Elucidating transmission parameters of African swine fever through wild boar carcasses by combining spatio-temporal notification data and agent based modelling

Supplementary Material

ODD MODEL DOCUMENTATION

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Model description

Overview

The ASF wild boar model is a compilation of a spatially explicit, stochastic, individual based demographic model for wild boars (*Sus scrofa*) in a structured landscape of habitat area. Superimposed is a transmission and disease course model for the ASFV. The model is documented following the ODD protocol (Overview, Design, Details; Grimm et al. 2006, Grimm et al. 2010).

Purpose

The model aims at assessment of ASF spread in Eastern European wild boar populations and the evaluation of reporting data from field surveys. Transmission of ASF infection is operated by direct contacts within groups of socialising wild boar hosts and with carcasses deposited in the habitat landscape.

Entities, state variables and scales

The model comprises three entities: spatial habitat units, connecting edges between these units and wild boar individuals.

All processes take place on a raster map of spatial habitat units. Each cell represents a functional classification of a landscape denoting habitat quality. The cells of the model landscape represent about 9 km² (3 × 3 km), encompassing a boar group's core home range (Leaper et al. 1999). State variables comprise wild boar habitat quality of the grid cells. At run time, habitat quality is interpreted as breeding capacity, i.e. the number of female boars that are allowed to have offspring (explicit density regulation; Jedrzejewska et al. 1997). Habitat quality may be applied to implement an external data set of spatial wild boar density distribution i.e. by reversely adjusted breeding capacity.

Habitat cells are connected by edges to the neighbouring eight cells. Connecting edges represent space between core habitat areas that is shared among neighbouring herds. Each habitat cell and each connecting edge handles a list of infectious wild boar carcasses.

The third model entities are the individual wild boars. State variables of host individuals are the age in weeks (where one week represents the approximate ASF infectious period in wild boar; (Blome et al. 2012)), resulting in age-classes: piglet (< 8 months ± 6 weeks), sub-adult (< 2 years ± 6 weeks) and adult. Accordingly, age class transition event is stochastic. Each host individual has a location, which denotes its home range cell on the raster grid as well as its family group. Further, the individual host animal comprises an epidemiological

status (*susceptible, non-lethally infected, lethally infected*, or *immune* after recovery or due to transient maternal antibodies). Sub-adult wild boar may disperse during the dispersal period (i.e. early summer) dependent on their demographic status (disperser or non-disperser).

Process overview and scheduling

The model proceeds in weekly time steps. Processes of each time step are performed as applicable: virus release, infection, dispersal of sub-adults, reproduction, ageing, mortality, hunting (for surveillance and depopulation), and control measures. Submodels are executed in the given order. In the first week of each year, mortality probabilities are assigned stochastically to the age classes representing annual fluctuations in boar living conditions; and boars are assigned to breed or not, according to the carrying capacity of their home range cell.

Design concepts

Wild boar population dynamics emerge from individual behaviour, defined by age-dependent seasonal reproduction and mortality probabilities and age- and density-dependent dispersal behaviour, all including stochasticity. The epidemic course emerges stochastically from within group transmission of the infection, individual disease courses, spatial distribution and decay of infectious carcasses, contact to carcasses as well as wild boar dispersal. Stochasticity is included by representing demographic and behavioural parameters as probabilities or probability distributions. Annual fluctuations of living conditions are realised by annually varying mortality rates. Stochastic realisation of individual infection and disease courses are modelled explicitly.

Details

Initialisation

The model landscape represents an area of approx. 200 km × 200 km along the border between Estonia and Latvia (see Figure 1 in the main paper). The local breeding capacity CC_{ij} of each cell is initialized from spatially structured wild boar density estimates of the region (source: FAO/ASFORCE, May 2015; see EFSA 2015; Figure 1 therein). The breeding capacity was calculated as CC_{ij} = 1.3455 * density_estimate [heads/km²] following the regression density_estimate = f(CC_{ij}). Non-integer values are randomly assigned to the adjacent integer values according to

$$\widehat{CC}_{ij} = \lfloor CC_{ij} \rfloor + \left(U(0,1) < \left(CC_{ij} - \lfloor CC_{ij} \rfloor \right) \right)$$

where $U(0,1)$ is a uniformly distributed random number in range 0…1.

Each cell is connected to eight neighbouring units (Moore neighbourhood). One boar group is released to each habitat cell, where initial group size is six times breeding capacity. Initial age distributions were taken from the results of a 100 years model run (see Table 1).

Table 1: Initial age distribution (Kramer-Schadt et al. 2009).

Upper age bound (years)	1	2	3	4	5	6	7	8	9	10	11
Proportion	0.38	0.24	0.15	0.09	0.06	0.03	0.02	0.01	0.01	0.01	0.00

Input

The applied model setup does not include any external inputs or driving variables.

Submodels

Submodels are described in the order of their execution. Parameters and their values are listed in Table 2 in section "Parameters".

Release of infection

The virus is released end of June of the 4th year of each model run to 10 hosts in the release location specific to simulation experiments. Release is scheduled in the 4th year in order to allow population dynamics to establish.

Transmission of infection

Transmission of infection with the ASF virus is modelled directly and carcass mediated.

Direct transmission: The mode refers to transmission between animals in direct animal-to-animal contact i.e. members of the same female group and males associated with the group. Direct transmission is modelled stochastically. Parameter $P_{inf}^{(i)}$ determines the probability of contracting the infection from an infectious group mate during one week. For each susceptible animal, the probability of becoming infected accumulates over all infectious animals within the group:

$$\Pi_i^{(i)} = 1 - \left(1 - P_{inf}^{(i)}\right)^{\lambda_i} \quad (1)$$

where λ_i is the number of infectious individuals in the same direct contact group as the receiving individual.

Carcass transmission: The mode refers to wild boar carcasses after ASF infected animals have died, laying down in the habitat area. Possible transmission is assumed to be associated with physical contact to the carcass i.e. no airborne or indirect mechanisms are considered relevant. Transmission through carcasses is modelled stochastically. Parameter $P_{inf}^{(c)}$ determines the probability of contracting the infection from an infectious carcass during one week. For each susceptible animal, the probability of becoming infected accumulates over accessible carcasses

$$\Pi_i^{(c,s)} = 1 - \left(1 - P_{inf}^{(c)}\right)^{\omega_i} \cdot \left(1 - P_{inf}^{(c)}\right)^{\Sigma_j \omega_{ij}} \quad (2)$$

where ω_i is the number of carcasses in the respective core home range, ω_{ij} is the number of carcasses in the connecting edges (i.e. shared areas).

Effective transmission: For every habitat cell an per time step, the transmission probability is accumulated from direct and carcass transmission probabilities

$$\Pi_i^{(t,s)} = 1 - \left(1 - \Pi_i^{(i)}\right) \cdot \left(1 - \Pi_i^{(c,s)}\right) \quad (3)$$

The model iterates over all individuals and stochastically sets each susceptible individual to infected if a uniformly distributed random number r drawn from U(0, 1) is smaller than $\Pi_i^{(t,s)}$ of the home cell.

Disease course

The disease course following infection is explicitly modelled for each infected individual. The probability of lethal infection is given by parameter p_L. Each host is infectious for t_{inf} weeks and thereafter either turns immune lifelong (probability 1-p_L) or dies (probability p_L). For the processing of the carcasses after death of infected animals see submodel 'Carcass distribution and persistence'.

Group splitting

Group splitting is performed in week 29 of the year. All groups containing more females than the cells breeding capacity and a minimum number of subadults to move N_{disp}, are processed. Groups are iterated randomly for the splitting submodel. From such groups, the

model collects subadult female yearlings without offspring. Then, an empty habitat cell is selected randomly among all accessible cells. All dispersing individuals of the group disperse as cohort and establish the new group on the target habitat cell. If no empty habitat is available, disperser females do not move. Accessible habitat cells are cells within Euclidean distance D_{disp} that can be reached accounting for landscape map structure (i.e. water bodies or other barriers). Accessible cells are determined using breadth-first search on the passable cells (nodes of a graph) and connecting edges in radius D_{disp}. Thus, the distance travelled to the target cell can be larger than D_{disp}, but the linear distance from the home cell does not exceed D_{disp} during search.

Male dispersal

Male dispersal is performed in weeks 25 to 30 of the year only (i.e. mid-June to end of July). Uniformly distributed over the weeks of the dispersal period subadult males start to disperse. During dispersal, a male moves from cell to cell along connecting edges. Each week, S_w steps are performed, until a total of S_t steps of dispersal are made. Each dispersal step can be either oriented (probability p_{ori}) or straight ahead (probability $1 - p_{ori}$). For oriented movement, the boar moves to the cell with the highest habitat value among the accessible neighbouring cells (Pe'er et al. 2013, Graf et al. 2007, Jeltsch et al. 1997). For straight movement, the previous direction is just continued. If the boar encounters a barrier edge or a blocked cell during straight movement, a random direction is taken as previous direction and movement continued with the next iteration.

Reproduction

Females reproduce only once a year if at least in subadult age. Individual females reproduce depending on the season with a peak in March (EFSA 2012). In the first week of the year, female individuals are checked whether they are able to breed. Starting with the oldest individuals and up to the breeding capacity CC_{ij} of the habitat cell, females are allowed to breed. The week of breeding is individually assigned by drawing of weekly probabilities, rooted of the data-based monthly probability distribution (Bieber & Ruf 2005, EFSA 2012, Figure 1a). Litter size is drawn from data-based truncated normal distribution (Bieber & Ruf 2005, EFSA 2012, Figure 1b). Litter size is reduced to a constant fraction for infected individuals. Litter size of transient shedders and lethally infected hosts is multiplied with the reduction factor α_f.

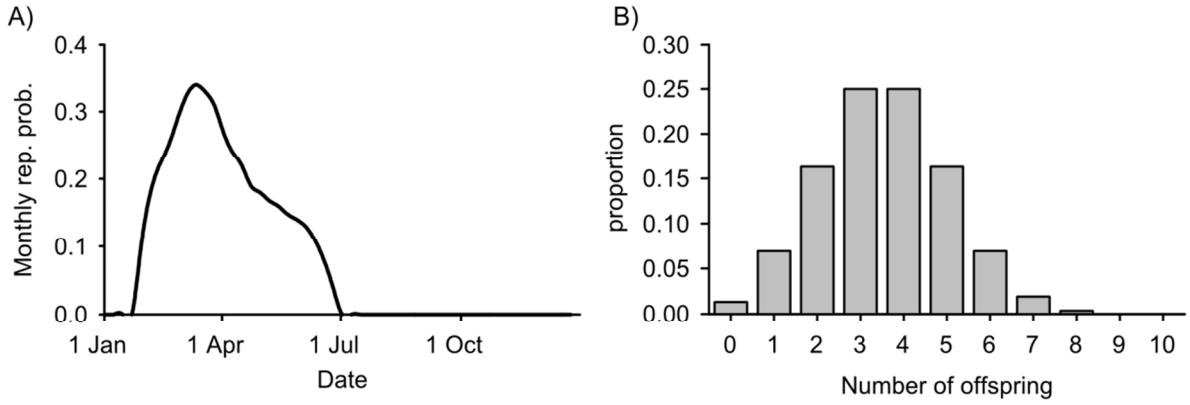

Figure 1 A) Monthly reproduction probabilities for wild boar. B) Breed count distributions for wild boar (Bieber & Ruf 2005, EFSA 2012).

Depending on the disease state of the breeding individual, its piglet's disease states have to be adjusted. The epidemiological data are not yet available for ASF in wild boar. Therefore the process was parameterised in accordance with existing evidence for Classical Swine Fever (CSF) in wild boar. However, at time of this study, lethality due to virus infections (p_L) was observed maximum and rather fast. Hence the knowledge gap does not conflict with the simulation rules: If assigned for reproduction, susceptible and infected but not yet infectious individuals produce susceptible offspring. However, non-lethally infected individuals (1-p_L) may potentially yield lethally infected offspring with probability of prenatal infection P_{PI}. Immune individuals produce offspring temporarily immune by maternal antibodies.

Mortality

Iterating over the entire population, each individual either stochastically dies with age class dependent mortality rates or after reaching a certain maximum age (T_{max}). Stochastic age class dependent mortality rates are adjusted to annual survival estimates from literature. Survival estimates and reported variability (see Table 2) determine a Gaussian distribution which is used in the model to draw the random annual survival (SP_{Year}). This stochastic effect resembles 'good' or 'bad' years for the host species, i.e. environmental noise. In the application the Gaussian distributions are truncated symmetrically around the mean. Per time step, the adjusted age-dependent mortality (PM_{Week}) was applied to the individual:

$$PM_{Week} = 1 - (SP_{Year})^{1/52} \quad (4)$$

Mortality due to infection is independently treated by the disease course submodel.

Carcass distribution and persistence

The carcass of an infected dead individual is accessible to non-group mates with certain probability p_{access}. Death of infected animals can occur either in the shared space between neighbouring groups (edges, probability p_{access}) or in the core area of the herd (probability $1 - p_{access}$). After death in the core area, the carcass is only accessible for the individuals associated with the respective cell. Otherwise, i.e. death in the shared area, the carcass is randomly assigned to one of the connecting edges of the habitat cell, so it is accessible for the individuals in the cell of origin as well as to the individuals of one neighbouring cell (8 possible neihgbours).

Ageing

The ageing process iterates over all individuals. For each individual k, age T_k is incremented one week. Consequent disease state transitions are performed following evidence from CSF: Non-lethally infected animals recover from the infection and are converted to immune after their individual infectious period t_{inf}. An offspring individual protected by maternal antibodies turns susceptible after reaching the maximum age of maternal immunity T_{immune}. Seropositivity due to maternal antibodies vanishes on reaching a maximum age of maternal antibody presence T_{anti}. Subsequently, the age of the infection is incremented by one week for all infected individuals.

Parameters, simulation experiments, analysis

Parameters

Model parameters of the transmission model are shown in Table 2.

Table 2: Model parameters

Name	Description	Value	Source / details
a) Wild boar ecology (used constant for the study)			
$T_{\max}$	Maximum age of boar	572 weeks	(Jezierski 1977)
$SP_{mean}^{(a)}$ / $SP_{\min}^{(a)}$	Mean / minimum annual survival rate adults (natural mortality + conventional hunting)	0.65 / 0.4	(Focardi et al. 1996)
$SP_{mean}^{(y)}$ / $SP_{\min}^{(y)}$	Mean / minimum annual survival rate yearlings (natural mortality + conventional hunting)	0.65 / 0.4	(Gaillard et al. 1987)

Name	Description	Value	Source / details
$SP_{mean}^{(p)}$ / $SP_{\min}^{(p)}$	Mean / minimum annual survival rate piglets (natural mortality + conventional hunting)	0.5 / 0.1	(Focardi et al. 1996)
b)	Dispersal and movement parameters (used as constant for the study)		
N_{disp}	Minimum number of subadult females for dispersal	2	technical assumption
D_{disp}	Maximum dispersal distance for subadult females	6 km	(Sodeikat & Pohlmeyer 2003)
S_t	Maximum dispersal steps of males	16 cells (48 km)	(Truvé & Lemel 2003)
S_w	Male dispersal steps per week	8 cells (24 km)	(Truvé & Lemel 2003)
p_{ori}	Probability of oriented movement during male dispersal	0.5	(Pe'er et al. 2013)
c)	ASF specific parameterisation		
p_L	probability of lethal infection	0.95	(Blome et al. 2012)
t_{carc}	Time of carcass persistence	4 weeks	(Ray et al. 2014)
$t_{\inf}$	Average period between infection and dead	1 week	(Blome et al. 2012, Guinat et al. 2014)
$P_{\inf}^{(i)}$	Infection probability by direct transmission within social groups	0.05	ad hoc, reflecting the limited transmission during physical contact of incubating (see Blome et al. 2012). In a contact group of 10-12 animals the resulting local R_0 is 4-6, see Guinat et al. (2014).
β_{carc}	Infection probability per carcass (including contact and transmission)	knowledge gap	Determined using spatial-temporal data of observed spread in wild boar
p_{core}	Probability of virus-induced death in core area	knowledge gap	Determined using spatial-temporal data of observed spread in wild boar
d)	Secondary disease course parameters (not relevant for ASF model variant due to short t_{inf})		
α_f	Fertility reduction if ill	0.625	Assumed like CSF 10/16 foeti aborted (Dahle & Liess 1992)
P_{PI}	Probability of prenatal infection	0.5	Assumed like CSF (Dahle & Liess 1992)
T_{anti}	Maximum persistence of maternal antibodies	15 weeks	Assumed like CSF (Depner et al. 2000)
T_{immune}	Maximum duration of immunity by maternal antibodies	12 weeks	Assumed like CSF (Depner et al. 2000)

Independent variables

The primary independent variables are the possible accessibility of dead animals for other wild boar groups P_{access}, and the infection pressure of their carcasses β_{carc}.

Simulation experiments

For each of the 121 possible combination of P_{access} and $\beta_{\text{carc}} \in \{0.01, 0.1, 0.2, \dots, 0.9, 0.99\}$, 100 model runs were conducted.

After establishment of population dynamics, 10 infected hosts are released in the vicinity of the first notified disease detections in June of the 4$^{\text{th}}$ year of the model run,.

After release, simulations were performed according the time horizon of the reference dataset until autumn of the subsequent year.

Dependent variables

Dependent variables are the record necessary to calculate the measures of fit described in the main document. These are spatial location and temporal occurrence of all infected individuals in the simulation area. For each model run all infected animals respective their carcasses were recorded with location, time of death and duration of presence. Per simulation run approx. 5,600 data points had to be recorded.

Analysis

Over all simulation runs with the same values of P_{access} and β_{carc} (100 runs), the measures of agreement were calculated and subsequently aggregated by arithmetic means. The resulting average values were smoothed using an artificial neural network (ANN). Neural networks can be used as an information processing approach with the ability to derive meaning from complicated and imprecise data. The network algorithm is an implementation of a Multilayer Perceptron (MLP, see main document, Figure 4), which is a feed-forward network that can be trained using the backpropagation of error algorithm.

The network has two hidden layers (12 and 8 nodes) with the log-sigmoid activation function

$$\phi(x) = \frac{1}{1 + e^{x}}$$

For the output layer (one node), a log-sigmoid activation function was used, and output variables were normalized to range [0, 1].

The network was trained to obtain a generalized regression of the measures of agreement as function of P_{access} and β_{carc}. Training was performed for 25,000 episodes (shuffled 121

averaged measures of agreement per episode) with an initial learning rate α of 0.3, exponentially decreasing by a rate of 1×10^{-4} per episode (final training rate approx. 0.02).

The trained network was used to estimate the measures of agreement for all input parameter combinations of P_{access} and $\beta_{\text{carc}} \in \{0.01, 0.05, 0.1, \ldots, 0.95, 0.99\}$. Figure 5 in the main document shows the good predictive performance of the network regression as observed vs. predicted plots.

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