Disease severity declines over time after a wild boar population has been affected by classical swine fever – Legend or actual epidemiological process?

Supplementary material

Appendix I: ODD model documentation

Martin Lange[1], Stephanie Kramer-Schadt[2], Hans-Hermann Thulke[1]

[1] Helmholtz Centre for Environmental Research Leipzig - UFZ, Dept. of Ecological Modelling, Permoserstraße 15, D-04318 Leipzig, Germany

[2] Leibniz Institute for Zoo and Wildlife Research, Alfred-Kowalke-Str. 17, D-10315 Berlin, Germany

Contents

1 Overview **2**

1.1 Purpose 2

1.2 State Variables and Scales 2

1.3 Process overview and scheduling 2

2 Design concepts **3**

3 Details **4**

3.1 Initialization 4

3.2 Input 4

3.3 Submodels 4

3.3.1 Virus release 4

3.3.2 Virus transmission 4

3.3.3 Disease course 5

3.3.4 Emerging disease patterns 6

3.3.5 Herd splitting 7

3.3.6 Reproduction 8

3.3.7 Death 8

3.3.8 Ageing 8

4 Simulation experiments, analysis, parameters **9**

4.1 Independent variables 9

4.2 Simulation experiments 9

4.3 Dependent variables 9

4.4 Analysis 9

4.5 Parameters 11

1 Overview

The Classical Swine Fever (CSF) wild boar model is a compilation of a spatially explicit, stochastic, individual based demographic model for wild boars (*Sus scrofa*) and an infection and disease course model for the Classical Swine Fever Virus (CSFV). The model is documented following the ODD protocol (Overview, Design, Details; Grimm et al. 2006, 2010).

1.1 Purpose

The model aims to demonstrate or disprove the possible decrease in CSF case fatality by pathogen evolution during the time horizon of a particular epidemic in a spatially structured wild boar population.

1.2 State Variables and Scales

The model comprises two major components, a wild boar demography model considering seasonal reproduction, dispersal and mortality, and a CSF virus model operating on the boar population. Boar population density and structure are influenced by the disease via virus-induced mortality and litter size depression (Laddomada, 2000).

All processes take place on a raster map, where each cell represents a functional classification of a landscape denoting habitat quality. The cells of the model landscape represent about 4 km^2, encompassing a boar group's core home range (Leaper et al., 1999). Habitat quality of the grid cells is translated into breeding capacity, i.e. the number of female boars that are allowed to have offspring (Jedrzejewska et al., 1997). Thereby, local host density is regulated in the model (i.e. increasing numbers of fertile females can breed only until breeding capacity is approached).

The crucial model entity is the individual wild boar, characterised by age in weeks (where one week represents the approximate CSF incubation time; Artois et al. 2002; Moennig et al. 2003), resulting in age-classes: piglet ($<$ 8 months $\pm$ 6 weeks), sub-adult ($<$ 2 years $\pm$ 6 weeks) and adult. Each host individual has a location, which denotes its home range cell on the raster grid as well as its family group. Further, the individual wild boar comprises an epidemiological status (susceptible, transiently infected, lethally infected with individual infectious period or immune by surviving the infection or maternal antibodies). Sub-adult wild boars may disperse during the dispersal period (i.e. early summer) dependent on their demographic status (disperser or non-disperser). Each individual has state variables for storing the virus characteristics in the event of infection, i.e. case mortality value (M) and the stochastic individual infectious period in case the disease course is lethal (m).

1.3 Process overview and scheduling

In the first week of each year, females are assigned to breed according to the carrying capacity of their family group's cell. Moreover, the model stochastically assigns natural survival figures to the

running year. Thereby, fluctuations in boar living conditions are represented, i.e. good and bad years.

Then, the model proceeds in weekly time steps. In each time step an ordered sequence of modules is executed: Infection and determination of the disease course, sub-adult dispersal, reproduction, death, and ageing. Figure 1 shows a flow chart of the scheduling of submodels.

2 Design concepts

Wild boar population dynamics emerge from stochastic individual behaviour, defined by age-dependent seasonal reproduction and mortality probabilities, and age- and density-dependent dispersal behaviour. The outbreak dynamics emerge from within and between group virus transmission, boar dispersal, individual stochastic disease courses and infectious periods for infected boars.

Demographic and behavioural parameters are represented by probability distributions to account for variations in the biological processes being modelled. Stochastic individual disease courses and infectious periods are modelled explicitly in accordance with the purpose of the modelling, i.e. emergent disease pattern on the population level.

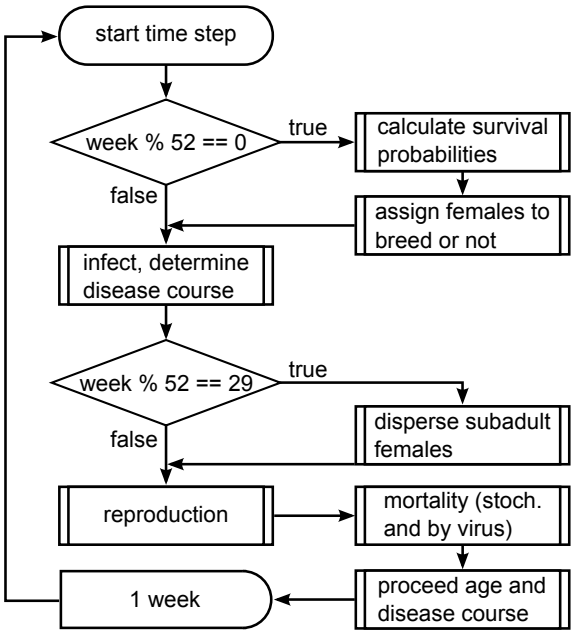

Figure 1: Flow chart of the scheduling of submodels.

3 Details

3.1 Initialization

The model landscape represents 200 $km \times 50$ km of connected boar habitat without barriers. The 2,500 grid cells are initialised randomly with uniformly distributed integer breeding capacity values $C_{ij} \in \{1, 2, \ldots, 9\}$. The average breeding capacity is 5 females per cell, resulting in approximately 20 boars per cell or a host density of 5 boars per km^2. Wild boar density reflects long-term average values of densely populated Central European habitats (Howells & Edwards-Jones, 1997; Sodeikat & Pohlmeyer, 2003; Melis et al., 2006; EFSA, 2008). One boar group is allocated to each habitat cell. Group size was initialised as three times the breeding capacity of the habitat cell. Initial age distribution was obtained from the results of a 100-year model run, conducted by Kramer-Schadt et al. (2009).

3.2 Input

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

3.3 Submodels

Submodels are described in the order of their execution. Parameters and their values are listed in table 1.

3.3.1 Virus release

The virus is released to the population by lethal infection of one randomly selected boar. The release is scheduled in a random week of the sixth year of the model run.

3.3.2 Virus transmission

Transmission is modelled stochastically (figure 2). The transmission parameter determines the weekly probability of being infected by an infectious group mate $P_{inf}^{(i)}$. The probability of being infected by an infectious animal in one of the eight neighbouring groups, $P_{inf}^{(e)}$, is fixed as one tenth of $P_{inf}^{(i)}$. For a susceptible animal the probability to become infected accumulates over all infectious animals within the group and in the neighbourhood according to

$$\Pi_i = 1 - (1 - P_{inf}^{(i)})^{\lambda_i} \cdot (1 - P_{inf}^{(e)})^{\Sigma_j \lambda_j} \tag{1}$$

where λ_i is the number of infectious group mates and λ_j is the numbers of infectious hosts in the jth neighbouring cell. The resulting probability value Π_i provides the parameter of a binomial chance process to decide on becoming infected. Infection might be translocated within the host population during dispersal of sub-adult females.

The transmission parameter $P_{inf}^{(i)}$ (and thereby $P_{inf}^{(e)}$) was calibrated in order to reproduce the spreading velocity observed in France (Rossi et al., 2010) with the simulated epidemic. The disease spread velocity of approx. 8 *km* per quarter was approached with the constant parameter value $P_{inf}^{(i)} = 2.08 \cdot 10^{-2}$ within and, hence, $P_{inf}^{(e)} = 2.08 \cdot 10^{-3}$ between groups.

3.3.3 Disease course

CSF shows a variety of disease courses on the individual level (Liess, 1987; Depner et al., 1997). Therefore, in our model the disease course is stochastically specified for each individual and determined by two parameters of probability distributions (figure 2): individual case mortality M defines a binomial and the mean infectious period of all lethally infected hosts μ an exponential distribution.

On infection the host is stochastically assigned as either lethally infected (with probability M) or as transiently infected $(1-M)$. M applies unchanged for yearlings $M^{(y)}$, is decreased for adults to $M^{(a)} = M^2$ and increased for piglets to $M^{(p)} = \sqrt{M}$ to represent age-dependent disease mortality (Dahle & Liess, 1992).

Transiently infected wild boars first pass through an infectious period of one week and subsequently become non-infectious but are protected against super-infection for the following three weeks. Afterwards anti-bodies are detectable and the animal is assigned to the immune state (Artois et al., 2002; Moennig et al., 2003; EFSA, 2008).

On infection lethally infected hosts receive their individual infectious period (m in weeks) drawn from the exponential distribution with the mean specified by parameter μ:

$$m = 1 + \lfloor -(\mu - 0.5) \cdot \ln(U(0,1)) \rfloor \tag{2}$$

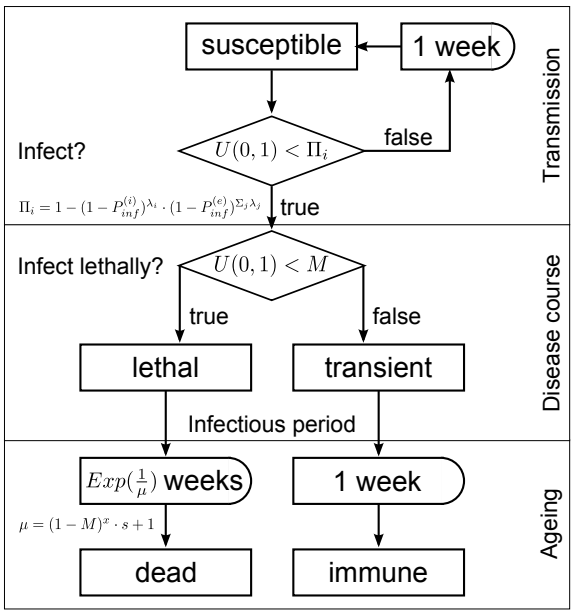

Figure 2: Flow chart of transmission and disease course.

where $U(0,1)$ is a uniformly distributed random number between 0 and 1. Lethally infected hosts remain infectious until death.

The total distribution of infectious periods resulting from the algorithms does not satisfy an exponential law (Wearing et al., 2005; Viet & Jacob, 2008). Example realisations of infectious periods for two case mortality values are shown in figure 3.

If the individual has maternal antibodies it is immune if its age T_k is below the number of weeks of immunisation due to maternal antibodies T_{immune}. Otherwise, mortality is decreased according

$$M_{MAB} = (1-M) + M \cdot \frac{1 + T_{anti} - T_k}{T_{anti} - T_{immune}} \tag{3}$$

where T_k is the individuals age in weeks, T_{anti} the number of weeks maternal antibodies persist and T_{immune} the number of weeks of immunisation due to maternal antibodies. A transient disease course is assigned stochastically by drawing a random number like described above.

3.3.4 Emerging disease patterns

A pathogen that causes high case mortality will produce few transient infections and short mean infectious periods in lethally infected hosts. Vice versa, longer mean infectious periods of lethally infected hosts should coincide with moderate case mortality (May & Anderson, 1983; Antia et al., 1994; Day, 2003; Ganusov & Antia, 2003). Therefore, in our model the mean infectious period of lethally infected hosts was represented as a function of the case mortality parameter M, according to

$$\mu = (M-1)^x \cdot s + 1 \tag{4}$$

The exponent x and the scaling factor s of the equation are chosen to represent standardised linear or quadratic relations (figure 4 a) (see section 'Simulation experiments').

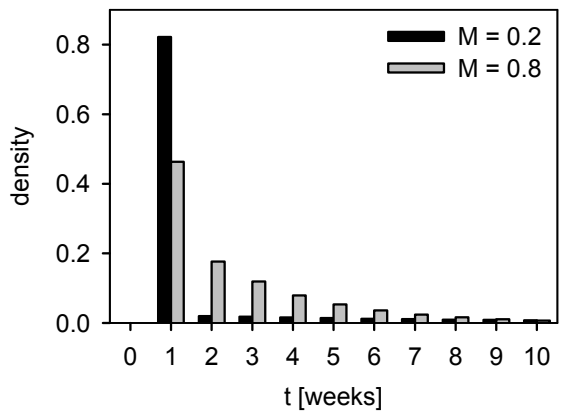

Figure 3: Realisation of infectious periods over lethally and transiently infected hosts for mortalities $M = 0.2$ (black) and $M = 0.8$ (grey).

At infection, the infected host inherits case mortality $M_{inherited}$ from the infecting host ($M_{transmitted}$). Case mortality was shifted by a random deviation from the ancestor case mortality $M_{transmitted}$:

$$M_{inherited} = M_{transmitted} + b_{virus} \cdot U(-1,1) \tag{5}$$

where b_{virus} is the maximum negative or positive deviation from $M_{transmitted}$ and $U(-1,1)$ is a uniformly distributed random number between -1 and $+1$. With the resulting $M_{inherited}$ it is stochastically decided whether the infection is lethal or transient. In case of lethal infection the corresponding mean infectious period $\mu_{inherited}$ is derived from equation 4 using $M_{inherited}$. Subsequently the individual's infectious period m is randomly drawn from an exponential distribution with mean $\mu_{inherited}$.

Case mortality of the initially released virus is $M = M^{(y)} = 0.8$. Case mortality of adults $M^{(a)}$ and piglets $M^{(p)}$ is determined according to the section 'Disease course'. With that, the average case mortality, measured over the entire population and one simulated year (according section 'Dependent variables') resulted in 0.76 with parameter $M = 0.8$.

3.3.5 Herd splitting

Herd splitting is performed in specified weeks of the year only. The model collects subadults able to move for each of the N habitat cells, which are all female yearlings without offspring. Afterwards, all herds to split are extracted, matching the conditions of containing a number of females exceeding the cells carrying capacity and containing at least a specified number of subadults to move (N_{disp}).

Splittable herds are iterated randomly. For each of them, an empty habitat cell (breeding capacity above 0) within an euclidean distance D_{disp} is selected randomly, excluding the source cell. All migratable individuals from the considered herd of the source cell form a new herd on that cell.

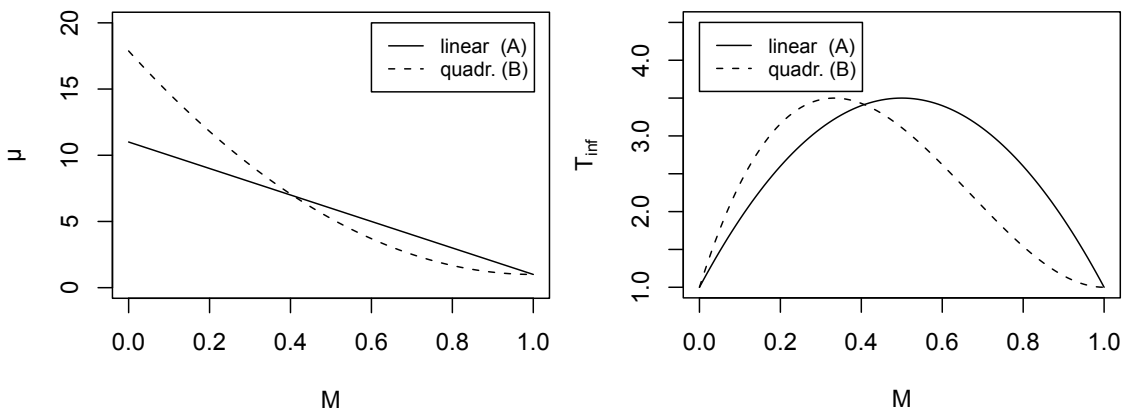

Figure 4: Mean infectious period μ [weeks] of lethally infected hosts b) effective mean infectious period T_{inf} [weeks] over all hosts, depending on case mortality M for different $M-\mu-$ relations. $(M-1)^x \cdot s + 1$. Solid: $x = 1$, $s = 10$ (linear), dashed: $x = 2$, $s = 16.875$ (quadratic).

3.3.6 Reproduction

Females reproduce only once a year, depending on their age class (they have to be at least subadult). Individual females reproduce depending on the season with a peak in April and no reproduction in winter from October to December (Boitani et al., 1995) (see table 4).

In the first week of the year, female individuals are checked whether they are able to breed. All females not exceeding their habitat cells carrying capacity, starting with the oldest individuals, are allowed to breed.

At each time step, for each individual allowed to breed and with currently no piglets, the model stochastically determines whether to breed this week by drawing a random number. The individual breeds, if the random number deceeds a weekly reproduction probability, derived from monthly probabilities and the number of weeks in the month (table 4).

Litter size is drawn from a precalculated truncated normal distribution (table 5) and reduced to a constant fraction for infected individuals. Litter size of transient shedders and lethally infected hosts is multiplied with the reduction factor α_f.

Depending on the disease state of the breeding individual, its piglets disease states are adjusted. Susceptible individuals and transient non-shedders produce susceptible offspring, immune individuals (no matter if due to preceding infection or vaccination) produce immune offspring with maternal antibodies. Transient shedders and lethally infected individuals yield offspring, each one lethally infected with a probability of prenatal infection P_{PI}.

3.3.7 Death

Iterating over the entire population, each individual either stochastically dies due to age class dependent mortality rates by drawing a random number, due to reaching a certain maximum age, or due to a lethal infection after a certain infection time span m (see section 'Infection').

Stochastic baseline mortality is age-dependent and adjusted to annual survival estimates found in the literature. These survival estimates together with the reported variability (see table 1) determine the Gaussian distributions we draw from the random survival in the model on a yearly basis (SP_{Year}). The stochastic effect resembles 'good' or 'bad' years for boars, i.e. environmental noise. In the application the Gaussian distributions are cut symmetrically around the mean. Per time step we apply the adjusted age-dependent mortality (PM_{Week}) to the individual:

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

3.3.8 Ageing

The ageing process iterates over all individuals. For each individual k, age T_k is incremented one week and disease state transitions are performed. Transient shedders are converted to transient non-shedders after a certain infectious period T_{trans}, losing their maternal antibodies if present. Transient non-shedder convert to immune after a certain latency period T_{latent}.

An individual k protected by maternal antibodies turns susceptible if reaching an age T_k of the protection time T_{immune}. Maternal antibodies themselves vanish on reaching an age of maternal antibodies persistence T_{anti}.

After disease state transition the age of the infection is incremented one week if the individual is not susceptible.

4 Simulation experiments, analysis, parameters

4.1 Independent variables

The primary independent variables of the study are the $M-\mu-$ relation and the maximum shift of case mortality per generation of infection (b_{virus}).

4.2 Simulation experiments

Simulations were performed assuming linear or quadratic $M-\mu-$ relations (figure 4 a) as these are two common types of relationship. Moreover, they represent two different strengths of the hypothetic relation. The parameters of equation 4 are:

- Linear relation: $x = 1, s = 10$
- Quadratic relation: $x = 2, s = 16.875$

where s was chosen to achieve equal maxima of the average infectious period over all hosts, $T_{inf} = 3.5$ weeks, for both relations (see figure 4 b). The respective case mortality value M^* where the maximum occurred was 0.5 and 0.33 for the linear and quadratic relation. Different values of the maximum shift of case mortality per generation of infection were simulated: $b_{virus} \in \{0.005, 0.025, 0.05\}$. For reference, simulations were performed without shift (i.e. $b_{virus} = 0.0$) for both linear and quadratic $M-\mu-$ relations.

All simulations were performed for 40 years or the until host or the virus went extinct. The virus was released into the boar population in a random week of the sixth year by infecting one randomly selected boar individual. For each scenario, 500 model runs were conducted to achieve a minimum precision of $\pm 5\%$ with 95% confidence for proportions.

4.3 Dependent variables

The average case mortality over the entire population of hosts and over each simulated year was calculated, dividing the number of new lethal infections by the number of new lethal and transient infections. Moreover, the time point of each virus extinction event was recorded.

4.4 Analysis

Case mortality measures were aggregated over 500 repetitions over the 52 weeks of each year. The effective mean infectious period T_{inf} over all hosts infected in a particular week was calculated

from aggregated case mortalities according to equation 4.

Survival curves of the virus in the population were derived by calculating the fraction of runs where the virus did not go extinct for each particular time step.

For the reference scenario without shift (i.e. $b_{virus} = 0$) and linear $M - \mu-$ relation, mortality (the fraction of present animals that die due to infection) was recorded per raster cell and per time step. Time series recorded for individual raster cells were aligned so that $x = 0$ became the week of the first infection of the cell. Mortality values were averaged over all cells and model runs.

Analysis was performed using GNU R 2.9.2 (R Core Development Team), plots were created with SigmaPlot ®10.0 (Systat Software Inc.).

4.5 Parameters

Table 1: Model parameters

Symbol	Description	Value	Source / details
α_f	Fertility reduction if ill	$10/16$	
P_{PI}	Probability of prenatal infection	0.5	
T_{max}	Maximum age of a boar	11 years (572 weeks)	Jezierski (1977)
T_{anti}	Maximum persistence of maternal antibodies	15 weeks	Depner et al. (2000)
T_{immune}	Maximum duration of immunity by maternal antibodies	12 weeks	Depner et al. (2000)
T_{trans}	Infectious period of transiently infected	1 week	Artois et al. (2002); Moennig et al. (2003)
T_{AB}	Period of transiently infected between infectious period and detectable anti-bodies	3 weeks	EFSA (2008)
$P_{inf}^{(i)}/P_{inf}^{(e)}$	Infection probability within / between herds	$2.08 \cdot 10^{-2}$ / $2.08 \cdot 10^{-3}$	Reversely fitted to the estimated disease spread velocity of approx. 8 *km* per quarter (Rossi et al., 2010)
M	Case mortality (subadults, see section 'Disease course')	0.8 (initial value)	
μ	Expectation value of the exponential distribution of life expectancies of lethally infected	3 weeks (initial value)	
D_{disp}	Maximum dispersal radius for subadult females	6 km (3 cells)	Sodeikat & Pohlmeyer (2003)
N_{disp}	Minimum number of subadult females for dispersal	2	
$SP_{mean}^{(a)}$ / $SP_{min}^{(a)}$	Mean / minimum annual survival rate adults	0.65 / 0.4	Focardi et al. (1996)
$SP_{mean}^{(y)}$ / $SP_{min}^{(y)}$	Mean / minimum annual survival rate yearlings	0.65 / 0.4	Gaillard et al. (1987)
$SP_{mean}^{(p)}$ / $SP_{min}^{(p)}$	Mean / minimum annual survival rate piglets	0.5 / 0.1	Focardi et al. (1996)
$u^{(a)}$ / $u^{(y)}$ / $u^{(p)}$	Bait uptake rates adults / yearlings / piglets	0.33 / 0.33 / 0.05	Piglet uptake rates were chosen low to represent difficulties in vaccination of juveniles, particularly younger than $4-5$ months (EFSA, 2008; Kaden et al., 2000).

Table 3: 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

Table 4: Monthly reproduction probabilities (Boitani et al., 1995)

Month	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Number of weeks	4	4	5	4	4	5	4	4	5	5	4	4
Reproduction prob.	0.06	0.1	0.23	0.34	0.07	0.08	0.06	0.03	0.03	0.0	0.0	0.0

Table 5: Breed count distribution (Bieber & Ruf, 2005)

Num. offspring	0	1	2	3	4	5
Probability	0.01306	0.06915	0.16290	0.24994	0.24994	0.16290
Num. offspring	6	7	8	9	10	
Probability	0.06915	0.01910	0.00343	0.00040	0.00002	

References

Antia, R., Levin, B., & May, R. (1994). Within-host population dynamics and the evolution and maintenance of microparasite virulence. *The American Naturalist*, *144*(3), 457–472.

Artois, M., Depner, K., Guberti, V., Hars, J., Rossi, S., & Rutili, D. (2002). Classical swine fever (hog cholera) in wild boar in europe. *Revue scientifique et technique (International Office of Epizootics)*, *21*(2), 287–303.

Bieber, C., & Ruf, T. (2005). Population dynamics in wild boar *Sus scrofa*: ecology, elasticity of growth rate and implications for the management of pulsed resource consumers. *Journal of Applied Ecology*, *42*, 1203–1213.

Boitani, L., Trapanese, P., Mattei, L., & Nonis, D. (1995). Demography of a wild boar (*Sus scrofa* l.) population in tuscany. *Gibier Faune Sauvage*, *12*, 109–132.

Dahle, J., & Liess, B. (1992). A review of classical swine fever infections in pigs: epizootiology, clinical disease, and pathology. *Comparative Immunology, Microbiology & Infectious Diseases*, *15*, 203–211.

Day, T. (2003). Virulence evolution and the timing of disease life-history events. *Trends in Ecology and Evolution*, *8*(3), 113–118.

Depner, K., Hinrichs, U., Bickhardt, K., Greiser-Wilke, I., Polenz, J., Moenning, V., & Liess, B. (1997). Influence of breed-related factors on the course of classical swine fever virus infection. *Veterinary Record*, *140*, 506–507.

Depner, K., Müller, T., Lange, E., Staubach, C., & Teuffert, J. (2000). Transient classical swine fever virus infection in wild boar piglets partially protected by maternal antibodies. *Deutsche Tierärztliche Wochenschrift*, *107*(2), 66–68.

EFSA (2008). Control and eradication of classic swine fever in wild boar - scientific report. *The EFSA Journal*, *932*, 1–18.

Focardi, S., Toso, S., & Pecchioli, E. (1996). The population modelling of fallow deer and wild boar in a mediterranean ecosystem. *Forest Ecology and Management*, *88*(1–2), 7–14.

Gaillard, J., Vassant, J., & Klein, F. (1987). Some characteristics of the population dynamics of wild boar (*Sus scrofa scrofa*) in a hunted environment. *Gibier Faune Sauvage*, *4*(1), 31–47.

Ganusov, V., & Antia, R. (2003). Trade-offs and the evolution of virulence of microparasites: do details matter? *Theoretical Population Biology*, *64*, 211–220.

Grimm, V., Berger, U., Bastiansen, F., Eliassen, S., Ginot, V., Giske, J., Goss-Custard, J., Grand, T., Heinz, S., Huse, G., Huth, A., Jepsen, J., Jørgensen, C., Mooij, W., Müller, B., Pe'er, G., Piou, C., Railsback, S., Robbins, A., Robbins, M., Rossmanith, E., Rüger, N., Strand, E., Souissi, S., Stillman, R., Vabø, R., Visser, U., & DeAngelis, D. (2006). A standard protocol for describing individual-based and agent-based models. *Ecological Modelling*, *192*, 115–126.

Grimm, V., Berger, U., DeAngelis, D., Polhill, J., Giske, J., & Railsback, S. (2010). The odd protocol: A review and first update. *Ecological Modelling*, *221*, 2760–2768.

Howells, O., & Edwards-Jones, G. (1997). A feasibility study of reintroducing wild boar *Sus scrofa* to scotland: are existing woodlands large enough to support minimum viable populations. *Biological Conservation*, *81*, 77–89.

Jedrzejewska, B., Jedrzejewski, W., Bunevich, A., Milkowski, L., & Krasinski, Z. (1997). Factors shaping population densities and increase rates of ungulates in bialowieza primeval forest (poland and belarus) in the 19th and 20th centuries. *Acta Theriologica*, *42*(4), 399–451.

Jezierski, W. (1977). Longevity and mortality rate in a population of wild boar. *Acta Theriologica*, *22*(20–29), 337–348.

Kaden, V., Lange, E., Fischer, U., & Strebelow, G. (2000). Oral immunisation of wild boar against classical swine fever: evaluation of the first field study in germany. *Veterinary Microbiology*, *73*, 239–252.

Kramer-Schadt, S., Fernández, N., Eisinger, D., Grimm, V., & Thulke, H.-H. (2009). Individual variations in infectiousness explain long-term disease persistence in wildlife populations. *Oikos*, *118*, 199–208.

Laddomada, A. (2000). Incidence and control of csf in wild boar in europe. *Veterinary Microbiology*, *73*, 121–130.

Leaper, R., Massei, G., Gorman, M., & Aspinall, R. (1999). The feasibility of reintroducing wild boar (*Sus scrofa*) to scotland. *Mammal Review*, *29*(4), 239–258.

Liess, B. (1987). Pathogenesis and epidemiology of hog cholera. *Annales de Recherches Veterinaires*, *18*(2), 139–145.

May, M., & Anderson, R. (1983). Epidemiology and genetics in the coevolution of parasites and hosts. *Proceedings of the Royal Society B*, *219*, 281–313.

Melis, C., Szafranska, P., Jedrzejewska, B., & Barton, K. (2006). Biogeographical variation in the population density of wild boar (*Sus scrofa*) in western eurasia. *Journal of Biogeography*, *33*, 803–811.

Moennig, V., Floegel-Niesmann, G., & Greiser-Wilke, I. (2003). Clinical signs and epidemiology of classical swine fever: A review of a new knowledge. *Veterinary Journal*, *165*, 11–20.

Rossi, S., Pol, F., Forot, B., Masse-provin, N., Rigaux, S., Bronner, A., & Le Potier, M.-F. (2010). Preventive vaccination contributes to control classical swine fever in wild boar (*Sus scrofa sp.*). *Veterinary Microbiology*, *142*(1–2), 99–107.

Sodeikat, G., & Pohlmeyer, K. (2003). Escape movements of family groups of wild boar *Sus scrofa* influenced by drive hunts in lower saxony, germany. *Wildlife Biology*, *9 (Suppl. 1)*, 43–49.

Viet, A.-F., & Jacob, C. (2008). Illustration of some limits of the markov assumption for transition between groups in models of spread of an infectious pathogen in a structured herd. *Theoretical Population Biology*, *74*(1), 93–103.

Wearing, H., Rohani, P., & Keeling, M. (2005). Appropriate models for the management of infectious diseases. *PLoS Medicine*, *2*(7).