora ramorum* on a landscape at regional level

Assumptions of the model:

The following sections describe the main assumptions made for the development of a first version (V1.0) for the epidemiological agent extension for LANDIS-II. The extension should approximate the transmission of one (or more) pathogen in the spatiotemporal landscape defined by host and weather variables. Its design should work well at typical regional extents used for LANDIS simulations.

1) Host landscape:

The spatial resolution of the model is a unit cell (or site, or forest stand) (e.g. >= 30m x 30m) and should be chosen compromising between useful amount of information and tractability. Cells are arranged on a square grid. The spatial distribution of known hosts of the pathogen "agent" is accounted for using a *relative host index, h_i*. This approach allows us to quantify the **susceptibility of each non-infected cell to become infected and the suitability of each infected cell to produce infectious spores of the pathogen**. The *absolute host index* of each cell *i*, H_i, is calculated using data on local composition (and density) of host species and a measure of relative susceptibility and infectivity of each host (e.g. competency score). As a proxy to density, we can use biomass information available in LANDIS-II for each species/age combination. Lethally infected non-sporulating hosts should not be included in the host index calculation. The relative index is obtained through division of the absolute index by its spatial mean over the study area (H_{mean}):

$$h_i = H_i / H_{mean}$$

and is incorporated in the model by multiplying the transmission rate β (see transmission section). The relative index allows us to compare the transmission rate β against homogeneous landscape conditions (where hi=1) and to interpret β as the rate of secondary infection of typical cells by a single infected typical cell in a non-infected landscape (see definition of dispersal kernel below)

2) Biology of the pathogen and host populations:

Parameter values relative to the lifecycle of the pathogen "agent" and to the host population must be summarized within the SpeciesParameter .txt file used by LANDIS-II. The dispersal of the pathogen is described by a probability function of the distance between source and target (dispersal kernel) that we assume to be ***anisotropic*** at the scale of the study area. This working hypothesis is meant to increase realistic dispersal dynamics of windblown pathogens over the study area. For simplicity and lack of data we do NOT explicitly account for topographic variables and features, such as cliffs and valleys, which could potentially affect pathogen dispersal but would be difficult to model.

3) Epidemiology:

Several assumptions are made to represent the epidemiological processes within the LANDIS-II extension. Once infection occurs in a unit cell, it spreads and intensifies within the cell, but in the first version (V1.0) of the extension, we should ONLY account for presence or absence of infection in each cell (0-1). This simplification ignores a transient effect, and amounts to considering an effective level of inoculum that is reached rapidly (but is below the maximum sporulating capacity of the cell), and which we use for calculating the force of infection exerted on other cells. However, we believe the approximation will not have affected significantly the model predictions for this system. We assume that infected cells **become immediately infectious**. If working with a pathosystems where the latent period is significant, a new component should be added to the model presented herein. Further, the proposed epidemiological framework for V1.0 does not include a limitation to the period for interruption of infection (thus "recovery" from infection is not accounted for).

4) Weather:

We use a **weather index** to account for the effect of weather conditions on the probability of non-infected hosts becoming infected and infected hosts sporulating and spreading the pathogen. For pathosystems that are not driven by weather conditions, this component should be removed (turned OFF) within the SpeciesParameter .txt file read by LANDIS-II. The weather index should be built and modified with predictors that are most relevant to the pathogen. As an example related to our case study, if annual Spring rainfall and mean temperature are thought to be the most significant, then the *annual weather index*, *w(t)*, can be defined as:

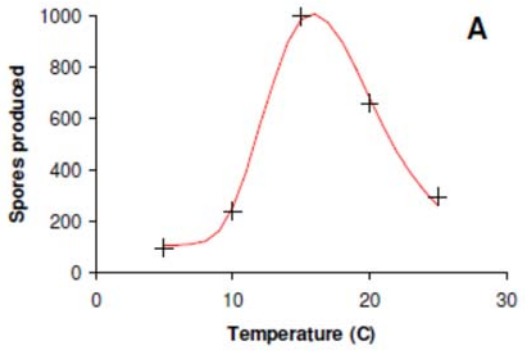
$$\beta(t) = w(t)\,\beta_0$$

Where β_0 is estimated by us (=mean rate at which an infected cell infects another cell) using aerial images etc. The *basic* weather index for year *t*, *W(t)*, comprises the joint cumulative effect of rainfall and temperature between April and June of each year, and is calculated as follows:

$$W(t) = \sum_{d \in \{Apr(t),\ldots,Jun(t)\}} r_5(d) f(T(d))$$

Where $r_5(d)$ is the cumulative rainfall over five days up to day *d*, *T(d)* is the average daily temperature on day *d*, and *f (T(d))* is a measure of the rate of sporulation under favorable conditions.

$$f(T) = 108.6 + 904.8 \exp(-0.5[\ln(T/15.87)/0.2422]^2)$$



The actual weather index, *w(t)*, is **normalized** by the mean (W_{mean}) over the decade of available historical data (e.g. 2000-2009)

$$w(t) = W(t) / \bar{W} .$$

This normalization follows the same spirit as that for the host index: it is such that β_0 can be interpreted as the annual transmission rate under average (or under constant) weather conditions. The weather index built this way is annually varying but spatially-uniform across the whole study area. **Maybe we could have a weather index that also varies spatially, maybe by ecoregion or something (in the LANDIS spirit)?**

<u>Mathematical formulation of the model</u>:

Here we provide a mathematical formulation and quantitative assumptions of the epidemiological model for the transmission of the pathogen "agent".

1) Basic Model:

We model the spread of a pathogen in the host and weather spatiotemporal landscape as a **dynamic process on a meta-population** comprising *N* contiguous subpopulations represented by cells (sites) arranged on a square lattice. Each cell mimics either a forest stand with its own vegetation composition or a patch without vegetation. Cells can be in one of the following states (Fig. S2): **Susceptible (S)**, **Infected (infectious but cryptic) (I)**, **Diseased (infectious and symptomatic) (D).**

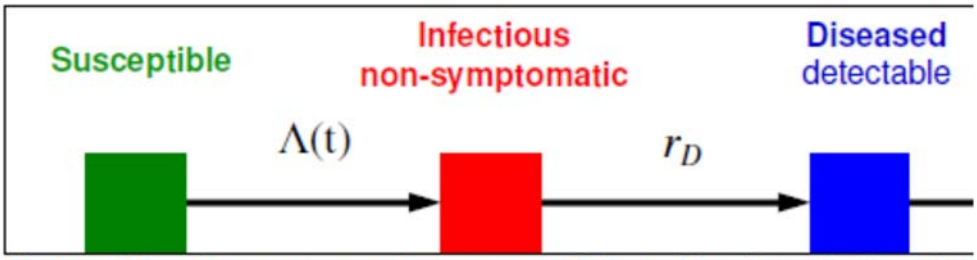


Figure S2 – Compartmental structure of the epidemiological model

2) Natural Dynamics:

A susceptible cell *i* can become *cryptically infected* subject to a force of infection $\Lambda_i(t)$ and once infected it can become diseased at rate r_D. Infected sites remain infectious for a very long period. In V 1.0 we will assume no recovery process involved. However, future version may include this as an additional state of a cell after diseased. Diseased cells, despite containing dead hosts have the same transmission rate, i.e. are as infectious. Within each diseased cell, specific tree species-cohort are affected by mortality with probability P_{mort} specified in the SpeciesParameter .txt table, depending on vulnerability. In V1.0, an entire cohort is removed once affected by mortality (Age-Only succession compatible LANDIS-II). Dead cohorts are subsequently recolonized as part of the LANDIS succession component by susceptible hosts leading to a newly computed Site Host Index H_i in [0, 1].

3) Predictive Model:

In order to make forward predictions regarding pathogen spread under natural dynamics, we used the following spatially-explicit, probabilistic formulation of the basic model described above. In

the spirit of LANDIS-II and similarly to the Dynamic Fire & Fuel extension, the model runs at annual time steps, although pathogen build-up and transmission processes happen at a much finer time scale (i.e. daily, weeks for weather index computation). The model shares features with spatially-structured metapopulation models. The probabilities that cell *i* is in each of the possible states, **Susceptible**, **Infected**, **Diseased**, i.e., $P_{i,S}$, $P_{i,I}$, and $P_{i,D}$, respectively, are governed by the system of differential equations:

$$\frac{\Delta P_{i,S}}{\Delta t} = -\Lambda_i(t) P_{i,S}$$

$$\frac{\Delta P_{i,I}}{\Delta t} = \Lambda_i(t) P_{i,S} - r_D P_{i,I}$$

$$\frac{\Delta P_{i,D}}{\Delta t} = r_D P_{i,I}$$

The initial conditions, at the estimated time of onset of the outbreak, are $P_{i,S} = 1$, $P_{i,I} = 0$, $P_{i,D} = 0$, except at the cell estimated to be the location of the first infection, where $P_{i,S} = 0$, $P_{i,I} = 1$, $P_{i,D} = 0$. The force of infection is given by:

$$\Lambda_i(t) = \beta(t) \sum_{j \neq i} A_j B_i(t) C_{j,I+D;i,S} K(d_{ij}) ,$$

Where $\beta(t) = \beta_0 w(t)$ is the transmission rate, with *w(t)* an annual index of weather fluctuation about a *N*-year average and β_0 the baseline rate; *K(d)* is the dispersal Kernel (see below) for a given distance between target and source cells; $A_j = h_j$ is the infectivity of source site *j* (as expressed by the Site Host Index), and $B_i(t)$ is the susceptibility of target cell *i* at time *t* (again, expressed by the Site Host Index. $C_{j,I+D;i,S}$ is the conditional probability that site *j* is infectious (with cryptic or symptomatic infection) given that site *i* is susceptible. To first order of approximation, we assume that $C_{j,I+D;i,S} \approx P_{j,I} + P_{j,D}$, which we expect to be a reasonable approximation to the infection pattern given that dispersal is not very localized, as indicated by the estimated exponent of the power-law dispersal kernel (> 1).

4) Dispersal Kernel:

Among several possible candidate distributions to express the probability that pathogen spores disperse a distance *d* from the source, we consider a power-law and negative exponential functional forms with generic form:

$$K_P(d) = C_1 / d^{\alpha}$$
$$K_E(d) = C_2 \exp[-d / \alpha]$$

where the constants C1 and C2 are such that the functions are normalized to 1 on the plane, excluding the area of the source cell. The latter condition means that we are only considering dispersal events where pathogen spores produced in a source cell are deposited in a different cell within the study area or further beyond, i.e. transmission in force of infection is conditional on spores being dispersed outside the source cell. The rationale for this choice is that we do not keep track of the infection process within a cell, which is below the resolution of the observations. In addition, we use an effective kernel that results from integrating the point kernel (A8) over the area of the target cell, accounting for all possible ways through which the target cell can become infected by a given source cell.