MOBILE BARRIERS AS EMERGENCY MEASURE TO CONTROL OUTBREAKS OF AFRICAN SWINE FEVER IN WILD BOAR

SUPPLEMENTARY MATERIAL

ODD MODEL DOCUMENTATION

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MATERIALS AND METHODS

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 to assess the performance of temporary erecting mobile barriers as contingency measures against wild-boar mediated spread of ASF, compared to local depopulation in the vicinity of detected infected animals. Transmission of ASF infection is operated by direct contacts within groups of socialising wild boar hosts and through carcass scavenging within and between groups.

State variables and scales: The model comprises three major components: 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 4 km² (2 × 2 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 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. 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 erection of barriers. Submodels are executed in the given order. In the first week of each year, mortality probabilities are assigned stochastically to represent 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 200 km × 200 km of connected wildlife habitat without landscape barriers. The local breeding capacity of each of the 10,000 grid cells is initialised randomly with uniformly distributed integer values drawn from $\{0, ..., 3\}$. Each cell is connected to eight neighbouring units (Moore neighbourhood). One boar group is released to each habitat cell, where 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".

Virus release: The virus is released to 10 hosts, randomly selected from the central 25 (5 × 5) habitat cells of the model landscape. Release is scheduled in the first week of the 4th year of each simulation in order to allow population dynamics to establish.

Virus transmission:

Direct transmission: Direct within-herd 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 herd.

Carcass transmission: 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

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

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). $\mathrm{a}_{core}^{(s)}$ and $\mathrm{a}_{shared}^{(s)}$ are sex specific (superscript *s*) probabilities to encounter a carcass in the group's core area, and the shared area respectively.

Total transmission: Total 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 its home cell.

Disease course: The disease course following infection is 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 virus-induced death, see submodel 'Carcass distribution and persistence'.

Group splitting: Group splitting is performed in specified weeks of the year only. All groups containing more females than the cells breeding capacity and at least a minimum number of subadults to move N_{disp}, are processed. The model then collects subadult female yearlings without offspring of these groups. Groups are iterated randomly for the splitting submodel. For each of them, an empty habitat cell is selected randomly among all accessible cells. All migrating individuals out of the considered source group establish the new group on the target habitat cell. If no empty habitat is available, disperser females do not move. Accessible habitat cells are all cells within Euclidean distance D_{disp} that can be reached accounting for between-cell barriers and blocked cells (i.e. water bodies). Accessible cells are determined using breadth-first search on the 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. 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.

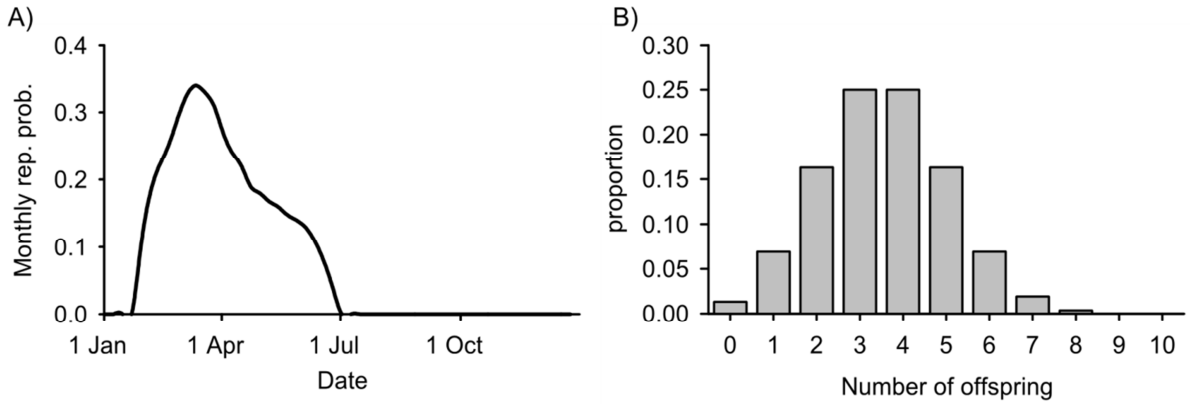

Fig. 1 A) Monthly reproduction probabilities for wild boar. B) Breed count distributions for wild boar (Bieber & Ruf 2005, EFSA 2012).

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 the study lethality due to virus infections (p_L) was maximum hence the uncertainty does rather not conflict with the simulations: If assigned for reproduction, susceptible and infected but not yet infectious individuals produce susceptible offspring, immune individuals produce offspring temporarily immune by maternal antibodies. Reproduction of transient (1-p_L) and lethally infected (p_L) individuals yields lethally infected offspring, each new-born with probability of prenatal infection P_{PI}.

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:

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

Virus-induced mortality is independently treated by the disease course submodel.

Carcass distribution and persistence: Virus-induced death can occur either in the core area of the herd (sex-specific probability $p_{core}^{(s)}$), or in the shared space between neighbouring

herds (edges, probability $1 - p_{core}^{(s)}$). After death in the core area, the carcass is only accessible for the individuals living in the respective cell. For 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 its origin as well as to the individuals from one of the neighbouring cells.

Carcasses are present in the cell or edge for a given number of weeks t_{carc}.

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: Transient shedders 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}. After finalising disease state transitions the age of the infection is incremented one week for all infected individuals.

Surveillance and management: A schematic diagram of management and surveillance is shown in Fig. 2. Surveillance and management are implemented as adaptive processes, based on virological and serological testing of randomly sampled wild boar individuals. The (unknown) actually infected area (grey area in Fig. 2) does not interfere with the simulations regarding design of zones and application of measures. Before detection of the first case of a simulation, spatially uniform disease surveillance is performed by sampling and testing in regular intervals t_m, where the sampling density is determined by the hunting parameter h_{base}. The parameter refers to the share of the population that is shot and tested for virus- and sero-positivity during one hunting campaign. Habitat cells containing animals with positive diagnostic are labelled infected (tests are assumed perfectly sensitive and specific). On detection of positive animals, control, fencing and monitoring measures are implemented (see Fig. 2 A) and performed during the subsequent interval t_m.

Around all detected virus- or sero-positive animals (black and hollow circles in Fig. 2A, resp.), a management area A_m is determined by buffering the respective cells with the width specified by radius r_m (hatched area). Under the barriers strategy, a fence is constructed around A_m (solid lines), while under the depopulation strategy, one population reduction campaign is performed in A_m (hatched area). Around A_m, an additional buffer with radius r_s is employed. The resulting area A_s is subject to increased hunting for monitoring purposes (dotted area with dashed outline in Fig. 2A). In the area A_s, hunting and testing is performed with increased hunting pressure parameter h_{inc}. For all other areas, the baseline hunting is maintained with h_{base}. Furthermore, from the first case detection onwards, each infectious carcass is detected in A_m and A_s with probability h_{carc} per surveillance campaign. Detected carcasses are removed from the model.

Once the initial campaign is performed, both strategies slightly deviate upon further case detections (Fig. 2B). In both strategies, any case (virus- or sero-positive or infected carcass) outside the management zone A_m triggers an extension of the zones (Fig. 2C). Under the barriers strategy, a fence is constructed to enclose the newly declared management zone ('additional barrier' in Fig. 2C). The surveillance zone is redefined by buffering the extended management zone by r_s (coarse dotted area in Fig. 2C).

Under the depopulation strategy, an additional update of the zones is triggered by subsequent detections of virus-positive animals or infected carcasses inside the management

zone. Buffering the new detections by r_m defines the depopulation zone for the next campaign (hatched area 'new depopulation zone' in Fig. 2C), and that area is added to the management zone. The surveillance zone is redefined by buffering the extended management zone by r_s (coarse and fine dotted area in Fig. 2C).

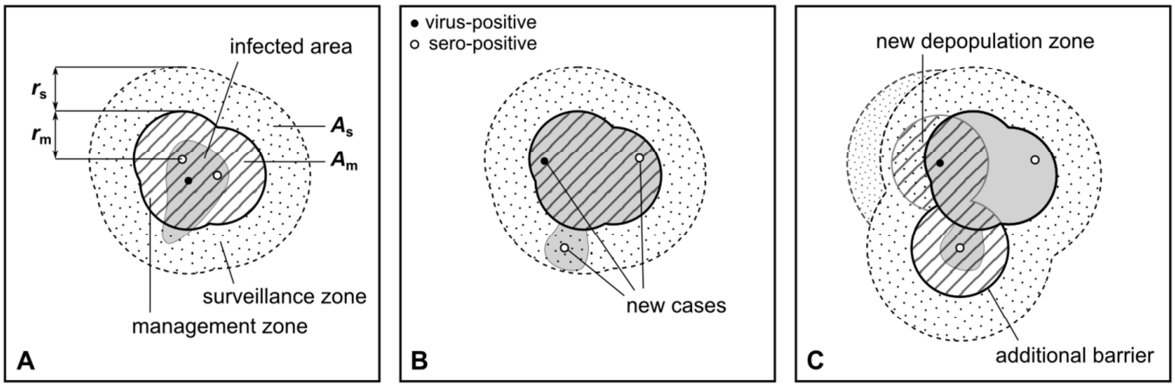

Fig. 2 Schematic diagram of management and surveillance. A) Initial case detections through virus-positive (filled dots) and sero-positive (hollow dots) animals, and measures implemented thereafter: management zone A_m, enclosed by barriers (solid lines) or with depopulation (hatched area). In both cases with associated intensified surveillance in a surrounding buffer (dotted area A_s). B) Situation at detection of cases after the initial campaign. C) Measures implemented thereafter: Any detection (virus- and sero-positive) outside the management zone A_m triggers the establishment of new barriers (solid lines) or a new depopulation area (lower part of hatched area). Additionally, virus-positive case detections inside the management zone trigger a new depopulation area (upper part of hatched area). Grey area: true infected area (unknown), circles: detected cases (filled: virus-positive, hollow: sero-positive); Solid lines: location of the movement barrier in radial distance r_f of the case detections; Hatched area: depopulation area; Dotted area with dashed edge: surveillance area of increased hunting in radial distance r_h of the movement barrier. Fine dotted area: additional surveillance zone under depopulation, triggered by virus-positive cease detection in management zone.

Depopulation is performed in each cell *i* by killing each individual with probability

$$p_{ki} = 1 - (d_{target} / d_i) \tag{5}$$

with target density d_{target} and the cell's actual density d_i. P_{ki} is constant cell-wise during a single campaign but varies between these.

Barriers reduce the probability of dispersers crossing between cells and deposition of carcasses. The remaining proportion of crossing events is determined by the barrier error rate p_{err}.

Disturbances due to depopulation activity are assumed to alter carcass deposition parameters $\hat{p}_{core}^{(s)}$, and the deposition probability of carcasses in the shared areas of neighbouring cells with probability p_{neigh}. The probability of deposition of a carcass inside the shared area of the individual's home cell is thus $1 - (\hat{p}_{core}^{(s)} + p_{neigh})$. Disturbances are applied to all cells where depopulation is performed in the week of the depopulation campaign and is continued for one more week.

Parameters, simulation experiments, analysis

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

Table 2. Model parameters.

Symbol	Description	Value	Source / details
p_L	probability of lethal infection	0.95	(Blome et al. 2012)
$a_{core}^{(s)}$	sex-specific probability to encounter one carcass in the core area	0.5 (females) 0.25 (males)	
$a_{shared}^{(s)}$	sex-specific probability to encounter one carcass in the shared area	0.5 (females) 0.75 (males)	
$P_{\text{inf}}^{(i)}$	Infection probability by direct transmission within herds	0.05	
$P_{\text{inf}}^{(c)}$	Infection probability per carcass	0.2	
t_{inf}	infectious period	1 week	(Blome et al. 2012)
t_{carc}	Time of carcass persistence	8 weeks	
p_{core}	Probability of virus-induced death in core area	0.9 (females) 0.8 (males)	
$\hat{p}_{core}$	Probability of virus-induced death in core area under disturbance	0.333	(Sodeikat & Pohlmeyer 2003)
p_{neigh}	Probability of virus-induced death in neighbouring cell under disturbance	0.333	(Sodeikat & Pohlmeyer 2003)
α_f	Fertility reduction if ill	0.625	
P_{PI}	Probability of prenatal infection	0.5	
T_{anti}	Maximum persistence of maternal antibodies	15 weeks	(Depner et al. 2000)
T_{immune}	Maximum duration of immunity by maternal antibodies	12 weeks	(Depner et al. 2000)
N_{disp}	Minimum number of subadult females for dispersal	2	
D_{disp}	Maximum dispersal distance for subadult females	6 km	(Sodeikat & Pohlmeyer 2003)
S_t	Male dispersal steps	8 (16 km)	
S_w	Male dispersal steps per week	4 (8 km)	
p_{ori}	Probability of oriented movement during male dispersal	0.5	
$T_{\max}$	Maximum age of boar	11 years (572 weeks)	(Jezierski 1977)
$SP_{mean}^{(a)}$ / $SP_{\min}^{(a)}$	Mean / minimum annual survival rate	0.65 / 0.4	(Focardi et al. 1996)
$SP_{mean}^{(y)}$ / $SP_{\min}^{(y)}$	Mean / minimum annual survival rate	0.65 / 0.4	(Gaillard et al. 1987)

Symbol	Description	Value	Source / details
$SP_{mean}^{(p)}$ / $SP_{\min}^{(p)}$	Mean / minimum annual survival rate	0.5 / 0.1	(Focardi et al. 1996)
r_m	Radius of management (fencing / depopulation) zone	4km, 8km, 12km, …, 40km	
r_s	Radius of outer (surveillance) zone	20 km	
t_h	Time between surveillance and hunting campaigns	4 weeks	
h_{base}	Proportion of population hunted and tested during each initial surveillance campaign (before first case detection)	0.001	
h_{inc}	Proportion of population hunted and tested during each surveillance campaign in the surveillance zone	0.01	
h_{carc}	Proportion of carcasses to be found during each surveillance campaign in the surveillance and management zones	0.01	
d_{target}	Target population density of depopulation	2 km^{-2}	
p_{err}	Failure rate of barriers, i.e. proportion of contacts and crossings not prevented by barriers	0.0, 0.1	

Independent variables: The primary independent variables are the management strategy (barriers or population reduction) and the radius of the management zone r_m.

Simulation experiments: Five management strategies were simulated:

1. N: reference scenario without any management
2. B00: perfect barriers, without permeability ($p_{err} = 0.0$)
3. B10: barriers with 10% permeability ($p_{err} = 0.1$)
4. H2-: depopulation to 2 heads/km² (culling of ≈ 60% of the population), no disturbances ($d_{target} = 2$, $p_{neigh} = 0.0$, $\hat{p}_{core}^{(f)} = 0.9$, $\hat{p}_{core}^{(m)} = 0.8$)
5. H2+: depopulation to 2 heads/km², with disturbances ($d_{target} = 2$, $p_{neigh} = 0.333$, $\hat{p}_{core}^{(f)} = \hat{p}_{core}^{(m)} = 0.333$)

Each strategies, except N, was simulated for different radii of the management zone $r_m \in \{4km, 8km, 12km, \dots, 40km\}$. The radius of the surveillance zone was fixed at $r_s = 20km$ for all strategies. Each combination of management strategy and radius of the management zone was simulated with 120 repetitions, resulting in a total of 4,920 simulations. All simulations were run until virus extinction (i.e. no infected animals and carcasses), but for a maximum of 20 years.

In addition, strategies N, B10 and H2+ were simulated under more pessimistic assumptions of the behaviour of moribund hosts. Here, a lower probability of carcass deposition in the core area was assumed with $p_{core}^{(f)} = 0.75$, $p_{core}^{(m)} = 0.33$. The radii of the management zone and the 120 repetitions were as described before.

Dependent variables: Dependent variables are the time to virus extinction / eradication, the total infected area, the length of erected barriers and the number of hunted animas.

Analysis: The outcome of the dependent variables of individual simulation was aggregated by management scenario and radius of the management zone. The distributions of the dependent variables are presented as boxplots.

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