EXTERNAL SCIENTIFIC REPORT

APPROVED: 26 October 2017

doi:10.2903/sp.efsa.2017.EN-1312

Simulation-based investigation of ASF spread and control in wildlife without consideration of human non-compliance to biosecurity

PG EcoEpi

Helmholtz Centre for Environmental Research GmbH – UFZ

Department of Ecological Modelling

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Abstract

African swine fever (ASF) is a devastating infectious disease of domestic pigs and wild boar. ASFV has spread in EU member states after entry through the eastern EU border. The objective of this assignment was to evaluate the ASF control measures that would be applied if the epidemic had already established along a large front rather than newly emerging foci; and whether EFSA's conclusions of 2015 regarding wild boar management options are still pertinent. A spatially-explicit individual-based simulation model was run on a 400x100km habitat landscape. The evaluated tools were population reduction measures and removal of carcasses. Alternative scenarios were modelled regarding timing of contacts of wild boar with carcasses and carcass removal. The simulated measures were applied with different levels of effectiveness upfront or within the ASF-affected part of the simulation landscape. The outcome considered was the eradication success. Additionally, the temporal ASF invasion into the control part was recorded by distance for all parameter combinations. The results indicated that single measures will be effective if applied very intensively, i.e. at or beyond the efficacy limits reported from the field. Measures were only effective if applied preventively, i.e. sufficiently far into the part of the simulation landscape without ASF infections. The efficacy of carcass removal was influenced by the time of contact with wild boar carcasses by con-species. Carcass removal time as reported from the field (2-6 weeks, median 4 weeks) did contribute marginally to success, whereas quick and effective carcass removal did improve the model outcome significantly, especially when first infectious contacts were assumed to take place a few weeks after death. The investigated measures were inadequate in model populations with higher local population densities than suggested for ASF affected areas before summer 2017. Representing real landscapes in the model tentatively improved the predictions but this would be at the cost of less general insights.

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Key words: African Swine Fever, individual-based, spatial-temporal simulation, control measures, hunting, carcass removal, wild boar

Question number: EFSA-Q-2017-00155

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Acknowledgements: The authors acknowledge expert input to model structure and simulation design by the ASF WG 2017. In particular the technical and interpretative comments by Vittorio Guberti (Bologna) were gratefully received.

Suggested citation: Helmholtz Centre for Environmental Research GmbH, Thulke H-H and Lange M, 2017. Simulation-based investigation of ASF spread and control in wild life without consideration of human non-compliance to biosecurity. EFSA supporting publication 2017:EN-1312. 30 pp. doi:10.2903/sp.efsa.2017.EN-1312

ISSN: 2397-8325

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1. Introduction

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

This contract was awarded by EFSA to:

Contractor: Helmholtz Centre for Environmental Research GmbH – UFZ

Department Ecological Modelling

Contract title: Simulation-based investigation of ASF spread and control in wildlife without consideration of human non-compliance to biosecurity

Contract number: NP/EFSA/ALPHA/2017/11

African swine fever (ASF) is a devastating infectious disease of domestic pigs and wild boar, and is usually fatal. No vaccine exists to combat this virus. It does not affect humans nor does it affect any animal species other than members of the Suidae family. From the beginning of 2014 up to 22/02/2017, ASFV has spread in Estonia, Latvia, Lithuania and Poland, mainly in wild boar, but also affecting the domestic pig sector. EFSA has issued 3 scientific opinions on African swine fever (2010, 2014 and 2015), an opinion on the role of ticks in the epidemiology of ASF in Eurasia (2010), an evaluation of possible mitigation measures to prevent introduction and spread of ASF virus through wild boar (2014), and a scientific report analysing epidemiological data collected on ASF in affected MS (2017). In the context of providing scientific and technical assistance on ASF to the European Commission, EFSA was asked to review the management options for wild boar identified in the EFSA scientific opinion of June 2015, and to indicate whether the conclusions of that scientific opinion regarding wild boar management options are still pertinent.

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

The main objective of the simulation study was to assess the possible effect of the control measures applied in ASF affected areas on the spread of the infection in wild boar populations.

The purpose of the study was to evaluate the preventive strategies proposed in the EFSA opinion of 2015 (EFSA, 2015) to stop the forward spread of an ASF epidemic in the wild boar population. The strategies were tested in an expert system model, building on expert and literature knowledge, including explanatory hypotheses about the ASF epidemiology (transmission, contact infection, role of carcass, population dynamics in the affected countries).

Human interferences leading to non-biological forward transmission of ASFV (i.e. cases without links to past reports of ASF in wild boar, considering biologically plausible movement behaviour of ASFV in the wild boar population in time and space) were not considered in the model analysis. Their unpredictable nature can lead to the occurrence of ASF cases in wild boar (e.g. in the Czech Republic) and outbreaks in domestic pigs (e.g. in Romania) within several hundred kilometres distance of the latest ASF case notifications.

In particular the study addressed the following questions:

1. Does new agreed knowledge or parameterisations change the simulation outcome as of EFSA opinion 2015?
2. Do the actually implemented measures meet the proposed control target?
3. Can the proposed option of intensifying carcass removal suggested by the EFSA 2015 opinion be supported, given the new insights about difficult practicalities related to carcass removal and possible duration of carcass remediation times?
4. Are the investigated strategies able to cope with population capacities in westward wild boar habitats?

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5. To what extent would more detailed knowledge of local habitat structure contribute to the success of the strategy, i.e. would improved density maps add to the understanding of control efficacy?

1.3. Technical background

The spatially-explicit and agent-based expert knowledge model applied for the EFSA 2015 opinion on ASF {EFSA 2015; Lange 2015} was updated, taking into account the most up-to-date evidence regarding ASF in wild boar in Eastern Europe. Sensitivity of resulting management recommendations to the most uncertain parameters was addressed using systematic range analysis. The modelling paradigm of the existing model tool followed the conceptual understanding of ASF transmission in wild boar, as determined by literature and WG experts at time of writing this report. Model flexibility is tailored to adequately consider unpublished data provided by the ASF working group or competent authorities involved in wild boar management in Eastern European member states (MS). Control and mitigation measures were proposed in detail through participatory conceptualisation involving subject-matter experts contacted by EFSA. Using these data different measures were implemented in the model platform. Alternative hypotheses were addressed by scenario analysis. The methods for quantitative comparison of the control and mitigation measures was determined and proposed to EFSA for approval; therewith further model amendments were determined and adopted. Following formal approval by EFSA, systematic simulations were performed. The multidimensional output was summarized, and conclusions proposed to EFSA and their implications discussed with the working group. After expert feedback on the first simulations, subsets of simulations were readdressed to answer specific details or strategic options including proposed alternative parameter scenarios.

2. Data and Methodologies

2.1. Data

ADNS case reporting data as of Sept 2017. Expert-based questionnaire regarding the implementation of carcass removal along with ASF control measures.

2.2. Methodologies

2.2.1. Model documentation

The complete model documentation published with the EFSA output in 2015 is still correct (minor adaptions of parameters are explicitly stated in the following). The documentation can be found also under http://ecoepi.eu/ASFWB.

2.2.2. Scenario definition

In order to address the objectives, several basic scenarios were implemented and assumed the efficacy of the suggested control measures varied from minimum (0%) to maximum (100%). The systematic approach was necessary as certain control measures may still be difficult to quantify with regard to the actually achievable efficacy in field implementation, e.g. what is the maximum proportion of carcasses removed within 2 weeks from an area subjected to control measures.

The scenarios cover:

(a) Method of population management, i.e. short-term drastic depopulation within 16 weeks [D1] vs. so-called soft measures targeting females with standard hunting efforts [D0];

(b) Time of first carcass contact by live wild boar, i.e. instantaneous as in 2015 [Default] vs. delayed by 2 weeks as suggested by Probst et al. (2017) [Delayed];

(c) Carcass remediation time, i.e. constant [Default] vs. seasonal [Seasonal]; and

(d) Habitat structure [Poisson].

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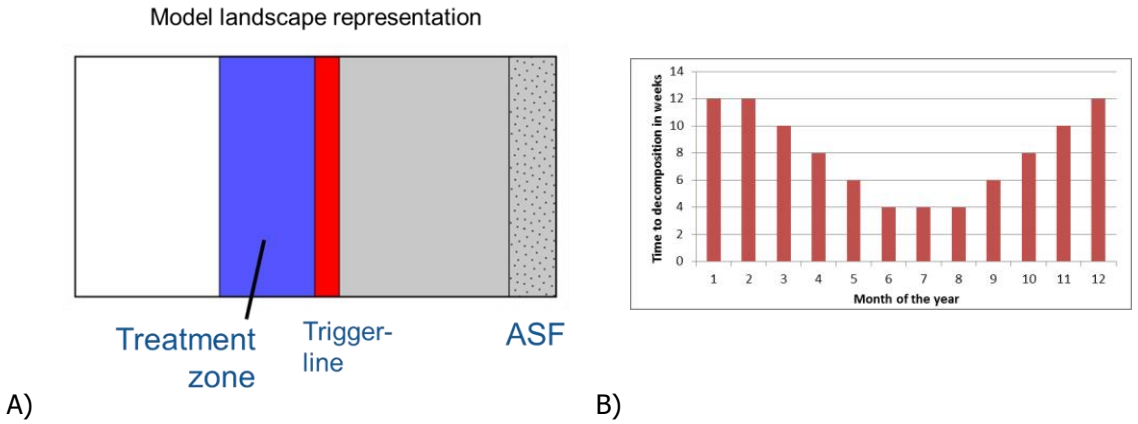
Simulations of any of the above alternative qualitative scenarios were repeated over parameter alternatives addressing uncertainty regarding:

- Width of the treatment zone (blue in Fig. 1) in front of ASF (50km; 100km; 200km)
- Efficiency of proposed measures in terms of percentage achievement of the targeted endpoint (one short-term depopulation, continued female reduction, carcass removal).
  - Percent (30%, 50%, …, 90%) depopulation within one single campaign of 4 months (no repetition) – [D1].
  - Percent (30%, 50%, …, 90%) reduction of next-season reproductive females using unchanged hunting bag size representing standard hunting pressure – [D0].
  - Percent (0%, 30%, 50%, …, 90%) carcass removed by a certain time after death – [Carc][1].
  - Combined application of Carc together with D1 or D0.
- Population density prior to ASF resp. alternative area size for wild boar groups (hypothesis) [Scaling factor (Sc) being 1, 2, 4 relative to the local density proposed by the affected member states hence set, doubled, quadrupled].
- Habitat structure changed from uniformly to Poisson distributed habitat quality, i.e. local group size (hypothesis of low population level mortality).
- Possible late contact with carcass. In 2015 it was assumed that carcasses are immediately contacted. A recent observational study may imply that the fresh cadavers are avoided until a certain degree of decomposition sets in (Probst et al. 2017). The hypothesis is implemented by discarding contacts to infectious carcasses for two weeks post death.
- Seasonal carcass decomposition rate to address the effect of seasonal differences in decomposition rate due to temperature (hypothesis; see Fig. 1B for details).

Model simulations are performed on the same landscape as used and described in the EFSA opinion 2015 (Fig. 1A). ASF spreads from the right border into the grey area until the infection reaches the trigger line (red), from that moment onwards the control measures as foreseen by a given scenario are applied according to the associated schedule (once or permanent) within the treatment zone (blue). The recorded output is the time at which the infection is eradicated (success) or breaks out of the treatment zone (failure).



A) B)

[1] Carcass removal implied the exclusion of carcasses after two weeks (default and CR2) or four weeks (CR4). The carcasses to be excluded are randomly drawn from all carcasses in the treatment zone using the carcass removal efficiency parameter as individual event probability. Carcasses not removed remain until decomposed.

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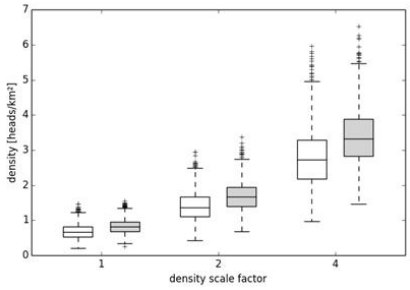

C)

Figure 1: A) Schematic representation of the spatial landscape of the simulations. ASF-infected animals occur from the right with continued forward spread towards the left. Once the simulated spatial spread reaches the trigger line, a prescribed control measure is applied to the blue zone. Different treatment zone (blue) widths - 50km, 100km and 200km - were simulated. Source EFSA, 2015. B) Seasonal distribution of carcass decomposition varies between 4 and 12 weeks. The function approximates observed decomposition speeds and is only indirectly dependent on temperature. C) Population density (y-axis) in the future control area (blue box in A) for the different density scenarios (x-axis), as resulting from alternative habitat landscapes used in the model simulation. Model density was measured at week 1 (white) and week 26 (shaded) of the calendar year. Variability is mainly due to stochastically volatile population dynamics. The graphs show the median (middle line), 26-75% range (box); 1.5 Interquartile range (whiskers) and outliers (crosses).

3. Assessment/Results

The complete results are presented in the contour plots in Fig.4 – Fig.9. The following example diagrams are presented to provide a principle understanding of the simulation outcome (Fig. 2 + 3). The differently coloured graphs represent alternative efficiency of carcass removal within one week. The efficacy of the assumed population measures was set to 70% which is already known to be technically difficult to achieve (EFSA, 2014). Three diagrams allow comparison of the strategy outcome for alternative levels of population aggregation.

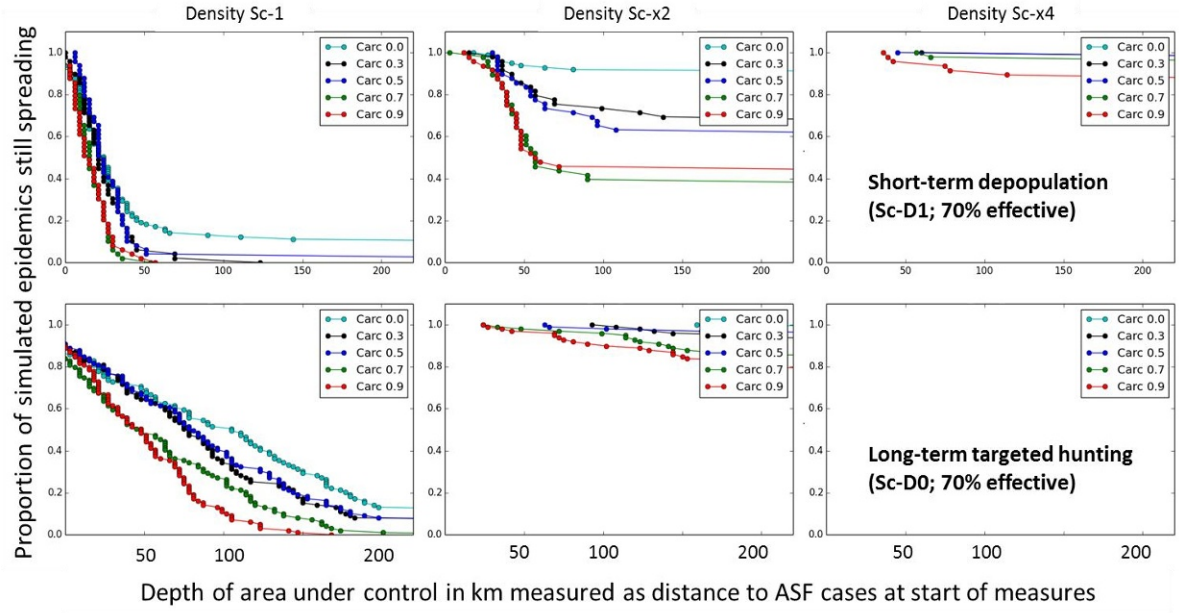

Figure 2 (top row) + Figure 3 (bottom row): Simulation output shown as the proportion of runs (y-axis) spreading beyond a given distance in km (x-axis) from the previous location of ASF at the moment the measures were started. The further the control zone stretches from the original ASF recordings, the lower the number of runs that continue to spread. The proportion on the y-axis is the success rate found with a given combination of measures in the model simulations. Top row (Fig. 2): Short-term depopulation assuming 70% effectiveness. Bottom row (Fig. 3): Targeted hunting of females over multiple years assuming 70% effectiveness. Alternative density scenarios (left column as reported for the Baltic MS; middle doubled; right quadrupled). Differently coloured graphs reveal the effect of increasing intensity of carcass removal within 1 week (turquoise no carcass

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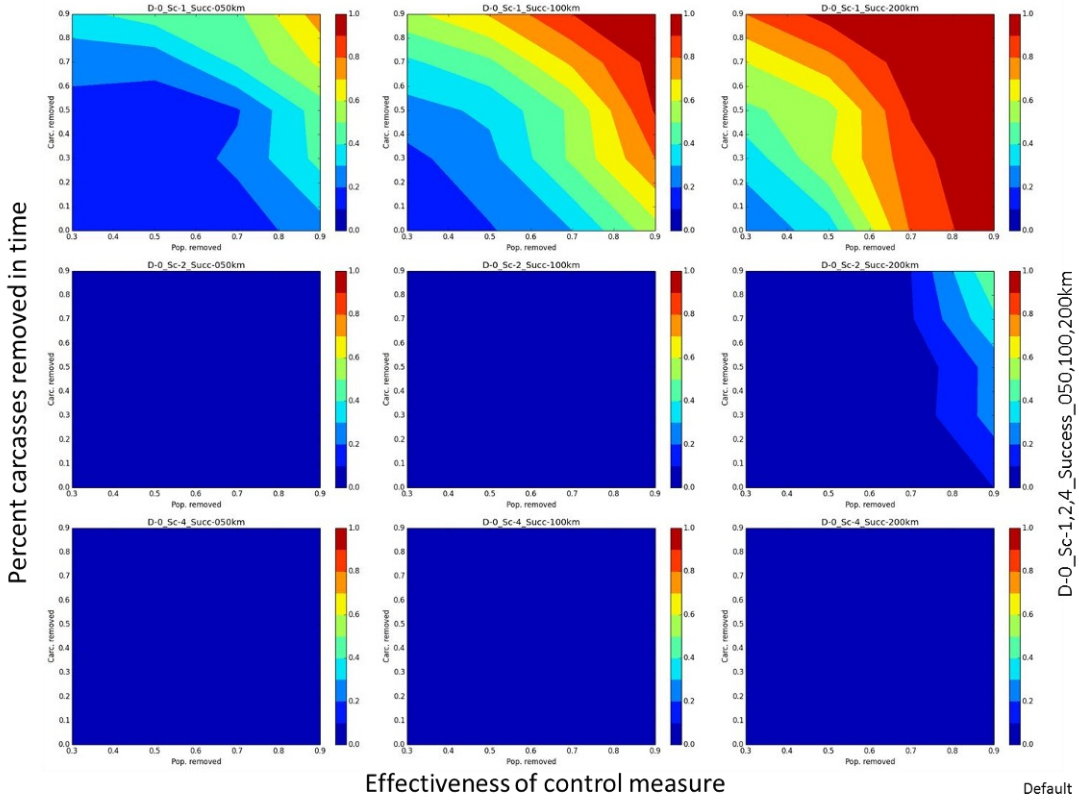
removal; red 90% removed within one week). Every dot represents the final distance made by one individual simulation run that was eliminated by the simulated control measures. The line graph therefore shows the risk to fail (y-axis) in eradicating the simulated epidemic with a given width of the control zone size (x-axis) and the measures applied respectively.

3.1. Default scenario

The diagrams of Fig. 4 summarize the standard simulation using updated transmission parameters according to Lange & Thulke 2017, Probst 2017 – no delay. The contours provide insight into the model prediction regarding containment of the simulated epidemic by a certain combination of measures, recorded as a proportion of simulation runs that stopped vs broke out of the treatment zone. The width of the treatment zone (see blue rectangle in Fig 1) was varied, measuring 50km (left), 100km (middle), and 200km (right). Density is doubled from 1 (top), 2 (middle) to 4 animals per sq km before reproduction. Colours beyond green (i.e. yellow, orange to red) refer to simulations that stopped the spread of ASF in the model by the respective treatment by more than chance outcome (>50%). Note: Contour lines should not be interpreted in an exact manner but rather by the principle curve because the parameter resolution was by steps of 0.2. The contours are smoothing interpolations.



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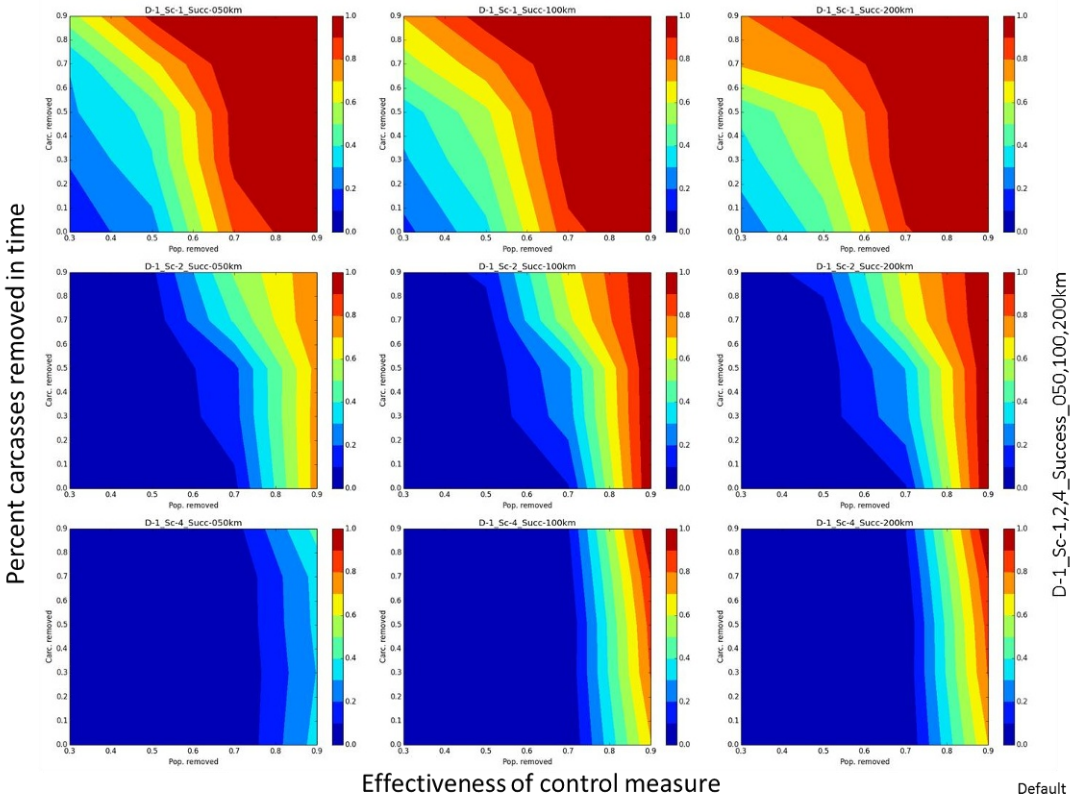

4.D

Figure 4: Success of simulated control strategies under the default scenario. Population measures use targeted hunting of females (top 9 diagrams) and depopulation (bottom 9 diagrams). Measures are applied to treatment zones of 50km (left), 100km (middle), 200km (right) in width determined as ASF-unaffected area at the moment the measures were started. Top row refers to the density reporting of the affected countries; then doubled (middle rows) or quadrupled (bottom row). Effectiveness of population measures is varied on the x-axis while the efficacy of carcass removal is varied on the y-axis. Reddish colours beyond the yellow segment indicate a success rate of at least 70% of simulated epidemics.

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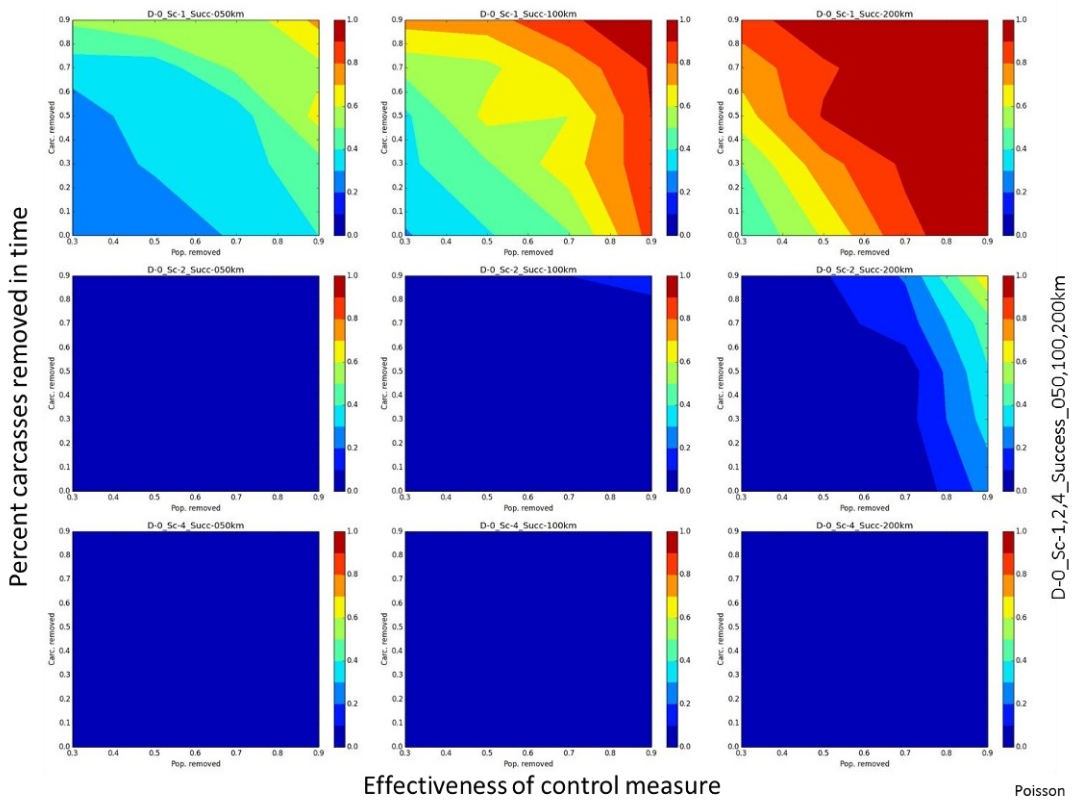
3.2. Non-uniform habitat quality distribution



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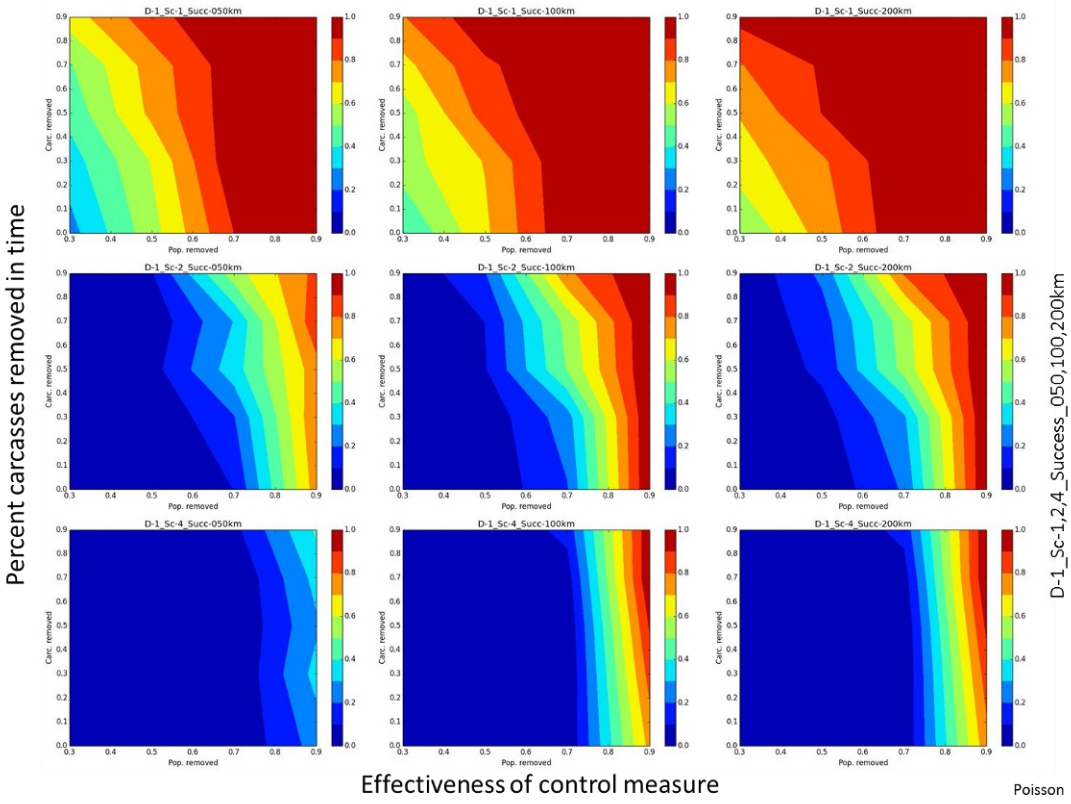

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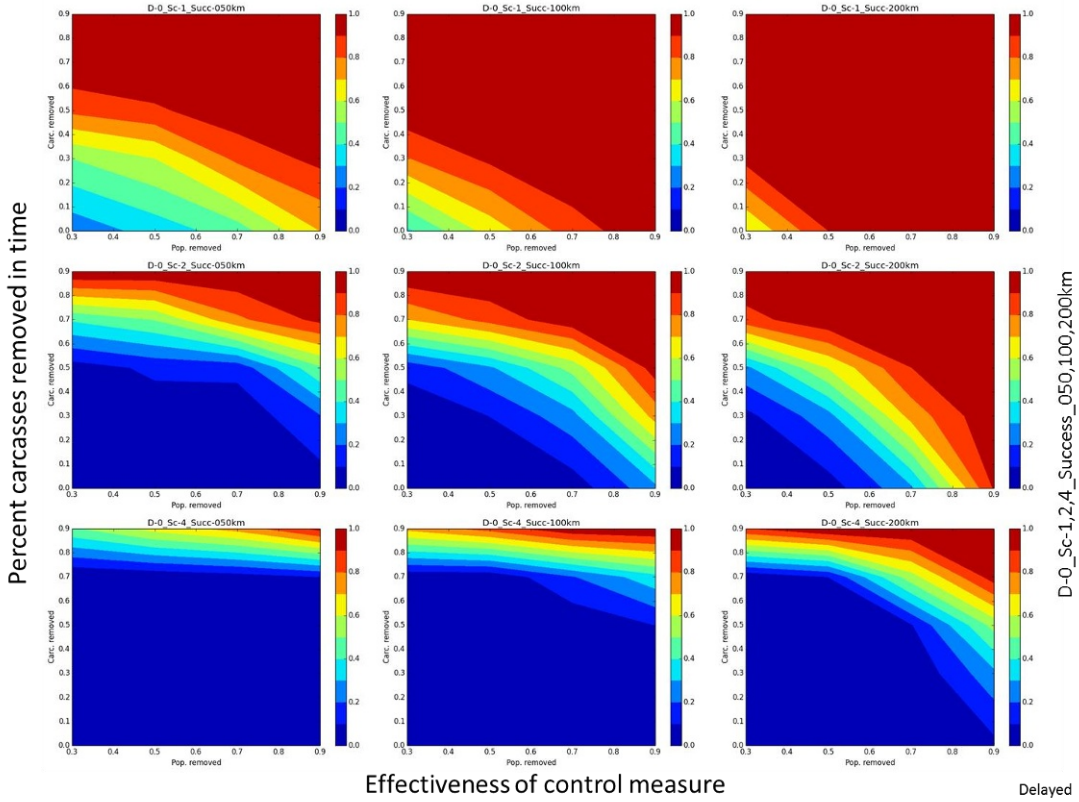
Figure 5: Success of simulated control strategies under the habitat structure scenario. The scenario is as in Fig. 4 but the habitat quality determining the group size is no longer assigned by uniform distribution but using Poisson law, creating more heterogeneous habitat. From ecology, it is expected that more structure should increase the success rate for small densities. Population measures use targeted hunting of females (top 9 diagrams) and depopulation (bottom 9 diagrams). Measures are applied to treatment zones of 50km (left), 100km (middle), 200km (right) in width determined as ASF-unaffected area at the time the measures are started. Top row refers to the density reporting of the affected countries; then doubled (middle rows) or quadrupled (bottom row). Effectiveness of population measures is varied on the x-axis while the efficacy of carcass removal is varied on the y-axis. Reddish colours beyond the yellow segment indicate a success rate of at least 70% of simulated epidemics.

3.3. Delayed contact with carcasses



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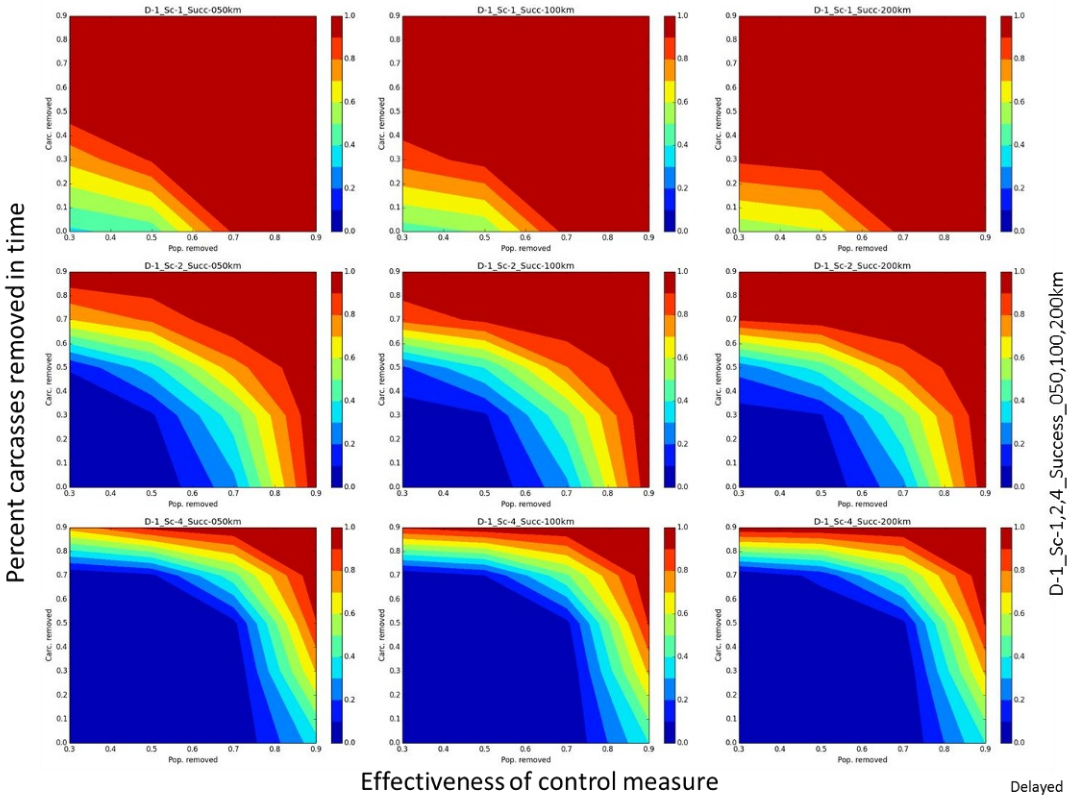

6.D

Figure 6: Success of simulated control strategies under the delayed scenario. The scenario is as in Fig. 4 but the first contact with carcasses is only enabled after two weeks as suggested by Probst et al. (2017). Logically it is expected that the instantaneous removal of carcasses will now be more useful. Population measures use targeted hunting of females (top 9 diagrams) and depopulation (bottom 9 diagrams). Measures are applied to treatment zones of 50km (left), 100km (middle), 200km (right) in width determined as ASF-unaffected area at the moment the measures were started. Top row refers to the density reporting of the affected countries; then doubled (middle rows) or quadrupled (bottom row). Effectiveness of population measures is varied on the x-axis while the efficacy of carcass removal is varied on the y-axis. Reddish colours beyond yellow segment indicate success rate of at least 70% of simulated epidemics.

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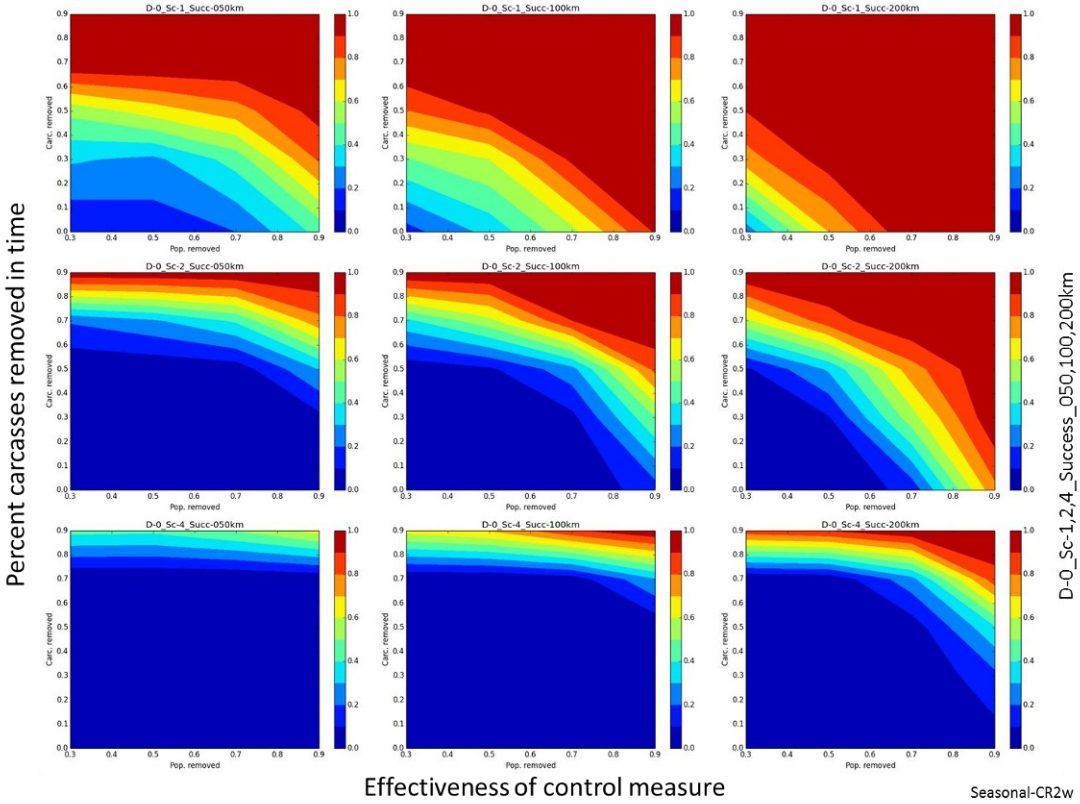
3.4. Delayed + seasonal decomposition + carcass removal after 2 weeks



7.T

Figure 7: Success of simulated control strategies under the delayed + seasonal + 2 weeks carcass removal scenario. The scenario is as in Fig. 6.T with first contact delay, but additionally seasonal decomposition of carcasses. Carcass removal is after 2 weeks (shorter than suggested by the MS data: 2-6 weeks, median 4 weeks). Logically it is expected that the later removal of carcasses will reduce efficacy but the seasonality counterbalances during the seasons with highest ASF incidence. Population measures use targeted hunting of females (top 9 diagrams). Measures are applied to treatment zones of 50km (left), 100km (middle), 200km (right) in width determined as ASF unaffected area at the moment the measures were started. Top row refers to the density reporting of the affected countries; then doubled (middle rows) or quadrupled (bottom row). Effectiveness of population measures is varied on the x-axis while the efficacy of carcass removal is varied on the y-axis. Reddish colours beyond the yellow segment indicate success rate of at least 70% of simulated epidemics.

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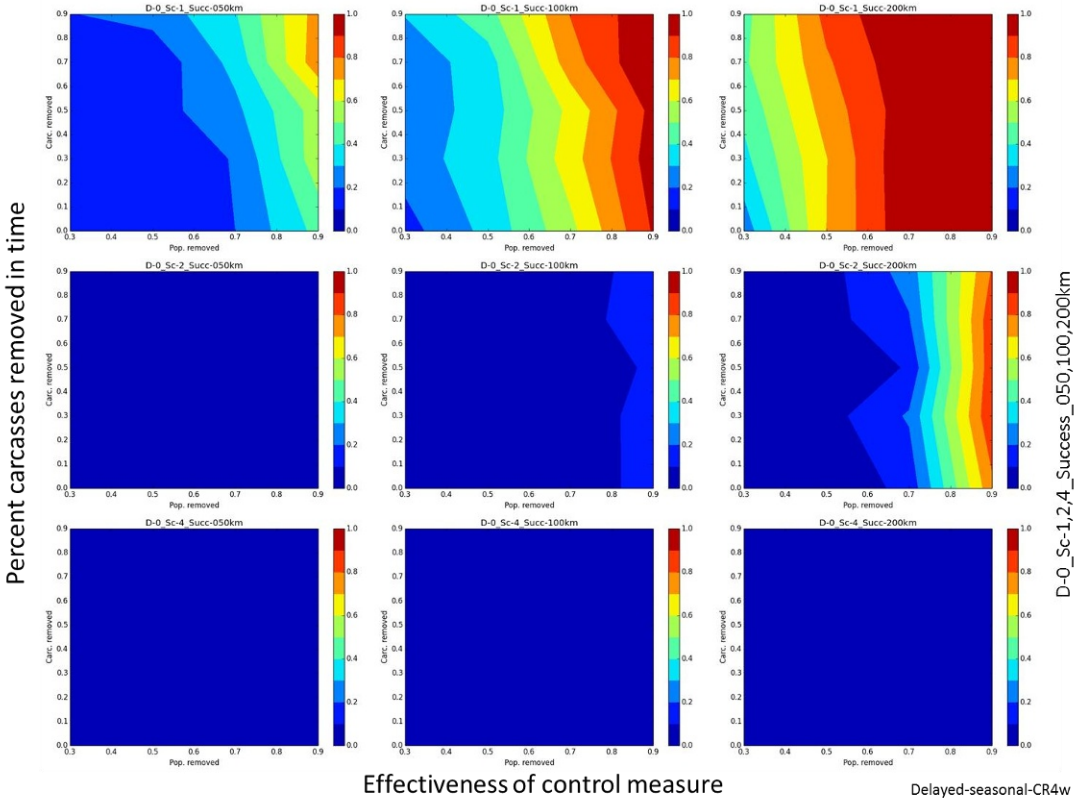
3.5. Delayed + seasonal decomposition + carcass removal after 4 weeks (field scenario)



8.T

Figure 8: Success of simulated control strategies under the delayed + seasonal + 4 weeks carcass removal scenario. The scenario is as in Fig. 7.T with first contact delay, seasonal decomposition of carcasses. However, carcass removal is after 4 weeks as suggested by the MS data: 2-6 weeks, median 4 weeks. Logically it is expected that the very late removal of carcasses will offset the effect of carcass removal. Population measures use targeted hunting of females (9 diagrams). Measures are applied to treatment zones of 50km (left), 100km (middle), 200km (right) in width determined as ASF-unaffected area at the moment the measures were started. Top row refers to the density reporting of the affected countries; then doubled (middle rows) or quadrupled (bottom row). Effectiveness of population measures is varied on the x-axis while the efficacy of carcass removal is varied on the y-axis. Reddish colours beyond yellow segment indicate success rate of at least 70% of simulated epidemics.

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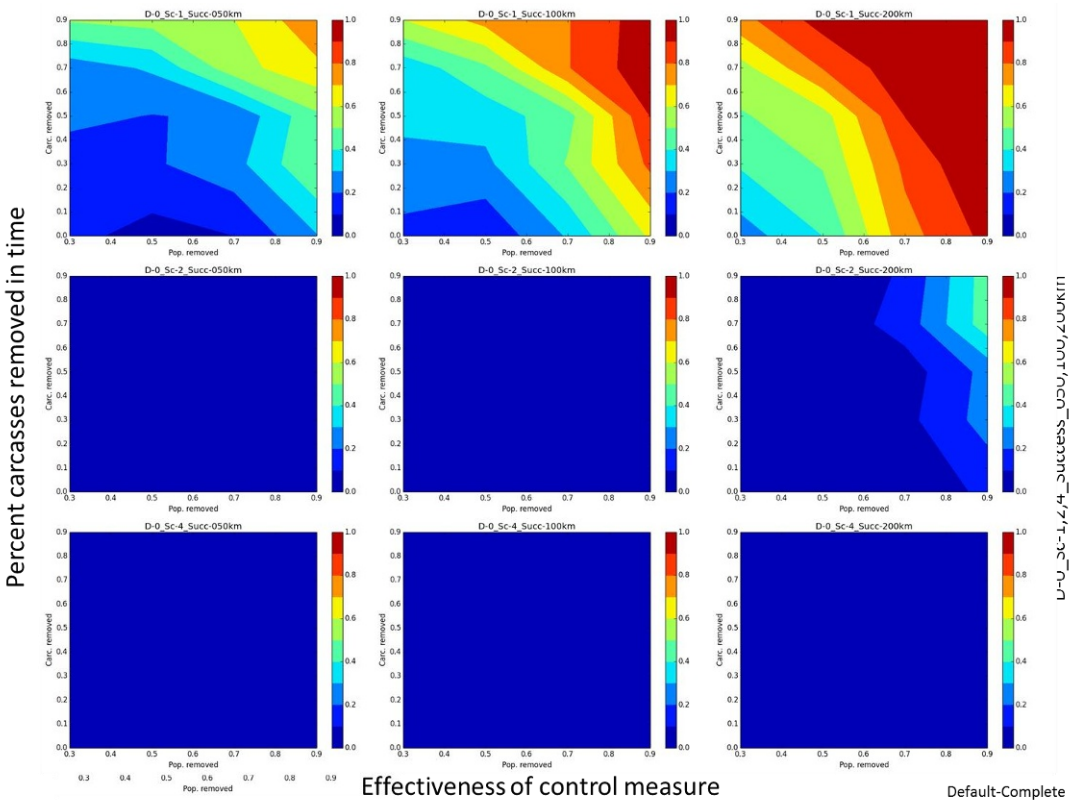
3.6. Default + control measures in all zones



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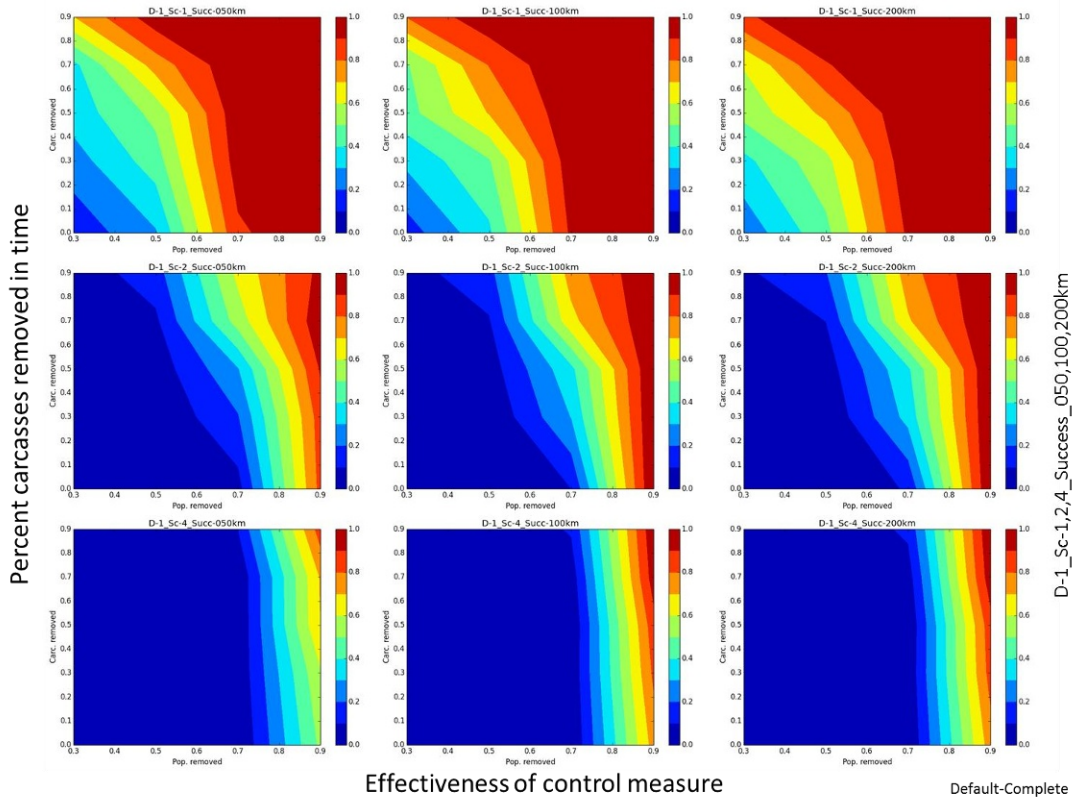

9.D

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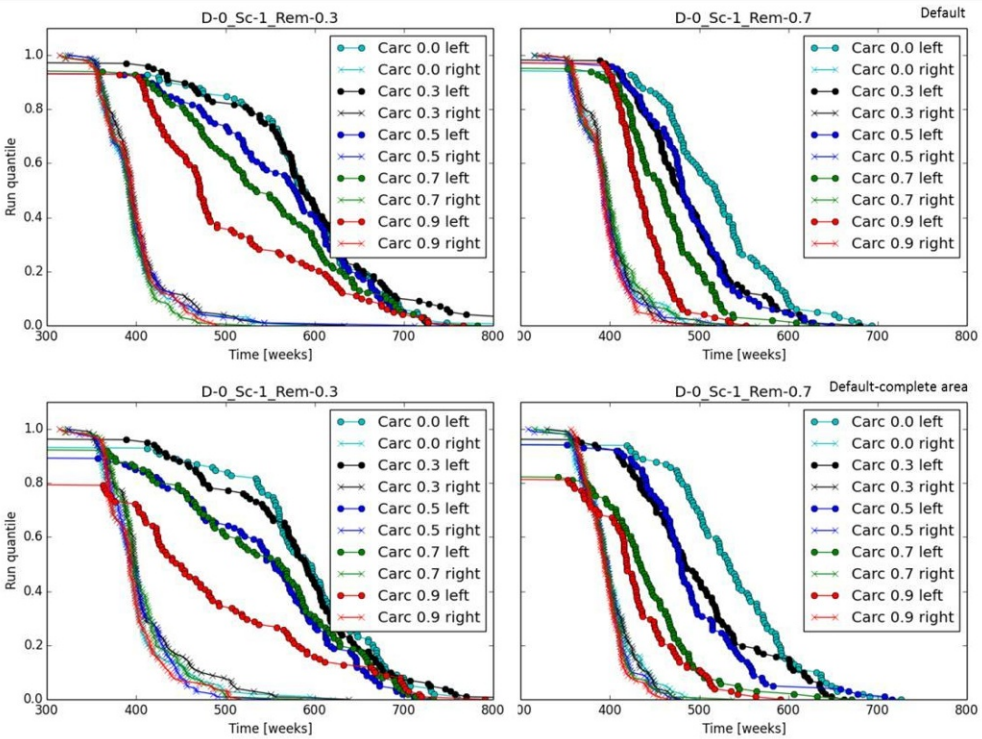
Figure 9: Success of simulated control strategies under the default + complete area scenario. The scenario is as in Fig. 4 but the measures simulated are simultaneously also applied to the left of the trigger line (grey part in Fig. 1). Logically, it is expected that the extra effort within the affected zone will improve the success rate. Population measures use targeted hunting of females (top 9 diagrams) and rapid depopulation (bottom 9 diagrams). Measures are applied to treatment zones of 50km (left), 100km (middle), 200km (right) in width determined as ASF unaffected area at the moment the measures were started. Top row refers to the density reporting of the affected countries; then doubled (middle rows) or quadrupled (bottom row). Effectiveness of population measures is varied on the x-axis while the efficacy of carcass removal is varied on the y-axis. Reddish colours beyond the yellow segment indicate a success rate of at least 70% of simulated epidemics.



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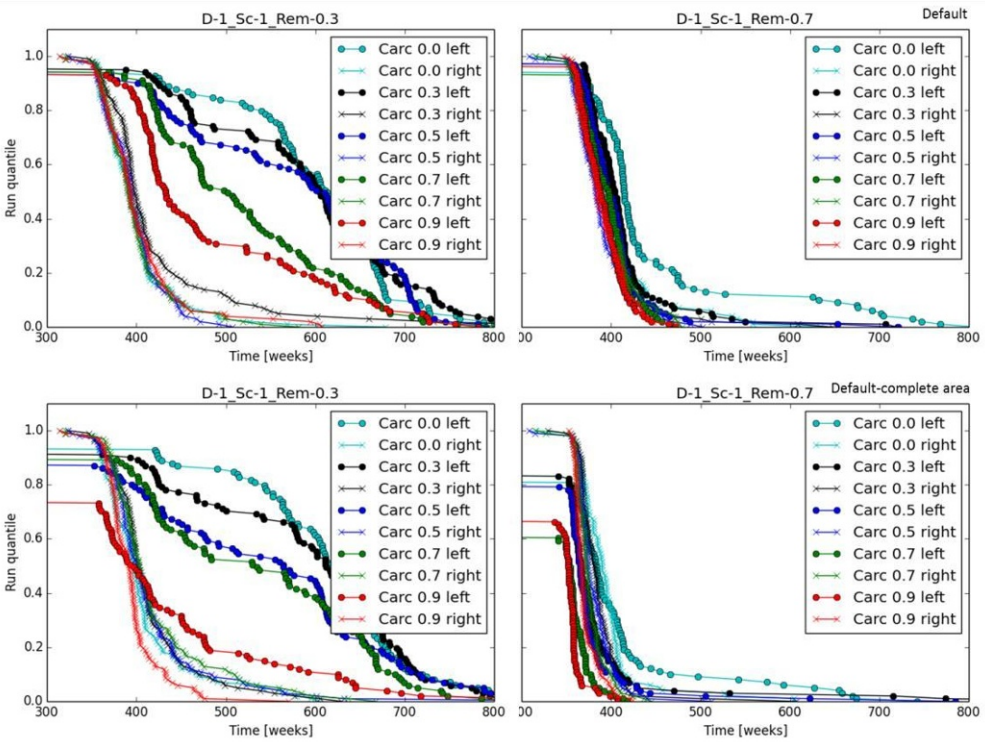

Figure 10: Cumulative success distribution of simulated control strategies under the default and the default+complete area scenario. The scenario is as in Fig. 4 and Fig.9. Population measures use targeted hunting of females (top 4 diagrams) and rapid depopulation (bottom 4 diagrams). Diagrams represent alternative efficacy of population measures (left 30% and right 70%). Line graphs represent alternative efficiency of carcass removal within one week. Every dot (cross) represents one individual simulation run which stopped perpetuating the infection in the treatment zone (dot-marked line) or in the affected zone (cross-marked line). The line graph therefore reveals the survival of the infection in the population (y-axis) over a certain duration of treatment (x-axis). Please notice that the diagrams cannot provide information about the success rate due to the limited size of the simulation landscape, but see the respective figures above. Comparison between the respective line graphs of first vs. second and third vs. fourth row reveals the effect of the additional application of measures in the affected zone (grey zone in Fig. 1A).

4. Interpretation

The intention of this simulation study was to evaluate the validity of the analysis reported previously in EFSA (2015) regarding the impact of certain control measures on the spread of ASF in spatially arranged wild boar populations. These further simulations were motivated by new insights and updated data sources since this earlier report (EFSA 2015; Lange 2015).

Relevant changes in our understanding of ASF in wild boar since early 2015 are related to the role, decomposition and contact source of carcasses. In reality, and also in the model, only carcasses from individuals that die while affected by the ASF virus are relevant. However, control measures to address the source of virus perpetuation will not be able to differentiate between "infectious" and non-infectious cadavers. Therefore we use "carcass" in the general sense of all cadavers, and "infectious carcass" for those that comprise live virus. Carcass removal in this document is related to the exclusion of cadavers from any possible transmission in the wild context, and could include burning, burying, removal etc.

In 2015, single, stand-alone measures were investigated, and combinations of measures (e.g. focal depopulation and distant application of the preventive population reduction by standard hunting methods) were not yet considered. At this time, carcass removal was not subjected to systematic study because no information was then available about key aspects of carcass removal, including the feasibility of different times to removal and the efficacy of this procedure (see EFSA 2015). At the time

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of writing of the current report, according to expert judgement from affected regions, it is practical to remove carcass within 2-6 weeks (median 4 weeks) after the death of the animal. This observation renders the simulations in 2015 impractical (see EFSA 2015; Lange 2015), noting that carcass removal was simulated at that time to improve understanding of the system rather than offering a control alternative. Removal was assumed to occur either instantaneously (CR0w) or one week post-death (CR1w). While the first (0w) led to nearly ideal success rate compared to all other possible options, the latter (1w) failed to improve beyond doing nothing. These insights underpinned the recommendation for further study on the role and importance of carcass removal in the context of ASF emergency management in wild boar.

> Does new knowledge or parameterisations change the simulation outcome as of EFSA opinion 2015? Does the simulated control approach in principle support the proposed control target of ASF eradication or halt of spread?

The current simulation study provides important insights after integrating new data about carcasses. Epidemiologically and empirically derived data substantiate the understanding that carcass contact in wild boar is rare (Lange & Thulke 2017; Probst et al. 2017) and scavenging probably negligible. Hence the new model simulations addressing the two main scenarios of 2015 (drastic short-term depopulation vs. long-term targeted population reduction) were repeated with all parameters up to the contact frequency per carcass. The impact of the measure on ASF is recorded by estimating the distance forward that the infection spreads after the control was started. In Figure 2a and 3a, the individual simulation runs are reported for a selected efficacy of population control measures (e.g. 70%). The turquoise graph (upper line) resembles the outcome with no carcass removal in place while the other lines represent an increasing efficiency of carcass removal within the first week after death (standard carcass removal scenario in EFSA, 2015). The main finding from these two diagrams is that even with an ambitious level of population reduction measures (70% depopulation in 4 months or multiple years of harvesting 70% of next-year's reproducing females), final success is not guaranteed, in support of the conclusions in EFSA (2015). Moreover, the strategy combining 70% effective targeted hunting with removal of up to 90% of all carcasses within one week provides substantial chance of success in treatment zones of less than 200km. However, both of these assumptions (hunting efficacy, 70%; carcass elimination, 90% in one week) cannot practically be implemented in the field.

The full picture of simulated combinations is summarized in Fig. 4.T and Fig. 4.D (T stands for Targeted hunting and D for Depopulation) top row for treatment zones of 50, 100, 200km in width. Clearly, the largest treatment zones of 200km would be necessary with the T strategy (top right diagram in Fig. 4.T). To achieve eradication success, hunting would need to be more than 70% efficacious (x-axis) and carcass removal nearly perfect within one week (y-axis). Drastic depopulation (Fig. 4.D) improves the picture slightly (population reduction of about 70% in only 50km), but is rather independent of efforts relating to carcass removal. The latter finding is logical, as depopulation will already prevent carcasses from being infectious. Nevertheless, the implied success requires greater depopulation efforts than would be deemed practical in the field (EFSA, 2014). Therefore, the new insights and altered parameters have not changed the outcome of the model analysis in 2015.

> Are the investigated strategies able to cope with population capacities in westward wild boar habitats?

Insights from simulations assuming different habitat quality (i.e. wild boar habitat with greater local density, or larger home ranges per wild boar group; Sc-2 and Sc-4) are even less favourable regarding the applied control measures. Here the simulated measures fail completely because substantial numbers of animals remain after the population reduction measures have been applied. If carcasses contribute to ASF epidemiology in the manner that is currently understood, then the entry of infection into regions with wild boar habitat more favourable than those in the Baltic Member States will likely mean that current control measures are of negligible effectiveness. Conversely, if carcasses do not play a relevant role, an alternative mechanism for virus perpetuation is as yet lacking.

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> Can the proposed option of intensified carcass removal suggested by the EFSA 2015 opinion be supported given the new insights about effectiveness of carcass removal and possible carcass remediation times?

The next point of the analysis addresses the effect of the hypothesis that carcass contacts do not occur immediately after an animal has died but are delayed by for a certain period, e.g. due to avoidance behaviour prior to decomposition, i.e. as long as the dead wild boar remains as such. The hypothesis derives from initial observations of contact behaviour in the field (Probst et al. 2017). A two week refractory period was suggested, and has been simulated in all scenarios named "Delayed" (see for example Fig. 6 and 7; these differ by the inclusion of a seasonal decomposition in the latter). As one would expect, if carcass contact transmission occurs only after two weeks, then any shorter removal period will improve the method. Idealistically, very promising results are achieved with the scenario of one week removal (e.g. Fig. 6). In Fig. 7, assuming two weeks for both the delay in carcass contact and the time to carcass removal, there is substantial improvement of the effectiveness of the measures, at least with basic habitat as reported for the North-Eastern MS (Fig. 7.T top row). Based on an expert report about the time of carcass removal under normal ASF control conditions, the data suggest a most-likely time horizon of 2-6 weeks post death, with a median value of 4 weeks. Using this median value for carcass removal time (Fig. 8.T), the results indicate that limited or no effectiveness when the measures were applied to the model landscape. Moreover, very reasonably, the success was again independent of carcass removal (y-axis in Fig. 8.T). The need for rapid removal of carcasses does conflict with observations from the field across all MS (2 vs. 2-6 weeks). Given our current understanding of carcass role in perpetuating the infection, carcass removal practice does not contribute substantially to the effectiveness of the simulated measures.

> To what extent would habitat structure contribute to the success of the strategy, i.e. would improved density maps add to the understanding of control efficacy?

The simulation of alternative landscapes regarding the homogeneity of structure of the wild boar habitat is in agreement with our understanding of the landscape ecology of infectious diseases (Fig 5): specifically, the success of simulated measures is improved with increasing landscape heterogeneity. Consequently, it will be possible to develop local predictions of the effectiveness of certain sets of measures using data, currently being collected, regarding wild boar presence at the level of the hunting ground or with detailed habitat prediction maps. However, such model improvements – already foreseen for the near future – will come at the cost of less general insights. Potentially, the results could no longer be extrapolated to any other wild boar region.

Finally, and in particular with regard to future European areas at risk of ASF entry, it seems reasonable to reconsider combinations of "soft" and "drastic" population reduction measures. If the objective is to prevent entry of the virus into territories neighbouring already affected areas, a targeted hunt combined with carcass removal as much as possible, would not satisfy the needs of other regions with much greater number of animals present.

The model uses common sense knowledge about ASF maintenance in wildlife, wild boar ecology, the disease course, transmission pathways and contact behaviour on the individual level. Although many of these aspects have been cross-validated or confirmed in field studies/laboratory experiments, there is still a substantial area of uncertainty. As one example, proof is currently lacking about the role played by carcasses in ASF transmission. Other uncertainties are more subtle. Even if the more general insights are robust and well-grounded in population ecology of infectious diseases, model predictions should be evaluated in terms of general trends between scenarios, rather than a strict interpretation of exact figures.

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5. Conclusions

- The analysed measures are only relevant if applied preventively to the unaffected part of the simulation landscape, i.e. including Part I areas and particular risk areas.
- The model analysis does not address the focal introduction scenario (e.g. Czech Republic; but see Proceedings SVEPM Gent 2015, 122-132, ecoepi.eu/ASFWB for such model scenarios).
- Either population measure alone is insufficient to halt the spread of ASF if parameters are assumed within a range that would correspond to those reported from field implementation (confirming 2015 with the new data).
- Carcass removal timing as reported by the MS (2-6 weeks, median 4 weeks) would not contribute to the success of the measures under the carcass related assumptions of the model simulations.
- In the model very early carcass removal (first week) improves the situation but may not be practical given the data reported by the MS on carcass removal time (2-6 weeks, median 4 weeks)
- The new hypothesis of a possible delay before first contacts of wild boar to carcasses (i.e. Probst et al. 2017) is a relevant uncertainty and the "time gain" does make adequate carcass removal more feasible.
- The investigated measures are inadequate for different populations with higher aggregation of wild boar, greater social groups or increased average number of animals per sq km because more individuals remain after treatment and ASF appears to have a frequency-dependent resistance to local fade-out.
- The use of uniformly distributed random habitat quality underestimates the performance of the measures. Detailed analysis using simulations on true landscapes with multiple habitat predictors would improve the understanding.
- Many assumptions are included, reflecting ad hoc expert discussions and preliminary laboratory insights regarding ASF transmission, perpetuation and maintenance by the wild boar host system found in central Europe. These include the role of carcasses as a reservoir, contact to dead animals, delay in contact, MAB in piglets from seropositive sows, artificial feeding, stringent and consistent application of measures, and exclusion of human-made transmissions.

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Annex A – ODD Model Documentation

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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).

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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

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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 model landscape represents an area of approx. 200 km × 200 km along the border between Estonia and Latvia (see Figure 1 in the main paper). The local breeding capacity CC_{ij} of each cell is 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} = 1.3455 * density_estimate [heads/km²] following the regression density_estimate = f(CC_{ij}). Non-integer values are randomly assigned to the adjacent integer values according to

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

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

Each cell is connected to eight neighbouring units (Moore neighbourhood). One boar group is released to each habitat cell, where initial group size is six times breeding capacity. Initial age distributions were taken from the results of a 100-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 2 in section “Parameters”.

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A.1.1.1.1. Release of infection

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

A.1.1.1.2. Transmission of infection

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

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

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

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

Carcass transmission: The mode refers to wild boar carcasses 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 indirect mechanisms are considered relevant. Transmission through carcasses is modelled stochastically. Parameter $P_{inf}^{(c)}$ determines the probability of contracting the infection from an infectious carcass during one week. For each susceptible animal, the probability of becoming infected accumulates over accessible carcasses

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

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

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

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

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

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

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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.

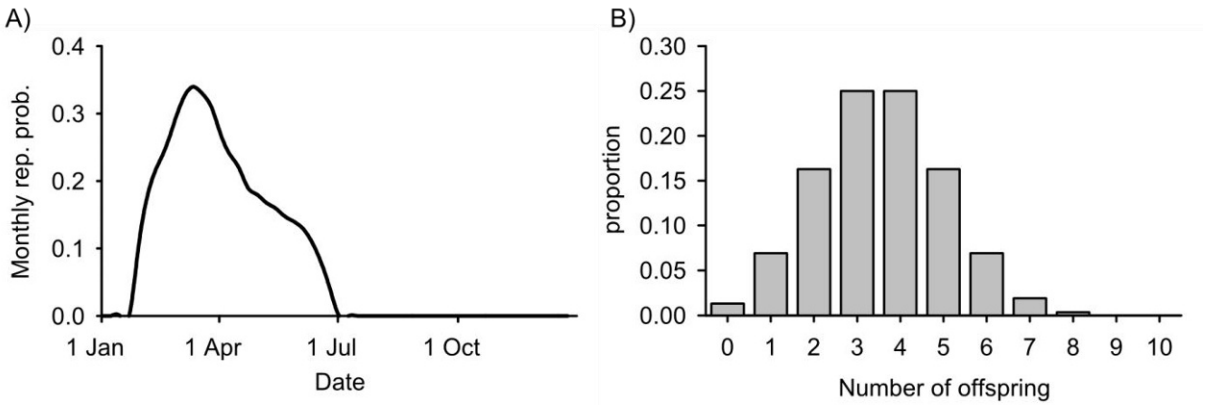

Figure 1 A) Monthly reproduction probabilities for wild boar. B) Breed count distributions for wild boar (Bieber & Ruf 2005, EFSA 2012).

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

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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:

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

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).

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

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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 2.

Table 2: 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)
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	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)

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Name	Description	Value	Source / details
$t_{\inf}$	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).
β_{carc}	Infection probability per carcass (including contact and transmission)	knowledge gap	Determined using spatial-temporal data of observed spread in wild boar
p_{core}	Probability of virus-induced death in core area	knowledge gap	Determined using spatial-temporal data of observed spread in wild boar
d)	Secondary disease course parameters (not relevant for 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)

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