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Understanding ASF spread and emergency control concepts in wild boar populations using individual-based modelling and spatio-temporal surveillance data

Martin Lange, Vittorio Guberti, Hans-Hermann Thulke

PG EcoEpi

Helmholtz Centre for Environmental Research GmbH – UFZ

Department of Ecological Modelling

Abstract

African swine fever (ASF) infection is circulating in Eurasia since a decade within wild boar populations without a demonstrated vector host. Further the infection was recurrently translocated by spatio-temporal dynamics that is incompatible with wild boar movement characteristics. Management actions are required in areas affected by ASF. Control measures address areas with recent focal introduction and areas with ASF circulating several seasons or endemic occurrence. In view of acknowledged gaps in understanding ecology and epidemiology of ASF in Eurasian wild boar, mechanistic modelling was applied. A comparative assessment of alternative control efforts in the focal situations was performed, considering pre-emptive hunting, carcass detection and removal as well as fencing of selected zones. The individual-based model was applied to test whether inclusion of natural barriers would affect the similarity with ADNS notification data when simulating the landscape scale spread in the Baltics and Poland. The tendency of barriers to improve the explicit space-time predictions of the model could not be proven, more research is needed here. The comparative assessment revealed that in the focal scenario the increasing removal of carcasses will provide the greatest return on investment (given carcasses being involved in the transmission). Culling of the inner zones, usually with ASF detections, should be organised early if biosecurity standards could be guaranteed. Preventive hunting around the zones affected by ASF was an inevitable measure to cope with the risk of undetected release of the infection, although the measure alone was unable to terminate the spread. Fences assumed not wild boar proof contributed only marginally to the success but may act as demarcation of management zones in practice. The focal approach appears useful and practical to address ASF after local introductions.

Key words: African swine fever, individual-based, spatial-temporal simulation, control measures, hunting, carcass removal, fencing, natural barriers, wild boar

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Correspondence: ahaw@efsa.europa.eu

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Table of contents

1. Introduction

1.1. Background and Terms of Reference as provided by the requestor

African swine fever (ASF) is a devastating infectious disease of domestic pigs and wild boar. From the beginning of 2014 up to now, Genotype II of ASF has been notified in Czech Republic, Estonia, Latvia, Lithuania, Poland and Romania causing very serious concerns.

EFSA has been requested by the European Commission to provide scientific and technical assistance on ASF (Mandate M-2017-0217). The latest scientific report of EFSA on ASF was published in November 2017 and it reviewed the epidemiological data on ASF from the Baltic States and Poland in order to update the analysis carried out in the EFSA opinion on ASF of 2015. The EFSA opinion and this technical report are supporting the Commission for reviewing the EU ASF strategy. The Commission is in need of an updated epidemiological analysis by EFSA of the latest ASF data related to both domestic pigs and wild boar.

Modelling the spread of the disease in Europe and an assessment of the effectiveness of the implemented measures by the MSs should be included in the next EFSA scientific report to reply to the Terms of Reference (TOR) of the mandate, in particular TORs 3-5:

TOR3. Review the control measures applied by the affected Member States for controlling the spread of the disease in wild boar and for eradicating it. Assess their effectiveness and review scientific literature addressing these measures.

TOR4. Review and assess the robustness and effectiveness of the different types of geographical artificial or natural boundaries used for the determination/demarcation of the restricted areas.

TOR5. Based on the latest science and epidemiological data, review the measures for managing the wild boar populations in four separate geographical areas:

· Disease free areas, far away from any ASF occurrence, which should take long term actions for preparing for a future possible incursion of the disease considering the human factor;

· Disease free areas neighbouring infected or restricted areas at higher risk of getting the infection mainly via natural spread of the disease through wild boar;

· Areas where the disease was recently introduced in wild boar;

· Areas where the disease has been present in the wild boar population for quite some time (more than one year).

In this context, EFSA had launched this procurement to obtain technical assistance in developing and validating an ASF spread model to assess the effectiveness of wild boar management options in preventing spread of ASF and/or its eradication.

Specific objective:

The objective of the contract is as follows:

Development, validation and use of an ASF spread model and to assess the effectiveness of control measures in preventing spread of the disease and/or its eradication

a) The spread of ASF in wild boar populations should be modelled for 3 different scenarios, namely:

(1) spread from a focal introduction of ASFV in a wild boar population located far from the current affected areas,

(2) spread of ASFV from an adjacent affected areas and

(3) spread within endemic areas (affected for more than 1 year).

The model should mimic the specific wild boar populations of the affected Member States (i.e. taking into account the abundance data provided by EFSA). It would be an asset if specific natural and

artificial landscape elements (e.g. rivers, canals, roads), land cover types (e.g. corine land cover) could be included and the effectiveness of natural and /or artificial barriers on the spread of ASF could be evaluated by the model.

b) Furthermore, the model should be able to assess different types of control measures that have been implemented to stop the spread and/or eradicate ASF: wild boar population reduction measures, changing the density or demographic composition of the wild boar populations with different levels of compliance/success. Also the removal of carcasses should be evaluated and their combinations with different levels of compliance/success in terms of detecting and removal of infectious carcasses from the environment. Finally, the model should be able to evaluate the effectiveness of the application of different combinations of the above listed measures.

The model should be validated using field data from local studies/epidemiological investigations (e.g. wild boar density, control measures), complementing data that will be provided to the successful tender by EFSA (i.e. laboratory results of active and passive surveillance of wild boar) and the ENETWILD consortium (i.e. data on wild boar occurrence and the number of harvested wild boar per month) from the ASF affected Member States.

1.2. Interpretation of the Terms of Reference, Objectives & Purpose

The background information addresses the following specific objectives:

1. Model development and validation
2. Demonstration of possible impact of natural barriers on the spatial spread of ASF in wild boar populations
3. Comparative assessment of alternative management measures regarding their effectiveness to limit spatial spread and/or finally eradicate ASF in wild boar using population reduction and carcass removal as options including targeted artificial fences.

The report details the research activities of the NP/EFSA/ALPHA/2018/01 addressing these objectives while contributing to ToRs of the EFSA mandate on Epidemiological reporting on ASF in wild boar foreseen by end of this year (EFSA 2018). The particular aspects considered with the model relate to the establishment of a fit-for-purpose model (3.1.), the relevance of natural barriers (3.2.) and the evaluation of design parameters of the currently debated/applied control programmes with particular reference to the measures proposed after the focal emergency strategy (3.3.). Further, it is deemed useful to test whether the proposed strategy to combat focal introductions of ASF into wildlife would be different if applied at the forefront of the large-scale region in North-East Europe affected by ASF notifications in wild boar (3.4.).

In this report, technical details of methodology and the structured output of the simulations including uncertainty discussion is provided. The interpretation in terms of the requested ToRs is not understood as in the remit of this report and the reader may refer to EFSA output (EFSA 2018).

1.3. Model development and parameterisation

1.3.1. Model validity

The module representing wild boar ecology was validated independent of ASF problems in terms of and habitat use predicted by the model rules, regarding reproduction, breeding capacity and sub-adult dispersal. Validity of predictions was field-verified with spatial distribution of opportunistic sighting of wild boar in Denmark (Moltke-Jordt et al. 2016). Moreover, the model was shown to accurately predict geographical disease spread and perpetuation time if the modes of infection and transmission are adequately understood (EFSA, 2012; Dhollander et al. 2016).

1.3.2. Modelling transmission of ASF infections in wild boar

The most important uncertainty when modelling ASF transmission processes in wild boar relates to the epidemiological relevance of different potential modes of ASF perpetuation. Wild boar is acknowledged to organise in a matriarchal structure with female groups of strong kinship and satellite solitary movement and temporary aggregation of males with sow groups. Consequently the wild boar-ASF-system comprises three potential modes of transmission, i.e. between live animals of the same social group (within group transmission), between live animals of different groups (between group transmission) and between carcasses of animals succumbed to the infection and live animals (carcass-mediated transmission).

Quantitative experimental data is accessible only for within-group transmission, i.e. animals in permanent contact with groupmates. Here, several transmission experiments with inoculated animals are documented (Guinat et al. 2014; Pietschmann et al. 2015; Oelsen et al. 2018). There is published literature available on trials studying transmission between domestic pigs in contact with each other, which propose estimates of the reproductive ratio of different magnitude, more or less clearly, excluding one from above (R0>1).

Very experimental evidence exists regarding the role of carcasses of animals dying consequent to ASF infection, including the possibly of contaminated soil thereunder. Given the assumption that carcass-mediated transmission is relevant, first insights exist on the mechanism (Lange & Thulke 2017; Probst et al. 2017). The frequency of live animal visits to point locations with a carcass over time of its decomposition or beyond was addressed in an observational study (Probst et al. 2017). The results underpinned the data-driven model analysis addressing the causalities of space-time-interaction of existing notifications of infected wild-boar (Lange & Thulke 2017). Based on both studies ubiquitous access to dead animals (i.e. no hiding or retreat due to morbidity) can be assumed but very seldom actual contacts that may warrant transmission (blood, secrets or body fluids). The critical result of Probst et al. (2017) regarding a potential delay period of about two weeks after death before contacts to carcasses were observed, was recently questioned in the light of latest field studies with carcasses (K. Depner & A. Viltrop pers.comm).

In preparation of this report, the uncertainty concerning between group transmission by live animals was addressed (i.e. transmission contact between adjacent social groups other than through dead animals). The related scientific debate is using plausibility arguments. Anecdotal evidence from epidemiological investigations in outbreak farms suggests a scattered and spatially limited distribution of infected pigs when barns are compared across an infected holding. Such observations may imply only limited transmissivity of infection if permanent contacts, as usual between socialising animals, are not possible. Anecdotal evidence suggests limited potential to transmit ASF between in-contact pigs as long as they are not showing strong clinical evidence involving blood excretion, i.e. contact pigs in several experiments that escaped infection until getting accidental access to blood of co-housed ill animals (S. Blome pers. comm.). Potential assembly points, e.g. watering or baiting places, could be a plausible source of between-group infectious contacts (EFSA SWG on ASF pers. comm.) and contradicted with the suggestion that animals with clinical disease quickly become heavily morbid preventing movements to assembly points but forcing erratic drop down of the individuals (S. Blome pers. comm.). Alternative model scenarios regarding the between-group contacts were compared with observational data to contribute to the debate (3.1).

1.4. Natural barriers

The relevance of natural barriers (3.2.) is addressed in response to the request to assess the effectiveness of geographical artificial or natural boundaries used for the determination of restricted areas {EFSA 2018b}. Unfortunately, potential quantitative experimental data of protective effect of large rivers and broad highways were not accessible. Therefore the objective was addressed in a reverse approach: If artificial or natural barriers delineating certain geographic area would impact the spatial spread of the ASF infection, then this impact has to be comprised in the set of ASF notifications recorded by the ADNS. Consequently, the continental scale simulation of ASF spread in the Baltic

Member State (MS) and Poland was performed either without or with the effect of such barriers (i.e. either line elements at position of highways and rivers were integrated in the model landscape or not). When the similarity of the resulting simulation outcome and the spatio-temporal notification data of the ADNS is calculated, the potential impact of these barriers should increase the similarity with barriers compared to no barriers. The increase in similarity between spatial model output and ADNS data is not a necessary consequence of barriers impact (i.e. impact could be weak, seasonal or not a general feature along a highway's or river's lengths). Hence failure to demonstrate a similarity increase cannot be reversely interpreted as proof of absence of any other barrier impact.

1.5. Control schemes

The focal emergency strategy (3.3.) refers to the localised emergency control measures as applied in the Zlin area (CZ). There, the focal introduction of infection into wild boar (no domestic pigs involved) was addressed with a circular approach combining narrow fencing of detected ASF-positive carcasses, a carefully managed buffer area encircling the core area while an intensive hunting area was assigned further around the buffer area (in the Zlin context the areas were differently named and were of slightly higher complexity; see P. Satran (2017). SVA report: PLAN OF THE ERADICATION OF AFRICAN SWINE FEVER IN THE POPULATION OF WILD BOARS for more detailed description). The approach was translated into a generalized strategy concept and alternative parameterisations were studied with the epidemiological model (Figure 1):

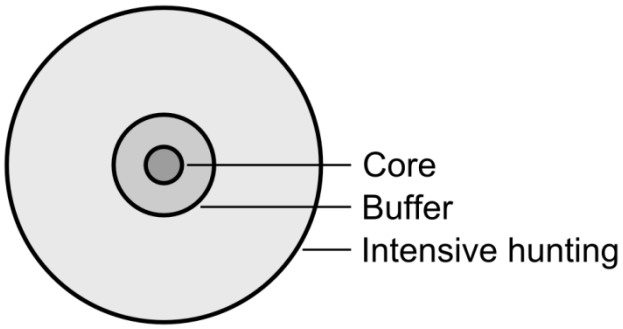


Figure 1. Different wild boar management zones considered in the model

The Core zone refers to the smallest circle around all detected ASF positive carcasses at the moment of the start of the application of zoned measures.

The Buffer zone is surrounding the core area and is meant to separate the Core and the Intensive hunting zone from each other to minimise disturbance of the former by the hunting activities in the latter.

The Intensive hunting zone is surrounding the buffer zone and demarcates the area to which intensified hunting measures are applied for the purpose of population reduction.

In this document population reduction measures inside core and buffer zone are called '**culling**' as non-conventional depopulation methods may be foreseen. Population reduction measures in the intensive hunting zone are called '**hunting**' as regular hunting tools are foreseen to be used.

Strategies were compared in terms of the overall success rate of alternative packaging of measures independently applied to the subareas, the spatial risk of ASF spread when control measures are applied accordingly, and the number of animals hunted or culled. Given the acknowledged uncertainties relating to the perpetuation of ASF infection in wild boar populations and the achievable intensity of control measures, maximum variation of parameters has to be explored. The interpretation of the findings addresses tendencies observed between alternative scenarios rather than the absolute specification of one general implementation value per parameter of the strategy design.

The adequate application of control tools forefront of the large-scale affected regions (3.4.) in North-East Europe (i.e. Baltic MS and Poland) was addressed in previous EFSA outputs sufficiently detailed (EFSA 2015; 2017; 2018a}. Here the question was if it is purposeful to apply the usefully parameterised focal strategy to non-affected areas at higher risk of ASF introduction via natural spread mediated by wild boar.

2. Data and Methodologies

2.1. Data

ADNS case reporting data as of October 2018 was provided by EFSA. The wild boar habitat model by Pittiglio et al. (2018) was converted into the breeding capacity raster according to BreedingCapacity [per cell] = 1.28 * Density [per km²].

Geodata on barriers were of different sources. Rivers were derived from European Environment Agency's WISE Large rivers and large lakes (https://www.eea.europa.eu/data-and-maps/data/wise-large-rivers-and-large-lakes); and roads of OpenStreetMap [highway=motorway] or [highway=trunk]. GIS data was download via QGIS plugin QuickOSM.

2.2. Methodologies

2.2.1. Model development and parameterisation

2.2.1.1. Language conventions

Infected carcass = Carcass from infected wild boar that succumbed due to ASF disease

Management zones = Zones with measures to control ASF spread, i.e. Core, buffer and intensive hunting zone (see section 1.5)

Zoning = Definition and set-up of management zones followed by start of scheduled measures

Core zone = Encircled detected ASF positive carcasses at the moment of zoning

Buffer zone **=** Surrounding the core zone.

Intensive hunting zone = Surrounding the buffer zone, dedicated to population reduction by regular hunting practices and carcass detection/testing/removal

Population reduction measures in the intensive hunting zone are called `hunting´ as regular **'hunting'** tools are foreseen to be used. Depopulation measures inside core and buffer zone are called **'culling'** as non-conventional depopulation methods may allow greater effectiveness

2.2.1.2. Model framework and documentation

The report is based on a spatio-temporally explicit individual-based model approach in structured geographic landscapes. The model framework has been developed and applied in the context of multiple infectious diseases of wild boar, i.e. CSF, FMD, ASF. The model compiles (i) an ecological component detailing processes and mechanism related to the ecology, sociology and behaviour of wild boar in natural free-roaming populations of the species Sus scrofa; (ii) an epidemiological component reflecting individual disease course characteristics and transmission pathways including direct contact transmission on different spatial scales and environmental transmission caused by ground contamination or contacts with carcasses of succumbed infected host animals; and (iii) a management component implementing surveillance and control scenarios in a spatio-temporal explicit manner. The model is stochastic in relation to all three components and parameterised using reported distributions from literature including variability and uncertainty. The model is simulated on heterogeneous landscapes of several thousand square kilometres, including real geographies derived from e.g. corine

land cover data. Model population emerges from birth and death probabilities depending on habitat quality on the level of individual social groups.

The model is documented according to the ODD protocol (Overview, Design, Details following Grimm et al. 2006; Grimm et al. 2010). The documentation is attached as Appendix A (http://ecoepi.eu/ASFWB). Model adaptations performed while approaching the objectives are additionally detailed in section 3.1 of the Assessment.

2.2.1.3. Amendments of the transmission model

The proposed epidemiological model of the wild boar-ASF-system comprises **three modes of transmission**: between live animals of the same social group (within group transmission; parameter $P_{inf}^{(i)}$), between live animals of different groups (between group transmission; parameter $P_{inf}^{(b)}$) and between live animals and carcasses of animals succumbed to the infection (carcass transmission; parameter $P_{inf}^{(c)}$).

The model was extended to reflect between group transmission mediated between live animals of different social groups, i.e. the mechanism applied with a CSFV variant of the model (Kramer-Schadt 2009; Lange et al. 2012). For each susceptible animal, the probability of becoming infected additionally accumulates over all infectious animals not in the same social group but in direct spatial neighbourhood of their home-range, i.e. social groups that are assumed to roam over a joined foraging range, i.e. overlapping movement range:

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

where λ_b is the number of infectious individuals in all contact groups relative to the receiving individual and the parameter $P_{inf}^{(b)}$ determines the probability of contracting the infection if one infectious animal lives for one week in any of the neighbouring group. The parameter $P_{inf}^{(b)}$ is bounded between 0 and $P_{inf}^{(ic)}$, i.e. the probability to contact infection from a group mate during the same week. The ecological data accessible in literature is scare, fragmented and heterogeneous. Therefore it does not suggest a direct parameterisation of $P_{inf}^{(b)}$. Instead a pattern-oriented approach was performed (Grimm et al. 2005).

2.2.1.4. Model analysis

The effect of alternative choice of the parameter $P_{inf}^{(b)}$ is measured by the speed of propagation and sustainable spatial spread. The first was implemented as the average distance the front wave was travelling across the landscape as function of time; the second by calculating the proportion of runs in which the infection covered 7/8th of the landscape. Both measures were valued against observational evidence from the field.

2.2.2. Modelling natural barriers

Alternative landscapes were applied in simulations referring to presence or absence of natural barriers reflecting expert debate in the SWG.

The conceptual understanding of the impact of natural barriers on the wild boar-mediated spread of ASF in geographical populations is still unclear. Therefore the modelling will provide a descriptive rather than explanatory understanding of the impact of natural barriers. The simulations mimic the continental spread of ASF on a map of the Baltic countries. Uncertainty of the approach is very large, noting that wild boar experts deny e.g. the impact of large rivers while anecdotal observations show certain slow-down of continental spread at e.g. particular segments of big rivers (Rossi et al. with CSF in wild boar at river Rheine).

The model introduced by the ODD Annex (see http://ecoepi.eu/ASFWB) is applied in combination with ADNS notification data (2.1.). The local spread velocity was estimated and likely human-mediated translocations identified based on the time and distance between notifications. Re-enforcing the likely human-mediated translocations (n=258), the continental spread is simulated on the habitat map of the region under study (Figure 2a) developed after the wild boar distribution map by Pittiglio et al. (2018). An alternative map was produced incorporating rivers and highways as suggested physical barriers to wild boar movement (Figure 2b). On the latter the continental spread of ASF infection was simulated with barriers preventing transmission events across the elements. Simulations assuming fully permeable barriers are compared to simulations with perfect barriers. The assessment will reveal whether considering barriers preventing transmission would improve the similarity of simulated epidemics with the spatio-temporal structure of the ADNS data.

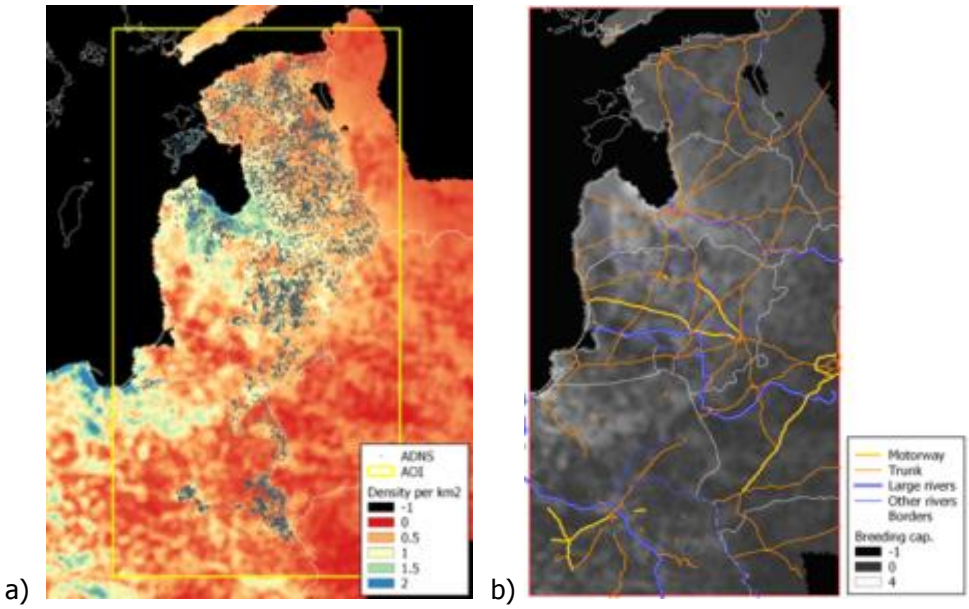


Figure 2. Habitat model of wild boar and physical barriers for the Baltic countries. a: Carrying capacity values (expressed in density per km2) according to the distribution model proposed by Pittiglio et al (2018). b: The carrying capacity overlaid with barriers

2.2.3. Modelling management options

2.2.3.1. Simulation setting

Simulation design implemented the proposed management structure comprising core, buffer and intensive hunting zone (Figure 1). One random realisation in shown in Figure 3a. Each simulation run used a particular set of parameters defining per zone the width, applied control measures, start of control measures, effort in implementing the measures, scheduling of the effort and potential side effects.

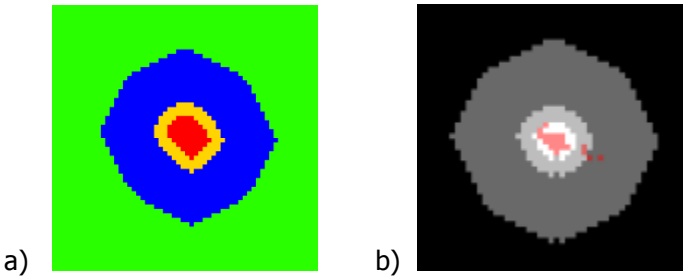


Figure 3. a) Outcome of the zoning sub-model for one arbitrary run. Zoning is based on random carcass detection during four weeks after the very first random notification. Red core zone; yellow buffer zone; blue intensive hunting zone; and

green landscape to protect. b) Example simulation snapshot showing true infection status of the wild boar groups at the moment of zoning (4 weeks after very first notification). Here, the zoning is based on perfect knowledge about distribution of infected carcasses and a 100% wild-boar proven fence is assumed around the core zone (white area). Zoning is based on carcass distribution, while newly infected live animals had entered the buffer zone (light grey area). As a consequence, even simulations assuming 100% wild-boar prove fences can fail.

The core zone (and the potential fence) is established by adding 2 home range diameters around all notified infected carcasses. Hence, the model output comprises the effect of zoning being based on carcass distribution estimate or knowledge and not on distribution of infected animals (Figure 3b).

2.2.3.2. Model analysis

Model analysis was performed using 100 runs per parameter scenario. Runs were only recorded if any management actions were triggered (i.e. spontaneous fadeouts prior to determination of zones were not included). The proportion of all runs in which ASF appeared beyond the buffer zone was assessed and plotted against the width of the intensive hunting zone. For every width of the intensive hunting zone the proportion of runs breaking off the respectively delineated zone was recorded. Results are shown as failure curves over width of the intensive hunting zone i.e. the proportion of simulation runs that crossed the core-buffer or a particular width of the hunting zone. Additionally, per simulation run the first involvement of every particular wild boar group home-range cell was recorded and infection probability heat maps were created.

2.2.3.3. Parameter variations

Parameters relevant for the implementation of the control measures are listed next and the standard parameterisation is given in red together with explored parameter variation covering possible aspects of the potential strategy.

Core zone encircles all carcasses detected within 4 weeks (pre-set) after first notification, plus 2 home ranges. Core zone is delineated according to random carcass detection (detection probability see below) within 4 weeks, or perfect knowledge about carcasses (not the same as those removed).

Buffer zone comprises the core zone and adds another 3 home ranges (variation 1, 2, 3, 4, 5) in each direction.

Intensive hunting zone encircles the buffer and adds another 0, 3, 6, 9, 12 home ranges around in each direction (e.g. width 0, 9, 18, 27, 36 km if home-range diameter is assumed to be 3km)

Zones are established based on carcass retrieval information, i.e. either notified carcasses from infected animals using 10% detection chance during four weeks after first notification (standard) or, as reference, based on perfect knowledge about all carcasses in the landscape (here, notified carcasses will not be removed automatically but according to carcass removal rate!).

Hunting protocols are implemented via campaigns:

- Campaigns have a duration over which a specified effort has to be delivered, i.e. 4 weeks
- Campaigns are performed 2 times per year (varied: 1, 2, 4, 13 times).
- Total number of campaigns during the simulation run, i.e. lengths of the control programme, was set unlimited for intensive hunting zone and to one culling campaign in core+buffer but following different waiting times (see below).
- Hunting proportions of live animals (DepopProp): Advice to hunt 75% (varied 0-99%) of the accessible animals per year. Effectively, animals are hunted per week with adequately adjusted partial probability x% considering the advice, the length of one campaign and the frequency.
- Hunting numbers per wild boar group (DepopNumber): Advice to hunt k animals (0, 5, 10, 20, 40) per wild boar group per year, i.e. distributed over all campaigns applied in one year.

- Core+buffer zone are culled after waiting 26 weeks (4, 8, 13, 26, 39, 52 weeks) with effort of 90% (varied 0-99%) in one 4-week campaign.

Hunting/Culling disturbance increases space use of infected animals by 1 home-range (1, 2, 3, 4 hr) and lasts 4 weeks after the hunting campaign.

Carcass detection rate 10% before zones are set up (see EFSA 2017).

Carcass detection rate after zones are set up lead to carcass removal rate of 1% (0%; 10%; 20%, 40%, 80%.

Carcass infection delay 2 weeks! (varied to 0)

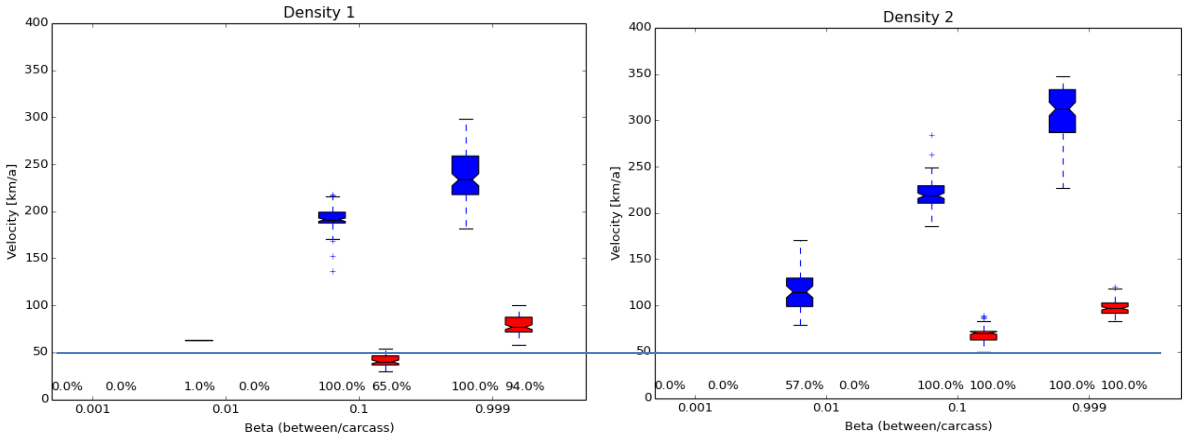
Fence has no effect on the system in the reported simulations where the fence was put as 100% permeable, i.e. being a line delineating core zone. Increasing the effect of the fence will limit movements/contacts of infectious animals and hence make all outcomes more optimistic while true fence efficiency is unknown. The simulated fences are built one week after zoning. Fences are assumed to be of varying permeability (90%, 50%, 10%, 5%, 0%). 100% equals the no-fence scenarios where the fence is a virtual line in the landscape; 0% is assuming wild-boar proof wall.

3. Assessment/Results

3.1. Model development and parameterisation

The following model output compares systematically changed assumptions regarding of transmission between social groups either by contact during live time of infected animals ($P_{inf}^{(b)}$; blue) or carcass-mediated ($P_{inf}^{(c)}$ red; no age-differences). Within-group transmission $P_{inf}^{(i)}$ was left unchanged using the standard parameterisations listed in the ODD protocol. The result is shown as box-plot across all scenario repetitions (stochasticity) valuing the annual speed of propagation conditioned on the actually achieved landscape spread (i.e. a spreading distance of at least 350km or 7/8th of the landscape). Observations from the field (horizontal line) urge to consider annual speed of propagation of about 25km (Podgorski et al. 2018) and hardly above 50km (EFSA 1818b; 3.1.1.4).

R1



R2

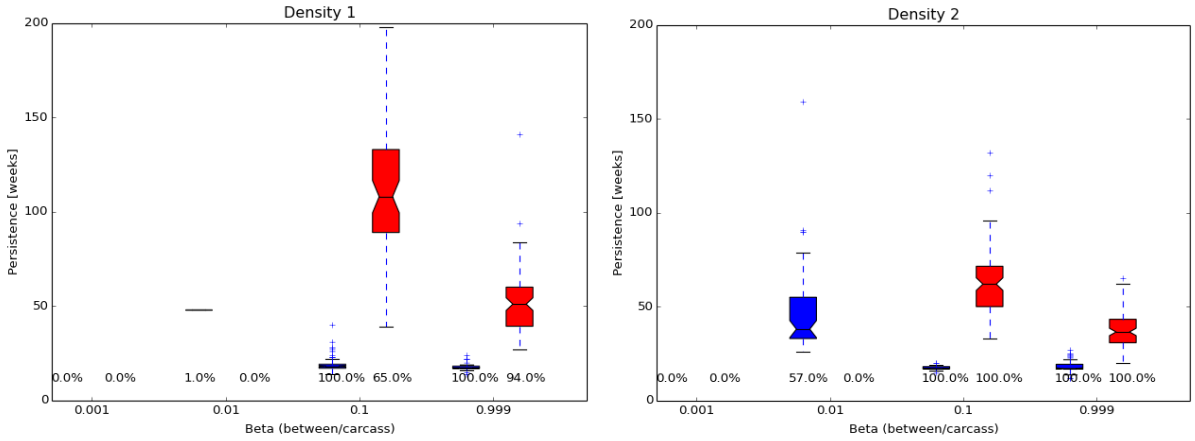


Figure 4. Speed of propagation and persistence time in ASF simulation runs applying alternative between group transmission modes i.e. between live animals (direct) vs. carcass-mediated (indirect) assuming alternative wild boar density (contact delay 1w, carcass decomposition 4w). Blue direct (i.e. $P_{inf}^{(b)}$), red indirect $P_{inf}^{(c)}$ between group transmissions. Numbers give proportions of runs that succeeded to cover 7/8 of the landscape (133x33 cells landscape dimensions; 100 runs). Box-plots summarize runs that covered the 7/8 of the landscape. Top row: Speed of propagation; Bottom row: Period of perpetuation after the whole landscape is covered once. Left panel standard Baltic density vs. doubled in the right panel.

All runs involving live-animal contacts between adjacent groups which achieved sufficient spatial expansion (blue in Figure 4 R2; i.e. no early fade out) did expand much faster than the suggested observational range (blue in Figure 4 R1). The impact the between live-animal transmission that could be pushed to not always exceeding the threshold for speed of propagation (e.g. 0.003 or less) is illustrated in Figure 5.

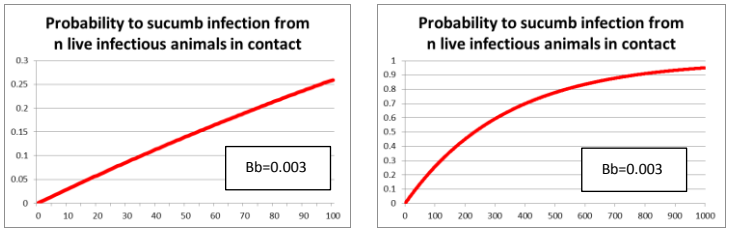


Figure 5. Illustration of the plausible values of between group transmission probabilities $P_{inf}^{(b)}$. The value allow transmission to a susceptible animal with 25% per week if 100 individuals in the neighbouring social groups are simultaneously infectious.

3.2. Natural barriers

The hypothesis was assess that simulated spatio-temporal spread on the continental scale could explain ADNS data better (i.e. in terms of matched spatio-temporal occurrence of ASF positive samples) if physical barriers are included in the simulation landscape compared to the simulation on a barrier free landscape. The barriers created new structures on the heat map of infection probability (Figure 6). However, the outcome valued in terms of similarity measures did not reveal the respective effect.

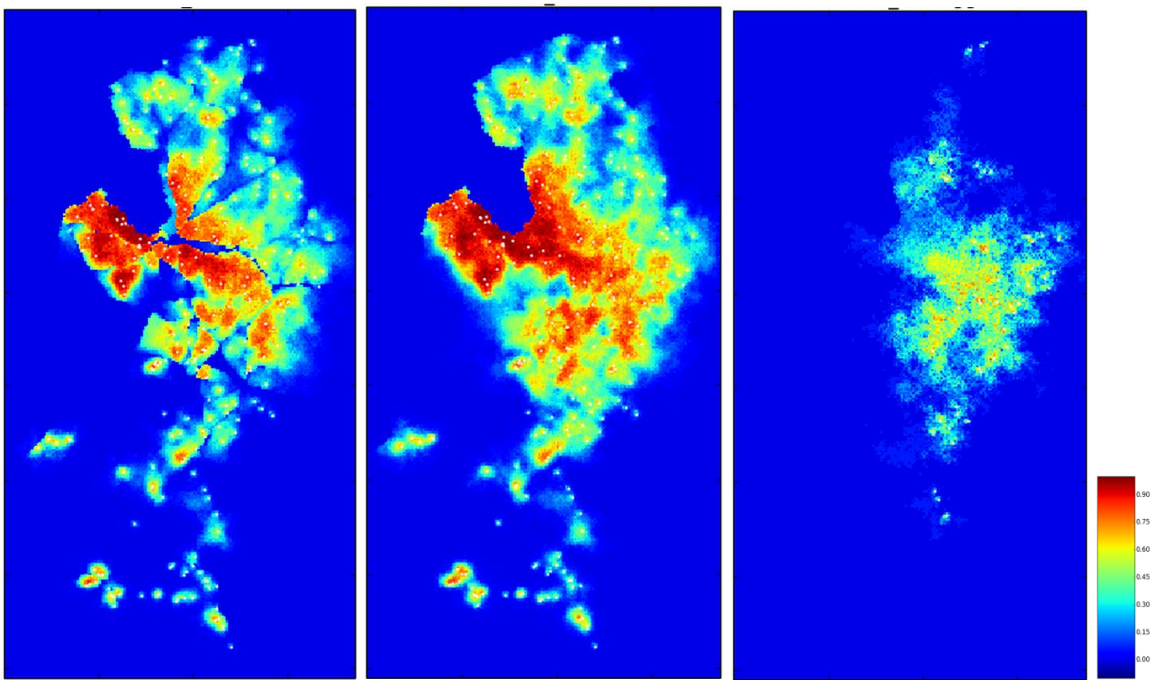


Figure 6. Probability to receive an ASF infection in wild boar dependent on physical barriers and likely human-mediated translocations. Heat map of 100 repetitions. Higher value (reddish) show greater probability. Left: Forced simulation assuming impermeable barriers (blocking walls); Middle: Forced simulation assuming fully permeable barriers; Right: As middle but without forcing from March 2015 onwards. White pixels represent forcing locations based on the excess values in local velocity.

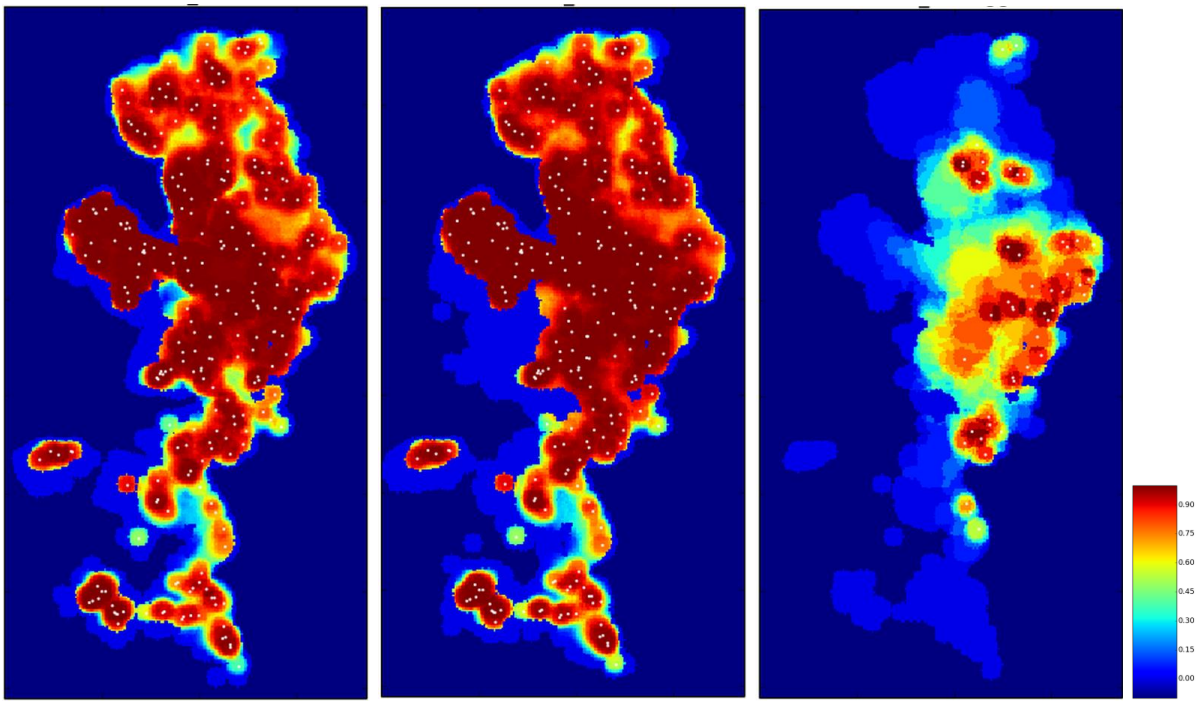


Figure 7. Jaccard Similarity map between ADNS and the final outcome of 100 repetitions considering combinations of physical barriers and likely human-mediated translocations. Higher value (reddish) show greater similarity. Left: Forced simulation assuming impermeable barriers (blocking walls); Middle: Forced simulation assuming fully permeable

barriers; Right: As middle but without forcing from March 2015 onwards. White pixels represent forcing locations based on the excess values in local velocity.

Uncertainty considerations: The investigations cannot provide absolute estimates of the effectiveness in terms of halting or slowing continental spread of ASFV due to the great variation in permeability of natural barriers in real life by e.g. bridges over rivers, variation in width or occurrence of shallow segments. The approach is pragmatic and the difference in matching performance between the compared simulations is not mandatory for barriers having certain effect on ASF spread, i.e. impact of barriers can be so small that the stochastic nature of the problem will hide the barrier effect. Further, the uncertainty of the practical implications is huge due to many processes acknowledged to alter continental spread of ASF in wild boar.

3.3. Control schemes

3.3.1. Simulation standard parameters

Parameters which were varied in certain experiment. Related changes are detailed under the experiment.

- BetaCarcass = 0.03
- DepopProp = 75%
- CarcassDetectionZones = 1%
- CarcassRemoval = 0.01
- InfectiousCarcassDelay = 2 w // carcass contact delay 2 weeks
- WaitingTimeDepopZones = 0 w
- WaitingTimeCullingCore+Buffer = 26 w
- CampaignIntervalZones = 26 w
- DisturbanceRadius = 1 home range
- Zoning based on sample information

3.3.2. List of Experiments:

Exp: General output presentation / Uncertainty quantification
Exp: BetaCarcass x DepopProp
Exp: BetaCarcass x DepopProp - no delay
Exp: BetaCarcass x DepopNumber
Exp: CampaignInterval x DepopProp
Exp: CarcassDetection x DepopProp
Exp: CarcassDetection x DepopProp - perfect info
Exp: CarcassDetection x DepopProp - perfect info - no delay
Exp: DepopPropCore x WaitingTimeCore
Exp: DisturbanceRadius x WaitingTimeCore
Exp: FencePermeability x CarcassDetection x DepopProp – perfect info
Exp: CarcassDetection x BufferZoneWidth
Exp: CarcassDetection x BufferZoneWidth - perfect info
Exp: CarcassDetection x DepopProp linear

3.3.3. Simulation outcome of Experiments

3.3.3.1. Output measures & uncertainty

*Exp: General output presentation / **Uncertainty** quantification*

Stochastic variation of the outcome measure is quantified by performing 60 repetitions of the standard simulation of 100 runs, but always using identical parametrisation (Figure 8). The remaining document uses only simulation package of 100 runs for each plotted line.

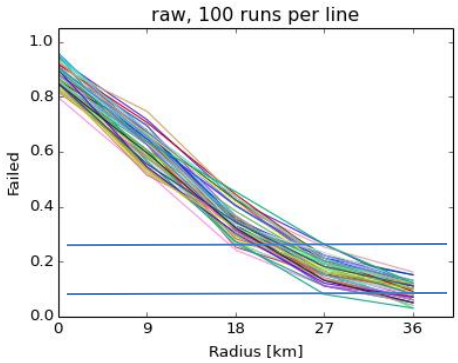


Figure 8. Variability of the simulation outcome addressing success rate of a control strategy.

The figure illustrates the principle way to represent the model outcome. Model outcome is presented as probability to fail using a given strategy combination. Failure is the breakout of the respectively sized intensive hunting zone. The x-axis shows the distance to the edge of the buffer zone in kilometres (i.e. number of group home-range diameters multiplied by 3km). Only runs that caused measures were included in the analysis. The y-axis reports the proportion of 100 runs that had ASF infected wild boar at the x-value distance from the buffer edge. One minus the zero value of the x-axis reveals the proportion of runs that were successfully terminated inside the core+buffer. For example, in Figure 8 between 10-20% runs terminated inside the core+buffer as the failure rate at value 0 is 80-90%. Furthermore, in the particular scenario about 75-90% of runs were stopped (failure between 10-25%) using a 27km wide intensive hunting zone.

3.3.3.2. Variation of hunting efforts – no carcass removal, no fence

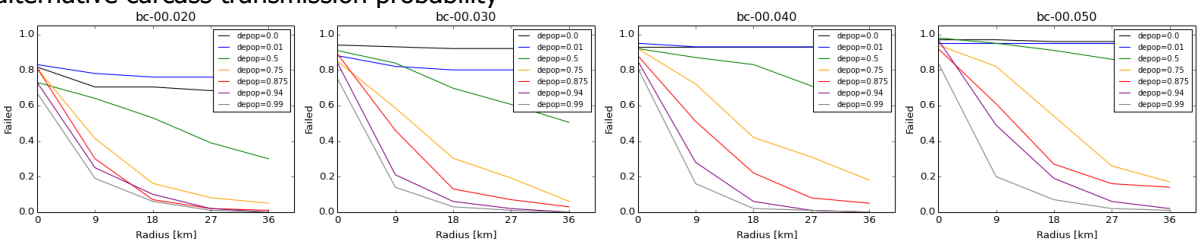
Exp: ***BetaCarcass x DepopProp***

- BetaCarcass [0.02, 0.03, 0.04, 0.05]
- DepopProp [0.0, 0.01, 0.5, 0.75, 0.875, 0.94, 0.99]

Variation of intensive hunting zone dimension (Radius/rad) from 0 to 36km width of the intensive hunting zone (i.e. for the Baltic scenario this would associate to 0 to 12 group home-range diameters – in regions with more densely wild boar occupation the extent of a group might be smaller (Keuling et al. 2008).

Variation of the hunting effort (DepopProp/depop) in the intensive hunting zone from 0% to 99% per year. The effort is distributed over all campaigns scheduled each year (see scenario variation number campaigns). The parameter value refers to the total effort over the year.

R1: By width of the intensive hunting zone (x-axis), hunting effort in intensive hunting zone and alternative carcass transmission probability



R2: By hunting effort in intensive hunting zone (x-axis), width of the hunting zone and alternative carcass transmission probability

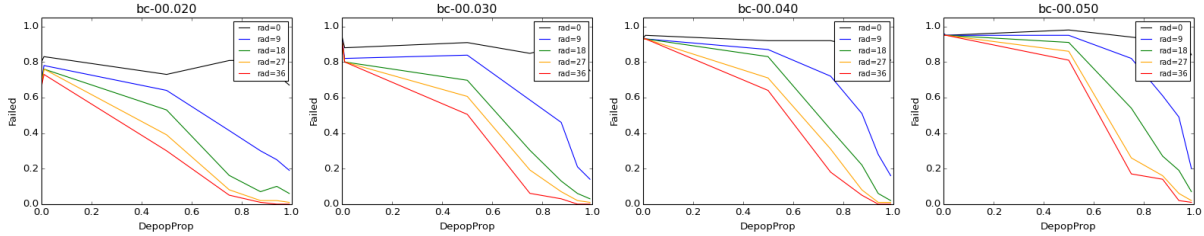


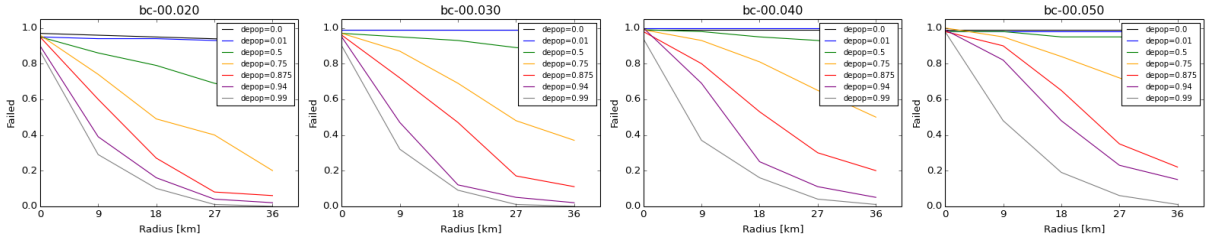
Figure 9. Simulation outcome regarding impact of hunting effort in differently sized intensive hunting zones. No carcass removal. No fence.

Exp: BetaCarcass x DepopProp - ***no delay***

- BetaCarcass [0.02, 0.03, 0.04, 0.05] (i.e. not adjusted)
- DepopProp [0.0, 0.01, 0.5, 0.75, 0.875, 0.94, 0.99]

Variation of time delay to carcass contacts (delay) 2-weeks contact delay with carcasses was suggested {Probst 2017} but recently was not confirmed in follow-up experiments (K. Depner & A.Viltrop, pers comm). The issue was addressed by an alternative scenario assuming contact since week of death.

R1: By width of the intensive hunting zone (x-axis), hunting effort in intensive hunting zone and alternative carcass transmission probability



R2: By hunting effort in intensive hunting zone (x-axis), width of the hunting zone and alternative carcass transmission probability

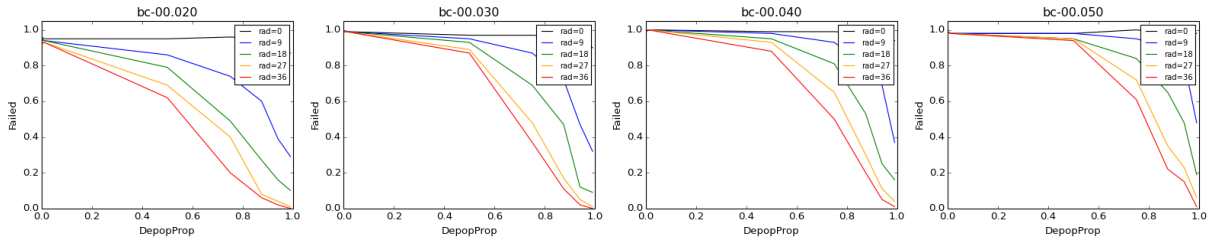


Figure 10. Simulation outcome regarding impact of hunting effort in differently sized intensive hunting zones – carcass contacts without delay after death. No carcass removal. No fence.
Note: Control success is lower assuming if 2-weeks delay in contact of live animals with carcasses was switched assuming immediate contact chance by week of death. Performance reduced by around one order of magnitude of the intensive hunting zone width compared to the standard scenario (Exp: BetaCarcass x DepopProp).

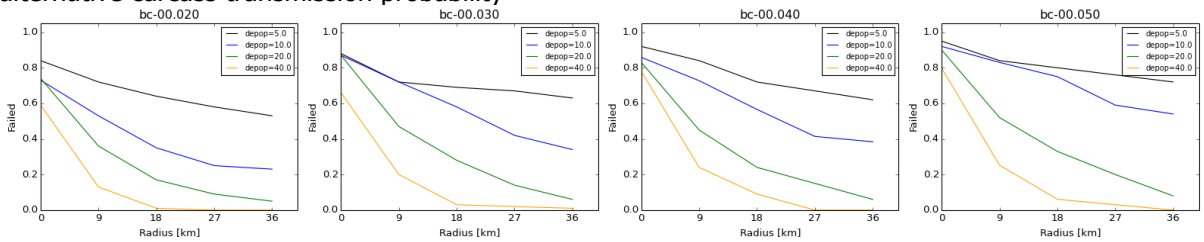
Exp: BetaCarcass x ***DepopNumber***

- BetaCarcass [0.02, 0.03, 0.04, 0.05]
- DepopNumber[5, 10, 20, 40]

Variation of alternative hunting effort advice (DepopNum/depop) in the intensive hunting zone from 0 to 40 animals per social group per year. The hunting advice is more practical than the standard DepopProp. Hunting clubs/hunters must target efforts at individual wild boar groups in their area. The

effort is distributed over all campaigns scheduled each year. The parameter value refers to the total effort over the year.

R1: By width of the intensive hunting zone (x-axis), hunting effort in intensive hunting zone and alternative carcass transmission probability



R2: By hunting effort in intensive hunting zone (x-axis), width of the intensive hunting zone and alternative carcass transmission probability

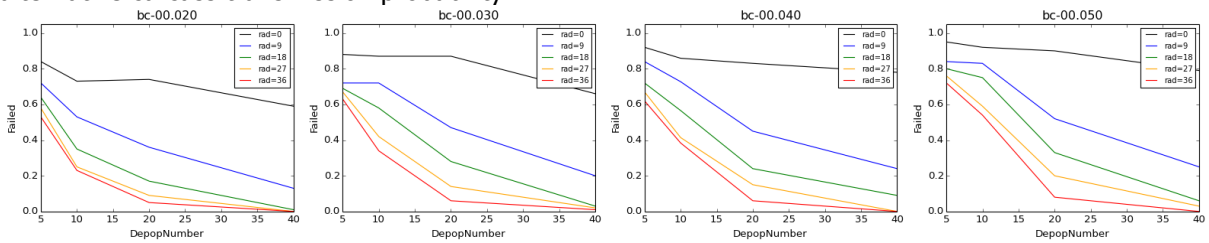


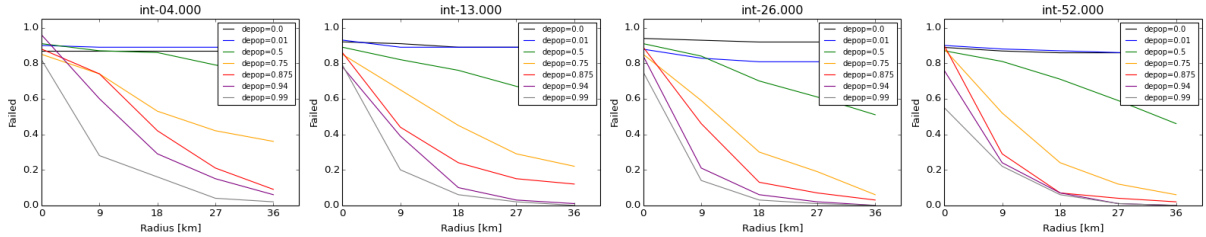
Figure 11. Simulation outcome regarding impact of hunting effort in differently sized intensive hunting zones – target definition per wild boar group. No carcass removal. No fence.
Note: The performance of the strategy based on the hunting of a number per wild boar group is more effective to combat break-outs from the core+buffer due to the patchy depopulation, i.e. smaller groups are nearly eliminated. E.g. comparing the two outcomes at carcass transmission of 0.03 (second column), correspondence was found at around depop%=75% vs. depopN=20 or depo%=99 vs. depopN=40 specification of hunting effort over the year using biannual campaigns.

Exp: ***CampaignInterval*** *x DepopProp*

- CampaignInterval [4, 13, 26, 52]
- DepopProp [0.0, 0.01, 0.5, 0.75, 0.875, 0.94, 0.99]

Varying temporal protocols of hunting effort in the intensive hunting zone (frequency of repetitions over the year) of four-week campaigns. Together these campaigns achieve equal overall annual hunting advice (from concentrated on one campaign, int=52, to scattered over 13 campaigns, int=4). The latter represents permanent efforts per week which therefore will be weak in the single campaign.

R1: By width of the intensive hunting zone (x-axis), hunting effort in intensive hunting zone and interval between 4-week campaigns (52/int = number of campaigns per year i.e. from left to right 13,4,2,1)



R2: By hunting effort in intensive hunting zone (x-axis), interval between 4-week campaigns and width of the intensive hunting zone

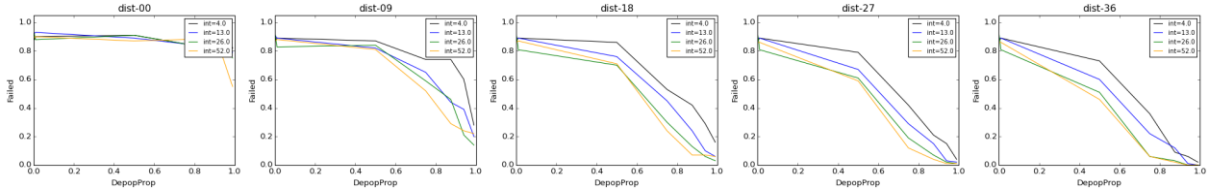


Figure 12. Simulation output regarding effect of alternative scheduling of hunting campaigns in order to achieve equal overall annual hunting effort (without carcass removal). The parameter interval (int; diagram title) refers to the interval in weeks between two campaigns i.e. 52 means 1 four-week campaign per year; 4 means 13 four-week campaigns per year (i.e. hunting always). Effort per campaign is adjusted to achieve annually the total hunting outcome as to DepopProp (x-axis; 0%; 50%; 88%). NB: Reasonably, the core+buffer induced set-off, (x-value zero for no-hunting) is equal over all simulations and about 10%.

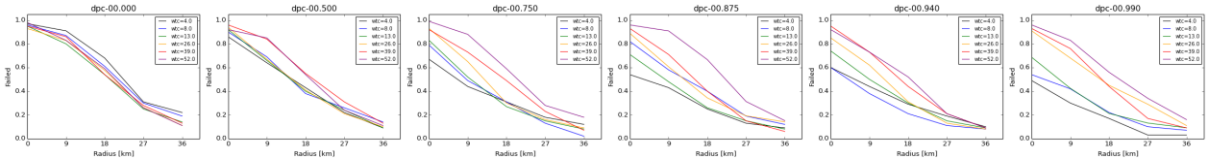
3.3.3.3. Changing the efforts attributed to the core+buffer zone – no carcass removal, no fence

Exp: ***DepopPropCore*** *x* ***WaitingTimeCore***

- DepopProp = 0.75
  - DepopPropCore [0.0, 0.5, 0.75, 0.875, 0.94, 0.99], annually (single campaign)
  - WaitingTimeCore [4, 8, 13, 26, 39, 52]

Varying waiting time till culling in the core+buffer zone (wtc)

R1: By width of the intensive hunting zone (x-axis), waiting time till culling of core+buffer (colours) and culling effort in core+buffer zone (diagrams)



R2: By width of the intensive hunting zone (x-axis), culling effort in core+buffer zone (colours) and waiting time to culling effort in core+buffer (diagrams)

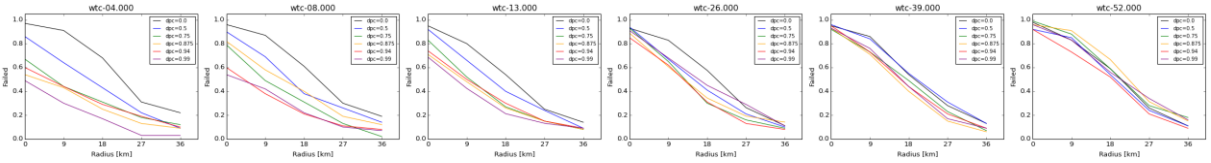


Figure13. Simulation outcome regarding impact of measures applied to the core+buffer zone on the overall success. No carcass removal. No fence.

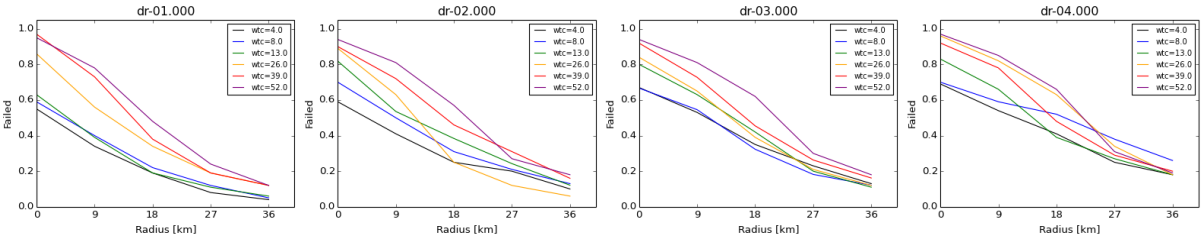
Note: The yellow line (i.e. waiting time wtc=26) of the scenario assuming 87.5% culling effort in the core+buffer (dpc=00.875) is close to the outcome of the reference parameterisation underlying other simulations (see Exp: BetaCarcass x DepopProp).

Exp: ***DisturbanceRadius*** *x WaitingTimeCore*

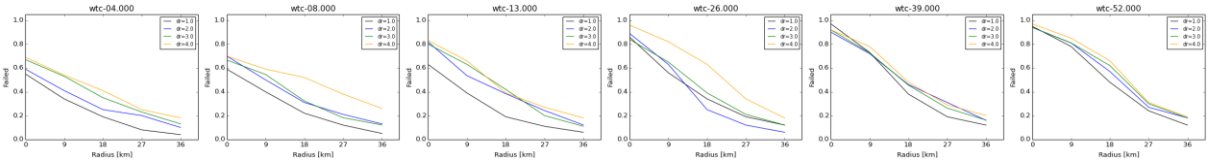
- DepopProp = 0.75
  - DisturbanceRadius [1, 2, 3, 4] cells
  - WaitingTimeCore [4, 8, 13, 26, 39, 52]

Varying spatial depth of the disturbance impact (dr). The disturbance effect, i.e. the outwards chasing of animals due to culling activities, includes movements of infected animals. Thus these infected animals were assumed to stochastically be able to move up to dr group home-ranges away from home (dr = 1, 2, 3 and maximum 4 group home-ranges) and for up to 4 weeks after the disturbance ended.

R1: By width of the intensive hunting zone (x-axis), waiting time to culling effort in core+buffer (colours) and radius of disturbance effect (diagrams)



R2: By width of the intensive hunting zone (x-axis), radius of disturbance effect (colours) and waiting time to culling effort in core+buffer (diagrams)



R3: By **<u>waiting time to culling effort in core+buffer (x-axis)</u>**, width of the intensive hunting zone (colours) and radius of disturbance effect (diagrams)

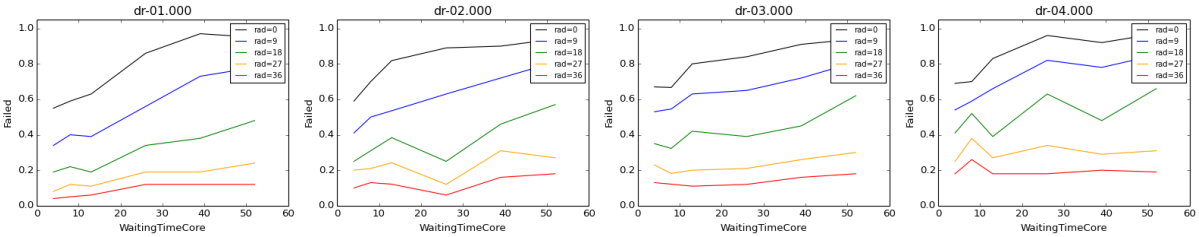


Figure 14. Simulation outcome regarding impact of disturbance modelling and waiting time till culling in core+buffer zone. No carcass removal. No fence.

Note: the proportion of runs that make it out of the buffer zone (zero value on x-axis) increases with increasing dimension of the disturbance effect (R2). The negative impact of an increasing waiting time till culling of core+buffer on the expected outcome (R1) remains valid no matter what disturbance we apply (R2).

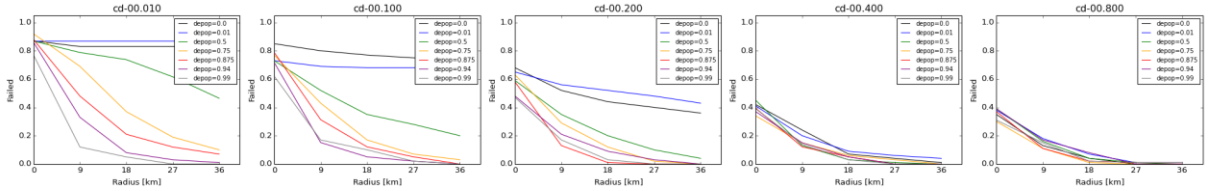
3.3.3.4. Variation of carcass removal efforts – added to intensive hunting, no fence

Exp: ***CarcassDetection*** *x DepopProp*

- CarcassDetection [0.01, 0.1, 0.2, 0.4, 0.8]
- DepopProp [0.0, 0.01, 0.5, 0.75, 0.875, 0.94, 0.99]

Varying carcass removal efforts (cd) in all zones. 10% (standard till zoning established), 20, 40, 80%.

R1: By width of the intensive hunting zone (x-axis), hunting effort in the intensive hunting zone (colours) and carcass removal rate in percent (diagrams)



R2: By width of the intensive hunting zone (x-axis), carcass removal rate in percent (colours) and hunting effort in the intensive hunting zone (diagrams)

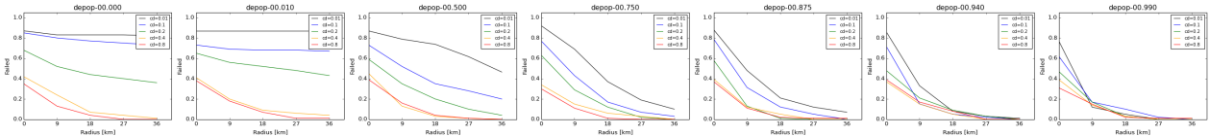


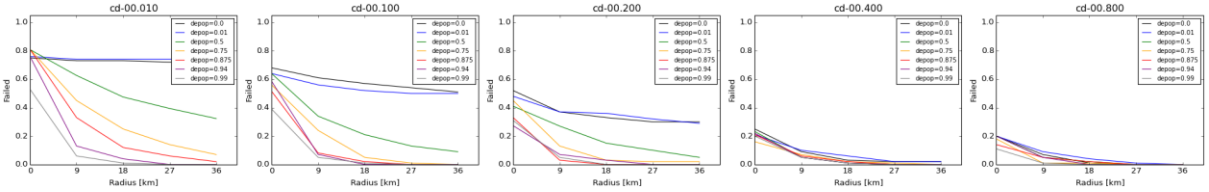
Figure 15. Simulation outcome regarding impact of hunting effort in differently sized intensive hunting zones supported by carcass removal in all zones. No fence.

Exp: CarcassDetection x DepopProp - perfect info

- Zoning based on **perfect** information
- CampaignInterval scenarios: 26
  - CarcassDetection [0.01, 0.1, 0.2, 0.4, 0.8]
  - DepopProp [0.0, 0.01, 0.5, 0.75, 0.875, 0.94, 0.99]

Varying carcass detection for zoning as perfect additionally to carcass removal efforts (cd) assuming 10% (standard till zoning established), then 10, 20, 40, 80%.

R1: By width of the intensive hunting zone (x-axis), hunting effort in the intensive hunting zone (colours) and carcass removal rate in percent (diagrams)



R2: By width of the intensive hunting zone (x-axis), carcass removal rate in percent (colours) and hunting effort in the intensive hunting zone (diagrams)

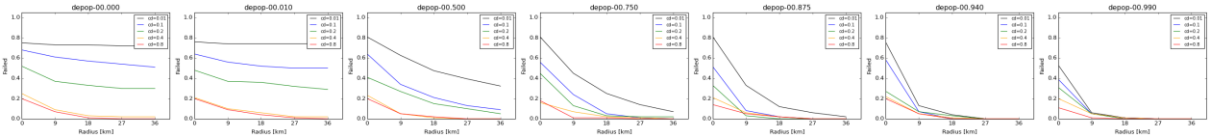


Figure 16. Simulation outcome regarding impact of hunting effort in differently sized intensive hunting zones supported by carcass removal in all zones – perfect knowledge on carcass distribution for zoning. No fence.

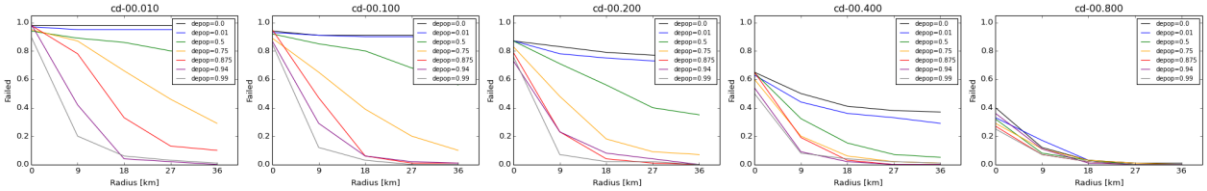
Exp: CarcassDetection x DepopProp - perfect info - ***no delay***

- Zoning based on **perfect** information
- InfectiousCarcassDelay = 0

- CarcassDetection [0.01, 0.1, 0.2, 0.4, 0.8]
- DepopProp [0.0, 0.01, 0.5, 0.75, 0.875, 0.94, 0.99]

Varying delay of carcass contact additional to carcass removal efforts (cd) assuming perfect carcass detection for zoning. 10% (standard till zoning established), 10, 20, 40, 80%.

R1: By width of the intensive hunting zone (x-axis), hunting effort in the intensive hunting zone (colours) and carcass removal rate in percent (diagrams)



R2: By width of the intensive hunting zone (x-axis), carcass removal rate in percent (colours) and hunting effort in the intensive hunting zone (diagrams)

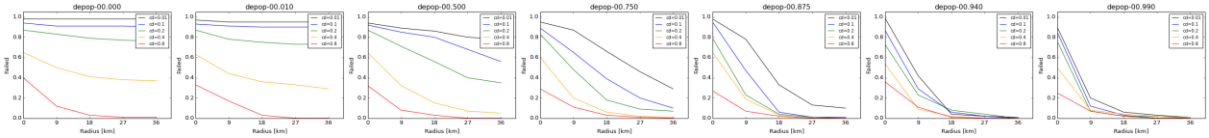


Figure 17. Simulation outcome regarding impact of hunting effort in differently sized intensive hunting zones supported by carcass removal in all zones – perfect knowledge on carcass distribution for zoning and no contact delay with carcasses. No fence.
Note: No delay costs about 20% success rate in core+buffer and even more in intensive hunting zone.

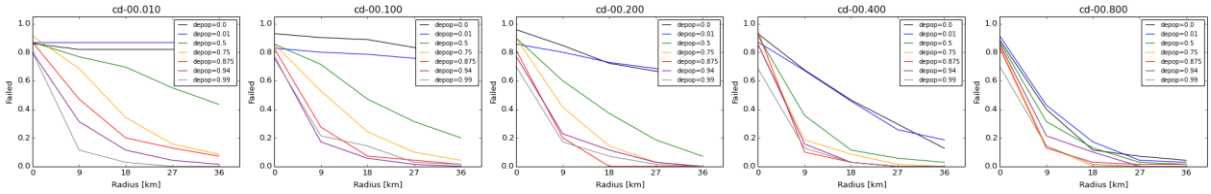
3.3.3.5. No carcass detection/removal in core and buffer – no fence

Exp: CarcassDetection x DepopProp - no carcass detection core+buffer

- CarcassDetection core + buffer = 0.01
  - CarcassDetection [0.0, 0.1, 0.2, 0.4, 0.8]
  - DepopProp [0.0, 0.01, 0.5, 0.75, 0.875, 0.94, 0.99]

Varying carcass removal in core-buffer (off).

R1: By width of the intensive hunting zone (x-axis), hunting effort in the intensive hunting zone (colours) and carcass removal rate in percent (diagrams)



R2: By width of the intensive hunting zone (x-axis), carcass removal rate in percent (colours) and hunting effort in the intensive hunting zone (diagrams)

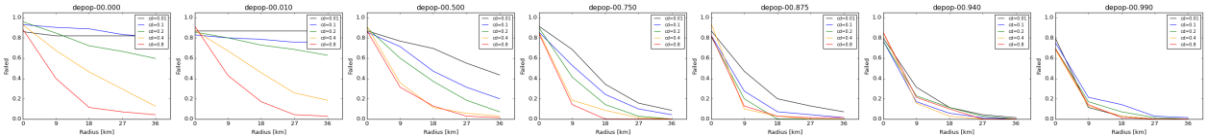


Figure 18. Simulation outcome regarding impact of hunting effort in differently sized intensive hunting zones supported by carcass removal in the intensive hunting zone – no carcass removal in core+buffer zone . No fence.
Note: Carcass removal is particularly relevant in core+buffer

3.3.3.6. Variation of the buffer width – no fence

Exp: CarcassDetection x BufferZoneWidth

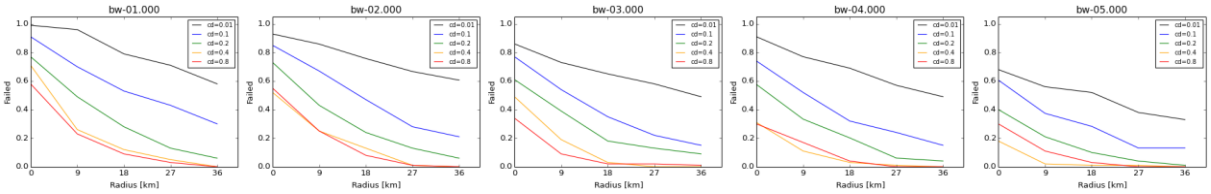
Exp: CarcassDetection x BufferZoneWidth - perfect info

- DepopProp scenarios: 0.5, 0.75
- PerfectInformation for zoning: 10% chance or perfect
  - CarcassDetection [0.0, 0.1, 0.2, 0.4, 0.8]
  - BufferZoneWidth [1, 2, 3, 4, 5] (cells)

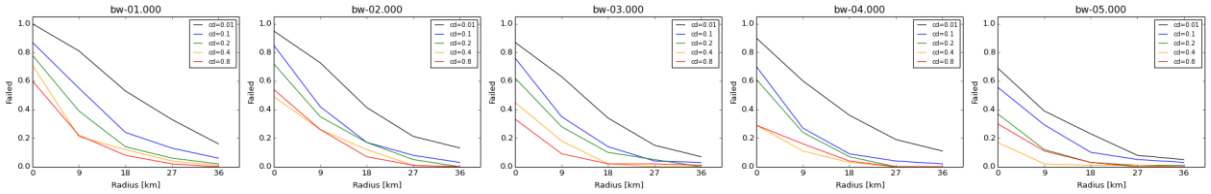
Varying width of the buffer zone.

By width of the intensive hunting zone (x-axis), carcass removal rate in percent (colours) and width of buffer zone in wild boar group home-range diameters (diagrams)

R1: Hunting effort 50% over two campaigns per year and culling of core+buffer zone 90% after 26w; zoning based on 10%-random detection of carcasses:



R2: Hunting effort 75% over two campaigns per year and culling of core+buffer zone 90% after 26w; zoning based on 10%-random detection of carcasses:



R3: Hunting effort 75% over two campaigns per year and culling of core+buffer zone 90% after 26w; zoning based on perfect knowledge about carcasses:

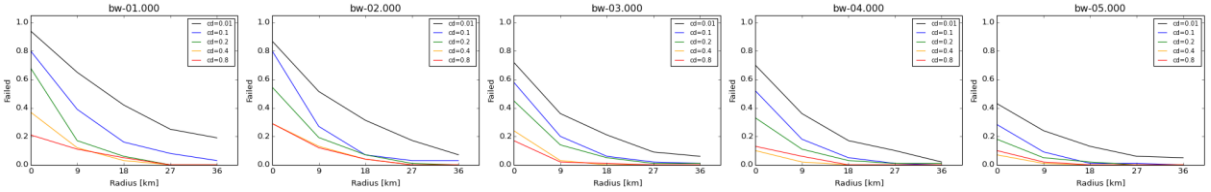


Figure 19. Simulation outcome regarding impact of hunting effort in differently sized intensive hunting zones supported by carcass removal in all zones – applying different buffer size either with or without perfect knowledge on carcass distribution for zoning. No fence.

Twofold presentation of hunting volume (blue box plots – left axis), success (1-failure, right axis) in the core+buffer (red line) or at least in the intensive hunting zone (black line), width of buffer zone in wild boar group home-range diameters (x-axis) and carcass removal efforts (diagrams)

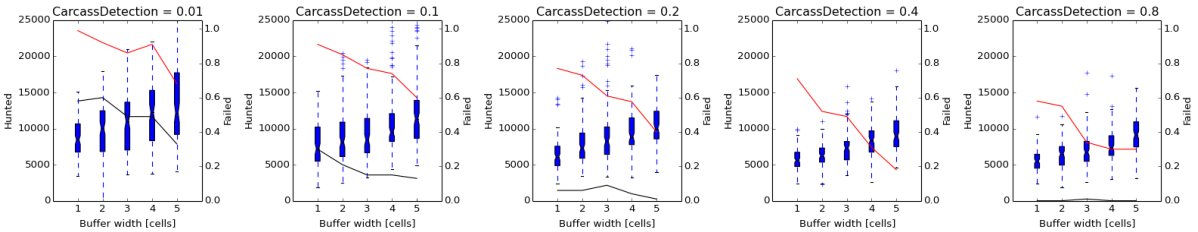


Figure 20. Simulation outcome regarding the volume of hunted animals versus the success rate dependent on the buffer zone width. No fence.

Note: Overall hunting effort (left axis) per buffer width (x-axis) implies enlarging buffer leads to more animals to shoot both in buffer and intensive hunting zone. Probability of breakout from core+buffer (red line, right axis) per buffer width scenario implies enlarging the buffer improves success rate (lowered failure rate). Final success (1-probability of failure; black line, right axis) per buffer width scenario, i.e. break out of even the maximum intensive hunting zone simulated (x-axis) implies that already the carcass removal intensity can solve the remaining problem as the black line disappears (null level) with 40% removed carcasses.

3.3.3.7. Variation of three control measures – intensive hunting, carcass removal, fences

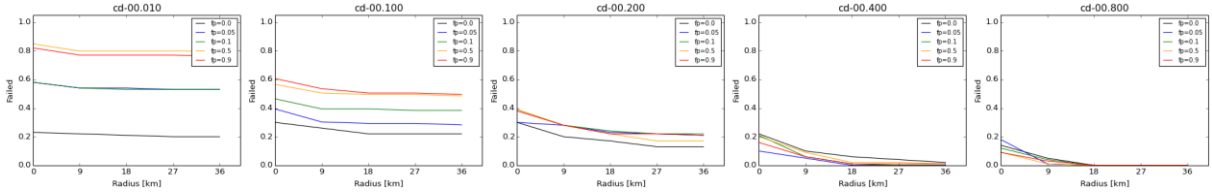
Exp: FencePermeability x CarcassDetection x DepopProp – perfect info

- Zoning based on **perfect** information
- DepopProp: 0, 0.5, 0.75
- CampaignInterval = 52w
  - FencePermeability [0.9, 0.5, 0.1, 0.05, 0.0]
  - CarcassDetection [0.0, 0.1, 0.2, 0.4, 0.8]

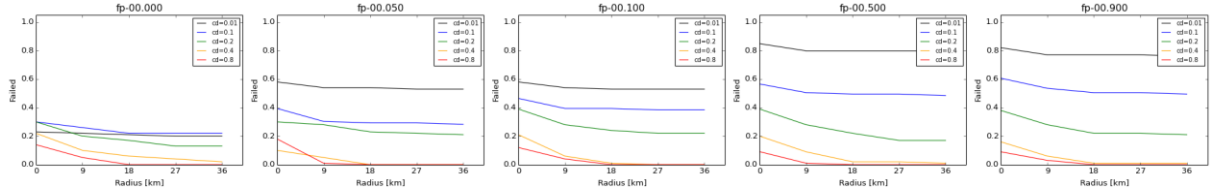
Varying the permeability of the fence line that surrounded the core area (fp). Simulation address the effect of fences vs. carcass removal vs. different intensive hunting effort (applied as one annual campaign immediate after zoning).

- **No hunting efforts in the intensive hunting zone and culling of core+buffer zone 90% after 26w:**

R1: By width of the intensive hunting zone (x-axis), permeability of the fence (colours) and carcass removal rate in percent (diagrams)

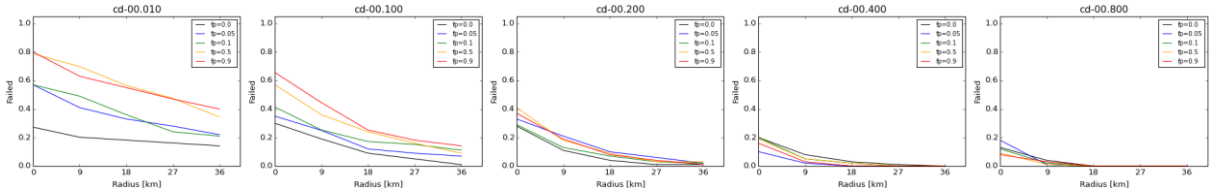


R2: By width of the intensive hunting zone (x-axis), carcass removal rate in percent (colours) and permeability of the fence (diagrams)

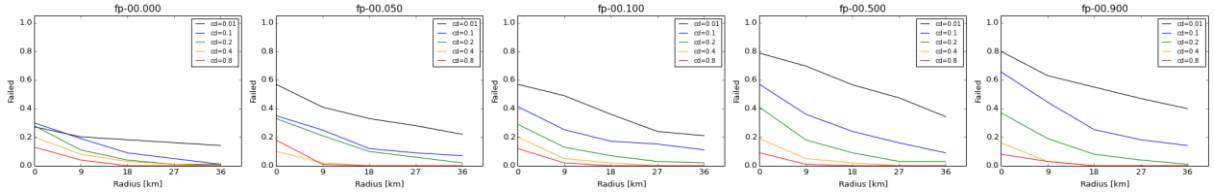


- **50% hunting effort in one immediate campaign applied to the intensive hunting zone and culling of core+buffer zone 90% after 26w:**

R3: By width of the intensive hunting zone (x-axis), permeability of the fence (colours) and carcass removal rate in percent (diagrams)

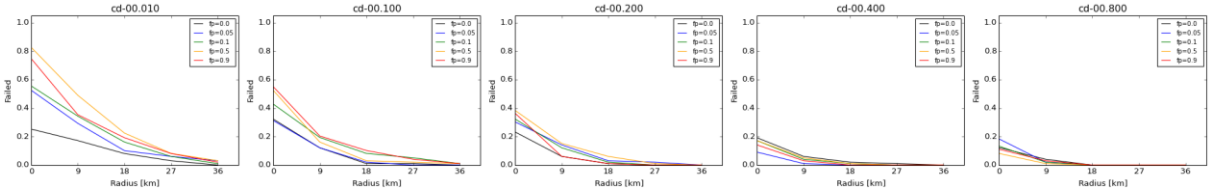


R4: By width of the intensive hunting zone (x-axis), carcass removal rate in percent (colours) and permeability of the fence (diagrams)



- **75% hunting effort in one immediate campaign applied to the intensive hunting zone and culling of core+buffer zone 90% after 26w:**

R5: By width of the intensive hunting zone (x-axis), permeability of the fence (colours) and carcass removal rate in percent (diagrams)



R6: By width of the intensive hunting zone (x-axis), carcass removal rate in percent (colours) and permeability of the fence (diagrams)

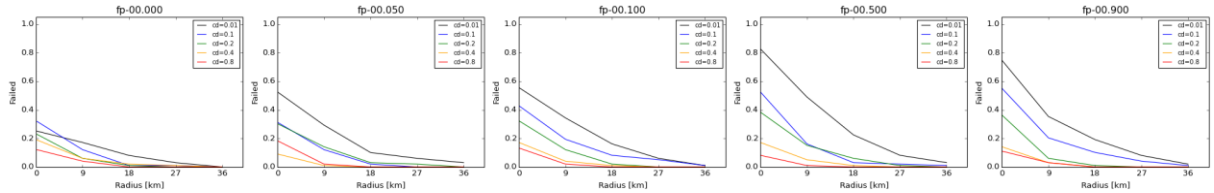
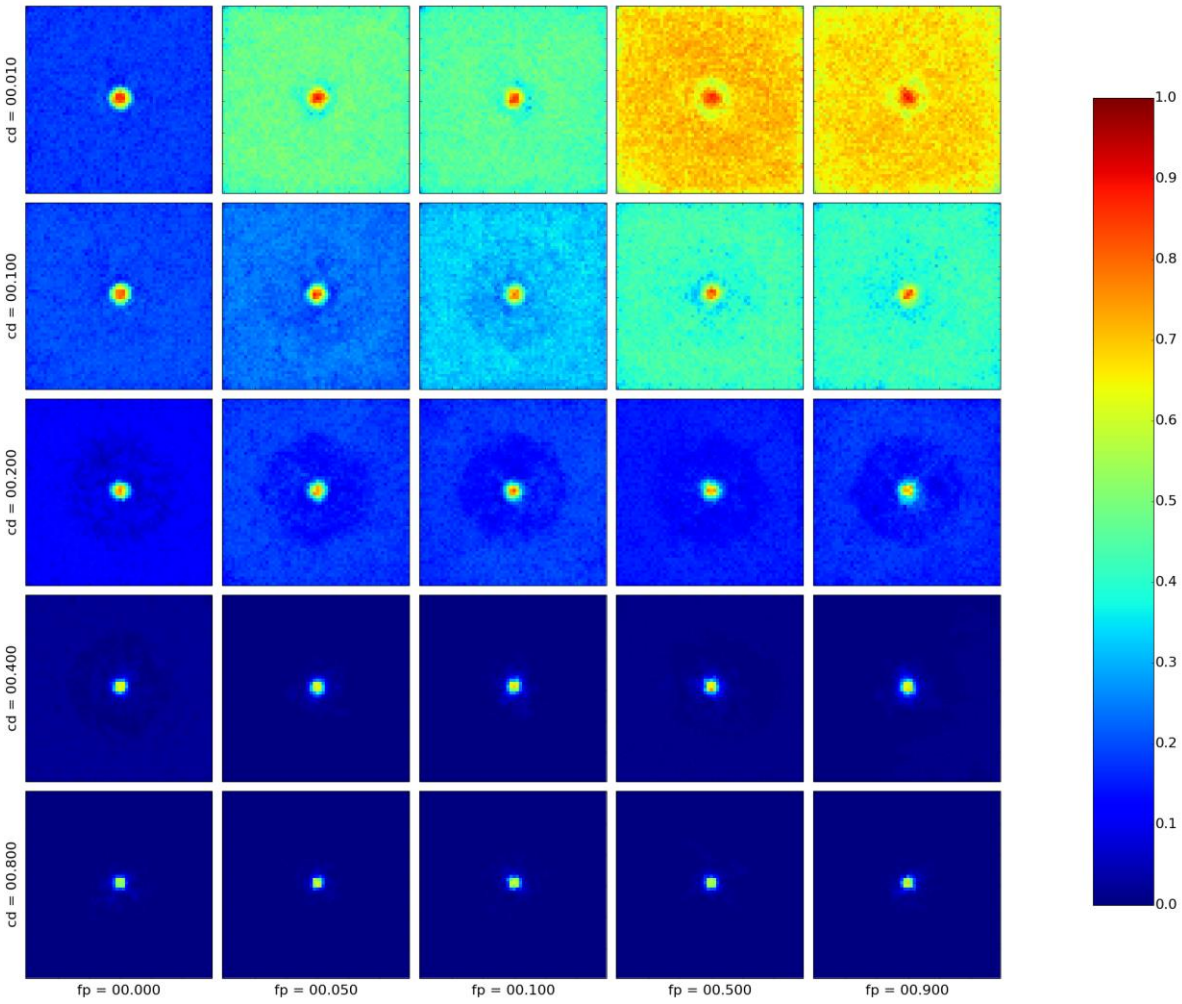


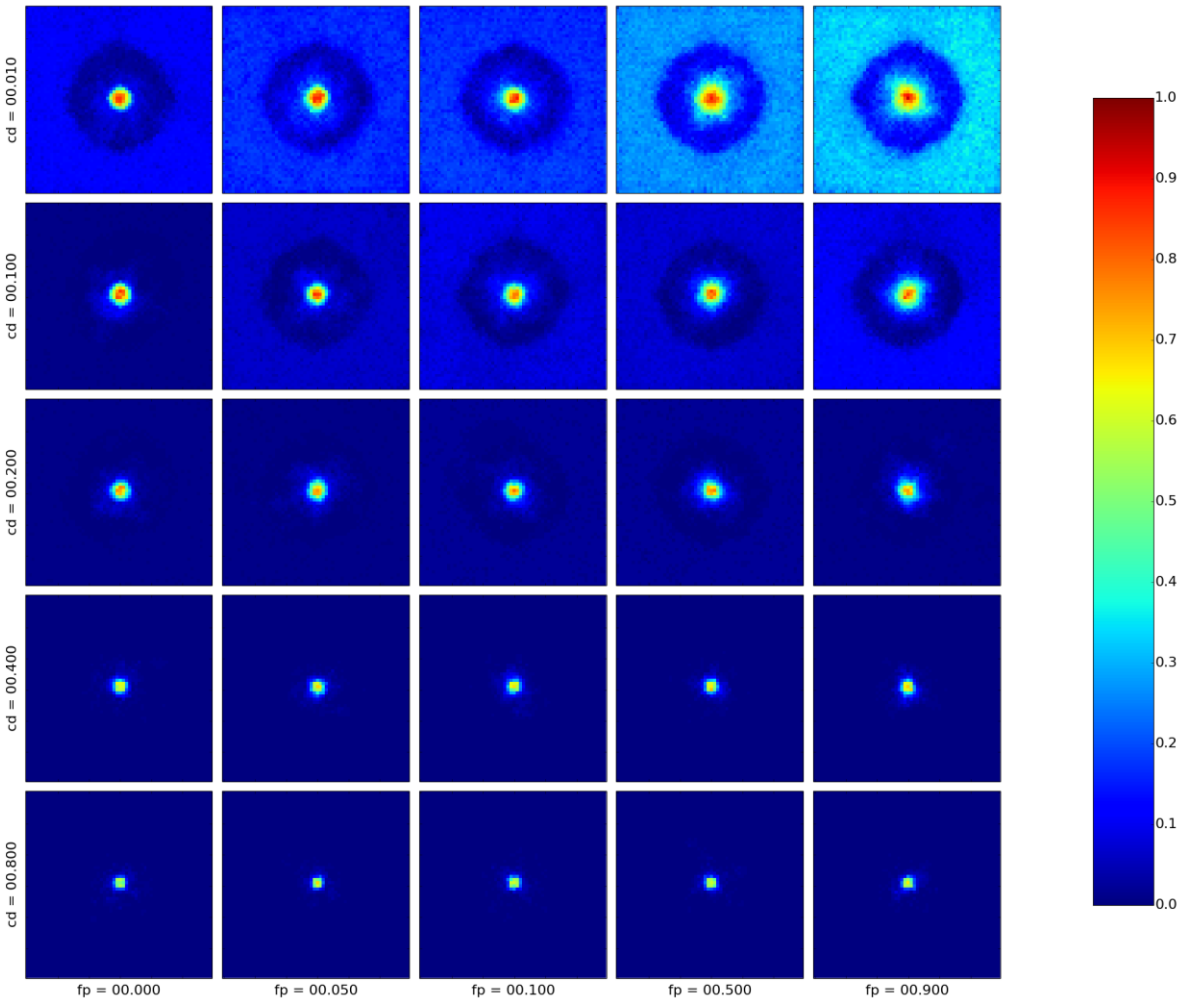
Figure 21. Simulation outcome regarding impact of hunting effort in differently sized intensive hunting zones supported by carcass removal in all zones and fencing of core zone of different permeability.

The following cross-tabulated charts provide an overview about the spatial impact of alternative assumptions regarding the carcass removal efforts (left, vertically increasing from top to bottom), fence permeability (bottom, horizontally increasing from left to right) and three different hunting efforts immediately and annually applied to the intensive hunting zone over 4 weeks. The diagrams show the heat map of the chance of certain wild boar groups being affected by ASF infection in the 100 runs per scenario (0.0 blue negligible; 1.0 red nearly sure).

R1: No hunting efforts in the intensive hunting zone and culling of core+buffer zone 90% after 26w:



R2: 50% hunting effort in one immediate campaign applied to the intensive hunting zone and culling of core+buffer zone 90% after 26w:



R3: 75% hunting effort in one immediate campaign applied to the intensive hunting zone and culling of core+buffer zone 90% after 26w:

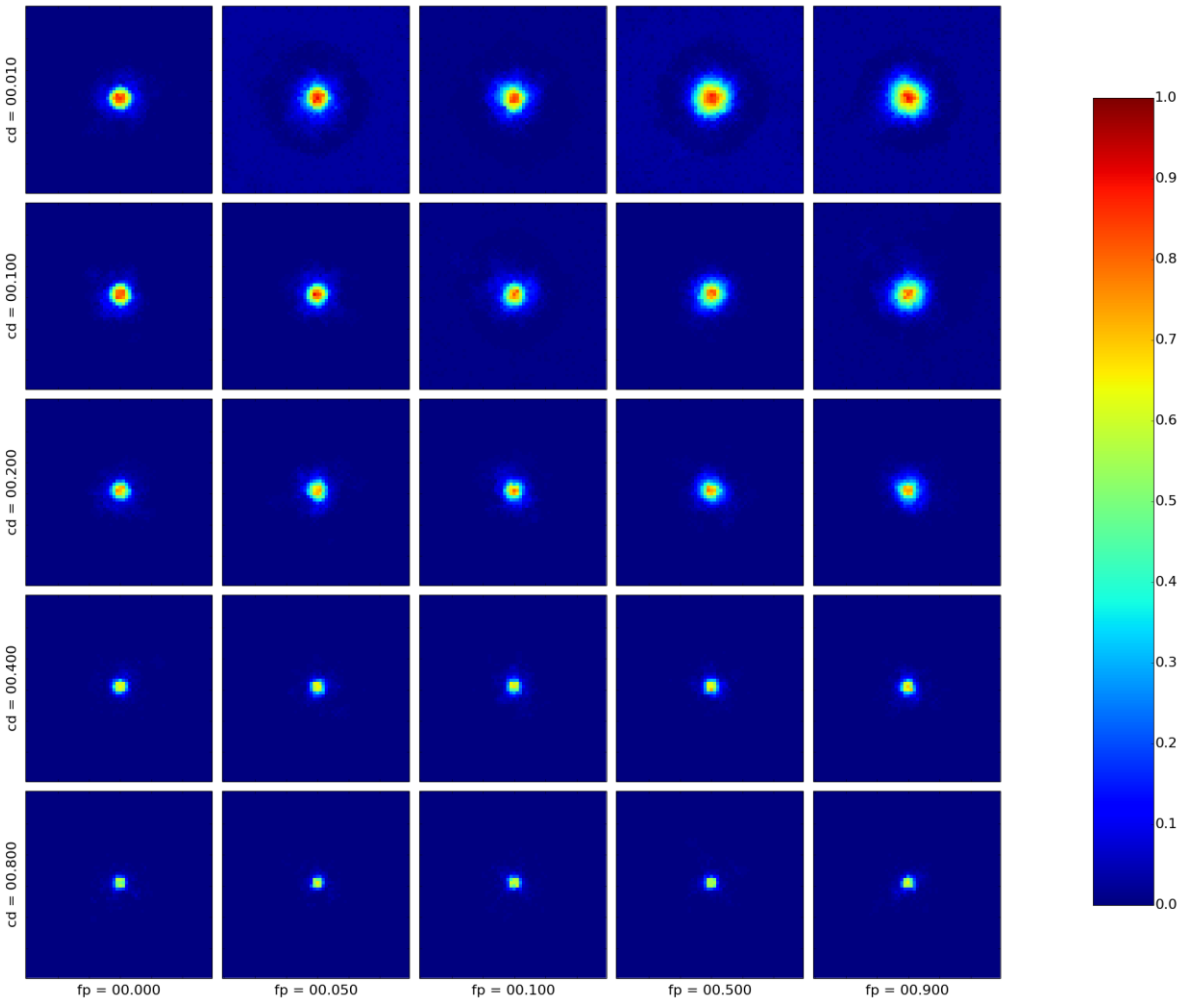


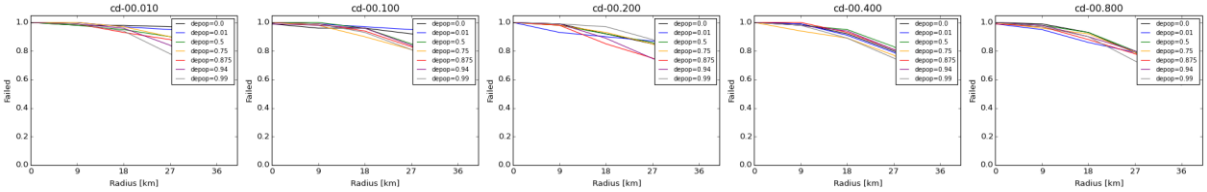
Figure 22. Spatial simulation outcome (heat map) regarding impact of hunting effort in intensive hunting zones supported by carcass removal in all zones and fencing of core zone of different permeability.

3.3.3.8. Large-scale application of focal strategy - variation of intensive hunting and carcass removal, no fences

Exp: CarcassDetection x DepopProp ***linear***

- Release of 300 individuals along the right/eastern edge of the landscape
- Minimum waiting time till first random notification was forced as 52 weeks resulting in burn-in simulation of large scale infection at the right edge of the simulation landscape
- InfectiousCarcassDelay = 0
    - CarcassDetection [0.01, 0.1, 0.2, 0.4, 0.8]
    - DepopProp [0.0, 0.01, 0.5, 0.75, 0.875, 0.94, 0.99]

R1: By width of the intensive hunting zone (x-axis), hunting effort in the intensive hunting zone (colours) and carcass removal rate in percent (diagrams)



R2: By width of the intensive hunting zone (x-axis), carcass removal rate in percent (colours) and hunting effort in the intensive hunting zone (diagrams)

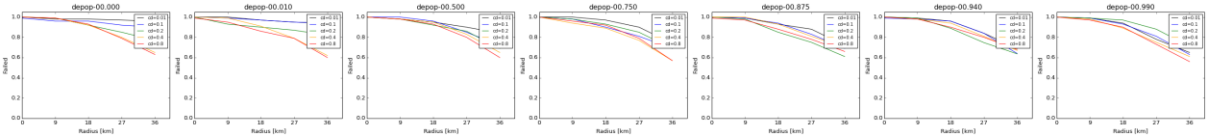


Figure 23. Simulation outcome regarding impact of applying the focal strategy design to a landscape level epidemic wave, i.e. hunting effort in differently sized intensive hunting zones supported by carcass removal in all zones and fencing of core zone with different permeability.

By probability per wild boar group home-range (pixel, cell) to be involved in ASF spread over 100 runs applying the cross-tabulated parameter values of hunting effort (bottom, increasing from left to right) and carcass removal rate (left, increasing from top to bottom). Probability scale 0.0 blue = negligible; 1.0 red = nearly sure).

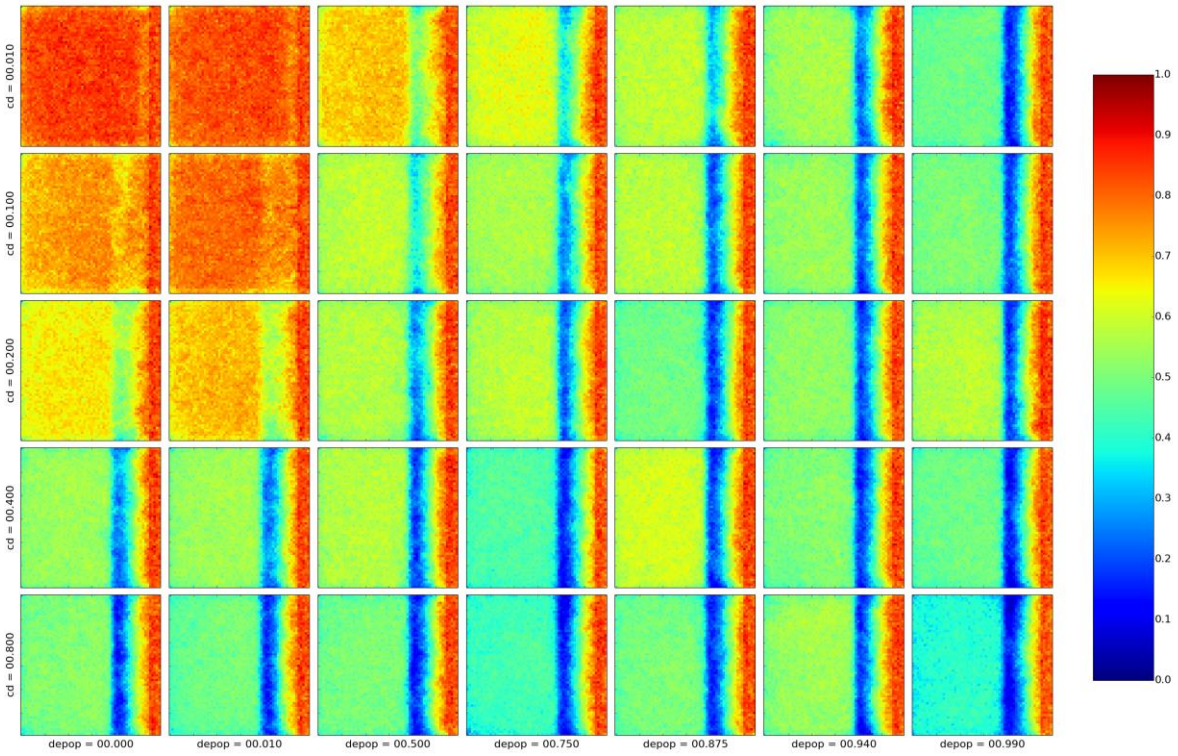


Figure 24. Spatial simulation outcome (heat map) regarding impact of the focal strategy design applied to landscape scale epidemic waves, i.e. hunting effort in intensive hunting zones supported by carcass removal in all zones and fencing of core zone of different permeability.
Note: Spatial simulation output shown as heat map of proportion of 100 repetitions of alternative scenarios addressing the large-scale control of ASF to stop wild-boar mediated spread into areas at higher risk of ASF introduction by wild-boar mediated spread. Scenarios differed by applied control measures. Column-wise: different hunting efforts in the intensive hunting zone (left 0%; 1%; 50%; middle 75%; 87%; 94%, right 99%) vs. row-wise different carcass detection rates (top 1%; 10%; middle 20%; 40%; bottom 80%). The more reddish a pixel is coloured the higher was the proportion of the 100 repetitions of the scenario in which the respective wild boar group became affected.

The focal approach does not involve adequate dimensions of the control elements to provide sufficient countermeasures to stop the ASF spread in an affected landscape. Even with perfect measures (99% hunting effort and 80% carcass removal) the intensive hunting zone (blue coloured reflecting low probability to have infected groups; reasonable as getting nearly emptied the better the hunting is performed) is not sufficient to withstand the continued spread from the ASF affected buffer (area right neighbouring the intensive hunting zone. The high failure rate is shown by the area left to the hunting zone which was affected in about 40% of all runs.

4. Uncertainty identification and discussion

- In general, it should be noted that there remains considerable epistemological uncertainty about many aspects of ASF epidemiology in wild boar, including the carcass contact rate, the contact rate between groups, and the role of insects. For this reason, the modelling results are presented as a comparison between scenarios rather than as prediction of absolute values.
- The potential age dependent probability of contacts with carcasses. According to observational studies with wild boar carcasses placed outdoor, piglets are suggested to form the dominant cohort involved in camera trapped contacts while adults more likely avoid such contacts (K. Depner & A. Viltrop, pers.comm.). The model was able to address the scenario with critical impact on the persistence time. The hypothesis has not yet been integrated in the model-based analysis presented in this report.

– The possible seasonal contacts between males and females during rut period. The hypothesis has not yet been integrated in the model-based analysis presented in this report.

– The possible involvement of insects as mechanical vectors in the transmission. The transmission ecology based on insects is not yet explicitly represented by the model. The critical aspect is the suggested seasonality. The transmission mode itself has several limitations that would not change the modelling outcome. First, insects would not host the virus thus their capability to act as "temporary reservoir" appears to be very limited. Second, the spatial scale of their activity might be rather limited compared to wild boar movements, e.g. group home-range dimension of 4-9km² might represent the better link between animals compared to vectors. It is uncertain if the model could match ADNS-like spread if carcasses are excluded and only insects would be modelled.

– Disturbance was of limited importance even for the length of the waiting time till culling of core+buffer zone which is unexpected given usual implications associated with disturbance by intensive culling activities. There are three plausible arguments why disturbance should decrease success only in few simulations compared to simulations without disturbance (i.e. culling of the core+buffer zone):

  - There are intentionally only few infected animals expected in core-buffer. Any of these few have to realise a long-distance escape both while being not too ill and prior the snippers finish the culling campaign (e.g. assumed to finish in 4 weeks' time in the simulations);
  - There must not yet have been infected animals outside the buffer before the disturbance chases an infected animal into the intensive hunting zone. Otherwise the "extra out" due to disturbance does not alter the overall assessment of the simulation run i.e. ASF beyond the core+buffer in both simulation runs;
  - Most important, and likely often forgotten, the problem considered is the chasing of animals across a certain management line (i.e. between inside and outside the core or buffer). Usually discussed with habitat-wide disturbance in wildlife are increased movement activities all-over the habitat maintaining transmission chain by increasing the contact rate. Here the issue is local and related to the border of the disturbed area only (i.e. inside the core+buffer infection can be perpetuated). Thus, when disturbing on one side of the management line only those locations close to the management line in terms of wild boar movements will actually contribute to a "disturbance effect" i.e. only those animals in fact have the chance to be chased over the management line, and these are few in numbers.

  It appears plausible that the negative impact of culling disturbance on the achieved control success might be less important compared to other strategy components. At least the intuitively suggested order of magnitude of the disturbance impact might be substantially overestimated.

– The disturbance effect, i.e. the modelled outwards chasing of animals due to culling activities, includes movements of infected animals. Thus these infected animals were assumed to be stochastically be able to move up to k group home-ranges away from home (sc: k = 1, 2, 3 and maximum 4 group home-ranges) and for up to 4 more weeks after the disturbance ended. This assumption does not influence the particular results regarding the waiting time before culling the buffer+core i.e. suggested to reduce disturbance related animal movements in the core+buffer. It might be possible that the uncertainty is rather related to a misconception traditionally related with disturbance of wildlife and the increased circulation of infectious diseases. Here the problem only is applicable for the question, whether infectious animals are chased over the demarcation line encircling the core+buffer. This chance of such events to happen is very unlikely as only limited number of animals could ever be chased across the demarcation line and those moribund will not move even if disturbed (S. Blome pers.comm) – so the critical event requires incubating animal close to the border of the core+buffer.

– Control success is highly sensitive to the hypothesis on 2-weeks delay in contact of live animals with carcasses (Probst et al. 2017). The issue was addressed by alternative scenarios assuming immediate contact chance by week of death. The predicted performance was different, e.g. by

around one order of magnitude of the intensive hunting zone in the standard scenarios. The issue affects all simulations both without and with carcass removal because less time is left to prevent secondary infections per carcass if immediate contact is possible. Particularly, carcass removal is less effective. The threshold by which the hunting in the intensive hunting zone is of marginal relevance moves from 20%/40% up to 40%/80% if no 2-weeks delay in contacts is modelled. The problem is rather difficult to solve using modelling. There is anecdotal reporting from new experiments that the delay in contact could not be reproduced (K. Depner pers. comm.)

- Carcass removal is an important measure in the model. The role assigned to carcass-mediated transmission is based on expert interpretation of knowledge regarding ASF perpetuation in wild boar (EFSA 2015, 2017, 2018a). Beyond its possibility due to contacts (Probst et al. 2017) and plausibility of infectiousness of blood-containing cadaver material this mechanism was neither biologically nor experimentally demonstrated. However, all current control concepts hinge on the truth of carcass-mediated transmission, so the model has to do the same. Indicative simulations have shown, that only live-to-live transmission (likewise CSF in wild boar), would make it impossible to match simulated ASF spread velocity with spread dynamics found in ADNS data (3.1). Hence, if not carcasses than certain other localised (limited movement range) environmental reservoir (staying for a while beyond an infectious period and allow minimal transmission either by seldom occurrence or limited effectiveness of contacts; Lange & Thulke 2017) might be required to explain ADNS-based spread dynamics. Simple live-to-live animal transmission is insufficient due to the very limited period of infectiousness between infection and death.
- The very limited number of infected carcasses (e.g. less than 40 within the early months post detection in BE) highlight the sensitivity of the outcome of the strategy to the carcass removal intensity, see 3.3.3.4. Therefore it seems reasonable that sufficiently effective carcass removal can override the impact of intensive hunting in the intensive hunting zone creating a rather safe strategy concept. Again, the role of carcasses is crucial.
- In the model configuration bio-security leaks by hunters or handling personal are not included. Hence, both carcass removal and culling in the core+buffer zone is assumed very clean and safe in the model. If the assumption of best practise would be violated in the focal approach then due to the very small affected area (often only core and parts of the buffer) it might have exaggerated impact on the outcome. Few misbehaviours/mistakes could have a drastic impact on the performance of the control strategy, e.g. one infected carcass not properly removed may cause infection outside the buffer that would not have happened otherwise.
- A waiting time till culling of the core+buffer was previously suggested in conjunction with the focal strategy plan. The reasoning refers to the prevention of greater numbers of infected animals to be culled in the core+buffer zone if the peak of infections had passed already. The model outcome does not support the suggestion due to continued spatial expansion of ASF infection inside and towards outside of the core+buffer area. Likely, the argumentation relies on the presence of a demarcation or fence that prevents movement and contact of animals between both sides of the fence-line. Even with semi-permeable fences (e.g. 90% usual events suppressed) the model outcome did not alter because prolonged waiting to culling would eventually cumulate new cases that "test" the fence permeability. However, uncertainty exists regarding the exclusion of human misbehaviour by which, during culling or removal of infected animals in the core zone contamination can sporadically happen. These negative occasions would be suppressed by timely reduction of the number of (infected) animals in the core area i.e. before they become infected.
- As discussed in previous EFSA outputs, the conceptualisation of carcass removal and intensive hunting as ASF control measures only for the zones with possible ASF notifications (i.e. buffer and core) is misleading. Particular, the intensive hunting zone treated immediately after zoning is a kind of insurance against semi-perfect success in the containment and culling activities addressing core+buffer. Intensified hunting and "preparatory" carcass removal (i.e. hopefully all negative) is an important component of the control package unless perfect fencing and super-efficient carcass removal are guaranteed. Independent of how large the buffer zone was chosen, and because the core+buffer was subjected to hunting ban until final culling, ASF did spread into the buffer zone

and subsequently could break out from the buffer before final culling is scheduled. During the time passing before final culling of the core+buffer zones, intensified hunting around the buffer could establish a more protective zone for the worst case. In that sense it might be arguable to include the intensive hunting zone into the "controlled area" to allow best hunting and carcass removal efforts with or without actual notifications of positive carcasses.

– Logically, arbitrary extension of the buffer is a pseudo-option because final culling of the core and the buffer is a component of the strategy. The larger the buffer, the more impractical are applications of drastic culling measures due to the quadratic relationship between buffer width and the number of animals to kill during final culling.

– The usefulness of fences as single measure in controlling spread of ASF in wild boar is not fully understood. Moreover, the permeability of fences, i.e. the probability that movements across the fence line or animal contacts at the fence are prevented, is also not quantified in field experiments (but see Lavelle et al. 2011). Here, possible permeability values were assumed to allow exploration of different effects on the overall strategy outcome.

5. Conclusions

• There remains considerable uncertainty about many aspects of ASF epidemiology in wild boar, including the carcass contact rate, the contact rate between groups, and the role of insects. For this reason, the modelling results are presented as a comparison between scenarios rather than providing absolute values for management.

• In the absence of carcass removal in the core area and assuming that carcass contact occurs immediately after death (no-delay scenarios), the probability of success can still exceed 80% if the intensity of hunting in the intensive hunting area is much higher than during times of sustainable wild boar management. This might be possible in practice because the intensive hunting area is limited (3 to 12 wild boar home ranges).

• Given acknowledged limits of intensive regular hunting, the sole application of intensive hunting zones around the buffer area may not be a solution-oriented option for focal emergency control measures.

• Lesser hunting efforts are required in the intensive hunting area when carcass removal is being implemented in the core area. With a carcass removal rate of 20%, the probability of success can exceed 80% with an intensive hunting area of limited size (3 to 12 wild boar home range diameters width) and less intensive hunting efforts (similar to that achieved during sustainable wild boar management). The probability of success is further increased if carcass removal rates are doubled and/or carcasses are removed more quickly following death.

• Following a focal introduction of ASF, the affected area is small and relatively limited numbers of wild boar are present that may subsequently become infected and die. In this situation, a high carcass removal rate in the core area can override carcass creation. Therefore, eradication could potentially be achieved without the need for intensive hunting efforts in the intensive hunting area.

• The impact of hunting on the wild boar population should be maximised over as short a time period as possible.

• A lengthy delay after the establishment of zoning and prior to culling in the core and buffer areas is detrimental with respect to maximising the probability of eradication success. This is contrary to expert opinion, where it is recommended – to avoid perturbation of the population during the epidemic – that culling commence only once the epidemic peak has been reached.

• The width of the buffer area will influence the probability of eradication success, with wider buffer areas leading to higher success rates. However, increases in the buffer area will also lead to a larger intensive hunting area, where intensive hunting efforts may be harder to achieve in practice, as well as to more animals finally to be culled in the buffer area.

- Even with wild boar proven fences around the core area, success might be imperfect because zoning must be based on carcasses, i.e. not on actual infected animals.
- Assumed protectiveness of fences only counts in if the other measures, as carcass removal and intensified hunting, fail in their efficient implementation.
- Still fencing of selected segments of demarcation would be a useful option to physically shelter parts of the "fence line" outside of which carcass removal or intensive hunting is difficult to put in action. Efforts could be concentrated to improve permeability problems.
- The focal emergency approach was not useful in the context of protecting areas bordering to large extent with areas affected by ASF in wild boar.

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Annex A – ODD Model Documentation (version Aug 2018)

Martin Lange & Hans-Hermann Thulke

UFZ – Helmholtz Centre for Environmental Research, Department of Ecological Modelling, PG Ecological Epidemiology, Leipzig, Germany

www.ecoepi.eu/ASFWB
Contact: martin.lange@ufz.de, hans.thulke@ufz.de

A.1. ODD Model documentation

A.1.1. 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).

A.1.1.1. 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.

A.1.1.2. 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, an 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).

A.1.1.3. 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. Sub-models 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.

A.1.2. 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.

A.1.3. Details

A.1.3.1. Initialisation

The local breeding capacity CC_{ij} of each cell is initialised 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} = 1.28 * density_estimate [heads/km²] following the regression density_estimate = f(CC_{ij}). Individual cell values $\widehat{CC}_{ij}$ are assigned by drawing from a Poisson distribution with $\lambda = CC_{ij}$.

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-year 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

A.1.3.2. Input

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

A.1.3.3. Submodels

Submodels are described in the order of their execution. Parameters and their values are listed in Table 3 in section “Parameters”.

A.1.1.1.1. Release of infection

The virus is released at the end of June of the 6th year of each model run to 5 (300) hosts in the release location of the focal (linear) simulation experiments. Release is scheduled in the 6th year in order to allow population dynamics to establish.

A.1.1.1.2. Transmission of infection

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

Direct transmission within groups: 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 within groups 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:

$$\Pi_i^{(i)} = 1 - \left(1 - P_{inf}^{(i)}\right)^{\lambda_i} \quad (2)$$

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

Direct transmission between neighbouring groups: The mode refers to transmission between animals of different female groups and males associated with those. Between group direct transmission is modelled stochastically. Parameter $P_{inf}^{(b)}$ determines the probability of contracting the infection from an infectious member of a neighbouring group during one week. For each susceptible animal, the probability of between group infections accumulates over infectious animals of all neighbouring groups:

$$\Pi_i^{(b)} = 1 - \left(1 - P_{inf}^{(b)}\right)^{\lambda_b} \quad (3)$$

where λ_b is the number of infectious individuals in the neighbouring groups of the one containing the receiving individual. Default neighbourhood addresses all groups with a common edge point of their grid cells.

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

$$\Pi_i^{(c,\mathrm{a},s)} = 1 - \left(1 - P_{inf}^{(c,a)}\right)^{\omega_i} \cdot \left(1 - P_{inf}^{(c,a)}\right)^{\Sigma_j \omega_{ij}} \quad (4)$$

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 s).

Effective transmission: For every habitat cell and per time step, the transmission probability is accumulated from direct, within and between, and carcass transmission probabilities for either age cohort a

$$\Pi_i^{(t,a,s)} = 1 - \left(1 - \Pi_i^{(i)}\right) \cdot \left(1 - \Pi_i^{(\mathrm{b})}\right) \cdot \left(1 - \Pi_i^{(,\mathrm{ac},a,s)}\right)\left(1 - \Pi_i^{(c,a,s)}\right) \quad (5)$$

The probabilities $P_{inf}^{(c\ \&\ b)}$ optionally might be increased during rutting season for female-male contacts.

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 $\Pi_i^{(t,a,s)}$ of the home cell.

A.1.1.1.3. 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 becomes 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'.

A.1.1.1.4. 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 sub-adults to move N_{disp}, are processed. Groups are iterated randomly for the splitting sub-model. From such groups, the model collects sub-adult female yearlings without offspring. Then, an empty habitat cell is selected randomly among all accessible cells. All dispersing individuals of the group disperse as a 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.

A.1.1.1.5. Male dispersal

Male dispersal is performed in weeks 25 to 30 of the year only (i.e. mid-June to the end of July). Uniformly distributed over the weeks of the dispersal period sub-adult 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 simply 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.

A.1.1.1.6. Reproduction

Females reproduce only once a year if at least at sub-adult 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 for their ability 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 in 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.

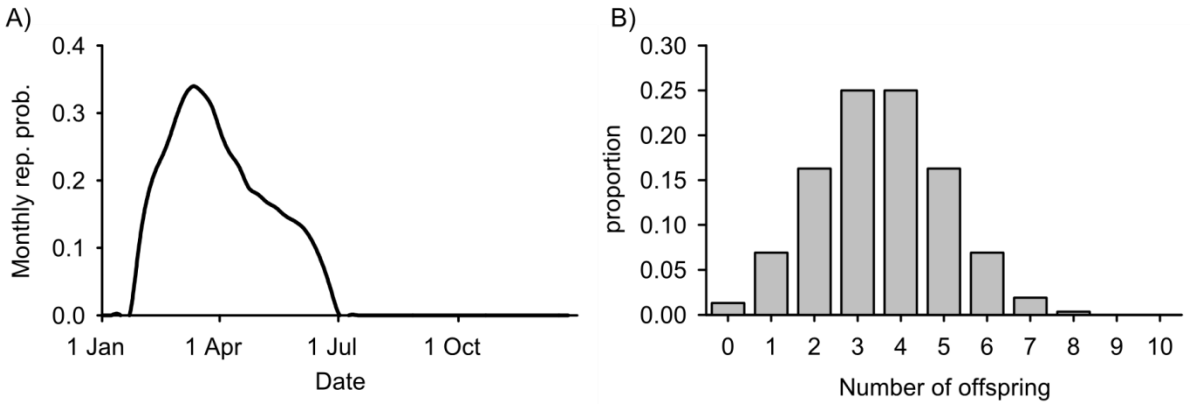

Figure 1 A) Monthly reproduction probabilities for wild boar. B) Breed count distributions for wild boar (Bieber & Ruf 2005, EFSA 2012).

Depending on the disease state of the breeding individual, its piglets' 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 to be at the maximum and rather fast. Hence the knowledge gap does not conflict with the simulation rules: If assigned to 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 a probability of prenatal infection P_{PI}. Immune individuals produce offspring that are temporarily immune due to maternal antibodies.

A.1.1.1.7. 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 the 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:

$$PM_{Week} = 1 - (SP_{Year})^{1/52} \quad (6)$$

Mortality due to infection is independently treated by the disease course sub-model.

A.1.1.1.8. Carcass distribution and persistence

The carcass of an infected dead individual is accessible to non-group mates with a 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 neighbours).

Carcass persistence is seasonal, with persistence times T_{carc} shown in Table 2. To always reflect the current persistence, each carcass has a persistence score, which starts with 1.0 and is decreased by $1/T_{carc}$ every step. The carcass is removed when the score reaches 0.0.

Table 2: Seasonal carcass persistence T_{carc}.

Month	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Persistence [weeks]	12	12	10	8	6	4	4	4	6	8	10	12

A.1.1.1.9. Ageing

The ageing process iterates over all individuals. For each individual k, age T_k is incremented by 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.

A.1.4. Parameters

Model parameters of the transmission model are shown in Table 3.

Table 3: Model parameters

Name	Description	Value	Source / details
a)	Wild boar ecology (used as constants 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)
$SP_{mean}^{(p)}$ / $SP_{\min}^{(p)}$	Mean / minimum annual survival rate piglets (natural mortality + conventional hunting)	0.5 / 0.1	(Focardi et al. 1996)
$T_{rutting\ start,\ end}$	Rutting season	Nov-Jan	Expert knowledge input
b)	Dispersal and movement parameters (used as constant for the study)		
N_{disp}	Minimum number of sub-adult females for dispersal	2	Technical assumption
D_{disp}	Maximum dispersal distance for sub-adult females	2 cells (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 death	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).

Name	Description	Value	Source / details
$P_{inf}^{(b)}$	Infection probability by direct transmission between different social groups	Less than $P_{inf}^{(i)}$	Range tested. Uncertain and motivated by expert discussion (EFSA SWG on ASF).
$P_{inf}^{(c,a)}$	Infection probability per carcass (including contact and transmission) dependent on the age *a* of the contacting animal	0.02-0.05	Range is derived from camera trapping (Probst et al. 2017) and data-driven model calibration (Lange & Thulke 2017) using spatial-temporal data of observed spread in wild boar
p_{access}	Probability of virus-induced death in shared area	0.8	Determined using spatial-temporal data of observed spread in wild boar

d) Secondary disease course parameters (not relevant for the 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)