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Incidence and persistence of classical swine fever in free-ranging wild boar (*Sus scrofa*)

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SUMMARY

Although veterinary authorities aim to limit persistence of classical swine fever (CSF) in wild boar (*Sus scrofa*), to avoid potential transmission to pigs, factors influencing CSF transmission and persistence are not clearly understood. Here we analyse incidence and persistence in a CSF epidemic that occurred in the French Vosges Forest. Higher incidence was found in large forests compared to smaller isolated ones, being highest near the starting point of the epidemic, but poorly related to the local density. We hypothesize that the spatial and social structure of wild boar populations may be responsible for this variability of incidence over space. Persistence was highest near the starting point of the epidemic and where initial density was highest. We hypothesize that persistence was favoured by the abundance of young wild boar, itself encouraged by CSF. Our results allow us to propose management measures aimed at limiting CSF persistence.

INTRODUCTION

Classical Swine Fever (CSF) is a viral disease affecting wild and domestic swine worldwide. Outbreaks occurring in domestic pigs entail severe losses to the pig-farming industry because the virus is highly contagious and may cause 20–90% mortality [1, 2]. Massive slaughtering is required to eradicate it and CSF-free countries restrict pig trade during outbreaks. For example, an outbreak in 1997 in The Netherlands caused more than 2.5 billion euros of losses [3]. Free-ranging populations of European wild boar (*Sus scrofa*) are regarded as potential reservoirs of CSF [4]. As CSF virus is able to survive in fomites and meat for several months [5], cross-contamination between pig and wild boar may occur either through direct contact between wild boar and domestic swine, or through the introduction of contaminated feed. In Germany, 46% of primary outbreaks recorded in domestic pigs between 1993 and 1997 were ascribed to contact with wild boar [6]. Thus, characterizing

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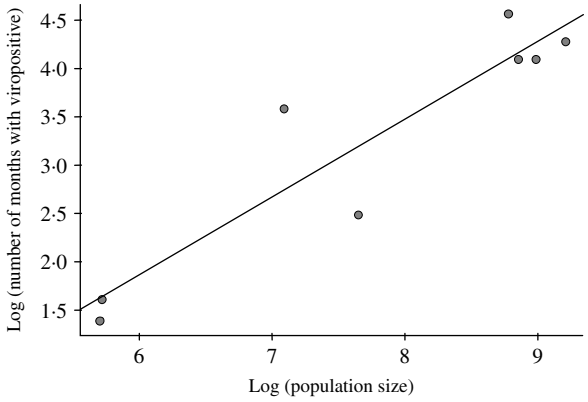

Fig. 1. Relationship between classical swine fever (CSF) persistence (months with virus isolation, log-transformed) and estimated population size (log-transformed) among epidemics of CSF in wild boar [7, 11–13, 18, 22]. Pearson $R^2 = 0{\cdot}860$, $P < 0{\cdot}001$.

CSF epidemiology in wild boar would be a preliminary step towards its control.

A first aspect of CSF epidemiology in wild boar that warrants attention is the variability of disease incidence. Incidence is supposed to depend on the rate of contact between susceptible and infected individuals [4, 7]. Under the assumption that transmission among wild boar occurs mainly through direct contact, mathematical models predicted that CSF incidence would increase with host density and that CSF would become extinct under a threshold density of susceptible hosts [8, 9]. We thus aimed to study the density-dependence of incidence. We also thought about the evolution of incidence in space and aimed to test the effect of connectivity among habitat patches which has been demonstrated to be of major importance in other host–virus systems [10].

A second aspect of CSF epidemiology in wild boar worthy of note is disease persistence. With the exception of Sardinia, where particular conditions of mixing occur between domestic and wild pigs, CSF is epidemic and unavoidably becomes extinct after one or several years [4, 7, 11, 12]. The comparison of different epidemics in wild boar shows that time to extinction is strongly correlated to population size (Fig. 1) [4, 7, 13]. Several mechanisms may explain this relationship between population size and disease persistence. A first mechanism may be related to population renewal, because CSF infection induces either rapid death or recovery with life-long immunity, so that new susceptible hosts are required for the virus to persist [14–16]. According to the concept of 'critical community size', larger populations would be at risk of longer persistence because they produce many piglets which increases the pool of susceptible individuals [2, 4, 14, 17, 18]. Alternatively, the largest wild boar populations can experience high density, therefore, density has to be analysed in addition to population size [2, 7]. Other factors may also alter persistence such as birth rate and connectivity among habitat patches. Field studies showed that populations subjected to intensive hunting pressure experienced long persistence, possibly because hunting pressure maintains a high birth rate through density-dependence [9, 11, 14]. Finally, connectivity among habitat patches may act on persistence by affecting the rate of infectious contacts among subpopulations [19].

In this paper, we aimed to investigate factors related to incidence and persistence at the level of one epidemic that occurred in France between 1992 and 2001 [20–22]. This epidemic emerged in early 1992 in the municipality of Philippsbourg and spread during 2 years. Incidence then slowly decreased; no viropositive case was detected after 1997 and the last young seropositive was detected in early 2001 [22] (Rossi et al., unpublished observations). From 1992 up to 2001, infection was monitored at the scale of the municipality, which allowed us to study incidence and persistence among spatial units. We tested whether incidence and persistence were influenced by wild boar density (D_t), distance from the starting point of the epidemic (*Dist*) and connectivity to a large forest (*Connect*) that we assumed as representative of connectivity among wild boar subpopulations.

MATERIALS AND METHODS

Study area and sampling

The study area is located in North-eastern France (Fig. 2) and was defined by veterinary authorities at the beginning of the epidemic (January 1992) in order to follow-up the progress of the epidemic. Motorways, rivers, urban zones and the borderline with Germany constitute the limits of the study area, which covers over 3030 km² including 1180 km² of woodland [22]. Data were obtained according the recommendation of the European Community [23]. Since January 1992, two zones were delineated: the *infected* zone included all municipalities where viropositive cases (see below for definition) had been encountered during the preceding 6 months, while

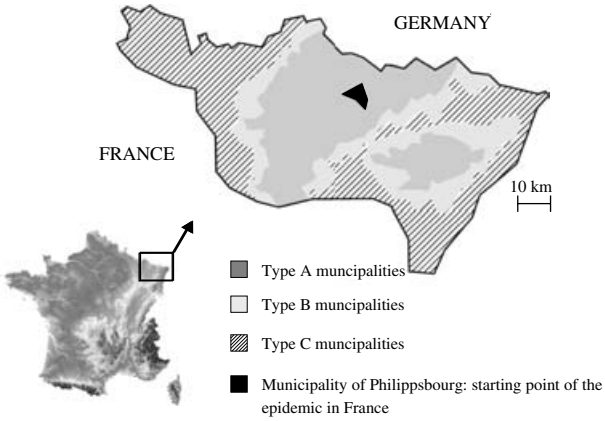

Fig. 2. Survey area and distribution of municipalities according the three types of connectivity to a large forested area (*Connect*).

other municipalities of the area constituted the *control* zone. The sampling plan intended to include all wild boar that had been shot in the *infected* zone, at least 30% of the individuals that had been shot in the *control* zone and all wild boar that had been found dead in the whole area. Sampling ended in January 2002 because no incident case (see below for definition) had been discovered during 1 year. For each individual boar, a questionnaire was completed, and the spleen and a blood sample were collected.

Laboratory diagnosis

A two-step procedure was adopted to detect CSF virus (CSFV) in the spleen. A first screening was performed using a commercial ELISA (Serelisa HCV Ag Mono Indirect, Synbiotics Europe, Lyon, France) to detect CSFV p-125. This first test was performed by local laboratories. All positive or doubtful ELISA samples were sent to the French reference laboratory for CSF [(AFSSA), French Agency for Food Safety, Maisons-Alfort, France] in order to perform virus isolation. Virus isolation is the reference method defined by the European Community as it maximizes both sensitivity and specificity [23]. Thus, individuals were considered definitely viropositive when showing a positive reaction to ELISA and virus isolation.

In sera, the French reference laboratory searched for anti-CSFV antibodies using a neutralization peroxidase-linked assay [23]. This test is more sensitive and specific than other available serological methods. Additionally the AFSSA differentiate CSFV and the closely related bovine viral diarrhoea virus by the comparison of serum titres regarding both viruses [24]. Seropositive cases included animals showing positive reaction at a 1/10 dilution with CSFV.

Definition of incidence and persistence

From March 1992 to February 2001, time (t in years) was partitioned into nine 12-month periods. This partition was done to include one reproduction period (from March to September) and the entire following hunting season (from October to February) in a given year (following recommendations of local game experts). Incidence and persistence were the two variables analysed in this study. We estimated incidence as the frequency of viropositive animals in our sample in a given year. This frequency estimates the proportion of wild boar that were infected a few weeks before being killed because the virus is no longer detectable in the spleen after a few weeks [2]. We studied risk factors of incidence at the individual level.

We estimated persistence as the number of years between the first and the last incident case detected in a given municipality. We classified as incident cases all individuals that had definitely been infected within a year, i.e. any viropositive individual, any seropositive individual during 1992, and any young seropositive after 1992. After 1992, seropositive individuals older than 1 year could not be classified as recently infected because antibodies are lifelong [2, 25]. We analysed the variability of persistence at the municipality level.

Explicative variables

Individual characteristics

Age (*Age*), gender (*Gender*) and capture method (*Cap*, hunted, found dead) were recorded in the questionnaires completed by hunters and taken into account as potential confounding variables in the analysis of incidence. We defined three age classes (*young*, *subadult*, *adult*) according to coat colour and body mass, in accordance with previous field studies [26]. *Young* individuals were all reddish-brown coated or strayed wild boar and were assumed to be aged <12 months. *Subadult* individuals were all black-coated wild boar whose carcass weighed <50 kg, supposedly corresponding to 13- to 24-month-old animals. Black-coated wild boar with carcass heavier than 50 kg were called *adult*, i.e. considered >24 months old.

Municipality characteristics

The spatial unit was the municipality and the location of a municipality was assimilated to its centroid coordinates (x_i, y_i). We aimed to study the influence of *Dist*, *Connect* and host density on incidence and persistence. We calculated the distance from the starting point of the epidemic (*Dist*, in km), i.e. the distance between the centroid of each municipality and the centroid of Philippsbourg municipality where the first infected wild boar has been reported [20].

Wild boar are most often found in forested areas, so the fragmentation of forests may lead to the fragmentation of wild boar populations into subpopulations [27]. Thus, we defined three classes of *Connect* in order to describe connectivity among subpopulations (Fig. 2). When completely included within large continuously forested areas (Vosges forest, 815 km²; Haguenau forest, 370 km²), municipalities were classified as *type A*. *Type B* included municipalities on the borderline between continuously forested areas and open areas. Outside of large forested areas, municipalities were classified as *type C*. In type C municipalities, forest cover is not continuous, therefore animal contacts and dispersal among municipalities are assumed to be limited compared to municipalities belonging to large forested areas (type A) [27]. Thus, we hypothesized that CSF transmission would be facilitated in type A compared to type C, entailing higher incidence and persistence in type A compared to type C. Type B municipalities would exhibit an intermediate situation.

Hunters declared their hunting bag per year and per municipality. In each municipality, we considered hunting bag per hectare as an estimator of the local density of wild boar (D_t at t). No data were available to compare hunting pressure among municipalities. We thus hypothesized that hunting pressure was identical in all municipalities. We used the hunting bag of the 1991–1992 hunting season as an estimator of density before the epidemic (initial density, D_0).

Statistical analysis

Factors related to CSF incidence

We analysed the effect of seven variables on incidence. The effects of *Age*, *Gender* and *Cap* were tested to account for individual differences in susceptibility to CSFV and adjust for discrepancies in the sample structure. Then we tested the effects of t, *Dist*, *Connect*, and D_0. We built all possible models including variables and interactions and selected the model with the lowest Akaike's information criteria (AIC) value. When models had close AIC we retained the most parsimonious model, i.e. with less parameters. We tested the goodness-of-fit of the chosen model by comparing its deviance to the deviance of the null model. The chosen model was also examined by testing differences between modalities of qualitative variables with Wald tests [28, 29].

We tested the effects of density measured before t (D_{t-3}, D_{t-2} and D_{t-1}) using a logistic regression in order to test the density-dependence of incidence. By testing the effects of density measured at t or after t (D_t, D_{t+1}, D_{t+2} and D_{t+3}) we aimed to test whether incidence during a given year affected D_t in the following years. We then represented model coefficients and their confidence intervals on a correlogram.

Factors related to persistence

Regarding persistence, we tested the effects of *Dist*, *Connect*, D_0. The effect of factors were tested with linear modelling, using an ascendant process. Models were compared using F tests and the quality of the chosen model was assessed by measuring the proportion of variance explained (R^2). All statistical analyses were performed with Splus 2000 software (© 1988–1999 Mathsoft), using a threshold P value of 0·05.

RESULTS

During the 9 years of survey, 215 spleens and 62 sera were collected and tested from wild boar that had been found dead. However, the sampling was not evenly distributed: 126 samples were collected from March 1992 to February 1993, and less than 30 per year thereafter. During the same period 15 593 spleens and 8013 sera were analysed from hunted wild boar. In average 1808 hunted wild boar were sampled each year, with a minimum of 925 in the second season (1993–1994) and a maximum of 2495 in the third year (1994–1995). In the first season (1992–1993) hunters sampled proportionally less young wild boar than in other hunting seasons ($P<0·001$).

Risk factors of infection

No viropositive case was discovered after 1997 [22], we therefore tested the effects of risk factors on incidence using data from 1992 to 1997, including

Table 1. *Coefficients of the final model chosen to explain incidence*

Variable class	Coefficients (S.E.)	*P* value (Wald test)
Intercept	−0·94 (0·57)	0·099
Capture method, Cap	**1·54 (0·41)**	**<0·001**
Time: t (year)	**−0·84 (0·13)**	**<0·001**
Age 1: subadults vs. youngs	**−2·17 (0·63)**	**<0·001**
Age 2: adults vs. youngs	**−3·89 (1·23)**	**<0·001**
Distance: Dist (km)	**−0·02 (0·01)**	**0·045**
Initial density: D_0 (hunting bag/ha)	−6·79 (10,01)	0·498
Connect 1: type B vs. type A	**1·80 (0·81)**	**0·026**
Connect 2: type C vs. type A	**−4·11 (1·35)**	**0·002**
Connect 1 × time t	−0·49 (0·30)	0·102
Connect 2 × time t	**0·79 (0·28)**	**0·005**
Connect 1 × D_0	−59·15 (32·99)	0·073
Connect 2 × D_0	**128·04 (49·25)**	**0·009**
Age 1 × Time	0·41 (0·22)	0·062
Age 2 × Time	**0·91 (0·29)**	**0·002**

The variables Age, *Age*; capture method, *Cap*; time, *t*; distance from the starting point of the epidemic, *Dist*; initial density, D_0; connectivity to a large forest, *Connect*; and three interactions: *Age* × *t*, *Connect* × *t*, *Connect* × D_0 had significant effects. Coefficients (log of the odd ratios) are given with their standard error (S.E.) and *P* values of the corresponding Wald tests. Significant Wald tests are in bold.

10 011 shot and 165 found dead wild boar. We retained a model including the effects of *Cap*, *Age*, *t*, *Dist*, *Connect*, D_0, and three interactions: *Age* × *t*, *Connect* × *t*, *Connect* × D_0 (Table 1). Considering the decrease of residual deviance as a measure of the goodness of fit, this model explained 28% of the total variation of incidence.

Regarding individual risk factors, there was no significant difference between males and females ($P=0·148$). As expected, animals found dead were on average more infected than hunted animals [odds ratio (OR) 4·66, 95% confidence interval (CI) 2·09–10·42]. Incidence was higher in young animals (<1 year) than in *subadult* (OR 8·76, 95% CI 2·55–30·11) and *adult* individuals (OR 48·91, 95% CI 4·89–545·01). But there was no significant difference between *subadult* and *adult* individuals ($P=0·197$). The difference of incidence among age classes decreased over time (interaction *Age* × *t*) (Fig. 3).

At the level of the whole survey area, incidence decreased over time ranging from 3·5% in 1992–1993 to less than 0·3% in 1997–1998. Incidence also decreased when *Dist* increased.

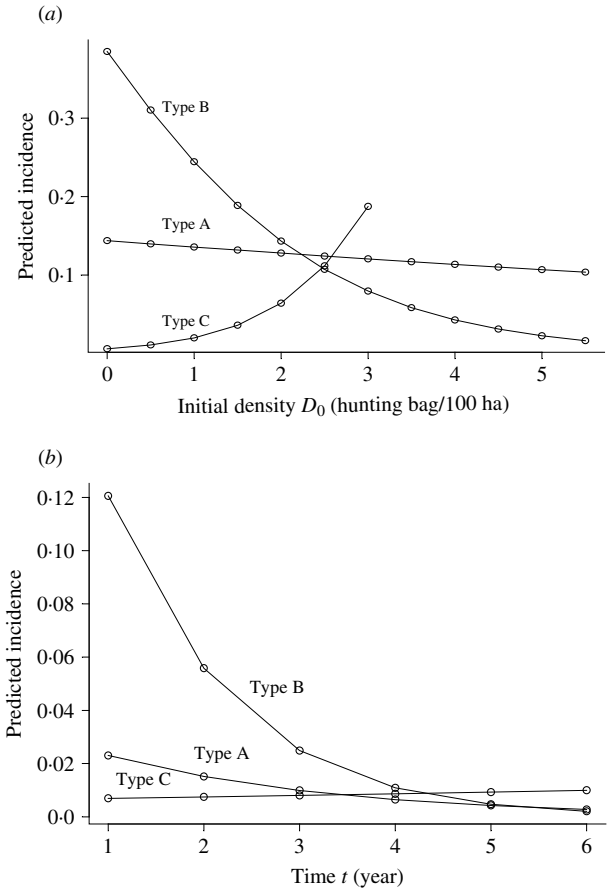

Fig. 3. Analysis of incidence: representation of the interaction between (*a*) Connectivity to a large forest (*Connect*), and initial density (D_0), (*b*) Connectivity to a large forest (*Connect*), and time (*t*).

Incidence was higher in type A and type B municipalities compared to type C municipalities when D_0 was below 2·5 shot individuals per 100 hectares, i.e. in most of the range of initial densities observed in type C municipalities ($OR_{A/C}$ 60·95, 95% CI 4·32–859·20; $OR_{B/C}$ 370·04, 95% CI 21·03–6509·90). Incidence was also higher in type B than in type A municipalities ($OR_{B/A}$ 6·05, 95% CI 1·24–29·59). However, *Connect* interacted with D_0 and *t* (Fig. 3). In type A and type B municipalities, incidence was not significantly related to D_0, but significantly decreased over time. On the contrary, in type C municipalities, incidence was positively related to D_0 but showed no temporal trend. The examination of incidence curves revealed that the survey period included only the decreasing phase of the epidemic in types A and B municipalities, but the complete epidemic curve in type C municipalities. For this reason we observed no significant temporal trend in type C municipalities.

Table 2. *Coefficients of the final model chosen to explain persistence*

Variables	Coefficients (S.E.)	*P* value (*t* test)
Intercept	**3·36 (1·06)**	**0·002**
Distance: Dist	−0·007 (0·008)	0·396
Initial density: D_0	**1·390 (0·391)**	**<0·001**
***Dist* × D_0**	**−0·009 (0·003)**	**0·008**

The distance from the starting point of the epidemic, *Dist*; initial density, D_0; and the interaction *Dist* × D_0, had significant effects. Coefficients (log of the odd ratios) are given with their standard error (S.E.) and *P* values of the corresponding Wald tests. Significant Wald tests are in bold.

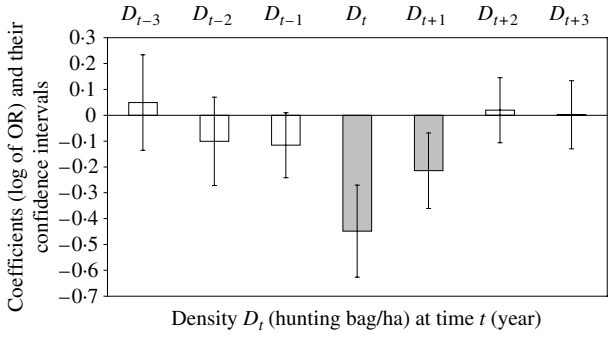

Fig. 4. Correlogram corresponding to the relationship between incidence measured at time *t*, and density measured at time *t*−3, *t*−2, *t*−1, *t*, *t*+1, *t*+2, *t*+3 (hunting bag/ha). Coefficients significantly different from zero are in grey.

Relationship between incidence and population density

Incidence at *t* was significantly and negatively related to D_t at *t* and D_{t+1} at *t*+1, but was not related to density at time D_{t+2}, D_{t+3}, D_{t-1}, D_{t-2} nor D_{t-3} (Fig. 4). So the relationship between density and infection was negative and was always contemporaneous or posterior to disease occurrence. These results suggest that infection depressed density, but do not support the hypothesis of density-dependence of incidence. The disappearance of a significant relationship at *t*+2 suggests that infection influenced D_t for no longer than 2 years (*t* and *t*+1).

Disease persistence

We analysed the persistence of CSF for 9 years among 60 municipalities. We retained the model including the effects of *Dist*, D_0, and the interaction between D_0 and *Dist* (Table 2). However, we cannot rule out the effect of *Connect* on persistence, because the effect of *Connect* was confounded with the effects of *Dist* and D_0. Our final model explained 44·3% of the persistence variability in our dataset ($R^2 = 0{\cdot}443$).

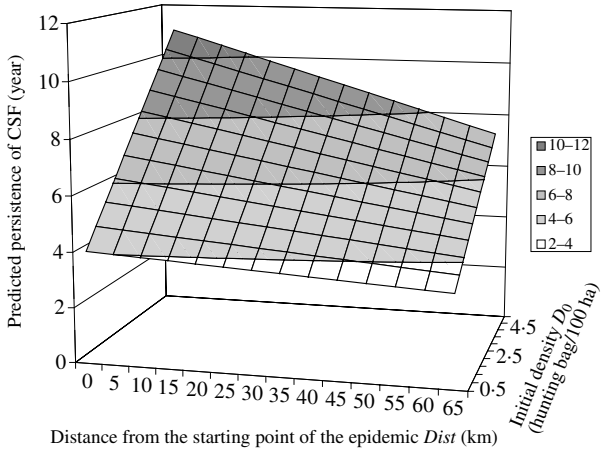

Fig. 5. Analysis of persistence: representation of the interaction between distance from the starting point of the epidemic (*Dist*), and initial density (D_0).

Persistence was positively correlated with D_0 (Fig. 5). Persistence was also negatively correlated to *Dist*. Finally, we observed a correlation between D_0 and *Dist*: D_0 had a stronger positive influence on persistence near the starting point of the epidemic (Fig. 5).

DISCUSSION

We first discuss which factors influence CSF incidence and whether disease has any influence on wild boar population dynamics. We then discuss how incidence, population dynamics and other factors may interact to influence disease persistence and which management strategies may help to prevent CSF persistence in the long term.

CSF incidence

We probably underestimated the true level of incidence because the ELISA-Ag test used for screening is less sensitive than virus isolation or PCR [24]. However, we assume this underestimation did not affect the analysis of risk factors. Concerning the variation of incidence in space and time, our analysis put forward the effects of *t*, *Dist*, *Connect*, D_0, and two interactions: *Connect* × *t* and *Connect* × D_0.

We observed a decreasing trend of incidence over time, which corresponds to the decline of the epidemic which was also observed in other CSF epidemics [4, 7, 12, 18, 30, 31]. Three hypotheses may explain the

decrease of incidence over space from the starting point of the epidemic. First, the effect of *Dist* may be confounded with the effect of D_0 which also decreased over space (R^2: $D_0 - Dist = 0{\cdot}217$, $P < 0{\cdot}001$). Such a decline of host density may alter the transmission of contagious parasites [32–35]. However, our data showed no indication that host density (either D_0, D_{t-3}, D_{t-2}, D_{t-1} or D_t) positively influences subsequent incidence, except in type C municipalities which exhibit particular isolation and low D_0. A second hypothesis is that the virulence of CSFV may decrease over time so that municipalities distant from the starting point of the epidemic should be infected by less virulent strains [7, 22]. However, virulence of CSFV strains remained stable during many other wild boar epidemics [36, 37]. Our third hypothesis is related to the spatial structure of wild boar populations. Wild boar are sedentary and live in social groups including females and young individuals while adult male are generally solitary [27]. We thus hypothesize that infectious contacts are more frequent within than between groups. This structure may correspond to the spatially designed epidemiological model developed by Bailey [38] that considers a host population structured in groups of equal density. This model predicts a decline of incidence from the starting point of an epidemic when the contact rate between groups is under a threshold level. Thus, although the effect of density cannot be ruled out, we advocate that host population structure is a major determinant of CSF incidence. The specific influence of social structure of host population could be tested through adapted mathematical modelling.

As expected, incidence was lower in small isolated forests (type C) than in large continuous forests (types A and B). Thus, besides the probable effect of wild boar social structure on incidence we also assume an effect of forest continuity by altering the connectivity among wild boar subpopulations. This effect of connectivity could be also tested through models.

Impact of CSF on host population dynamics

Our analysis of incidence revealed the impact of CSF on wild-boar population dynamics. We first hypothesize that CSF caused important mortality in wild boar because incidence was higher in animals found dead than in those shot [31, 39]. This hypothesis is supported by the negative correlation observed between incidence and density at t and $t+1$. Incidence was not related to density at $t+2$, which suggests that CSFV depressed D_t for up to 2 years. However, the impact of CSF on D_t may have been overestimated because intensive hunting was requested of the hunters in 1992 which possibly aggravated the density crash in 1992 [20–22]. Hunters then planned to allow the population to renew after the epidemic peak, therefore, hunting pressure may have declined in 1993 and 1994. Thus, the decline of the hunting bag probably overestimated the true decline of density so we cannot accurately estimate the importance of the decline of density.

Incidence was also higher in young than in older individuals, which is consistent with the high receptivity of young wild boar to CSFV [2, 17, 18, 31]. The high incidence in the young combined to the high lethality of CSF in piglets [2, 17] suggests that young (<1 year old) died more frequently of CSFV than older animals. This assumption was sustained by the low proportion of young animals shot during 1992 compared to all other hunting seasons. Thus, besides its influence on density, CSF probably modified the age structure of the population. The effect of age on incidence decreased over time so that we hypothesized that the impact of CSF on age structure was also temporary. Two mechanisms may explain the decrease of incidence in young wild boar over time. First, newborn piglets were protected by maternal antibodies by as soon as 1993. CSFV may then have exerted a selective pressure during the following years by favouring the survival of individuals resistant to CSFV.

We conclude that CSF temporarily modified density and age structure in the wild boar population, importantly in municipalities where incidence reached the highest values. These modifications of age and density might entail an increase of the birth rate during 1 or 2 years [40]. We thus hypothesize a probable increase of the birth rate after a CSF epidemic. Below, we ask whether this mechanism may explain variability in persistence, and why.

CSF persistence

We probably underestimated persistence by removing from incident cases the seropositive individuals older than 1 year. However, we assumed this underestimation did not affect the analysis of risk factors. In our analysis persistence was negatively correlated to *Dist*, and positively to D_0. The effect of density was strongest near the starting point of the epidemic, and the effect of habitat connectivity *Connect*, was

confounded with the effects of *Dist* and D_0. We propose that, by depending on the availability of new susceptibles, persistence was influenced by population size and birth rate. First, we cannot rule out the effect of *Connect*, and population size. Persistence was longest in types A and B municipalities where connectivity among subpopulations was assumed to be highest, i.e. where a large number of wild boars could get in contact. This interpretation agrees with the concept of critical community size (Fig. 1) [13, 41], but it does not account for the highest persistence of CSF near the starting point of the focus, above all in municipalities with highest D_0. We propose the following scenario: we hypothesize that municipalities with the highest D_0, corresponded to wild boar subpopulations close to habitat-carrying capacity, where density-dependence negatively affected the birth rate before disease arrival [40]. In those municipalities the birth rate may have increased when a large number of animals died of CSF, i.e. in municipalities with highest incidence peaks. Such an increase in the birth rate may have favoured disease persistence because the piglets born after the epidemic increased the pool of new susceptible individuals.

Obviously our analysis is not complete and other factors remain to be explored like the social structure of wild boar, the seasonal distribution of births, particular hunting methods, or the clustering of wild boar on permanent feeding places, that may influence CSF persistence by influencing the network of infectious contacts [41].

Conclusion

For veterinary authorities the key point of CSF management in wild boar is to control persistence. According to our previous discussion any measure limiting the availability of new susceptible boars should favour a rapid extinction of CSF. Thus, a first objective should be to limit population size and birth rate by shooting a large part of the population including reproductive females. However, this method may be dangerous if insufficient animals are shot or it is not continued over the long term because a temporary decrease of density may positively affect the birth rate and persistence through density-dependence. A limitation of artificial feeding could be proposed in order to limit fertility and survival, but artificial feeding has not yet been demonstrated as a determinant factor of population dynamics in wild boar. Moreover, measures could be implemented to limit connectivity among forest patches in order to limit population size. This could be achieved by reinforcing existing barriers or positioning artificial fences [4, 7, 9, 11, 40]. Moreover, other management methods like oral vaccination should at least be explored in the field.

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