ODD of the host-pathogen model "SwiFCoIBM"

Cédric Scherer[1*], Martin Lange[2], Volker Grimm[2], Hans-Hermann Thulke[2], Stephanie Kramer-Schadt[1]

[1] Department of Ecological Dynamics, Leibniz Institute for Zoo and Wildlife Research, Berlin, Germany

[2] Department of Ecological Modelling, Helmholtz Centre for Environmental Research-UFZ, Leipzig, Germany

* scherer@izw-berlin.de

The classical swine fever (CSF) pathogen – wild boar host model is a combination of a spatially explicit, stochastic, agent-based model for wild boars (*Sus scrofa* L.) and an epidemiological model for the CSF virus interacting with the host's life-history, thus the system or community consist of two species, with the pathogen species never occurring outside the host (no indirect transmission and survival outside alive hosts). The model was implemented in NetLogo (Wilensky, 1999), has been parameterized with knowledge from field experiments and clinical studies and is documented in each of the publications mentioned below following the ODD protocol (Grimm et al., 2006, 2010).

1 Purpose

The original model (Kramer-Schadt et al. 2009) was used to assess intrinsic (system immanent host-pathogen interaction and host life-history) and extrinsic (spatial extent and density) factors contributing to the long-term persistence of the disease and has further been used to assess the effects of intrinsic dynamics (Lange et al. 2012a) and indirect transmission (Lange et al. 2016) on the disease course. In an applied context, the model was used to test the efficiency of spatiotemporal vaccination regimes (Lange et al. 2012b) as well as the risk of disease spread in the country of Denmark (Alban et al. 2005).

2 Entities, state variables and scales

The model comprises two major components, a wild boar host demography model considering seasonal reproduction, natal dispersal (of both, subadult females and males) and mortality, and a CSF virus model operating on the boar population via within- and between-group infection. Wild boar population density and structure are influenced by the disease via virus-induced mortality and litter size depression.

The model entities are spatial units, or grid cells, and wild boars. Grid cells are characterized by habitat quality in terms of breeding capacity, i.e. the number of female boars that are allowed to have offspring (Jedrzejewska, Jedrzejewski, Bunevich, Milkowski, & Krasinski, 1997). Thereby, local host density is regulated in the model, i.e. increasing numbers of fertile females can breed only until breeding capacity is reached. A grid cell represents about 2 km × 2 km, encompassing an average home range area of a boar group (Leaper, Massei, Gorman, & Aspinall, 1999). The landscapes are heterogeneous with random distributed habitat qualities ranging between 0 and 9 breeding females.

The crucial entity, individual wild boars, are characterized by sex, age in weeks, location, demographic status (e.g. breeding, dispersing and ranging) and health status. Three age classes are distinguished: piglet (< 34 weeks), subadult (between 34 weeks and < 1 year for females and < 2 years for males) and adult. Location is defined by the grid cell the wild boar inhabits. The health status of the individuals is described by an SIR epidemiological classification (susceptible; transiently infected; lethally infected with individual infectious period; immune by surviving the infection or by maternal antibodies). Females, which are at least subadult, may be assigned as breeders according to the breeding capacity of their family group's cell. Subadult wild boars may disperse during the dispersal period dependent on their sex and demographic status (disperser or non-disperser).

One time step corresponds to the approximate CSF incubation time of one week (Artois et al., 2002; Moennig, Floegel-Niesmann, & Greiser-Wilke, 2003) while simulations usually run for 12 years (624 weeks) with the virus being released in the second year (week 53–104) to a defined boar group to ensure the same distance to the model borders and an established spatial population structure. The model landscape consists of 100 km × 50 km (50 × 25 grid cells).

3 Process Overview and Scheduling

Each time step, the following procedures are executed by the wild boars in the given order (Fig. 1): pathogen transmission, natal dispersal of males and females, respectively, reproduction, mortality, ageing, and disease course. In the first week of each year, females are assigned to breed. Natal dispersal of males and females was limited to week 17 and week 29 of the year, respectively.

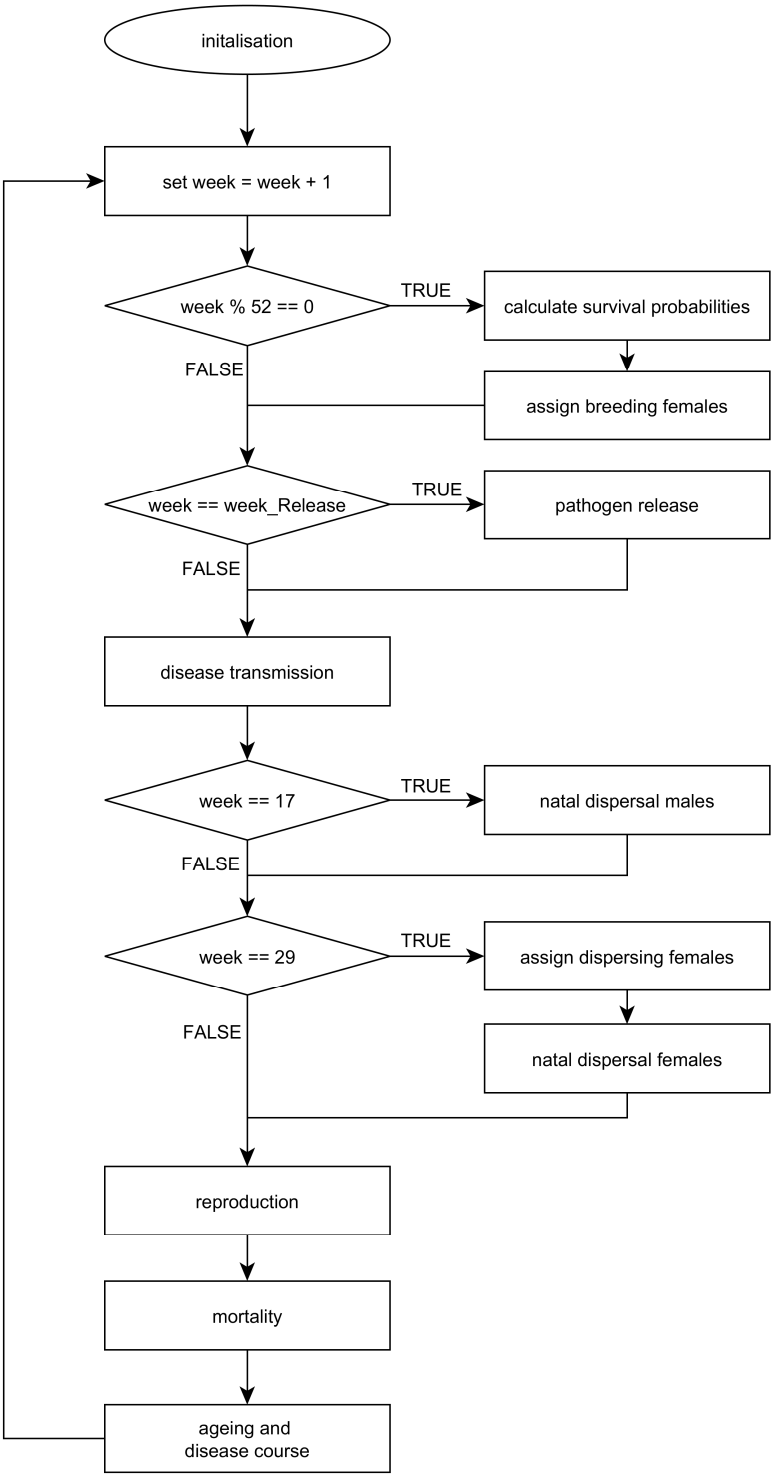

Fig. 1. Schedudling of the model's processes.

4 Design concepts

Basic principles

Both processes of the host and the pathogen are simulated with a given stochasticity which resembles natural conditions. Disease transmission for groups is modelled density-dependent (see Eq. 1 and 2).

Emergence

Wild boar population dynamics emerge from individual behaviour, resulting from age- and sex-dependent movement behaviours (natal dispersal) as well as age-dependent seasonal reproduction and survival probabilities. The epidemic course emerges from different virus transmission probabilities as well as individual stochastic disease courses and infectious periods.

Adaptation

The number of breeding females per cell is determined by (a) the habitat quality of the cell and (b) the number and age of the reproductive females within the cell.

Objectives

Dispersing individuals search for empty (unoccupied) cells (in case of female dispersers) or join other groups that are below their cells' carrying capacity (in case of male dispersers) within their search range.

Learning

There is no learning implemented in our model.

Prediction

Since the landscape structure is static there is no need to predict future environmental conditions.

Sensing

Individuals can sense if and how many other animals are within a cell in their search range when dispersing.

Interaction

Based on the transmission rates, interactions between individuals might lead to virus transmission from an infected to a susceptible individual. Reproduction is density-and age-dependent with the oldest females giving birth first. Thus, if the number of reproducible females exceeds a cell's breeding capacity, interaction leads to repressed reproduction.

Stochasticity

Demographic and behavioural parameters are imposed via probability distributions to account for variation in the biological processes. Stochastic individual disease courses and infectious periods are modelled explicitly because variation in the disease outcome between individuals was identified to be essential for virus endemicity without reservoirs (Kramer-Schadt, Fernández, Eisinger, Grimm, & Thulke, 2009).

Collectives

Individuals form groups within their cell that experience density-dependent transmission probabilities. During natal dispersal, male subadults may join those groups while female subadults may form new groups.

Observation

To evaluate model outcomes, we measured several properties of the host-virus system at each time step of the simulation. These outputs include the overall number of individuals as well as

per health status (susceptible, infected, recovered) and their combinations. Furthermore, the last week of infection are recorded. From this output, duration of an outbreak as well as probability of disease persistence (i.e. the virus being present in the system until the end of simulations) can be estimated.

5 Initialization

One boar family group was allocated to each cell with an average breeding capacity B of 4.5 females, resulting in the reported density of approximately 20 boars per cell or 5 boars per km² (EFSA (European Food Safety Authority), 2009; Howells & Edwards-Jones, 1997; Melis, Szafranska, Jedrzejewska, & Barton, 2006; Sodeikat & Pohlmeyer, 2003). We simulated heterogeneous landscapes with a breeding capacity B between 0 and 9 assigned to each of the 2500 grid cells ($B \in \{0;9\}$). In case B is a floating point number, the number of individuals is determined stochastically based on the remainder. Initial age distribution was obtained from the results of a 100-year model run conducted by (Kramer-Schadt et al., 2009); Table S2), the sex ratio was balanced (i.e. probability of 0.5 to be either male or female). Wild boar density reflects long-term average values of densely populated Central European habitats (EFSA (European Food Safety Authority), 2009; Howells & Edwards-Jones, 1997; Melis, Szafranska, Jedrzejewska, & Barton, 2006; Sodeikat & Pohlmeyer, 2003). Group size was initialised according to the cells carrying capacity K_i that is 4.5 times its breeding capacity B_i.

6 Input

The model does not include external input representing environmental conditions changing over time.

7 Submodels

Initial infection (pathogen release)

The virus is released to the population by infection of one wild boar group (i.e. cell) in middle of the upper row of home ranges (i.e. cell with the coordinates x = 1 and y = 13) to allow for comparison between runs. The release is scheduled in a random week of the second year of the model run.

Pathogen transmission

Virus transmission is modelled stochastically. The transmission parameter determines the weekly probability of being infected by an infectious group mate β_w. Weekly infection pressure λ_i for each susceptible individual in cell i is determined by the probability of being infected by an infectious group mate β_w (within-group transmission probability) and the probability of being infected by an infectious individual in one of the eight neighbouring cells β_e (between-group transmission probability):

$$\lambda_i = 1 - (1 - \beta_w)^{I_i} \cdot (1 - \beta_e)^{\Sigma_j I_j} \quad [1]$$

where I_i is the number of infectious group mates and I_j is the numbers of infectious hosts in the j^{th} adjacent cell. The resulting probability value λ_i provides the parameter of a binomial chance process to decide whether a susceptible animal will be infected.

The transmission parameters β_w (and thereby β_e since it was fixed as one tenth of β_w) was calibrated in order to reproduce the spreading velocity observed in France (Rossi et al., 2010) with the constant parameter value β_w = 0.0208 within and, hence, β_e = 0.00208 between groups (Kramer-Schadt et al., 2009).

CSF shows a variety of disease courses on the individual level (Depner et al., 1997; Liess, 1987). Therefore, in our model the disease course is stochastically specified for each individual. The disease course submodel is described by two parameters: individual case mortality M and the mean infectious period of lethally infected hosts μ. Upon infection the host is stochastically assigned either as lethally infected (with probability M) or as transiently infected ($1 - M$). M is age-specific (Dahle & Liess 1992): for adults the probability is decreased to $M_a = M^2$ and for piglets increased to $M_p = M$ while it is unchanged for yearlings $M_y = M$. Transiently infected wild boars first pass through an infectious period of one week and subsequently becomes non-infectious and gain life-long immunity (Artois et al. 2002; Moennig, Floegel-Niesmann, & Greiser-Wilke, 2003; EFSA 2009). The individual infectious period (m_i in weeks) of lethally infected hosts is drawn from an exponential distribution with the mean specified by parameter μ:

$$m_i = 1 + floor(-(\mu - 0.5) \cdot ln(U(0,1))) \quad [2]$$

where $U(0,1)$ is a uniformly distributed random number between 0 and 1. To avoid unrealistically long infections, m_i was stochastically assigned until $m_i \leq 10 \times \mu$. Lethally infected hosts remain infectious until death. Offspring from immune female breeders gets maternal antibodies and is thus immune for the first eight to twelve weeks after birth (t_{anti}). The number of weeks of immunisation due to maternal antibodies is randomly assigned for those piglets.

Natal dispersal

Herd splitting, where subadult individuals may move together to search for or form new groups, is performed in specified weeks of the year only. The timing of these events is sex-dependent: Subadult females without offspring perform their natal dispersal in the 29th week of the year, while subadult males disperse during the 17th week. In the given week of the year, all herds to split are extracted, matching the conditions of containing at least a specified number of subadults N_{disp} to move (either male or female). For female dispersal, only cells exceeding the breeding capacity B are evaluated. Splittable herds are iterated randomly. For each of them, a habitat cell not exceeding carrying capacity K (for males) or without any family group (for females) within a Euclidean distance d_{disp} is selected randomly as new cell for the group, excluding the source cell. If there is no cell fulfilling these conditions, subadults stay within their group's cell.

Reproduction

Females reproduce once a year, depending on their age class. Individual females, which are at least subadult, reproduce depending on the season with a peak in March and no reproduction in winter from October to December (Boitani et al. 1995; Table S2). In the first week of the year, female individuals are checked for their breeding status. All females not exceeding their habitat cells breeding capacity, starting with the oldest individuals, are allowed to breed. The week of the year to breed is assigned in the first week of each year according to weekly reproduction probabilities, derived from monthly probabilities and the number of weeks in the month (Table S2). Litter size is drawn from a pre-calculated truncated normal distribution (Table S3 based on

Bieber & Ruf 2005) 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_i.

Depending on the disease state of the breeding individual, its piglets disease states are adjusted. Susceptible individuals produce susceptible offspring, immune individuals produce immune offspring with maternal antibodies (see section "Pathogen state transition"). Transient shedders and lethally infected individuals yield offspring, each one lethally infected with a given probability of prenatal infection p_{pi}.

Mortality

Stochastic baseline mortality is age-dependent and adjusted to annual survival estimates found in the literature (Focardi, Toso, & Pecchioli 1996; Gaillad, Vassant, & Klein 1987; Table S1). Per time step we apply the adjusted age-dependent mortality (m_{week}) to the individual:

$$m_{week} = 1 - (s_{year})^{1/52}. \quad [3]$$

In addition to the stochastic baseline mortality, each individual may die due to reaching a certain maximum age, or due to a lethal infection after a certain infection time span m_i (see section "Pathogen transmission").

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. Females become subadult and adult at an age of 34 and 52 weeks, respectively, while males enter the subadult and adult age groups at an age of 21 and 104 week, respectively.

Disease course (pathogen state transition)

Transient shedders convert to immune after a certain latency period t_{latent}. An individual i protected by maternal antibodies turns susceptible if reaching an age T_i of the protection time of t_{anti} (see section "Pathogen transmission"). After disease state transition the age of the infection is incremented by one week if the individual is not susceptible.

Tables

Table S1: Parameter setup

Table S1: Parameter setup used in the spatially-explicit Classical Swine Fever-wild boar model. Each of the 60 combinations (3 movement strategies × 4 landscape scenarios × 5 case fatality ratios) was repeated 200 times while generating new underlying landscapes depending on the scenario input, resulting in 12.000 runs in total.

Parameter	Value	Reference(s)
Longevity	11 years (572 weeks)	(Jezierski, 1977)
Sex ratio	1:1	(e.g. Durio, Gallo Orsi, Macchi, & Perrone, 2014; Fernández-Llario, Carranza, & Mateos-Quesada, 1999; Moretti, 2014)
Survival probability of adults and subadults	s_{mean}= 0.6; s_{min} = 0.4	(Focardi, Toso, & Pecchioli, 1996; Gaillard, Vassant, & Klein, 1987)
Survival probability of piglets	s_{mean}= 0.5; s_{min} = 0.1	(Focardi, Toso, & Pecchioli, 1996)
Reproduction probability	see Table S2	(Boitani, Trapanese, Mattei, & Nonis, 1995)
Breed count distribution	see Table S3	(Bieber & Ruf, 2005)
Maximal natal dispersal distance d_{disp}	3 cells (6 km)	(Sodeikat & Pohlmeyer, 2003)
Minimum number of dispersers N_{disp}	2 for females; 3 for males	

Case fatality ratio M	{0, 0.25, 0.5, 0.75, 1}	
Mean infectious period μ	4 weeks	
Within-group transmission probability β_w	0.0208	(Kramer-Schadt et al., 2009)
Between-group transmission probability β_e	0.00208	(Kramer-Schadt et al., 2009)
Fertility reduction due to infection	0.625	(Kramer-Schadt et al., 2009)
Probability of prenatal infection	0.5	(Kramer-Schadt et al., 2009)
Transient period t_{trans}	1 week	(Artois et al., 2002; Moennig et al., 2003)
Period of maternal antibodies t_{anti}	12 weeks	(Depner, Müller, Lange, Staubach, & Teuffert, 2000)
Simulated years	12 (624 weeks)	
Pathogen release	random week of the 2nd year	
Mean number of reproductive females cell^{-1}	4.5	(EFSA (European Food Safety Authority), 2009; Howells & Edwards-Jones, 1997; Sodeikat & Pohlmeyer, 2003)
Habitat quality range	[0, 9]	
Initial mean density	5 individuals/km^2	(Kramer-Schadt et al., 2009)
Initial age distribution	see Table S4	(Kramer-Schadt et al., 2009)
Age blur in initial individuals	± 3 weeks	

Table S2: Monthly reproduction probabilities

Table S2: Monthly reproduction probabilities (Boitani et al., 1995) used to stochastically determine the number of breeding females.

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 probability	0.0	0.1	0.23	0.34	0.07	0.08	0.06	0.03	0.03	0.0	0.0	0.0

Table S3: Breed count distribution

Table S3: Breed count distribution (Bieber & Ruf, 2005) used to estimate litter sizes.

Litter size	**0**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Probability	.01306	.06915	.01629	.24994	.24994	.01629	.06915	.01910	.00343	.0004	.00002

Table S4: Initial age distribution

Table S4: Initial age distribution (Kramer-Schadt et al., 2009) used to initialize each model run.

Age (years)	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Proportion	0.38	0.24	0.15	0.09	0.06	0.03	0.02	0.01	0.01	0.01

References

Alban, L., Andersen, M.M., Asferg, T., Boklund, A., Fernandez, N., Greiner, M., Kramer-Schadt, S., Stockmarr, A., Thulke, H.-H., Uttenthal, Å., Ydesen, B. (2005). Classical swine fever and wild boar in Denmark: A risk analysis. Project Report, Danish Institute for Food and Veterinary Research DFVF. ISBN: 87-91587-01-8. 118 pages.

Artois, M., Depner, K. R., Guberti, V., Hars, J., Rossi, S., & Rutili, D. (2002). Classical swine fever (hog cholera) in wild boar in Europe. *Revue Scientifique et Technique*, *21*(2), 287–303. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/11974616

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. *The Journal of Applied Ecology*, *42*(6), 1203–1213. doi:10.1111/j.1365-2664.2005.01094.x

Boitani, L., Trapanese, P., Mattei, L., & Nonis, D. (1995). Demography of a wild boar (*Sus scrofa*, L.) population in Tuscany, Italy. *Gibier, Faune Sauvage / ONC, Office National de La Chasse*, *12*(2), 109–132.

Depner, K. R., Hinrichs, U., Bickhardt, K., Greiser-Wilke, I., Pohlenz, J., Moennig, V., & Liess, B. (1997). Influence of breed-related factors on the course of classical swine fever virus infection. *The Veterinary Record*, *140*(19), 506–507. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/9172299

Depner, K. R., 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 Tierarztliche Wochenschrift, 107(2), 66–68. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/10743336 Durio, P., Gallo Orsi, U., Macchi, E., & Perrone, A. (2014). Structure and monthly birth distribution of a wild boar population living in mountainous environment. *Journal of Mountain Ecology*, *3*(0). Retrieved from http://www.mountainecology.org/index.php/me/article/view/112

EFSA (European Food Safety Authority). (2009). Control and eradication of Classical Swine Fever in wild boar. *European Food Safety Authority Journal*, *932*(EFSA-Q-2007-200), 1–18. Retrieved from https://efsa.onlinelibrary.wiley.com/doi/abs/10.2903/j.efsa.2009.932

Fernández-Llario, P., Carranza, J., & Mateos-Quesada, P. (1999). Sex allocation in a polygynous mammal with large litters: the wild boar. *Animal Behaviour*, *58*(5), 1079–1084. doi:10.1006/anbe.1999.1234

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), 7–14. doi:10.1016/S0378-1127(96)03804-2

Gaillard, J. M., Vassant, J., & Klein, F. (1987). Quelques caractéristiques de la dynamique des populations de sangliers (*Sus scrofa scrofa*) en milieu chassé. *Gibier, Faune Sauvage / ONC, Office National de La Chasse*, *4*, 31–47.

Grimm, V., Berger, U., Bastiansen, F., Eliassen, S., Ginot, V., Giske, J., … DeAngelis, D. L. (2006). A standard protocol for describing individual-based and agent-based models. *Ecological Modelling*, *198*(1), 115–126. doi:10.1016/j.ecolmodel.2006.04.023

Grimm, V., Berger, U., DeAngelis, D. L., Polhill, J. G., Giske, J., & Railsback, S. F. (2010). The ODD protocol: A review and first update. *Ecological Modelling*, *221*(23), 2760–2768. doi:10.1016/j.ecolmodel.2010.08.019

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*(1), 77–89. doi:10.1016/S0006-3207(96)00134-6

Jedrzejewska, B., Jedrzejewski, W., Bunevich, A. N., Milkowski, L., & Krasinski, Z. A. (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*, *4*(42). Retrieved from https://www.infona.pl/resource/bwmeta1.element.agro-article-f12b76e6-383a-4497-809a-9ca2f1205a93

Jezierski, W. (1977). Longevity and mortality rate in a population of wild boar. *Acta Theriologica*, *22*(24), 337–348. Retrieved from http://rcin.org.pl/dlibra/docmetadata?id=10471

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*(2), 199–208. Retrieved from https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1600-0706.2008.16582.x

Lange, M., Kramer-Schadt, S., Thulke, H.-H. (2016). Relevance of indirect transmission for wildlife disease surveillance. *Frontiers in Veterinary Science 3*, 110. doi: 10.3389/fvets.2016.00110

Lange, M, Kramer-Schadt S, Thulke HH (2012a). Efficiency of spatio-temporal vaccination regimes in wildlife populations under different viral constraints. *Veterinary Research*, *43,* 37.

Lange, M, Kramer-Schadt, S., Blome, S., Beer, M., Thulke, H.-H. (2012b). Disease severity declines over time after a wild boar population has been affected by Classical Swine Fever – Legend or actual epidemiological process? *Preventive Veterinary Medicine 106(2),* 185-195.

Leaper, R., Massei, G., Gorman, M. L., & Aspinall, R. (1999). The feasibility of reintroducing Wild Boar (*Sus scrofa*) to Scotland. *Mammal Review*, *29*(4), 239–258. doi:10.1046/j.1365-2907.1999.2940239.x

Liess, B. (1987). Pathogenesis and epidemiology of hog cholera. *Annales de Recherches Veterinaires. Annals of Veterinary Research*, *18*(2), 139–145. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/3304111

Melis, C., Szafranska, P. A., Jedrzejewska, B., & Barton, K. (2006). Biogeographical variation in the population density of wild boar (*Sus scrofa*) in western Eurasia. *Journal of Biogeography*, *33*(5), 803–811. doi:10.1111/j.1365-2699.2006.01434.x

Moennig, V., Floegel-Niesmann, G., & Greiser-Wilke, I. (2003). Clinical signs and epidemiology of classical swine fever: a review of new knowledge. *Veterinary Journal* , *165*(1), 11–20. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/12618065

Moretti, M. (2014). Birth distribution, structure and dynamics of a hunted mountain population of Wild boars (*Sus scrofa* L.), Ticino, Switzerland. *Journal of Mountain Ecology*, *3*(0). Retrieved from http://mountainecology.org/index.php/me/article/view/110

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. doi:10.1016/j.vetmic.2009.09.050

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*(1), 43–49. doi:10.2981/wlb.2003.063

Wilensky, U. (1999). NetLogo (Version 6.0.4). Retrieved from http://ccl.northwestern.edu/netlogo/