Modelling advanced knowledge of African swine fever, resulting surveillance patterns at the population level and impact on reliable exit strategy definition

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Abstract

African swine fever (ASF) is an infectious lethal disease affecting domestic pigs and wild boar. For more than a decade ASF infection by genotype II has been circulating in Eurasian wild boar populations. It can be transmitted via direct animal contact or contact with contaminated carcasses in the environment. After several years of virus circulation in European wild boar population ongoing disease surveillance suggests regional fade-out of the infection. With this study an exit strategy based on routine surveillance procedures had to be informed and tested using an eco-epidemiological spatially explicit individual-based transmission model. The model simulations were performed on the geographical wild boar habitat map of Estonia and analysis referred to the administrative unit level of 2.500 km² on average (LAU1 units). The analysis addressed the temporal profile of virus- and sero-positive animals in different age cohorts and the abundance of carcasses of animals succumbed to the disease in conjunction with regional virus-fade out. Alternative scenarios were tested for their impact on the duration of virus circulation in a limited area. Finally, different criteria to decide on the final status ASF circulation in a region were tested for their reliability by mimicking routine volumes of passive and active surveillance. The temporal profiles confirm the limited chance of a direct demonstration of virus absence. Therefore, the exit decision requires monitoring periods of several months to years. In order to keep efforts down a two-phase exit protocol does combine a longer phase with routine surveillance (the Screening phase) and a shorter (minimal) phase (the Confirmation phase) with increased surveillance (the maximum possible under field conditions). The two-phase exit strategy facilitates trade-off between invested surveillance efforts and total time to the final decision. Sensitivity of the exit decision is tested for uncertain aspects of ASF epidemiology and revealed performance decrease with increasing natural mortality, but improvement with increasing wild boar density and more distant translocations e.g. due to human activity. Survivors with extreme long-term infectiousness (years), if they would exist, do render the tested exit decision as unreliable. However, there is no evidence in support of such an assumption. Virus attenuation in terms of increased proportion of survivors (up to 20% infected animals) and longer infectious periods (up to 4 weeks post infection) was congruent with the proposed exit strategy. The input to an exit decision by active surveillance i.e. testing of hunted animals, in particular for serology, was negligible.

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1. Introduction

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

African Swine Fever (ASF) is an infectious lethal disease affecting domestic pigs and wild boar. It can be transmitted via direct animal contact or via dissemination of contaminated food or equipment. ASF has serious economic implications for the pig meat and related sectors, including indirect costs related to trade restrictions. The persistence of the disease in wild boar and the limited number of control measures available represents a challenge for the whole EU agricultural sector, in particular the pig farming industry. There is no vaccine or cure despite active ongoing research.

The latest EFSA report stated that ‘in areas where ASF has been present in the wild boar population for more than 1 year, active surveillance is the suited approach to monitor the effect of interventions on the prevalence of infected animals and building evidence to regain ASF-free status’. To explore the options for an ‘exit strategy’, the European Commission (EC) mandated EFSA to evaluate all the necessary elements and specific measurements of prevalence (through active and passive surveillance). Through this procurement, EFSA seeks assistance for these tasks, by developing or modifying an existing (simulation) model, replicating the spread of ASF in wild boar populations in Estonia or/and Latvia where the model predicts the efficiency of the existing surveillance strategy to detect potential circulation of ASFV in affected countries/areas, taking into account the specific ecological and epidemiological conditions.

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

Does the ASFV infection circulate in wild boar population although one does not see it in the routine surveillance output? Models (Gervasi et al., 2020) and field data (Schulz et al. 2020) show that there is a window of uncertainty when a country has to be declared free after ASF circulation in wild boar. Can the length of this window reasonably be estimated, i.e. the necessary duration of a monitoring period of virus search be determined, to remove this uncertainty about actual freedom? Naturally acquired antibodies persist lifelong. The time needed to declare a country free using hunted animals will be determined by the life span of seropositive animals. Could the dynamics of decline in sero-prevalence be interpreted for an exit decision? Or, following the young stock window approach to confirm freedom from certain livestock diseases, could missing detection of sero-positive sub-adults be interpreted in terms of fade-out of virus circulation? How could these different sources of information be combined to construct the most reliable and robust criteria list for an exit strategy based on field surveillance?

The background problem is cut into the following three specific objectives:

- Understanding the temporal profile of a wild boar population with ASFV in terms of population numbers, virological and serological positive animals and number of carcasses present at any time after virus introduction into a region, and around the time-point of fade-out from the region.

- Evaluation of ecological and epidemiological mechanisms not yet considered by ASFV modelling (EFSA 2018 resp. Lange et al. 2018), which potentially could prolong the time of virus circulation in a region.

- Assessing the performance of proposed approaches for an exit protocol within a wild boar region with simulated ASFV circulation and fade-out.

In this report, technical details of methodology and the structured output of the simulations including uncertainty discussion is provided. The methodology and assessment sections of this report are structured according to the three objectives. The interpretation boxes address issues, conclusions and limitations of the study.

The report details the research activities of the NP/EFSA/ALPHA/2020/09 addressing these objectives and contributing to the EFSA output on an ASF exit approach (EFSA 2021).
2. Data and Methodologies

2.1. Data

The wild boar habitat model by Pittiglio et al. (2018) was converted into the breeding capacity raster according to parameter BreedingCapacity [per cell] = 1.28 * Density [per km²].

Events of human-mediated ASF translocations were applied according to Lange et al. (2018) and base on ADNS notification data as of October 2018 provided by EFSA.

Diagnostic outcome of testing wild boar samples is dependent on day post infection and was provided by EFSA SWG on ASF (S. Blome, FLI Germany; pers. comm.).

Table 1: Time since infection and interpretation of diagnostic results in shot wild boar (after Blome et al 2020)

| Time since infection | Diagnostic outcome | Infection status in model |
|----------------------|--------------------|---------------------------|
| Week 1 (3-10 dpi)    | Virus positive, PCR+ and sero-negative | Infectious |
| Week 2-8 (10-60 dpi) | Virus positive, PCR+ and **sero-positive** | Not infectious and immune* |
| Week 9-14 (60-100 dpi) | Virus negative, PCR+ and sero positive | Not infectious and immune |
| From week 15 (>100 dpi) | Virus negative, PCR- and sero positive | Not infectious and immune |

*: In the scenario 'prolonged infectiousness of survivors' the status is infectious in week 2-4 followed by recovery.

Virus titre starts at about 10^3 and declines till day 30dpi (titre 20dpi still 1/3 above cut-off, 42dpi not titratable). Infectiousness ends between day 21 and day 35 post infection. From day 28 there were only minority reports.

Implementation: surviving animals will follow the above principles regarding diagnostic result and a prolonged infectious period for surviving animals will be tested as alternative scenario.

2.2. Methodologies

2.2.1. Model

The reported study is based on an established spatially explicit eco-epidemiological model of ASF transmission and control in wild boar (http://ecoepi.eu/ASFWB). The model was designed to support evaluation of landscape related spread of ASF infection in wild boar populations in different geographical contexts and to support evaluation of control measures in great detail. The model comprises a wild boar ecology module and a disease and transmission module. The module representing wild boar ecology was validated independently of ASF problems in terms of 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 time of infection circulation if the modes of infection and transmission are conceptually understood (EFSA, 2012; Dhollander et al. 2016).

Lange et al. (2018) provides details of the model and demonstrates usefulness in investigating spread and control of ASF in wild boar. The associated model documentation (ODD following Grimm et al. 2006 Grimm et al. 2010) is available from http://ecoepi.eu/ASFWB. The updated ODD protocol is attached as Appendix A.

The model is spatio-temporally explicit, individual-based and operationalised 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 mechanisms 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 or habitat models (e.g., Pittiglio et al 2018). Model population emerges from birth and death probabilities depending on habitat quality on the level of individual social groups.

2.2.1.1. Transmission model of ASF infections in wild boar

The basic principle of transmission relates to the number of adjacent/in contact animals and carcasses using event probabilities, i.e. each infectious object provides a chance of transmission to every susceptible animal sufficiently close.

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). The conceptual framework of multi-modus transmission was established during past usage of the model (Kramer-Schadt et al. 2009; Lange & Thulke 2017; Lange et al. 2018) and recently validated by an ecological study of contact frequency within and between social groups (Podgorski et al 2017). Details of the modes of transmission related to ASFV were studied also by Pepin et al. (2020).

Parameterisation of the modes of transmission is based on multiple sources. Quantitative experimental data is accessible for within-group transmission, i.e., animals in permanent contact with groupmates (transmission trials; see review in EFSA 2021). Between group transmission was parameterised relative to the within group transmission and reversely calibrated against the speed of propagation (Lange et al. 2018). Evidence regarding the role of carcasses of animals dying consequent to ASF infection is very experimental, including the potentially contaminated soil thereunder. Given the assumption that carcass-mediated transmission is relevant, insights exist on the likely frequencies of carcass-based transmission (Probst et al. 2017). Based on the reverse parameterisation procedure by Lange & Thulke (2017), ubiquitous access to dead animals (i.e. not hiding or retreating due to morbidity) but very seldom actual contacts that may warrant transmission (blood, secrets or body fluids) is modelled.

2.2.2. Simulation protocol

The simulations were performed on real habitat geography for wild boar in Estonia. Habitat map is derived from the habitat model according to Pittiglio et al (2018). The landscape is further calibrated to the reported overall population density. Hence, the local abundance is derived from geography and the total population calibrated with the reported average density of wild boar in the simulated area (e.g., Estonia). Further, the simulation tracks administrative sub regions according to LAU1 level (see Fig. 1).
Figure 1. Wild boar habitat geography following Pittiglio et al. 2018) and calibrated to the overall population density estimate of wild boar in Estonia prior to ASF incursion. The darker a grid cell is coloured the more wild boar would be sustained within the location due to assumed habitat quality i.e., feed resources. Administrative sub-regions (LAU 1) are delineated in red and numbered for further reference. The total area covered is about 45 thousand km² and sub-regions having an area of about 2.5 thousand km² varying from 1.000 to 5.000 km². Source: OpenStreetMap contributors.

The infection was released according to the early notifications in 2014 and continued spread simulated. Two different simulations protocols were applied either ignoring or considering the human-mediated translocations of the infection identified from notification data (Lange et al. 2018). The simulations ignoring human-mediated translocations simulate the stochastic spread only by wild boar contacts with infectious live animals or carcasses. The simulations considering human-mediated translocation additionally seed infections at time and location of translocation events identified from notification data.

2.2.3. Analysis of model output

Model analysis was performed using 100 runs per simulation scenario.

Evaluation focused either the whole simulation area (i.e., Estonia), or 13 individual LAU1 units (two LAU1 units of Estonian islands were excluded, number 6 & 14 in Figure 1).

Model runs were recorded in full to facilitate different analyses (proposed alternative exit protocols). Fade-out of infection was determined for the whole simulation area and for each individual LAU1 unit. “Extinct runs” count simulations with global fade-off, i.e. after 25 years of simulation no more infectious animals or carcasses from animals succumbed to ASF (aka infectious objects) were present. Fade-out events on LAU1 unit level were recorded if the last infectious object disappeared followed by three years without any ASF infection in the respective LAU1 unit. Hence, during a complete simulation an individual LAU1 unit could have multiple introduction and fade-out events.

In order to derive population and diagnostic profiles over time the model output data per unit of analysis was standardised in time to first infected animal and subsequently to the time point when last the infectious object disappeared (i.e., all infectious animals died and all carcasses from individuals that succumbed to the infection are decomposed).

Model output was analysed for different strata, i.e. infectious animals, carcasses from animals succumbed to ASF, virus and PCR positive, sero-positive, sub-adult animals (6-24 months), adult animals (older than 24 months), hunted animals and carcasses collected.
2.2.4. Duration of virus circulation (aka persistence)

The model was assessed for several alternative mechanisms hypothesised to affect the duration of virus circulation within a given wild boar population. It was investigated how long the infection would circulate in Estonian wild boar when simulating the model used in previous EFSA output (see EFSA 2018 and Lange et al. 2018), hereafter referred as standard model. Then the model was amended to include alternative mechanisms proposed to prolong the duration of virus circulation by EFSA (2021). With the amended model versions spread simulations were repeated on the same wild boar habitat map. This approach enables comparative analysis without and with the proposed mechanism regarding whether the duration of ASF circulation is actually prolonged, and if so, whether and how the temporal profiles of population and diagnostics change.

2.2.4.1. Model amendments

- Scenario 1a: Variation of the proportion of animals surviving the infection (aka "attenuation"). It was assumed that the lethality of the infection may be lower. Thus, more animals are surviving the infection. Hence, a greater reproductive potential after an ASF outbreak would increase population recovery and thereby prolong time of virus circulation. In order to address the issue, the proportion of animals surviving the infection was amended in the model.

  Proportion surviving to immune = [5%; 10%; 20%] of infected animals

- Scenario 1b: Variation of the infectious period of animals surviving the infection. Recent experiments evidenced virus genome detection in surviving animals beyond the short infectious period reported for succumbing animals. Subsequent to virus isolation until 10dpi, relevant PCR titres are reported till 28dpi with strong decline in-between (Table 1, S. Blome pers. comm.). In order to address the issue, the infectious period was extended to four weeks in combination with scenario 1a.

  Duration of infectiousness in surviving animals = [1 week; 4 weeks] after infection

- Scenario 2: Variation of the durability of protection by acquired immunity. There is concern that acquired natural immunity in ASF surviving wild boar may not protect the animal for life time (EFSA 2021). Therefore, in this scenario the protection by immunity is limited.

  Immunity loss = [never; on average after 1y (0.5y; 1.5y)]

- Scenario 3: Variation of time of protective immunity due to maternally acquired antibodies (MAB). Three variations were simulated assuming alternative periods of protection by respective detection of MAB in piglets.

  (MAB-immunity, MAB-presence) = [(0, 0); (7, 9); (12, 15)] in weeks.

- Scenario 4: Long-term infectious survivors of ASF (aka carrier animals). The discussion on existence and epidemiological relevance of animals which do not succumb to ASF but develop an extremely long infectious period was addressed by hypothetically assuming life-long infectiousness for a proportion of infected animals. Those animals would have succumbed to the infection after one week in the standard model. In the amended model those animals are assigned with lifetime infectiousness till they die from natural mortality.

  Proportion = [0%; 0.1%; 1%] of animals normally would succumb to the infection

The animal finishes its normal live cycle but as infectious animal (i.e., no extra lethality by the status of life-long infectiousness – worst case assumption) and additional to the 5% surviving animals that recover to immunity.

2.2.5. Performance test of exit strategy concepts

Exit strategy protocol were developed by the EFSA SWG on ASF in terms of sampling effort in the hunted and carcass cohort (active and passive surveillance branch), and relative to diagnostic signals like time of last virus detection (PCR+ animal) or last serological finding in sub-adult sample stratum. The overall
performance of proposed exit strategies was tested on repeated simulations of stochastic ASF spread in the simulation area. The sampling strategy and criteria defining a proposed exit strategy were applied from the beginning of the simulations and continuously evaluated. If the simulated surveillance did satisfy the proposed exit strategy criteria, meaning the tested area would be declared as free from ASF in wild boar, the decision (sample-based knowledge) was compared with the true status in the model population (perfect knowledge). The evaluation was based on a factorial design for the lengths of the two different monitoring phases. One deliberately long phase with routine efforts (screening, 4-24 months) and a second with shorter duration and maximum intensive search efforts (confirmation, 0-14 months). The performance of the investigated exit strategy was measured as the proportion of correct exit decisions. For every decision, the time between true virus fade-out and exit decision was measured and the actually achieved sample size recorded.

2.2.5.1. Simulation protocol of evaluation of exit protocols

Table 2 details the simulation experiments with regards to exit protocols (Exit strategy I & II).

Table 2: Specification of the exit strategy protocols tested with the model. Simulated surveillance efforts are given for the Screening and the Confirmation phases of the tested exit protocol (Exit strategy I and II). Moreover, tested alternatives are provided. These variants were simulated to understand the possible impact on the performance of the exit protocol.

| Surveillance efforts applied with | Exit strategy I                                  | Exit strategy II                                  |
|--------------------------------|-------------------------------------------------|-------------------------------------------------|
| Screening Phase                |                                                 |                                                 |
| Passive surveillance           | Carcass collection                              |                                                |
| Requested outcome              | 2 % of hunting bag per LAU 1 prior to ASF       | 1 per 1,000km² of area surface per year         |
| Tested alternatives            | All samples PCR negative                        | All samples PCR negative                        |
| Active surveillance:           | Hunting effort for sampling                     | All hunting harvest tested                      |
| Requested outcome              | 100 WB hunted per LAU1 unit                     |                                                |
| Tested alternatives            | 60 or 0 WB hunted per LAU1 unit                 | All subadults sero-negative                    |
| Confirmation Phase             |                                                 |                                                 |
| Passive surveillance           | Carcass collection                              |                                                |
| Requested outcome              | 2 % of hunting bag per LAU 1 unit prior to ASF  | 2 per 1,000km² of area surface per year         |
| Tested alternatives            | All samples PCR negative                        | All samples PCR negative                        |
| Active surveillance            | Hunting effort for sampling                     | All hunting harvest tested                      |
| Requested outcome              | 100 WB hunted per LAU1 unit                     |                                                |
| Tested alternatives            | 60 or 0 WB hunted per LAU1 unit                 | All subadults sero-negative                    |

Alternative scenarios
- Annual mortality (ODD Table 3) split by 80%/20% instead of 90%/10% between hunting harvest & natural mortality.
- Density prior to ASF doubled by increasing the breeding capacity of habitat landscape.
- Exclusion of human-mediated translocation events during 2014-2018 (EFSA 2018).
- Assume one out of hundred infections results in a long-term (natural life cycle finished) infectious animal.
2.2.5.2. Model implementation of routine surveillance protocols

Calculations per simulation run are performed individually for each administrative unit.

Active surveillance - hunting volume

The weekly hunting target is calculated from the number of annually targeted samples divided by 52 (non-integer value).

Weekly target values are accumulated over simulation steps and every week discounted by the number of samples taken from the available hunting bag of the current week. If more hunted animals are available than necessary, the required number of samples is selected randomly from the available hunting bag. At the end of the year, the target is reset to zero, no matter if it was satisfied in the past year.

Passive surveillance – carcasses numbers

Approach I: The average annual hunting bag prior to virus introduction to Estonia is recorded for every LAU1 unit over 3.5 years of simulations immediately before introduction. 2% of this hunting bag sets the annual target of carcass collection for surveillance. Corresponding weekly target values calculated by dividing the annual target by 52 (non-integer value).

Weekly target values are accumulated over simulation steps and every week discounted by the number of carcasses actually sampled. All carcasses available in a week are collected. If more carcasses than required were available, the required number is selected randomly among those. At the end of each calendar year, the carcass target is reset to zero, no matter if it was satisfied in the past year.

Approach II: Based on preliminary simulations the expected number of animals removed from the population over time was estimated. According to literature these animals were split into a portion representing the hunting harvest and a proportion representing natural mortality other than ASF (e.g., 90% vs 10% following Focardi et al. 1996). Animals dying either from natural mortality or virus induced death represent the carcasses distributed in the landscape at a given time considering the duration of decomposition.

The number of animals dead not from disease were the reference source from which a required number of carcasses (0, 1, 2, 6) needs to be sampled per 1,000 km² and year; and hence the necessary individual carcass detection probability can be calculated (see Table 3 below). Figure 30 below, shows that from the individual sampling probability the emerging number of actually sampled carcasses per year and 1000 km² fits the target. Intentionally, excess mortality due to disease (if the virus did not yet fade-out) increases the number sampled and therefore also the chance to detect ASFV positive carcasses.

To realize unbiased carcass sampling the process need to integrate the duration of carcass decomposition. To achieve this, all individual carcasses are reconstructed from simulation outputs (i.e., weekly deaths due to baseline and virus-induced mortality) with their seasonal duration of decomposition. Then for each carcass, its weekly detection probability \( p_w \) is calculated from the given carcass detection probability \( p \) corresponding to the surveillance target, and the time \( t \) (in weeks) the carcass is decomposing:

\[
p_w = 1 - (1 - p)^{\frac{1}{t}}
\]

Iterating over the weeks of carcass sampling, each of the carcasses available in that week is randomly selected as sample with its weekly detection probability \( p_w \). If selected the carcass is removed from the list of next week’s available carcasses.
3. Results

The exit protocol needs to be built on detailed understanding of the dynamics of ASF in wild boar towards a potential end of an epidemic in an area. First, there is a need to understand the temporal course of diagnostic and epidemiological characteristics for affected population. These are total number of animals over time, the virological and serological status of the population as well as the number of carcasses distributed (which subsequently may be detected; Section 3.1). Second, the expected time horizon the virus would circulate in a certain area is important for an exit strategy but may be influenced by recently discussed knowledge on ASF in wild boar. Therefore, the impact in time of circulation was tested with the model for mechanisms identified from a narrative literature survey (Section 3.2). Finally, conceptually designed protocols confirming extinction of the ASF infection in an area were tested for their reliability within the simulation model (Section 3.3).

3.1. ASF spread simulated in geographical landscapes

The ASFV infection is introduced in the wild boar population of Estonia and detailed quantities recorded from the model runs. The resulting patterns provide understanding how well the model reflects several descriptors recorded in the field. Instead of imprinting to the model, temporal patterns of population dynamics and diagnostic status emerge through simulation of model rules of individual life-histories and host-to-host and carcass-to-host transmission.

3.1.1. Population dynamics

Figure 2 A-B. Temporal dynamics of the model population. ASF infection was introduced in year 5. A: Summary of 100 runs with weekly values as data points. B: Hundred individual runs with annual average as data points.

Figure 2 shows the total counts of the simulated population of Estonia (without islands) over a duration of 25 years. Annual fluctuation results from the short mean life expectancy i.e., high chance to die from hunting or natural mortality and the enormous reproductive capacity of the population. The pre-ASF population estimate from hunters’ data for Estonia ranged between 35-40 thousand animals which aligns with the starting situation in the model simulation (year 0 to 5). Along with the spatial spread of ASF infections (expansion of the affected area) and due to the high case-fatality (95%) the total simulated population collapsed by up to 60% to 90% (36 to 12 thousand in the central tendency – black line). For comparison, 4820 animals were hunted during 2019/2020 hunting season (~0.12 WB/km² hunting ground including ASF free islands). According to the Environmental Agency the wild boar density estimate by the end of winter 2020 was 0.13-0.16 wild boar per km². Hunters’ prediction for 2020/2021 was an increase of population by 35%. The footprint index actually shows almost two times increase in winter 2020 compared to 2019 (from 0.18 to 0.34) (Estonian Environment Agency, 2020). In the model
simulations subsequent recovery rebuilt the population with comparable dynamics but apparently not yet finished within ten years.

Figure 3. Estimated population density of the Eastern and Western half of Estonia. One early (E 2014/15) and one later affected (W 2016/2017) by the spreading infection. The figure confirms the simulated population collapse within 2-4 years after introduction and the reference density between 0.5 and 1 animal per km² of wild boar habitat (Schulz et al. 2019). NOTE: The data was not used to calibrate the emergent model dynamics.

The following paragraphs address temporal characteristics of ASF spread through Estonia. The resulting temporal profiles are based on 100 replicate runs of the standard model parameterisation (EFSA 2018; Lange et al 2018) for 20 years after infection introduction i.e., corresponding to the time horizon 2014-2034.

3.1.2. Epidemic curve

First the epidemic curve is shown in absolute numbers i.e., the number of carcasses accumulated in the simulation landscape respecting decomposition, the number of virus positive animals, the number of serologically positive animals by age cohort, and the number of current deaths due to ASF infection relative to all deaths per week. Prevalence profiles are detailed in following section.

All data are shown per LAU1 unit i.e., 13 LAU1 units comprise the whole territory of Estonia without islands. Data are standardised on the day of first ASF positive animal within the respective LAU1 unit (time 0.0 on the x-axis) and variability is shown by week-wise percentiles over 100 simulation, times 13 LAU1 units (excluding islands). The actual number of LAU1-level data series is specified with each diagram. Theoretically, 1300 series are expected i.e., 13 LAU1 units times 100 runs. However, the number is modulated by (i) runs that did not start due to very early extinction of the infection, (ii) runs in which ASF never entered a certain LAU1 units and (iii) LAU1 units may undergo two independent reintroduction events with minimum three years break between first fade-off and second introduction. Summary diagrams are plotted for all 13 LAU1 units (in total about 38.000 km² hunting grounds surface). Additionally, the 100 runs per two individual LAU1 units are shown (unit 1 and 4 in Figure 1) average size 3.000 km² and about 2.000-3.000 animals pre-ASF.
Figure 4 A-C. Epidemic curve in terms of carcasses from infected wild boars distributed in the simulation area: 13 LAU1 units of Estonia (exclusive islands), B: example LAU1 unit 1; C: example LAU1 unit 4.

The number of carcasses rises steeply reaching a first peak already during the first year (Figure 4). In line with field observations, the model reproduces the early increase and the usual continuous decline following the maximum cases. The pattern is compatible with a rising epidemic within a finite landscape. There is annual peak in number of carcasses present which is triggered by the seasonal temperature curve causing slower or faster decomposition of the carcasses depending on the season (see ODD for the distribution of decomposition time).

The contribution of ASF to the deaths occurring per week in the model is explicitly read from the simulations and shown with Figure 5. Noteworthily, the excess mortality due to ASF raises early in the epidemic but ceases very quickly (e.g., after 1.5 years in the Estonian landscape configuration).

Figure 5. Epidemic curve in terms of percentage of weekly deaths which are due to ASF infection across all 13 LAU1 units of Estonia (exclusive islands), N=1389.

The excess mortality due to infection translates in a drastic population decline of more than 50% (Figure 6). The variation in population size is contributed by stochasticity of the model runs but less due to differences in LAU1 (compare Figure 6A all units with 6B or 6C single unit). The median population decline following ASF circulation in a LAU1 unit was about 60% (2,400 to 800 Figure 6A) and did plateau about 2.5 to 3 years after introduction of the infection in a LAU1 unit.
Figure 6 A-C. Epidemic curve in terms of weekly population abundance of: A: 13 LAU1 units of Estonia (exclusive islands), B: example LAU1 unit 1; C: example LAU1 unit 4. The resulting collapse of the population in the simulated LAU1 units is due to the excess death caused by ASF infection. NOTE: Different to Figure 2A here all data series are adjusted to the day of first virus positive animal per LAU1 unit. Therefore, the annual population cycle is no longer visible (although still emergent from the model and visible if series are adjusted to a fixed day in the calendar year as in Figure 2).

The number of infectious animals present in the simulation area rises steeply reaching an initial peak during the first year post introduction (Figure 7). The model output is in agreement with field observations, of an early increase and usually continuous decline following the maximum of cases. The pattern is compatible with a rising epidemic in a finite landscape. The number of infectious wild boar reaches its climax earlier than the peak in carcasses. The reason is that alive infected animals do not accumulate in the landscape due to the short infectious period (on average 1 week, i.e., 4 to 10 dpi) compared to carcass presence after dead (4 to 12 weeks dependent on season).

Figure 7 A-C. Epidemic curve in terms of (alive) infectious wild boars present in the simulation area A: all 13 LAU1 units of Estonia (exclusive islands), B: example LAU1 unit 1; C: example LAU1 unit 4.
For comparison the next figure resembles the PCR positive animals (virus genome detectable up to 60-100 dpi) from the same simulations (see Table 2).

![Figure 8 A-C. Epidemic curve in terms of PCR-positive wild boars present in the simulation area A: all 13 LAU1 units of Estonia (exclusive islands), B: example LAU1 unit 1; C: example LAU1 unit 4. NOTE: Consider the changed y-axis scaling. The PCR positive testing increases the number of animals by about 50% compared to those deemed infectious.](image)

Final temporal diagnostic profile refers to the serologically positive animals in the simulated wild boar population. The number of seropositive animals present in the simulation area rises continuously reaching a plateauing peak after about two years post introduction (Figure 9).

![Figure 9 A-C. Epidemic curve in terms of seropositive wild boars present in the simulation area A: all 13 LAU1 units of Estonia (exclusive islands, N=1389), B: example LAU1 unit 1; C: example LAU1 unit 4.](image)

### 3.1.3. Prevalence of virus positive and seropositive wild boar

The purpose of this section is to accompany the above considerations in terms of numbers by the relative view of prevalence. The discussions of the model output revealed the need for understanding...
the absolute as well the relative dynamics. To support the comparison either view is addressed by dedicated chapters.

Prevalence values were calculated every week using the actual population number as denominator i.e., all animals alive at the moment of calculation. Therefore, the relative output data resembles the source of sample-based surveillance data over time intervals.

The maximal virus prevalence of 1.5% is approached around 1.5 years after introduction of the infection in the LAU1 unit in the median of all simulations (75-percentile 3%; 95-percentile 6%; cf. Figure 10A below for infectious animals.

The prevalence of PCR+ animals (Figure 10B) is about one third higher than that of infectious animals (median peak about 1%, 75-percentile close to 3%, 95-percentile 6%). This effect is due to the experience from infection experiments which proposes longer PCR+ detectability in recovering animals with virus titres already below those associated with excretions of sufficient infectious dose.

The sero-prevalence profiles reveal the typical difference between the “long-term sero-positive” adult age-cohort (Figure 11A) and the “freshly sero-positive” sub-adults (Figure 11 B).

The sero-prevalence profile of the sub-adult cohort reacts slower to the rising infection, remains substantially lower and shows a steeper decline compared to the adult sero-prevalence profile. The reason for the first observation is the continuous influx of sero-negative animals by annual birth. The other two observations relate to the time surviving animals can stay in their cohort which is less than 18 months for sub-adults and until death for adults. The natural turn-over of the sub-adult cohort with sero-negative animals results in a rapid decline of sero-prevalence in the young animals over time (principle of the young-stock window). In adult animals, the sero-positives remain in the cohort until the end of their life, contributing to a slower decline in overall sero-prevalence.
3.1.4. Possible fade-out of virus circulation in wild boar

The last aspect of the general characterisation of an ASF affected wild boar population addresses the eventual fade-out of the infection in a finite landscape. The time horizon of possible fade-out of infection from the simulation area was investigated to enable comparative evaluation of time of virus circulation (persistence) if other mechanisms are introduced in the model that were suggested to prolong the duration of virus circulation. Obviously, the exact time between introduction of the infection and the last infectious object being resolved, varies substantially. Over all simulation runs the infection circulated between five and more than 20 years (Figure 12A). Hence, the mean or average view is not helpful.

Figure 11 A-B. Temporal sero-prevalence profile of A: adult animals (older than 24 months) and B: sub-adult animals (6-24 months) on the LAU1 unit level. Data are standardised between units to the week of first ASF incursion into the unit (t=0). Weekly prevalence was calculated given the current population size. The data series summarise 100 runs and 13 LAU1 units. Region: 13 LAU1 regions of Estonia (exclusive islands), N=1389.

Figure 12 A-B. A: Temporal dynamics and point-wise variation of infectious objects in simulated wild boar populations of Estonia i.e., live animals within the infectious period plus carcasses of animals succumbed to disease, between introduction (year 4) and fade-out (variable between year 8 and 25; 10% of the runs did not fade out in the horizon of the simulation). B: Duration of circulation of ASFV infection in simulated wild boar populations of Estonia. Since time of introduction of the infection in the simulation area (t=0, x-axis) the graph shows the percentage of simulated outbreaks out of 600 runs (y-axis) which still contained infectious objects (either infectious animals or carcasses from animals that succumbed to ASF). Region: Estonia (exclusive islands).
The (apparent) end of virus circulation in regional wild boar populations was observed in different parts of the Baltic MS including suspicion of country wide ASF extinction in Estonia prior to summer 2020. The observed quasi fade-off of the infection from Estonia might be supported by the particular geographical layout. Estonia was first affected at the southern border, followed by 4-5 years of regional spread towards north, where the Baltic Sea provides most of the border line.

The model simulations of ASF on the wild boar habitat geography of Estonia resulted in virus fade-out starting about 5 years after introduction (~2019) and reaching 50% chance 7 years after introduction (Figure 12B). Finally, in 5% of the simulations infection did still circulate after 20 years following introduction.

NB: Longer-lasting virus material in the soil or elsewhere in the environment was assumed to not result in new infections of live animals. Although representation of purely environmental infections is technically possible with the model (see Dhollander et al. 2016; Lange et al. 2016), the evidence of infections causally attributable to infectious material for long after the carcass is fully decomposed does not exist. Hence, assuming a prolonged infectiousness of carcasses after the decomposition period, already varying in length by season, would be completely speculative. This would be as reliable as assuming infectious meat in the fridge of hunters that later is swill-fed to animals.

3.2. Impact of proposed mechanisms on the duration of circulation of infection in a finite area (Persistence)

The following four sub-sections (3.2.1-3.2.4) address estimation of time of circulating ASF infection. However, here the patterns are compared between different model versions/scenarios developed to reflect proposed mechanisms (EFSA 2021, chapter 4.3 & 4.4) discussed, e.g., in literature, as impactful on the time the infection is circulating in a given region (aka persistence). It is well acknowledged that the time of virus circulation depends on a lot of regional specifications like habitat structure, location of introduction, larger epidemiological constellation, connectedness of the meta-population and reproductive performance. Therefore, the useful experimental design has to elucidate the impact on time of virus circulation per mechanism relative to the standard model without the particular mechanism. Starting from the survival curve data for the original model (see Fig 3.2.1.4.i B), the following figures highlight the quality of changed virus circulation when a particular mechanism is introduced in the model and after rerunning the simulations.

3.2.1. Scenario 1: Reduced case fatality rate + prolonged infectious period of surviving animals

The scenario addresses uncertainty regarding lower case fatality rate (aka attenuation). Figure 13 shows the effect of reduced case fatality from 95% [Reference] of all infected animals to 90% respective 80% on the time interval with circulating infection (Figure 13A). Additionally, the same effect is shown assuming a prolonged infectious period of succumbing animals of 4 weeks (Figure 13B) instead of 1 week [Reference] (Figure 13A).

The scenario reflecting uncertain proportion of animals that survive an infection after about one week of infectiousness did alter the duration of virus circulation only marginally and the fade-out graphs did not differ substantially (Figure 13A). Within the investigated range, i.e., fourfold chance of infected animals to survive to immunity, the time horizon of circulating infections does not change qualitatively. The fade-out after 20 years seems not affected.

The scenario with additionally prolonged infectious period (Figure 13B) reveals a recognised change to the fade-out graph if simulated in conjunction with 20% surviving animals. The temporal diagnostic and population profiles for this scenario need to be investigated to exclude changes in patterns formed around fade-out.
3.2.2. Scenario 2: Immunity loss

The scenario assumes that wild boar surviving the infection and turning immune will not be lifetime protected against infection. While being continuously positive in serological diagnostics contact with the virus will again lead to infection and infectiousness after variable time periods. The duration of protective immunity was varied between 1 year and lifelong. The following graph resembles the two scenarios assuming either no loss of immunity (black line) versus loss of immunity on average after 1 year post infection (blue line) i.e., between 0.5 and 1.5 years.

Figure 14. Fade-out probability by time since introduction of ASFV infection in the wild boar population of Estonia. The simulations compare the standard model of no loss of acquired immunity (black line, \(t(imm)=0\)) with the scenario assuming immunity loss between 0.5 and 1.5 years, mean 1 year after infection (blue line \(t(imm)=52\)). Since time of introduction of the infection in the simulation area (t=0, x-axis), the graph shows at a given value x the proportion of simulations (y-axis) which still contained infectious objects either infectious animals or carcasses from animals succumbed to ASF.
The simulated outbreaks do not reveal different time horizon of circulating infection if surviving animals lose acquired immunity on average after 1 year. Between the two extreme assumptions, the time horizon of circulating infections does not change (Figure 14).

3.2.3. Scenario 3: Shortened protection by maternal antibodies

The scenario assumes different age of piglets from which onwards maternally acquired immunity is no longer protective respective detectable by antibody diagnostics. The pair of values was varied as (0 weeks, 0 weeks), (7w, 9w), (12w, 15w) [R].

![Figure 15. Fade-out probability by time since introduction of ASFV infection in the wild boar population of Estonia.](image)

The simulations compare the standard model wherein MABs are protective for 12 weeks (red line, \(t(\text{MAB})=12.0\)) and 15 weeks detectable with scenarios assuming shorter durations (blue line 7 of protection \([t(\text{MAB})=7.0]\) and 9 weeks detectable; black line 0 and 0 weeks \([t(\text{MAB})=0]\). Since time of introduction of the infection in the simulation area \((t=0, x\text{-axis})\), the graph shows at a given value \(x\) the proportion of simulations \((y\text{-axis})\) which still contained infectious objects either infectious animals or carcasses from animals succumbed to ASF.

The simulated time horizon of circulating infection is independent of the assumed duration during which piglets are post-natal protected from ASFV infection. The changes of the interval of protection by maternally acquired immunity does not alter the related time horizon of circulating infections (Figure 15).

3.2.4. Scenario 4: Life-long infectious period (aka carriers)

The scenario hypothetically assumes that among all wild boar that succumb to the infection certain proportion survives as infectious animal for prolonged time horizon (i.e., life-long infectious period for few animals, aka carriers). These animals will be continuously positive in virological and serological diagnostics. Contact with susceptible animals will lead to transmission equivalent to the standard transmission process and the carcass is infectious. The proportion of disease courses with lifelong infectiousness was varied and the value covered 0 [R], 0.001, and 0.01.

The simulated outbreaks reveal substantially prolonged time horizon of circulating infection the more infected animals develop life-long infectiousness (carrier status). If per hundred infected animals one develops this disease outcome (Figure 16; red line graph), then the majority of outbreaks (more than 80%) did no longer fade out within the simulation time compared to less than 10% of outbreaks if no infected animal develops into long-term infectiousness (Figure 16).
The simulations compare the standard model without "carrier animals" (black line, \(p(\text{carr})=0.0\)) with scenarios assuming alternative proportions of infections that develop into life-long infectiousness (blue line 1 in 1,000 \(p(\text{carr})=0.001\); red line 1 in 100 \(p(\text{carr})=0.01\) of all infected animals). Since time of introduction of the infection in the simulation area \(t=0\), the graph shows at a given value \(x\) the proportion of simulations \(y\)-axis which still contained infectious objects either infectious animals or carcasses from animals succumbed to ASF.

**Interpretation:** In summary, to inform general criteria for an exit approach based on the temporal population and diagnostic profiles at time without virus findings by routine surveillance two scenarios require consideration. These are low lethality in combination with 4 weeks infectious period (Scenario 1B) and life-long infectiousness (aka carrier, Scenario 4). Both scenarios were discussed in literature (see EFSA 2021 Chapter 4.3 & 4.4) and a corresponding immediate model implementation was developed i.e. lower lethality together with prolonged infectious period in surviving animals, and extreme "carrier concept". In fact, however, the scenarios are related to each other addressing greater proportion of infection outcomes with longer infectious period (Note: the reduction of lethality alone does not create issues (Figure 13A), only if more animals surviving the infection also are infectious longer than 1 week (Figure 13B) fade-out was changed.). The critical scenarios can be compared by the proportion of all infections that result in longer infectious period and the associated length of the period.

| Proportion of all infections | length of infectious period (in weeks) |
|-----------------------------|--------------------------------------|
| Null model                  | 5%                                   | all 1 week                           |
| Sc.1B red line              | 20%                                  | all 4 weeks                          |
| Sc.4 red line               | 0.95%                                | average survival ("natural")        |

Figure 16. Fade-out probability by time since introduction of ASFV infection in the wild boar population of Estonia.
3.3. Population and diagnostic profiles around time of fade-out

The exit criteria refer to the field situation when no virus detection was notified for few times and hence virus fade-out or eradication could be hypothesised. Thus, the problem addresses the potential patterns of temporal diagnostic profiles around the time point when the infection stopped spreading (fade-out). In order to generalise temporal profiles of population and epidemiological status across multiple area units (here 13 LAU1 units of Estonia), we further consider data standardised to the time point of last infectious animal succumbed. The mechanistic model provides full knowledge about all individuals and therefore the correct time of infection fade-off is known. Contrary, field surveillance must decide on potential virus extinction on sample basis. Nevertheless, field surveillance data also can be standardised to the date of last virus detection as in EFSA (2021, chapter 4.1). The simulated (or observed) data patterns suggest possible general and testable criteria to be met by a population (likely) free of ASFV circulation, the exit strategy. The following diagrams show the outcome of simulations of the standard ASF model in terms of temporal profiles of virus-prevalence, sero-prevalence and number of carcasses retrievable, all adjusted to the time point (t=0) of the disappearance of the very last infectious living animal. Additionally, the three features are explored by their temporal profile under the scenarios of prolonged infectiousness both by longer infectious period of increased number of surviving infected animals (Sc.1B) and assumed life-long infectiousness of “carrier animals” (Sc.4).

Profiles are shown as summary distribution of 100 simulation runs and 13 LAU1 units. Per LAU1 unit, multiple events of virus introduction and subsequent fade-out were included as individual data series. Between consecutive series, a minimum of 3 years without infection in the respective LAU1 unit was required. Before aggregation, individual data series are adjusted to the time when the last infectious animal succumbed (t=0). Weekly prevalence was calculated given the current population size. (Note: Different figures below represent the output of the same profile using the standard model. These figures represent an equivalent outcome but are based on independent model simulation. Hence, by comparing different figures for the standard model the stochastic variability of the temporal profiles can be assessed.)

3.3.1. Temporal profile of virus prevalence around fade-out

3.3.1.1. Standard model

Figure 17. Temporal profile of number of infectious animals on the LAU1 unit level plotted for all 13 LAU1 units of Estonia (exclusive islands, A), and individual LAU1 unit 1 (B), and 4 (C, see Figure 1). Individual data series are adjusted to the time when the last infectious animal succumbed (t=0). The data series of A include those of B and C.
The number of infectious animals in simulations with the standard model (Figure 17) illustrates the steep decline in possible virological findings around the virus fade-out. Only about one year before virus fade-out the peak number of infectious animals is passed, and decline starts. As the numbers per LAU1 unit were already rather low i.e., 12 in the median line (4 to 30 in the 50% central interval), the reliable detection of virus positive animals around fade-out quickly renders impractical.

3.3.1.1. **Ad Scenario 1B – Reduced case fatality & prolonged infectious period**

![Figure 18](image1.png)

Figure 18. Temporal profile of the prevalence of infectious animals on the LAU1 unit level plotted for all 13 LAU1 units of Estonia (exclusive islands). From left to right the diagrams show the prevalence for different intensities of scenario 1B i.e., prolonged infectious period for transiently diseased animals (4 instead of 1 week). Left: standard model 5% survivors, middle: 10% survivors, right: 20% survivors.

Figure 18 shows the effect of increasing the proportion of transient infections (Figure 18 left to right) on the prevalence of infectious animals (less than PCR+, Table 1) close to fade-out. The mechanism, which did prolong the duration of the circulation of infection (Figure 13B), has no noteworthy effect of the percentage infectious animals close to fade-out. This implies that the chance to hunt virus positive animals is unchanged, hence, an exit strategy based on the profile of the standard model would also be congruent with scenario 1B.

3.3.1.2. **Ad Scenario 4 - Life-long infectious period (aka carriers)**

![Figure 19](image2.png)

Figure 19. Temporal profile of the prevalence of infectious animals on the LAU1 unit level plotted for all 13 LAU1 units of Estonia (exclusive islands). From left to right the diagrams show the prevalence for different intensities of scenario 4 i.e., long-term infectiousness. Left: standard model, middle: 1 in 1.000 infected animals, right: 1 in hundred infected animals.

The increasing proportion of life-long infectious survivors (animals which do not succumb to the disease; Figure 22, left to right) flattens the curve of prevalence of infectious animals close to fade-out. Hence, the limited detectability of virus positive animals by hunting-based (active) surveillance is shifted away from the point of final virus fade-out (Figure 19, t=0). Therefore, the construction of reliable exit criteria gets more difficult as no pronounced changes around the fade-out occur if many such extreme disease courses would exist. This implies that an exit strategy based on the profile of the standard model would be incongruent with scenario 4.
3.3.2. Temporal profile of sero-prevalence around fade-out

### 3.3.2.1. Standard model

Figure 20. Temporal profile of the number of sero-positive animals on the LAU1 unit level plotted for all 13 LAU1 units of Estonia (exclusive islands, A), and individual LAU1 unit 1 (B), and 4 (C, see Figure 1). Individual data series are adjusted to the time when the last infectious animal succumbed (t=0). The data series of A include those of B and C.

The number of sero-positive subadult animals in simulations with the standard model (Figure 20) illustrates the decline in possible serological findings in subadults around the virus fade-out. Prior to fade-out there are equal few numbers of sero-positive subadults than infectious animals. As the maximum numbers per LAU1 unit were already rather low i.e., 13 in the median line (6 to 25 in the 50% central interval), the reliable detection of sero-positive subadult animals around fade-out is quickly rendered impractical. Nonetheless, the time interval between virus fade-out (t=0) and the disappearance of seropositive subadults is limited to 1 year in median (3/4 to 1.5 years in 50% central interval).

#### 3.3.2.1. Ad Scenario 1B – Reduced case fatality & prolonged infectious period

Figure 21 shows the effect of increasing proportion of transient infections (Figure 21 left to right) on the sero-prevalence in two age cohorts close to fade-out. The mechanism, which did prolong the duration of circulation of the infection (Figure 13B), does strengthen the dynamics of sero-positive animals close to fade-out. This implies that the chance to hunt sero-positive animals is consolidated, hence, an exit strategy based on the profile of the standard model would also be congruent with scenario 1B.
3.3.2.2. Ad Scenario 4 - Long-term infectious period (aka carriers)

Figure 22. Temporal profile of sero-prevalence on the LAU1 unit level plotted for all 13 LAU1 units of Estonia (exclusive islands), first row adult cohort (older than 24 months), and second row subadult cohort (6-24 months old). From left to right the diagrams show the prevalence for different intensities of scenario 4 i.e., long-term infectiousness. Left: standard model, middle: 1 in 1,000 infected animals, right: 1 in hundred infected animals.

The increasing proportion of life-long infectious survivors (animals which do not succumb to the disease; Figure 22, left to right) flattens the curve of prevalence of sero-positive animals close to fade-out. Therefore, the construction of reliable exit criteria gets more difficult as no pronounced changes around the fade-out occur if many such extreme disease courses would exist. This implies that an exit strategy based on the sero-prevalence profile of the standard model would be incongruent with scenario 4.
3.3.3. Temporal profile of carcasses abundance around fade-out

3.3.3.1. Standard model

The number of carcasses from animals succumbed to disease in simulations with the standard model (Figure 23) illustrates a steep decline comparable to the virus positive animals’ findings around the virus fade-out. However, the number of carcasses per time is greater by nearly an order of magnitude compared to infectious animals (100 vs 12 in median, 30-250 vs 4-30 by the 50% central interval). Although reasonable by comparing 1 week infectious period and 4-12 weeks of decomposition, the difference highlights the different odds to find a positive sample in either stratum close to fade-out.

3.3.3.1. Ad Scenario 1B – Reduced case fatality & prolonged infectious period

Figure 24 shows the effect of increasing proportion of transient infections (Figure 21 left to right) on the number of carcasses close to fade-out. The mechanism, which did prolong the duration of circulation infection (Figure 13B), has no noteworthy effect of the carcass abundance. This implies that the chance to detect positive carcasses is unchanged, hence, an exit strategy based on the profile of the standard model would again be well congruent with scenario 1B.

Figure 23. Temporal profile of carcass abundance on the LAU1 unit level plotted for all 13 LAU1 units of Estonia (exclusive islands, A), and individual LAU1 unit 1 (B), and 4 (C, see Figure 1). Individual data series are adjusted to the time when the last infectious animal succumbed (t=0). The data series of A include those of B and C.

Figure 24. Temporal profile of carcass abundance on the LAU1 unit level plotted for all 13 LAU1 units of Estonia (exclusive islands). From left to right the diagrams show the prevalence for different intensities of scenario 1B i.e., prolonged infectious period for transiently diseased animals (4 instead of 1 week). Left: standard model 5% survivors, middle: 10% survivors, right: 20% survivors.
3.3.3.1. Ad Scenario 4 - Long-term infectious period (aka carriers)

Figure 25. Temporal profile of carcass abundance on the LAU1 unit level plotted for all 13 LAU1 units of Estonia (exclusive islands). From left to right the diagrams show the prevalence for different intensities of scenario 4 i.e., long-term infectiousness. Left: standard model, middle: 1 in 1,000 infected animals, right: 1 in hundred infected animals.

The increasing proportion of life-long infectious survivors (animals which do not succumb to the disease; Figure 22, left to right) flattens the curve of carcass abundance close to fade-out. The ASF independent lifetime of these long-term infectious animals determines the time horizon of presence of infections in the region and therefore builds a very long tail with only few detectable animals but circulation prolonged for years. This implies that an exit strategy based on the profile of the standard model would likely be incongruent with scenario 4.
Interpretation: The two scenarios which did impact the duration of circulating infection have different properties regarding the temporal profiles. While the assumption of extremely long-infectious periods changes the temporal profiles towards incompatibility with the dynamics of the standard model (sc. 4), the fourfold longer infectious period in every fifth infected animal does not (sc. 1B). This difference implies that an effect on the surveillance data around virus fade-out could be expected only with the worst case assumption of extremely long infectious period (i.e. death of the life-time infectious animal is independent of disease, sc.4). Vice versa, there is uncertainty about the possible attenuation of ASF virus and the frequency of transient courses as well as the infectious capacity and epidemiological role of PCR+ animals reported 30 to 70 days post infection in animal experiments. Nevertheless, their consideration in the model (sc.1B, 4 week infectious period) revealed that the elaboration on the exit strategy based on temporal profile of virus positive, sero-positive and carcass abundance is equally valid and applicable. This finding resolves substantial portion of uncertainty about epidemiology of ASF in wild boar and the exit strategy protocol. Nonetheless, if extremely long infectious periods e.g. for several years must be respected as relevant under field conditions even only with low frequency (below 1% of infections) the exit strategy based on criteria from the above profiles may not be robust.

The principle dynamics of the temporal profiles as built on LAU1 unit level are independent of the global outcome of the simulation. In this report figures on temporal profiles show simulation output for all simulations no matter if the ASF infection did fade-out globally; while in EFSA (2021) similar figures were presented showing data of subset of runs in which the infection did fade-out globally. There is no difference between these two ways of output analysis. Therefore, the LAU1 unit level might reasonably considered as an adequate spatial dimension to test for virus fade-out. However, it should be considered that a reintroduction from adjacent LAU1 units cannot be excluded as it was repeatedly seen in the simulations when multiple exit decisions were taken on the same LAU1 unit i.e. introduction followed by fade-out followed by reintroduction etc.

### 3.4. Epidemiological understanding of a two-phases exit strategy

#### 3.4.1. General principles

The final objective addresses the performance evaluation of proposed approaches for an exit protocol within a wild boar region with simulated ASFV circulation and fade-out. Indicators/parameters determining a proposed exit strategy refer to active and passive surveillance and temporal and spatial distribution of samples. Therefore, it is reasonable to use the explicit model simulations of ASF in real geographic landscapes and apply the proposed exit protocol to simulated spatio-temporal surveillance data. The following paragraphs introduce the general approach of the exit strategy and highlight uncertainties to be considered when interpreting the outcome of the exit simulations. The derived exit criteria and strategy recommendations are presented by EFSA 2021.

The exit approach, in general, will consider two phases. First, a Screening Phase with a focus on virus detection, using routine surveillance. This phase is meant to be continued for a longer time dependent on possible virus detections. The approach only switches to a Confirmation Phase if no virus is found

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for a certain duration of the Screening Phase. During the Confirmation Phase, the aim is to maximise the surveillance effort without finding evidence of virus circulation. It is important to consider the different aims of the two phases i.e., long-term with routine effort vs minimum duration with maximum extra effort.

3.4.1.1. Motivation of the “epidemiological switch” (Olsevskis pers comm.)

The motivation of the two-phase approach is based on the logic that an exit scenario will be happen during a period where there are very few infected animals (with these animals being difficult to detect) and few virus positive carcasses. This is indicated in the virological and serological prevalence profiles, as reported above. Further, in wildlife populations, such as wild boar, representative sampling is difficult to achieve and unbiased sample-based testing against design prevalence cannot be guaranteed. Given this, the evidence to demonstrate absence of ASFV circulation has to be accumulated over time (likewise practiced with rabies in Thulke et al 2000). Thereby relying on the paradigm that possible ongoing circulation of a lethal infection will in reasonable time resurge to case numbers that facilitate passive detection. Obviously, the duration and surveillance effort proposed with an Exit strategy must be sustainable under field conditions. Hence, a longer phase with routine surveillance effort (the Screening Phase) and a shorter (minimal) phase (the Confirmation Phase) with increased surveillance effort (the maximum possible under field conditions) is considered.

3.4.1.2. Approach of analysis and reporting of exit strategy evaluation

The model was used to evaluate the two-phase exit strategy by considering the failure rate. The failure rate is defined as the percentage of false exit decisions that were obtained by deciding freedom from ASF while (undetected) infectious objects (live animals and carcasses attributable to ASF infection) were still present in the simulation area.

In the evaluation, combinations of phase lengths were considered systematically. The Screening phase was tested for 4 to 24 months in two-monthly steps. The Confirmation phase was varied between 0 and 14 months in 3.5 monthly steps. Thus, the shortest investigated exit strategy would require 4 months and the longest 38 months. The simulation data are presented using a contour plot representing the simulated parameter combinations by circles (Figure 26).

The plot in Figure 26 illustrates the failure rate for combinations of differing monitoring periods during the Screening (x-axis) and Confirmation (y-axis) phases. The failure rate was calculated across multiple simulations (100 per combination). Failure here refers to the percentage of trials in which the exit strategy would have failed, i.e., obtaining a false negative result by proposing freedom from ASF while (undetected) infectious objects were still present in the simulation area.

The plot allows selection of a purposeful confirmation phase if, for example (light dot), for 9 months no ASF virus was notified and minimum 1 carcass was collected from the target region per year and per multiple of 1.000 km². In order to achieve less than 2% failure rate the previous 9 months (screening, x-axis) should be combined with another 10 months (confirm, y-axis) where 2 carcasses need to be collected per year and multiple of 1.000 km².
3.4.1.1. Failure reasons of exit decisions

What is the reason for the failing decisions of the exit strategies? The model output revealed two processes resulting in a false negative decision dependent on the parameters chosen for the waiting times in phases A and B.

The first reason why exit decisions did fail refers to an inadequate sampling horizon i.e., the lengths of the screening and confirmation phase which by chance did not result in virus positive or serologically positive sub-adult samples (shaded area in Figure 27A). The cumulated monitoring time simply was too short (10 months) to find the low prevalent virus. These failures can be prevented by more appropriate phase lengths as in Figure 27B, e.g., in total 19.5 months. Checking with Figure 26, a screening phase of 12 months and a confirmation phase of 7.5 months still creates a failure rate about 2% and Figure 27B reveals that none of these failures was due to continuously circulating but undetected infection. Indeed, in all simulations where the extended exit strategy still failed the infection was reintroduced into a LAU1 unit during the confirmation phase (below the light grey line in Figure 27B). E.g., more than 80% of the failing decisions relate to a reintroduction during the last 3 months of the 19.5 months long application of the exit strategy. Such late reintroductions close to the exit decision are problematic because case numbers will grow slowly in the spot, cannot be easily detected by routine surveillance and thus, leave the exit strategy protocol uninformed. However, it should be considered that a reintroduction from adjacent, still affected, units cannot be excluded on the level of compartmental units (e.g., LAU1) no matter how long the individual waiting periods are chosen but requires larger context of evaluation to epidemiologically preclude the risk of ASF reintroduction from neighbouring units.
3.4.2. Assessing the importance of human mediated translocations

Human mediated translocations are suggested as an important component of ASF spread in the geographical context. Human mediated translocations are a data-driven concept and refer to notifications that are close in detection date but far apart by spatial distance from any older detection. Hence such ASF notifications are unlikely to be established via contact transmission chains built on wild boar movement ecology (EFSA 2018). The illustrating examples are the earliest notifications in CZ or BE which had no plausible wild-boar-mediated relationship with existing infected areas. The inclusion of human-mediated translocations into the spatial spread of ASF in wild boar of Eastern Europe facilitates the adequate replication of large-scale spatio-temporal spread history comprised in the ADNS notification data (EFSA 2018). The question arose whether the simulation of ASF spread in wild boar with and without human-mediated translocations has an impact on the performance of the exit strategy. The hypothesis was that human translocations make the spatial dynamics more comprehensive compared to completely stochastic transmission between wild boar groups. This synchronisation would lead to more stringent exit decisions.

Figure 28 compares the two outcomes of the same exit strategy (here 1 carcass per screening and 2 carcasses per confirmation phase) without (same as Figure 26) and with human mediated translocations.

In accordance with the hypothesis, the exit strategy simulations that include human-mediated translocations (Figure 28B) provide the same performance as simulations without human mediated translocations (Figure 28A) but with shorter monitoring periods both in the screening and the confirmation phase.
### Figure 28 A-B. Comparison of the exit strategy evaluation simulating either none (A) or all human-mediated translocations between 2014-2018 (B). The applied surveillance effort was 1 carcass per 1000 km² per year during the Screening phase (x-axis) and 2 carcasses (doubled intensity) during the Confirmation phase (y-axis). Active surveillance included virological investigation of hunted animals and serological investigation of subadults in both phases. The colours represent the percentage of trials in which the Exit strategy would have failed, i.e., obtaining a false negative result by proposing freedom from ASF while (undetected) infectious objects were still present in the simulation area. Lines show isoclines for failure rate of 2% (solid), 5% (dashed) and 10% (dotted).

**Interpretation:** Human-mediated translocations lead to more coherent outcome in the spatial spread and therefore also to more precise exit strategy performance (same failure rate with shorter monitoring periods in both phases). The benefit of the synchronising effect of human-mediated translocations cannot be exploited for the exit strategy parametrisation due to a-priori ignorance. However, vice versa, if an exit protocol is parameterised sole based on wild boar mediated spread the predicted performance of the exit strategy is conservative, but potential human-mediated translocations do not violate the strategy. In case of uncertainty about human-mediated translocations, it is recommended to assess the exit strategy parameterisation based on wild boar mediated spread. The outcome are longer monitoring phases but a safer decision.

#### 3.4.3. Assessing the importance of natural mortality in a wild boar population

Assumptions with respect to the ‘estimated relative mortality not attributed to hunting’ in a wild boar population has implications for passive surveillance. The increased contribution of natural mortality will lead to increased carcass numbers, which in turn will decrease ASFV detection probability per carcass if the passive surveillance requirements remain unchanged (e.g., testing a single carcass per 1,000 km² per year). In other words, there is a dilution of the effectiveness of passive surveillance due to increased numbers of non-infected carcasses.

Simulations of the wild boar ecology follow the literature regarding the proportion annually removed from the population (Table 3 in the ODD Appendix A; 35-60%; Focardi et al. 1996; Gaillard et al. 1987). To split the data into hunting harvest and natural mortality, the following information was retrieved (J. Vicente pers comm), highlighting variation in estimated proportions of removed animals:

- 90% hunting harvest and 10% natural mortality (Focardi et al. 1996),
- 84% hunting harvest and 16% natural mortality (Keuling et al., 2013), and
- 80% hunting harvest and 20% natural mortality (Toïgo et al. 2010).
Table 3: Targets and efforts necessary to collect carcasses per annum per multiple of 1.000 km² for the implementation of an exit strategy dependent on reported mortality figures in managed populations.

| Phase          | % hunting harvest / % natural mortality | Target number of carcass per year per 1.000 km² | Number of carcasses available | % carcasses to be found = individual carcass detection |
|----------------|----------------------------------------|-----------------------------------------------|-------------------------------|-----------------------------------------------------|
| Screening (1)  | 90% / 10%                              | 1                                            | 12.5                         | 8%                                                  |
| Confirmation (2)| 90% / 10%                              | 2                                            | 12.5                         | 16%                                                 |
| Confirmation (6)| 90% / 10%                              | 6                                            | 12.5                         | 48%                                                 |
| Confirmation (12)| 90% / 10%                             | 12                                           | 12.5                         | 96%                                                 |
| Screening (1)  | 80% / 20%                              | 1                                            | 25                           | 4%                                                  |
| Confirmation (2)| 80% / 20%                              | 2                                            | 25                           | 8%                                                  |
| Confirmation (6)| 80% / 20%                              | 6                                            | 25                           | 16%                                                 |

Figure 29 shows the extent to which the performance of the exit strategy is influenced by the dilution effect of increased natural mortality i.e., more negative carcasses in the landscape.

Figure 29 A-B. Comparison of the exit strategy evaluation simulating natural mortality contributing 20% (A) and 10% (B) to the annual removal prior to ASF (including human-mediated translocations). The applied surveillance effort was 1 carcass per 1000 km² per year during the Screening phase (x-axis) and 2 carcasses (doubled intensity) during the Confirmation phase (y-axis). Active surveillance included virological investigation of hunted animals and serological investigation of subadults in both phases. The colours represent the percentage of trials in which the Exit strategy would have failed, i.e., obtaining a false negative result by proposing freedom from ASF while (undetected) infectious objects were still present in the simulation area. Lines show isoclines for failure rate of 2% (solid), 5% (dashed) and 10% (dotted). Figure 29B equivalent to 28B.

The exit strategy simulations that use 20% natural mortality out of annual removal (Figure 29A) provide the same failure rates as simulations assuming 10% natural mortality (Figure 29B equivalent to Figure 28B) but with shorter monitoring periods both in the screening and the confirmation phase.

If there is uncertainty about natural mortality rates in a region, a more conservative exit criteria would be advisable.

**Interpretation:** The lower natural mortality component is under annually removed animals of a population, the more precise exit strategy performance is (same failure rate with shorter monitoring periods in both phases). The reason is that with larger natural mortality more negative carcasses are abundant in the region under surveillance, hence the chance to collect the ASF positive carcasses is reduced. If an exit strategy is parameterised without data on natural mortality then the assumption of higher natural mortality leads to a conservative but more reliable exit decision.
3.4.4. Assessing the importance of the passive surveillance component

The efficiency of passive surveillance for ASF detection has previously been reported (EFSA 2010, 2018, Gervasi et al. 2020). It was hypothesised that greater effort in passive surveillance would enhance the performance of an exit strategy. Therefore, alternative exit decisions were tested based on varying number of carcasses retrieved per year and per multiple of 1000 km² of a sampled area.

![Figure 30. Comparison of different Exit Strategy options given different level of passive surveillance during the Confirmation Phase. The scenario replicates 1 carcass collected per year and 1000 km² in the Screening phase but varies the intensity of carcass detection during the Confirmation phase (bottom legend), namely 0, 1, 2, or 6 carcasses per 1,000 km² per year (corresponding to 0%, 8% 16% and 48% carcass detection probability, Table 3). The boxplots present the variation in the number of carcasses that were actually collected per year and per multiple of 1000 km² in the LAU1 units and across model runs. The data is shown only for one specific combination of monitoring periods: Screening phase of 6 months, Confirmation phase of 7 months (a total of 13 months). At the top of the figure, the resulting strategy performance is shown in terms of the probability of a false decision. The accuracy of the Exit strategy in supporting a decision on viral extinction is increased with an increasing number of carcasses that are routinely collected and tested. This is illustrated in Figure 30 where the percentage of false Exit decisions (the probability of false negative results) decreases (15.57%, 5.41%, 3.91% and 2.44%) concurrent with an increase in the percentage of all carcasses that are collected during the Confirmation Phase in the wild boar area (resulting in 0, 1, 2, and 6 carcasses per year and 1,000 km², respectively).

3.4.5. Assessing the importance of active surveillance component

According to the sero-prevalence profiles around fade-out (3.3.2), the response among adult animals to virus fade-out is slow and does not provide information. Adult seropositive animals will remain positive throughout life. Therefore, the sampling for serologically positive adults isn’t relevant for the exit decision. In contrast, the response in virus-positive and subadult sero-positive profiles occurs at virus fade-out or within about 1 year following viral extinction respectively (3.3.1 + 3.3.2).

The impact of different components of active surveillance on the performance of the Exit strategy can be demonstrated through a comparison of model simulations that do or do not include active surveillance within the two-phase approach. Figure 31 summarises this information.

First, the performance of an exit strategy is improved by prolonged monitoring periods. The total monitoring period increase from the left to right panel in Figure 31 covering 7, 23 and 38 months respectively. In accordance the failure rate (top values) associated with each panel decreases from roughly 11-18%, 1-2%, to 0.3-0.8% i.e., longer monitoring periods improve the reliability of an exit decision. However, the improved performance by longer monitoring comes at the cost of longer time span between point of virus fade-out and time when an exit decision can be reached (‘time free’). This
trade-off may require an economical evaluation of surveillance efforts compared to commercial costs of prolonged restrictions.

Figure 31 A-C. Comparison of different strategy options given differing levels of active surveillance during the Screening and Confirmation phases. Boxplots reflect either the inclusion or omission of active sampling of subadults for serology during the Screening Phase (sero+ done; sero- omitted) and of any active surveillance during the Confirmatory Phase (hunt+ all tested for viral genome and antibody; hunt- no shot animals are tested). The boxplots present a summary of the length of time that a LAU1 unit was free of ASFV infection before the Exit strategy came to a decision for that LAU1 unit. The data is shown only for specific combinations of monitoring periods during the Screening or Confirmation Phase: (A) 3 + 4 (a total of 7) months; (B) 12 + 11 (23) months; (C) 24 + 14 (38) months. At the top of the Figures, the resulting strategy performance is shown in terms of the probability of a false decision.

Second, the four performance values on the top of each individual diagram inform about the impact of active surveillance within the broader exit strategy. In each of these three diagrams, the value at the far left reflects the maximum application of active surveillance (in addition to passive surveillance). Hunted animals are tested for viral genome [hunt+] and subadult animals are tested for serology [sero+] in both the screening and confirmation Phases. In contrast, the value at the far right of each diagram reflects the exit strategy performance where hunted animals are virologically investigated only in Screening Phase [hunt-] and subadult serology is omitted in both phases [sero-] and that is, active surveillance was not performed at all in the confirmation phase. Using active surveillance data with the confirmation phase does create only marginal effect on the performance (comparing the two left and two right performance values in either diagram of Figure 31).

Third, adding subadult serology during the screening phase did regularly improve the performance of the exit decision (comparing the first with third and second with fourth performance values in either diagram of Figure 31). However, the gain in performance by adding subadult serology was marginal compared to improvements achieved by lengthening of the monitoring periods. To illustrate this, consider Figure 31A, where the choice of the monitoring periods (screening plus confirmation phases) is extremely short (7 months in total). With this scenario, the difference between the performance value at the far left (11.28%, Figure 31A) and the far right (17.44%, Figure 31A), i.e., the effect of active surveillance, is minimal compared to the improvement that is achieved when the monitoring period is prolonged, either to 23 months, resulting in a failure rate between 0.94% and 1.77% (Figure 31B), or 38 months, with a failure rate between 0.30% and 0.81% (Figure 31C). In conclusion, the contribution of active surveillance to an efficient exit strategy is less important than either passive surveillance (carcasses collected) or the lengths of the monitoring period i.e., the screening and confirmation phases.

Finally, the cost of serological surveillance was considered. Here Figure 31 reveals that adding active surveillance data to the confirmation phase (hunt+) left the interval between virus fade-out and exit decision unchanged. On the other hand, applying subadult serology in the screening phase (sero+) prolonged that interval meaning in most cases the extra effort introduced prolonged time to exit decision although the infectious already did fade-out. More systematically, Figure 32 addresses the effective time horizon without infectious animals and carcasses attributed to infection for all correct exit decisions. The applied monitoring periods in this experiment combined a screening phase of e.g., 12 months with a confirmation phase of 7 months i.e., 19 months in total (Figure 32).
Figure 32 A-B. Correct exit decisions with 12 months screening and 7 months confirmation phase. The graph shows for each exit decision the time between virus fade-out from the LAU1 unit and the decision (‘time free’). Light grey horizontal indicates the time horizon of confirmation phase (7 months), and dark grey horizontal indicates the length of confirmation and screening phase (7m + 12m = 19 months).

For the majority of the correct decisions (left 95% in Figure 32A) the resulting periods without the virus circulation were adequate and fade-out occurred actually already during the screening phase (central 90% where the graph is in-between the two grey lines). In less than 5% of the decisions the actual virus fade-out occurred during in the confirmation phase (below the lower line). However, for another 5% of correct decisions (right 5% in Figure 32A) the area was much longer free from infection than the exit approach intended (i.e., longer than 19 months). Figure 32B reveals that all these imperfect decisions were caused by a reset of the confirmation phase after serologically positive subadult findings.

**Interpretation:** In contrast to the suggestive temporal profile of subadult sero-prevalence, detailed testing of exit strategies has shown that this information is of marginal use in this regard. In hindsight, this conclusion is logical given the greater efficiency of passive compared to active surveillance for case finding, as highlighted previously (Gervasi et al. 2020). Specifically, information from subadult serology will be redundant in the presence of robust passive surveillance.

Active (serological) surveillance of subadults is not only of limited value but comes at an extra cost for the exit strategy. If it were to be included within the protocol, the detection of seropositive subadults would result in a return to the start of the exit procedure. This will prolong the period between viral extinction and the exit decision.

### 3.4.6. Assessing the importance of population density prior to ASF

Wild boar population density is always an issue of concern when investigation surveillance or control related measures in epidemiological models. Here additional simulation output was used to test the effect of doubled density on the performance of the exit strategy. Assuming the same geographic wild boar habitat but doubling the ecological capacity of the landscape, the set of simulations already investigated was repeated. The direct comparison of the performance panel of the exit strategy is achieved in Figure 33.
Figure 33 A-B. Comparison of the exit strategy evaluation simulating spread in wild boar habitat structure as to Estonian data (A) and assuming doubled capacity (B) (including human-mediated translocations). The applied surveillance effort was 1 carcass per 1000 km² per year during the Screening phase (x-axis) and 2 carcasses (doubled intensity) during the Confirmation phase (y-axis). Active surveillance included virological investigation of hunted animals and serological investigation of subadults in both phases. The colours represent the percentage of trials in which the Exit strategy would have failed, i.e., obtaining a false negative result by proposing freedom from ASF while (undetected) infectious objects were still present in the simulation area. Lines show isoclines for failure rate of 2% (solid), 5% (dashed) and 10% (dotted). Figure 33B equivalent to 29B.

Figure 33 supports the interpretation that more conservative exit criteria are determined for low density populations as the fade-out in landscapes with greater density was more reliable and therefore exit decisions more often correct.

**Interpretation:** Estimates of population density for particular wild boar areas often are understating the true abundance and create uncertainty to quantitative characteristics of disease management. Regarding the exit strategy this problem is less acute because greater population density prior to ASF would lead to more reliable application of the exit protocol.

### 3.4.7. Assessing the importance of long-term infectiousness

The virological and serological profiles of ASF affected regions are similar in simulations with or without an increase in survival of infected animals, with surviving animals having a longer (but still transient) period of infectiousness (Sc. 1; 3.2.1 & 3.3). Similarly, this scenario does not influence the outcomes of the exit approach proposed here.

In contrast, virus circulation is substantially influenced by life-long infectious survivor animals (Sc. 4; 3.2.4 & 3.3). At virus fade-out the slower decline in virus prevalence is observed, and seropositive and virus positive animals may be present, albeit in very small numbers, over long periods. Similarly, there will be a low number of carcasses from infected animals over an extended period. In this scenario, exit is a ‘trial and error game’, if life-long infectious animals contribute for years to ASF spread in an affected region. As a consequence, an exit approach will repeatedly result in a restarting of the screening phase due to ongoing infection circulation, and the low number of permanent infectious animals led many false exit decisions. As reflected in Figure 34, the respective performance is disappointing, both in terms of high failure rate (Figure 34B) and overly long time free (Figure 34B). In conclusion, the exit strategy is problematic in the presence of life-long infectious survivor animals.
Interpretation: Long-term infectious animals (e.g. for CSF the chronic infections or persistently infected [PI] piglets, Kramer-Schadt et al., 2007), if associated with an infection, facilitate continuous or endemic circulation of infection in wildlife populations (Kramer-Schadt et al. 2009). The epidemiology of ASF and CSF are not compatible, in part due to the large difference in case-fatality between the two diseases. Nonetheless, the potential impact of prolonged duration of the infectious period on the outcome of the exit decision needs extra consideration. But only if regularly animal infections turn into a year-long of virus shedding. With such an extremely modified disease course the proposed exit approach would not lead to robust decisions. That said, it should be emphasised that based on current knowledge the existence of such life-long infectious survivors of an ASF infection is speculative and not required to reproduce the observed spatio-temporal spread of ASF in wild boar (e.g. EFSA 2015, Pepin et al. 2020). The sole decrease in lethality with few weeks longer infectious periods does not corroborate the approach to an exit decision.
4. Discussion

The development of an exit strategy recommendation for ASF in wild boar (see EFSA 2021) passed different conceptual steps. In order to highlight the review of the presented model output (chapter 3) by the ASF expert group and the AHAW Panel of EFSA revealed several concerns, questions and discussion points. As far these were addressed by additional analysis of the model or its output, the answers are provided in the following chapter, or otherwise archived in the subsequent chapter as recommended research to do.

4.1. Origination of the proposed approaches of the exit strategy

From Chapter 3.3 it is known that virus prevalence is extremely low for a relatively short time prior to local viral extinction. For this reason, zero virus detection is a useful trigger for an exit trial, even if undetected virus circulation may stay still occur. The two phases have to be passed while satisfying the criteria e.g., listed in Table 2. For example, the length of the screening and the confirmation phase was set to 52 weeks (12 months) and 30w (7.5 months) (the choice for the crossover parametrisation still reflects that stage by using 3-monthly and 3.75-monthly bins).

During the two phases of an exit trial, continued surveillance must result in no new virus detection and a defined number of carcasses must be collected and tested routinely. Moreover, the total monitoring time should be minimised to allow the free status to be achieved, on the assumption that ASF has actually faded-out from an area. The EFSA WG proposed two different approaches to specify surveillance efforts underlying two alternative exit strategies (I and II).

Exit strategy I: The first approach did follow the idea of maximising the carcass collection during the monitoring phases. The epidemiological unit for a useful exit decision was associated with an area size of about 2500 km² (i.e., the average size of LAU1 units of Estonia). The important surveillance component of the exit strategy was the identification of a practicable rule that could lead to sufficient carcasses being collected. Therefore, the first exit strategy required:

- Passive: Collection and diagnostic testing of a minimum number of carcasses equivalent to 2% of the hunting bag prior to ASF incursion into the LAU1 unit. The hunting bag estimate per LAU1 usually is available from hunting organisations. For model analysis the number of hunted animals was determined from simulations without ASF following Focardi et al. (1996).

- Active: 100 hunted animals have to be shot per annum per LAU1 unit i.e., distributed continuously over the year according to availability, for virological and serological diagnostic.

Figure 21 shows the performance of Exit Strategy I for different combinations of duration of the screening (x-axis; 4-24 months) and the confirmation (y-axis; 4 to 15 months) phase. The simulated surveillance effort led to the performance panel (% of failed exit trials) in Figure 35A.
Figure 35 A-C. A: Performance of different parameterizations of the Exit Strategy I. Every exit trial is based on per-annum collection of carcasses equivalent to 2% of the average annual hunting bag prior to ASF and 100 samples from the hunting bag. The Exit Strategy I criteria were applied during the Screening Phase (time without virus detections) and Confirmation Phase (time without virus detection AND without serological positives among sampled subadults). The colour contours represent the percentage of trials in which Exit Strategy I would have failed, i.e., obtaining a false negative result by proposing freedom from ASF while (undetected) infectious objects were still present in the simulation area. Lines show isolines for failure rate of 2% (solid), 5% (dashed) and 10% (dotted). B: As for A but excluding the active component – no sampling from hunted animals. C: As for A but excluding the passive component - no carcass collection.

According to Figure 35A the heuristic combination proposed above, applying a 12-month Screening phase and a 7.5-month Confirmation phase (a total of 19.5 months since last virus detection) resulted in a probability of only 1.4% of a false negative decision. Doubling the length of the monitoring period (24 + 15 months, i.e., more than 3 years in total after last virus detection) can only marginally improve the failure rate to 0.9%.

Two observations caused additional debate: First, the Exit strategy I revealed very nice results, and second, it appeared dominated by the carcass-based surveillance branch confirmed by Figure 36.
Figure 36. Comparison of alternative variants of the Exit Strategy I with regards to the surveillance effort. The four box plots represent varied sample size from the hunting bag (hunt 100, 60, 0) referring to 100, 60 and 0 hunted wild boar tested per annum per LAU1 unit) and carcass testing either switched on or off (carc 2%; carc 0%). The boxplots present a summary of the length of time that a LAU1 unit was free of the infection before the exit strategy came to a decision. The data is shown for the phase length combination 12 + 7.5 months. At the top of the Figure, the resulting strategy performance is shown in terms of the probability of a false decision.

The diagnostic investigation of hunted animals was without noteworthy impact on the performance (Figure 36, values on top of the diagram) or the time between actual freedom and exit decision (median 18 months). To the opposite omitting the carcass search (left plot in Figure 36, carc 0%) corrupts the performance of the Exit Strategy I. The translation of the carcass retrieval request, 2% of pre-ASF hunting bag, into physical volume revealed the substantial number of carcasses required for collection and testing i.e., between 20-40 per LAU1 unit per annum or equivalently about 10-15 per multiple of 1,000 km² of an area. Given that the ASF affected wild boar population around fade-out is on a low level in animal numbers, collecting 10-15 fallen animals per year implies about 100% detection probability per every carcass.

Here it was recognised that such intensive carcass collection procedures might be applicable for focal introductions (e.g., CZ, BE, DE) where in small, confined management zone and over limited time (e.g., several months) highly intensive carcass collection is feasible according to field reports. However, under situations where a potential exit was preceded by a widespread infection over thousands of square kilometres (likewise Baltics or eastern Poland) practical experience suggested 1 carcass per 1.000 km² and year as reasonable routine effort. Expert knowledge suggested further that for short time an intensified level of 2-6 carcasses per 1000 km² per year might be achievable. With this constellation a second proposed exit strategy was necessary addressing large, affected areas.

**Exit Strategy II:** The second exit strategy is less reliant on ineffective carcass searches and based on the sampling and collection efforts as currently implemented and achieved in affected Member states. The principle of the two phases is unchanged. According to EFSA's DCF one carcass per year per 1.000 km² reflects actual field practice. According to regulations routine diagnostic testing is conducted on all hunted animals and retrieved carcasses, including viral genome (PCR) testing of samples from hunted wild boar and carcasses and antibody testing of samples from hunted subadult animals. If either virus genome or antibodies are detected in these samples, the procedure is reset to start again. After successful completion of the Screening Phase, the Exit Strategy II is continued to the Confirmation Phase, where carcass sampling efforts are intensified for a limited period of time.
Specifically, carcass collection efforts are intensified to 2 or 6 carcasses collected per annum per 1,000 km².

Figure 37 A-B. Performance of different parameterizations of the Exit Strategy II. Every exit trial combines a Screening phase (duration x-axis) with a Confirmation phase (duration y-axis). The Screening Phase was applied with the request to test for virus (PCR) one carcass per 1000 km² per year and all hunted animals. The Confirmation Phase was applied with no (A), one (B), two (C) and six (D) carcasses tested for virus (PCR) but no testing of hunted animals. The colour contours represent the percentage of trials in which the exit decision would have failed, i.e., obtaining a false negative result by proposing freedom from ASF while (undetected) infectious objects were still present in the simulation area. Lines show isoclines for failure rate of 2% (solid), 5% (dashed) and 10% (dotted).

Figure 37 summarizes the effect of passive surveillance efforts during the Confirmation phase on the performance of the exit decision at different length of the total monitoring phase. Without carcass testing (Figure 37A) the exit decision is spoiled, continuing the effort in carcass testing as in the Screening phase (Figure 37B) made the exit decision reliable, and more intensive carcass testing enhanced the reliability of the procedure also for shorter monitoring periods (Figure 37C+D).

4.2. Issues of the exit strategy addressed with the model

**Issue: Possible small pockets of infection in part of a single LAU or spanning two connected LAUs may be difficult to address with the evaluation of the exit approach. Additionally, these would argue for a risk-based approaches?**

The spatial distribution of the infection close to fade-out was investigated for the performed simulations. The objective was to map how often the individual wild boar groups were involved in the last 52 weeks of circulating infection. The outcome is plotted for runs finally fading out during the simulation (Figure 38A) and those runs with infection circulation till the end of the simulation (Figure 38B).
The spatial distribution of late infections across different simulations involves different locations (Figure 38). The two outcomes suggest that there is overarching risk-structure for the final location. In those runs that end with global fade-out, the infection spread from south to north of the landscape and fade-out concentrated along the northern coastline (LAU1 unit 4, 9 and 13; Figure 38A). However, in runs where the infection did re-emerge behind the original epidemic wave fade-out locations are scattered across the habitat landscape (Figure 38B).

The last infections in the ASF simulation in wild boar do not occur as sparkling pattern all over the previously affected area but clumped in local spots (in contrast to e.g., persistent BVD infections in livestock farms at the end of a control programme); and there are spots obviously more prone to become the final one (orange regions in Figure 38B) - maybe “pockets”. The first recognition is reasonable for infectious diseases spreading in finite free roaming wildlife populations - to have virus cases over few weeks (i.e., to the end of the simulation) there is need for local association between these last cases - the “pockets of last infections”. Eventually there will be one last “pocket” while before several might have been there towards the end. The second observation of clustered locations of fade-out over multiple simulations is due to landscape structure and would lead to kind of support for risk-based efforts if causality could be determined and relative risk measured. The presented evaluation of different exit strategies did cover both processes.

**Interpretation:** The spatially explicit simulations of ASF spread does comprise the situations of small pockets as well as possible overlap between two LAU1 units. The consequence of the last pockets therefore is integrated with the evaluation of exit protocols as presented above. In a wider sense the Figure 38B shows that the model output does cover multiple (re-)introductions of the infection on the LAU1 unit level. That is, at the scope of individual LAU1 units the infection can fade-out regionally and resurge after e.g. three years without any virus circulation due to reintroduction from adjacent LAU1 units (or even by human translocation). This effect should be considered when applying the exit approach to a regionalised area (e.g. the LAU1 unit) without global fade-out of the infection (e.g. whole country around that LAU1 unit).
Issue: Spatial dimension of simulation of the exit approaches. If the model evaluates ASF surveillance at the level of the country, the infection will be “detected”. How about smaller area which size are not known a-priori with ASF exit approaches in wild boar?

The issues were leading to the evaluation of the model simulation on the LAU1 unit level and later to the scaling of all surveillance efforts per 1.000 km². Interestingly, the evaluation at the LAU1 unit level showed a good coverage by dynamics and variability in data series of individual LAU1 units matching that of the overall summary. Hence, the stochastic part of the variability (due to multiple simulation runs) was greater than that between LAU1 units. In the study reported here, the LAU1 unit covered areas of 1000 to 5000 km² (average 2500 km²) and had the advantage of being administrative so field data of disease surveillance were at the same scale facilitating matched interpretation. Administrative regions are also easily converted onto a spatial explicit model landscape because usually related GIS layers are already available.

Issue: For test of the first exit procedure the number of actively sampled wild boar was prescribed and chosen as 100 actively shot animals per year per LAU1 unit i.e., on average 2.500 km² and 2.000 to 3.000 animals. Are 100 samples per annum entirely realistic in all LAU in all affected member states on an ongoing basis? If not, sensitivity analysis would be required using the same exit protocol but lower intensity of active surveillance.

The choice of 100 hundred actively hunted wild boar samples was proposed by the WG matching the 2.000-3.000 animals per LAU1 unit. Hence, in member states with smaller LAU1 units a different active sample size might be advisable, or several LAU1 units should be monitored at once. The required hunting bag with the exit strategy concept was assessed additionally for 60 and 0 (Figure 35B) samples from hunted animals included in the surveillance.

Whether 100 hunted animals were realistic could be read from the simulation because with exit strategy concept II all hunted animals were subject to testing (Table 2) and therefore the model data are available (Figure 39).

Figure 39 A-B. Annually hunted wild boar per 1000 km² of 13 LAU1 units of Estonia (area size 1000 to 5000 km²) during the Screening (A, 12 months) and Confirmation phase (B, 7.5 months). Data summarize 100 simulation runs. Note: The hunting bag is taken during exit strategy application which implies that the ASF related lethality already reduced the population by about 75%, Figure 6).

In about 75% of the simulation (lower edge of the box to top in boxplots of Figure 39) the hunting bag per 1000 km² which is generated by model simulations provides more than 100 hunts for all LAU1 units. Comparing screening (Figure 39A) with confirmation phase (Figure 39B) the plots also reflect the beginning recovery of population with increasing hunting bags per 1000 km² of LAU1 units. Figure 39 also shows that the active surveillance component, if included in the screening or confirmation phase of exit strategy approach II, exceeds the effort foreseen with exit strategy approach I which requires...
100 hunted samples per LAU1 unit. Nonetheless, in both approaches and when applying sufficiently extended monitoring periods, the active surveillance branch does not contribute much to inform the exit decision.

**Issue: Do the required sample numbers have practical magnitudes i.e., 2% of hunting bag prior to ASF collected as carcass**

While surveillance effort in exit strategy II was intentionally parameterised in agreement with field experience from affected member states (e.g., EFSA's DCF database on actually recorded carcasses, or expert knowledge), the design of exit strategy I did not a-priori test the request of 2% hunting bag prior ASF to be collected as carcasses. The expected number of carcasses (from infected and non-infected animals) in the standard density scenario was 10-15 carcasses per 1000 km² in an exit situation i.e., at virus fade-out. This estimate is based on the population number per LAU1 unit, the annual mortality and reduction of the model population due to ASF lethality of about 75%. The model simulations of exit strategy I facilitate the comparison of available number of carcasses in the simulation with the required sample of 2% of the hunting bag prior to ASF (Figure 40).

![Figure 40. Fulfilment of required sample size for passive surveillance i.e., 2% of the hunting bag prior to ASF determines the number of carcasses which should be included in the sample of the exit strategy. The data is shown for the correct (blue) and false (red) exit decisions. The simulated exit decisions (x-axis) are ordered according to the fulfilment level of the sample (y-axis) measured per LAU1 unit as the ratio of found carcasses divided by the target number. Maximum 20% of the simulated exit decisions (left quintile of the x-axis in Figure 40) were based on unsatisfied surveillance target, while only 10% tested less than 90% of the target number. Interestingly, the issue of carcass availability was not related with the outcome of the exit decision i.e., correct (blue) and failed (red) exit decisions (Figure 40) have parallel distribution of carcass sample size fulfilment.](image)

**4.3. Pertinent queries to the principles of the exit strategy**

Based on data and modelling output this document addresses a principal approach and general structure of a phase-wise exit strategy through cumulating plausibility of ASF absence using standard surveillance outputs over time. The development of the approach, however, also led to undiscussed issues and new questions that cannot yet answered by the data collected within the time frame of this report. For the improvement of the strategy recommendation these queries should be tackled by further elaborations.

Q1: The greater contribution of natural deaths vs hunting harvest to the overall mortality prolongs the purposeful duration of the monitoring phases of the proposed exit strategy. Does there exist a lower bound across European wild boar population that would enable adjusting monitoring periods in the strategy phases?
Q2: If case fatality of ASF is lower than the reported 90%, less carcasses of ASF infected animals may reduce the sensitivity of passive surveillance. Does this in turn increase the importance of active surveillance components for the exit strategy which for now was found as less important?

Q3: The assumption of long-term surviving infectious animals (e.g., more than years) does harm the performance of the proposed exit strategy. Such animals are neither reported to exist nor is their potential role for the spread of the infection in the field unequivocally understood. However, the problem with the exit strategy still remains and needs to be addressed in more detail e.g., searching other indicators along with an exit trial that would simultaneously exclude that a situation without notifications is driven by long-term surviving infectious animals rather than normal viral extinction?

Q4. Intuitive relevance of serology-based surveillance for the exit strategy is contradicted by the model outcome. First, adult serology does not promise useful extra information to a timely and performant exit approach because there are no fast changes in the serology profile of adults close to (true) virus fade-out. Only by generation turn over, sero-positive adults disappear from the population. The definition of a correct exit decision is not about finding sero-positive animals but about NOT finding them any longer. Therefore, also the end of detection of seropositive sub-adults is very slow as criteria compared to the information source based on carcass retrieval. Moreover, model outcome (3.3.2) and data (EFSA 2021) suggest that seropositive sub-adults form such a small proportion of all sub-adults as infectious carcasses do among dead animals. Consequently, serology-based criteria are rather irrelevant for an exit approach when the latter is based on intensive passive sampling (not road kills but animals found dead in the habitat e.g., forest, Gervais et al. 2020).

Q5. The actual duration of effective virus transmission from carcasses in the environment to live animals is uncertain. It is widely accepted that carcasses from animals succumbed to infection represent the environmental transmission component of ASF-spread in wild boar, including supporting evidence of intra-species contacts to carcasses (Probst et al. 2017). When we reduce their role, maybe the other mechanism can become more important?

Q6: Carcasses attributable to an ASF infection are required to mimic the disease dynamics observed in the field (e.g., Lange & Thulke 2017). However, it is possible that the ‘need for carcasses’ is due to something else e.g., other environmental contamination. It would be useful to test whether duration of infectiousness of the carcasses shorter than the time till decomposition would affect the dependence of the exit strategies on carcass retrieval.

5. **Conclusions**

- The exit protocol is built on detailed understanding of the ASF dynamics in wild boar towards a potential end of an epidemic in an area.

- Due to simulated spatial spread of ASF infections and the high case-fatality (95%) the total population collapsed by up to 60% to 90% within three years. ASF related excess mortality peaks within 1.5 years after introduction and ceases quickly.

- In simulated wild boar populations in Estonian habitat, the probability of continued virus circulation falls to approximately 50% at 7 years after virus introduction, reducing to approximately 10 % after 10 years.

- Throughout the simulated ASF epidemic, a low virus prevalence is observed with a median of about 2 % at the peak of epidemic (1%-4% for the 25th and 75th percentile), and prevalence is very low six months prior to virus extinction in a LAU 1 region in Estonia (median virus prevalence below 0.5 %; 25th and 75th percentile of 0.1%-2%).

- The median seroprevalence in sub-adults declined to 0% within 1 year (9 and 18 months for the 25th and 75th percentile) after local extinction of ASFV in a LAU 1 region in Estonia. In adults, this decline took more than 3 years.
The median number of wild boar deaths attributable to ASF is around 150 carcasses per LAU 1 at the peak of the epidemic (100-300 central 50% interval across runs and LAU1 units) and about 40 carcasses (10-150 central 50% interval across runs and LAU1 units) a year prior to local extinction.

Loss of protective immunity and reduced duration of protection by maternal antibodies does not prolong the duration of infection circulation in the simulation area.

A case fatality rate as low as 20% and a four-week period of transient infectiousness among surviving animals did not prolong circulation of infection in the simulation area.

The hypothetical presence of life-long infectious survivor animals resulted in ASF circulation beyond 20 years following introduction with about 90% probability. Under such worst case scenario the temporal profiles of virus and seroprevalence and carcass abundance are less informative around fade-out due to the substantially slowed dynamics in the presence of life-long infectious survivor animals.

Based on marked decline in virus- (and subadult sero-) positive animals around the virus fade-out missing virus detection during routine surveillance protocol suggests an epidemiological switch towards virus absence that must be attested by maximised surveillance efforts over minimum time interval. This general principle of a two-phase approach (Screening Phase, Confirmation Phase) combines the need for potentially longer phase of sustainable screening with a shorter phase of temporarily maximised confirmation efforts.

Applying the exit decision strategy to LAU1 units of average size of 2.500km² (1.000 to 5.000 km²) gave robust results. However, compartmentalised exit decisions maybe affected by reintroduction from neighbouring areas even if a fade-out had occurred previously. The epidemiological context of the area needs to be considered.

Greater wild boar density in the same simulation context improved the performance of the exit decision due to more stringent spread.

Increasing the number of carcasses being routinely collected and tested improves the performance of the exit strategy for the same choice of the lengths of the two phases; or does allow to adjust the duration of the phases.

However, the exit strategy will only be feasible if the intensity of the passive surveillance can be sustained under field conditions. This has to be achieved as priority and in case of inconsistencies in carcass collection outcome the monitoring phases should be prolonged.

In general, the inclusion of active surveillance in the exit decision has very limited impact on the performance compared to a lengthening the overall monitoring period.

A declining seroprevalence in sampled sub-adults can add information about the fade out of the epidemic and trigger the decision to initiate the exit strategy, however, including this surveillance activity in the exit decision only marginally improves its performance. This is because information from subadult serology (absence of detections of sero-positive subadult samples) will be redundant in the presence of robust passive surveillance (absence of virus-positive carcasses).

The exit strategy is problematic in the presence of life-long infectious survivor animals. That said, it should be emphasised that the existence of such animals is - based on current knowledge - speculative. Moreover epidemiological evidence exist that epidemic curve, spatio-temporal spread of ASF and duration of virus circulation congruent with field observations can be modelled without the need for long-term infectious survivors.

Higher natural mortality that is not caused by ASF or hunting reduces the probability of finding infected carcasses in an affected area, and therefore reduces the performance of passive
surveillance. If there were uncertainty about natural mortality rates in a region, a conservative exit criterion would be advisable.

- Human-mediated translocations do not reduce the performance of an exit decision due to their structuring effect on the spatial spread and hence on the time interval till fade-out.

- The role of infectious carcasses in the spread of ASF in wild boar in the field context is still based on plausibility argumentation rather than factual evidence. It would be useful to study the performance of an exit decision protocol under the hypothetical assumption that contact to carcass of an animal that succumbed to disease does not cause new infections.

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Annex A – ODD Model Documentation (version Jan 2021)

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A.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. Purpose

The purpose of the model is to investigate various diagnostic profiles over time in an ASFV affected wild boar population. The model aims at assessment of ASF spread in Estonian 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.1.2. 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 runtime, 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.1.3. 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.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.3. Details

A.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 Figure 3 in EFSA 2015, Pittiglio et al 2018). The breeding capacity was calculated as $CC_{ij} = 1.28 \times$ density_estimate [heads/km²] following the regression density_estimate = $f(CC_{ij})$. Individual cell values $\bar{CC}_{ij}$ 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 with positive breeding capacity, 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 4: 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.3.2. Input

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

A.3.3. Submodels

Submodels are described in the order of their execution. Parameters and their values are listed in Fehler! Verweisquelle konnte nicht gefunden werden. in section “Parameters”.

A.3.3.1. Release of infection

Release is scheduled according to time and locations extracted from the ADNS dataset. The first year of introduction into EU member states (2014) corresponds to the 4th year of each
Model-based analysis of ASF surveillance and exit decision principles

A.3.3.2. 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 $P^{(i)}_{inf}$ determines the probability of contracting the infection from an infectious group mate for one week. For each susceptible animal, the probability of becoming infected accumulates over all infectious animals within the group:

$$\Pi^{(i)} = 1 - \left(1 - P^{(i)}_{inf}\right)^{\lambda_i}$$  \hspace{1cm} (1)

where $\lambda_i$ 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 groups direct transmission is modelled stochastically. Parameter $P^{(b)}_{inf}$ determines the probability of contracting the infection from an infectious member of a neighbouring group for one week. For each susceptible animal, the probability of between group infections accumulates over infectious animals of all neighbouring groups:

$$\Pi^{(b)} = 1 - \left(1 - P^{(b)}_{inf}\right)^{\lambda_b}$$  \hspace{1cm} (2)

where $\lambda_b$ 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 (i.e., eight neighbours).

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 $P^{(c,a)}_{inf}$ 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

\[
\Pi^{(c,a,s)}_i = 1 - \left(1 - P^{(c,a)}_{\text{inf}}\right)^{\omega_i} \cdot \left(1 - P^{(c,a)}_{\text{inf}}\right)^{\sum_{ij} \omega_{ij}}
\]

(3)

where \(\omega_i\) is the number of carcasses in the respective core home range, \(\omega_{ij}\) 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

\[
\Pi^{(t,a,s)}_i = 1 - \left(1 - \Pi^{(i)}_i \right) \cdot \left(1 - \Pi^{(b)}_i \right) \cdot \left(1 - \Pi^{(ac,a,s)}_i \right) \cdot \left(1 - \Pi^{(c,a,s)}_i \right)
\]

(4)

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 \(\Pi^{(t,a,s)}_i\) of the home cell.

A.3.3.3. 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 \(t_{\text{inf}}\) 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.3.3.4. 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 \(N_{\text{disp}}\) 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 \(D_{\text{disp}}\) 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 $D_{\text{disp}}$. Thus, the distance travelled to the target cell can be larger than $D_{\text{disp}}$, but the linear distance from the home cell does not exceed $D_{\text{disp}}$ during search.

A.3.3.5. Male dispersal

Male dispersal is performed in weeks 20 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 does move 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 $p_{\text{ori}}$) or straight ahead (probability $1 - p_{\text{ori}}$). 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.3.3.6. 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 $\alpha_f$. 


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.3.3.7. 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:

\[
PM_{week} = 1 - (SP_{Year})^{1/32}
\]

Mortality due to infection is independently treated by the disease course sub-model.
A.3.3.8. 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 5: 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.3.3.9. 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.3.4. Parameters

Model parameters of the transmission model are shown in Table 6. The present document has been produced and adopted by the bodies identified above as authors. This task has been carried out exclusively by the author(s) in the context of a contract between the European Food Safety Authority and the authors, awarded following a tender procedure. The present document is published complying with the transparency principle to which the Authority is subject. It may not be considered as an output adopted by the Authority. The European Food Safety Authority reserves its rights, view and position as regards the issues addressed and the conclusions reached in the present document, without prejudice to the rights of the authors.

### Table 6: Model parameters

| Name | Description | Value | Source / details |
|------|-------------|-------|-----------------|
| **a) Wild boar ecology (used as constants for the study)** | | | |
| $T_{\text{max}}$ | Maximum age of boar | 572 weeks | (Jezierski 1977) |
| $SP_{\text{mean}}^{(\alpha)} / SP_{\min}^{(\alpha)}$ | Mean / minimum annual survival rate adults (natural mortality + conventional hunting) | 0.65 / 0.4 | (Focardi et al. 1996) |
| $SP_{\text{mean}}^{(\gamma)} / SP_{\min}^{(\gamma)}$ | Mean / minimum annual survival rate yearlings (natural mortality + conventional hunting) | 0.65 / 0.4 | (Gaillard et al. 1987) |
| $SP_{\text{mean}}^{(p)} / SP_{\min}^{(p)}$ | Mean / minimum annual survival rate piglets (natural mortality + conventional hunting) | 0.5 / 0.1 | (Focardi et al. 1996) |
| $M_{\text{nat}}$ | Proportion of natural mortality/ 1-M_{\text{nat}} proportion of conventional hunting | 80%-90% | (Focardi et al. 1996, Toïgo et al. 2010, Keuling et al. 2013) |
| **b) Dispersal and movement parameters (used as constant for the study)** | | | |
| $N_{\text{disp}}$ | Minimum number of sub-adult females for dispersal | 2 | Technical assumption |
| $D_{\text{disp}}$ | Maximum dispersal distance for sub-adult females | 2 cells (6 km) | (Sodeikat & Pohlmeyer 2003) |
| $S_{\gamma}$ | 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) |
| $p_{\text{ori}}$ | 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) |
| $t_{\text{carc}}$ | Seasonal Time of carcass persistence | 4-12 weeks | Table 2; (Ray et al. 2014) |
| $t_{\text{inf}}$ | Average period between infection and death | 1 week | (Blome et al. 2012, Guinat et al. 2014) |
| Name | Description | Value | Source / details |
|------|-------------|-------|-----------------|
| $p^{(i)}_{\text{inf}}$ | Infection probability by direct transmission within social groups | 0.05 | In a contact group of 10-12 animals the resulting local $R_0$ is 4-6 (Guinat et al. 2014). Also reflecting the limited transmission during physical contact during incubation (Blome et al. 2012). |
| $p^{(b)}_{\text{inf}}$ | Infection probability by direct transmission between different social groups | 0.0003 | Data-driven model calibration (Lange et al. 2018) using spread and velocity of ASF in wild boar |
| $p^{(c,a)}_{\text{inf}}$ | Infection probability per carcass (including contact and transmission) | 0.05 | Data-driven model calibration (Lange & Thulke 2017) using spatial-temporal data of notified ASF cases in wild boar |
| $p_{\text{access}}$ | Probability of virus-induced death in shared area | 0.8 | Data-driven model calibration (Lange & Thulke 2017) using spatial-temporal data of notified ASF cases in wild boar |
| $t_{\text{delay}}$ | Proposed delay of animals contacting carcasses | 2 weeks | (Probst et al 2017) |

**d)** Secondary disease course parameters (not relevant for the ASF model variant due to short $t_{\text{inf}}$)

| Name | Description | Value | Source / details |
|------|-------------|-------|-----------------|
| $\alpha_f$ | Fertility reduction if ill | 0.625 | Assumed like CSF 10/16 foeti aborted (Dahle & Liess 1992) |
| $P_{ri}$ | Probability of prenatal infection | 0.5 | Assumed like CSF (Dahle & Liess 1992) |
| $T_{\text{anti}}$ | Maximum persistence of maternal antibodies | 15 weeks | Assumed like CSF (Depner et al. 2000) |
| $T_{\text{immune}}$ | Maximum duration of immunity by maternal antibodies | 12 weeks | Assumed like CSF (Depner et al. 2000) |

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