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Source: Journal of Wildlife Diseases, 45(4) : 1121-1137

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-45.4.1121

Journal of Wildlife Diseases, 45(4), 2009, pp. 1121–1137

RABIES IN NORTHEASTERN EUROPE—THE THREAT FROM INVASIVE RACCOON DOGS

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ABSTRACT: The red fox (*Vulpes vulpes*) is the main terrestrial wildlife rabies vector in Europe. However, recently the raccoon dog (*Nyctereutes procyonoides*)—an invasive species originating from East Asia—has become increasingly important as secondary host, especially in the Baltic States. This imposes problems on neighboring rabies-free countries (such as Finland), where the density of each of the two vector species on its own might be too low to sustain a long-term rabies epizootic, but the community of vectors could be large enough to support a rabies epizootic. In this modeling study, we analyzed rabies epizootics in a community of foxes and raccoon dogs. We focused on the impact of density and behavioral differences (hibernation) between the two vector species. We found that rabies could persist in the community, even if the disease would not spread in the single vector species because its density was too low. Epizootics in the community were stronger than expected for single species, and raccoon dogs were usually the major rabies host. If raccoon dog territory density was high, invasive raccoon dogs could even outcompete native foxes because of apparent competition via the rabies virus. The enhancement in disease risk and disease intensity caused by raccoon dogs suggests that current strategies to control wildlife rabies in Europe should be reviewed, and that oral rabies vaccination also should target raccoon dogs after they emerge from hibernation.

Key words: Climate, cross-species transmission, disease introduction, *Nyctereutes procyonoides*, rabies, raccoon dog, simulation.

INTRODUCTION

Effective large-scale vaccination campaigns have successfully eliminated terrestrial wildlife rabies from most parts of Western Europe, where it primarily has been spread by the red fox (*Vulpes vulpes*). With strong efforts from Poland and other eastern European countries, the "rabies free" zone is being quickly pushed further to the east (Niin et al., 2006; Potzsch et al., 2006; Matouch et al., 2007). However, pockets of endemic wildlife rabies still remain (e.g., in Russia, the Baltic States, and Belarus). One reason for these persistent foci could be the high cost of vaccination campaigns, but for example in the Baltic States (countries that currently experience major rabies epizootics), the presence of a new rabies vector, the raccoon dog (*Nyctereutes procyonoides*), could be complicating any effort to control the disease.

Invasive raccoon dogs have quickly established populations in eastern and northeastern Europe since their introduction to western Russia (and other parts of the former Soviet Union, e.g., Estonia and Ukraine) in the first half of the 20th century (Lavrov, 1971; Nowak, 1984; Helle and Kauhala, 1991). Omnivory, large litter size, and hibernation were suggested as explanations for their high reproductive success (Helle and Kauhala, 1995; Kauhala, 1996; Kauhala et al., 1998). Raccoon dogs start hibernation in November and wake up in March in southern Finland (Kauhala et al., 2007). They usually stay in their dens when temperature is below −10 C, snow depth >35 cm and day length <7 hr. Sometimes, however, they wander around in midwinter if the weather is mild. They may also change their den site during winter. During hibernation, the average body temperature decreases by 1.4–2.1 C (Mustonen et al., 2007). Raccoon dogs gather large fat reserves in autumn and lose about 43% of their body mass during winter (Kauhala, 1993; Mustonen et al., 2007).

Raccoon dogs are known rabies hosts in eastern Asia (Kim et al., 2006), from where they originate (Ognev, 1962; Nasimovic and Isakov, 1985). But they also were involved in zoonoses in western Russia and eastern Europe (Kantorovich, 1975; Cerkasskij, 1980; Wlodek and Krzywinski, 1986; King et al., 2004; World Health Organization [WHO], 2008) and they are highly susceptible to the European fox rabies strain.

The presence of a second rabies vector poses several potential problems. Disease control and elimination might become more difficult for several reasons; new paths of disease introduction may occur. Additionally, cross-species infection can reintroduce the virus into a vector species from which it has been eliminated. Generally, a larger vector reservoir has to be managed, and vector species behavior and interactions might impact on the epizootiology.

In addition, the effect of raccoon dog hibernation on rabies epizootiology is relatively unclear. It has been argued that a hibernating, thus mostly immobile, animal will not transmit virus, except to its den-sharing mate (Nyberg et al., 1992). However, even in the north, active raccoon dogs have been observed in warm periods during winter. Temperature, snow depth and day length affect their winter activity (Kauhala et al., 2007). Hibernation may also alter rabies pathogenesis. Studies have indicated that the period of rabies incubation is prolonged while animals hibernate. Botvinkin et al. (1985) demonstrated in the laboratory that small rodents would not fall ill during hibernation, but would show rabies signs shortly after becoming active again. A similar effect is expected for rabies incubation in hibernating raccoon dogs (Cerkasskij, 1980; Holmala and Kauhala, 2006).

In this study, we analyzed the dynamics of a rabies outbreak in a previously disease-free community of foxes and raccoon dogs. We focused on the area of southern Finland. Finland is free of rabies, but experienced a short outbreak of sylvatic rabies in 1988–1989. During the outbreak, more verified rabies cases (73%) were recorded in raccoon dogs than in foxes (18%) (Westerling, 1991; Nyberg et al., 1992).

The rabies virus needs a minimum fox density to be sustained in an area (Anderson et al., 1981). The Finnish fox density is lower than the estimated threshold density (except that of the southwestern part of the country). However, it recently was argued that the community of carnivores in southern Finland could serve as a sufficient reservoir for wildlife rabies (Holmala and Kauhala, 2006; Kauhala et al., 2006). In previous studies, we showed that neither the southern Finnish fox nor the raccoon dog population would be sufficient to support a rabies epizootic in isolation, but that the community of both vectors could be a potent reservoir (Singer et al., 2008). We simulated rabies dynamics for a wide range of (disease-free) vector densities of both species under different assumptions on raccoon dog hibernation. We then assessed the risk of disease persistence after arrival of infected raccoon dogs and calculated the strength of the subsequent epizootics. Although mainly concentrated on southern Finland, the wide-ranging parameter variation allows conclusions on rabies epizootics for larger regions of eastern Europe.

MATERIALS AND METHODS

Two-species disease model

A stochastic model was constructed to simulate rabies spread in each of the two species. Construction of the submodels for each of the two vector species followed approaches for models on rabies epizootics in the red fox (Smith and Harris, 1991; Smith and Wilkinson, 2003; Eisinger et al., 2005). They comprised population dynamics of each of the host species, rabies transmission, and pathogenesis. The submodels were parameterized independently for each of the vector species. Parameters for the fox component were based on data from Poland (Goszczynski, 1989, 1999; Eisinger et al., 2005). Data to model raccoon dog dynamics was collected in extensive studies in southern Finland (Helle and Kauhala, 1993, 1995; Kauhala and Holmala, 2006; Kauhala et al., 2006).

To allow disease spread in the community of vector species, cross-species transmission was introduced as an additional process. It was assumed that transmission would occur from random encounters of rabid animals of one species with susceptible animals of the other species. Estimates of transmission rates were not available from field studies; therefore, we adjusted the cross-species transmission rate (0.0016) so that rabies was highly likely to spread in a community of raccoon dogs and foxes in southern Finland, independent of raccoon dog hibernation. The short rabies outbreak in the southeastern part of Finland in the late 1980s (Nyberg et al., 1992) suggested that the disease could spread in the area.

In each model simulation run, the disease was introduced in summer of year 26. This allowed time for the disease-free dynamics to establish. A simulation run either ended after extinction of both vectors or of the disease, or if 100 yr was reached.

Variation of vector densities

In Finland during winter, population densities of both vector species are controlled by hunting and by availability of food. In particular, the mortality rate of raccoon dog juveniles increases if berries are scarce in late summer and autumn, when raccoon dogs accumulate their fat reserves for winter (Kauhala and Helle, 1995). Climate is also important: If the winter is too long, juveniles especially do not survive because they did not accumulate enough fat for the long winter. These winter conditions limit population growth. For the model, winter mortality was split into a density-independent part from hunting and a linearly density-dependent part from resource limitation. The limiting capacity was modeled as territory density (which in the model was proportional to density of a disease-free population in winter, a parameter available from field studies). See Appendix I for a detailed description.

We varied territory density for both species to assess the impact of (disease-free) population density on the epizootiology of rabies in the community. We sampled territory densities of foxes and raccoon dogs 5,000 times from a joint uniform distribution. Territory densities ranged from 0.001 to 1 raccoon dog territory per km^2 and 0.001 to 0.4 fox territories per km^2.

Radio tracking and studies of snow tracks in winter in southern Finland found population density of foxes between 0.35 and 0.44 adults per km^2. Raccoon dog density ranged between 0.38 and 0.77 adults per km^2 (Kauhala et al., 2006). For both species it was assumed that 1 pair of adults shared a territory (Kauhala et al., 1993). The southern Finnish raccoon dog densities were at the higher end of densities found in different areas of Europe, while densities of red foxes were comparably low. Thus the range of variation in this study covered current densities in southern Finland and other regions in northeastern Europe, although higher raccoon dog densities have been recorded in recent years (Kauhala, unpubl. data).

Hibernation scenarios

The effects of hibernation on rabies epizootiology are uncertain. We therefore took five reasonable assumptions of hibernation and analyzed their effects on rabies epizootiology. The hibernation scenarios are listed in Table 1. Scenario Hib0 assumed that raccoon dogs did not hibernate in winter, as may occur in warmer parts of Europe or following climate change. Hib1 was inspired by the observation that juvenile raccoon dogs cannot gather enough resources in autumn and must continue foraging during winter, but that adults hibernate and thus have a prolonged incubation period. Hib2 assumed that hibernation would impede transmission and prolong incubation period for all raccoon dogs; however, foxes would still infect raccoon dogs. This scenario was unlikely, as foxes rarely share dens with raccoon dogs (Wlodek and Krzywinski, 1986); Consequently, Hib3 was the same as Hib2 except that no cross-species transmission occurred in winter. Hib4 added the assumption that incubation period of raccoon dogs was not prolonged, so that hibernating raccoon dogs became rabid and died during winter.

Analysis of model simulations

For each parameter set, the simulation was repeated 1,000 times. For each of the repeti-

TABLE 1. Potential effects of hibernation on disease progression.

Hibernation type	Hibernation	Raccoon dogs become rabid	Transmission in winter
Hib0	No	Yes	Yes
Hib1	Only adults	Only juveniles	Juvenile raccoon dogs spread rabies
Hib2	Yes	No	Raccoon dogs do not transmit rabies; foxes transmit the virus into the raccoon dog population
Hib3	Yes	No	No transmission from or to raccoon dogs
Hib4	Yes	Yes	No

tions, the times of extinction events of vectors or disease were recorded.

Risk of disease persistence was measured 5 yr after the disease was introduced into the population as the ratio between simulations in which the disease persisted in the community of vectors and the total number of simulation runs (1,000). To increase our understanding of the disease extinction process, a population viability method (Grimm and Wissel, 2004; Singer, 2006) was applied that permitted separation of the extinction risk during the initial transient and the established phase of dynamics.

We then smoothed the persistence metrics: We classified the joint territory density of raccoon dogs and foxes by windows of 0.02 fox territories per $km^2 \times 0.02$ raccoon dog territories per km^2. Persistence metrics from corresponding parameter sets within one window were averaged. To display the result, we applied contour lines from the statistical software R (R-Development-Core-Team 2006).

For simulations in which the disease was not extinct, the extent of disease was estimated. Metrics included the number of infected animals, prevalence, and population size before disease introduction and at the end of a simulation run. All metrics were calculated for the individual vector species, estimated as temporal statistics (mean and variance) from the last eight simulated years. The metrics were further averaged over all 1,000 simulation runs per parameter set. Results for different hibernation scenarios were treated separately.

RESULTS

A full sensitivity analysis has been performed looking for first-order and total effects, but changing parameter values (other than those stated below), produced smaller effects than those reported (A. Singer and G. Smith, unpubl. data).

Risk of rabies persistence

Figure 1 depicts the risk of rabies persistence dependent on territory densities of both vectors for three different hibernation scenarios (columns). The three metrics allowed a complementary view on risk of disease persistence: In the top row, contours indicate the risk that rabies was still present in the community 5 yr after disease introduction, a realistic time frame for disease management. However, minor epizootics still could be present after 5 yr. In order to distinguish minor from major epizootics, persistence metrics t_m and c_1 (Grimm and Wissel, 2004; Singer, 2006) are displayed in the following two rows. Metric t_m is the intrinsic mean time to extinction of an established disease. Thus a high value of t_m indicated long disease persistence. Metric c_1 indicated disease survival during the phase of disease establishment: a low value of c_1 (around 0) but a high value of t_m (several 1,000 yr) meant that the disease could not establish in the population in most simulation runs—it remained a minor outbreak. However, if the disease became established, it persisted for long time. The opposite case of high c_1 (higher than 1) and low t_m (some decades) indicated that the disease did not produce long-term establishment in the vector community, but could be present for years.

With this in mind, rabies risk after 5 yr could be understood. We give a detailed explanation for the case of Hib0 (the left column in Fig. 1), before pointing to differences between hibernation scenarios.

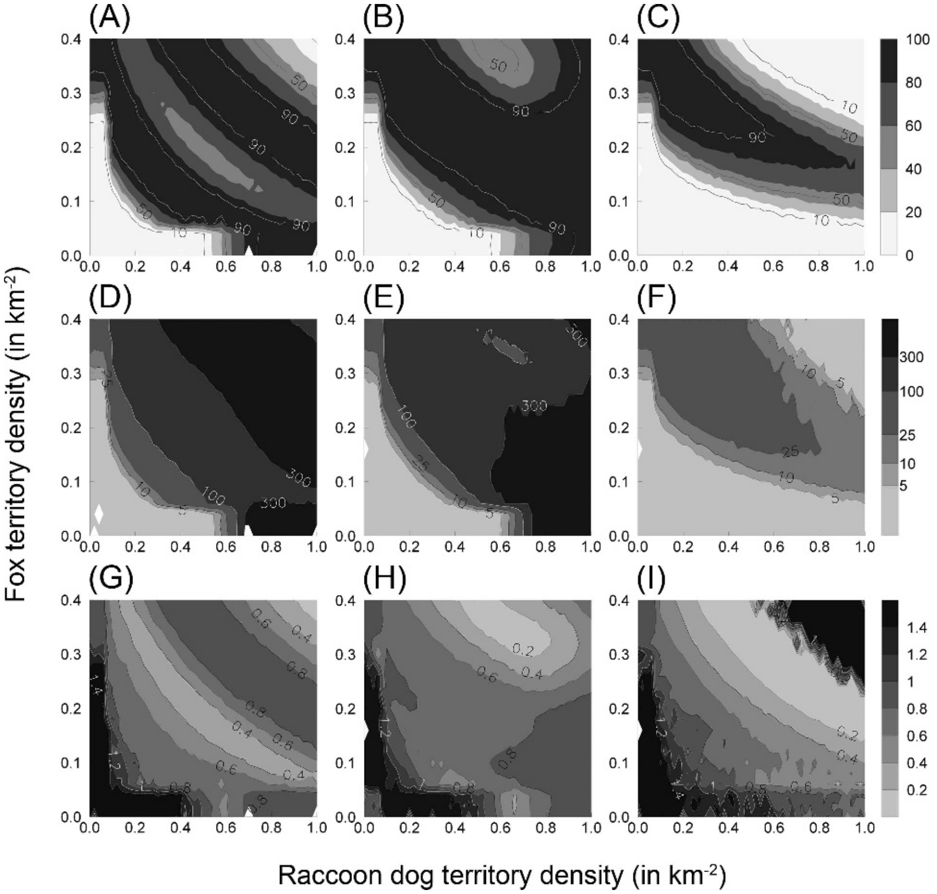

FIGURE 1. Risk of rabies persistence (grey shades) dependent on winter raccoon dog (x-axis) and fox (y-axis) territory density. Top row (A–C): rabies persistence risk after 5 yr (0–100%). The middle row (D–F): intrinsic mean time to extinction t_m (in years). Bottom row (G–I): persistence metric c_1 (see text for details). These last two rows indicate the long-term risk of establishment and chance of extinction after rabies introduction, respectively. Hibernation scenarios (Hib0, Hib3, and Hib4) are displayed in the three columns.

The isolines in Figure 1A denote the probability that rabies was still present 5 yr after being introduced. At low territory densities, the risk of rabies was very low (white area in bottom left corner of Fig. 1A). The corresponding low values of the intrinsic mean time to extinction t_m (<100 yr) in Figure 1D indicated that the disease did not persist in the community of vector species. Thus, below a joint threshold density, there was no risk of a persistent rabies outbreak. The threshold appeared as a steep increase in rabies risk for increasing vector densities. (Note: The threshold density discussed here was defined by the risk of the disease to persist at least for 5 yr: This is not the standard definition of a disease threshold [R_0] widely used for deterministic models [R_0>1].)

The joint threshold depended nonlinearly on territory densities of both vector species (note the curved shape of threshold isolines in Fig. 1A), which suggested synergies from rabies virus transmission between species. A slight increase in territory density of one species could strongly reduce the territory density need-

TABLE 2. Threshold densities for 50% risk of rabies.[a]

	Minimum territory density for disease in single species[b] mean (standard error)		Minimum joined territory density[c]		
	Raccoon	Fox	Raccoon	Fox	Total
Hib0	0.59 (0.00)	0.29 (0.00)	0.10	0.16	0.26
Hib1	0.60 (0.01)	0.29 (0.00)	0.10	0.16	0.26
Hib2	0.68 (0.01)	0.29 (0.00)	0.10	0.17	0.27
Hib3	0.69 (0.01)	0.29 (0.00)	0.10	0.20	0.30
Hib4	–	0.29 (0.00)	0.10	0.22	0.32

[a] Values derived from threshold isolines as displayed in Figure 1.

[b] Estimated from isoline points for which the territory density of the other vector was too low to sustain the disease-free population in at least 90% of simulations. That is for fox territory densities below 0.05/km^2 or raccoon dog densities below 0.06/km^2

[c] Total=Raccoon dog territory density+fox territory density. Values were estimated from isoline points, where total was lowest. In case of equality, the point with lowest raccoon dog density was chosen.

ed for the other vector to sustain rabies in the community. It became obvious that rabies could persist at lower vector densities than expected from spread in a single species vector population (Table 2). Independent of hibernation, the total density (summed densities of both vector species) was less than half the raccoon dog threshold density. But the minimum joint threshold was about the same as the fox threshold density. On average, 6.4 times higher raccoon dog territory density and 1.6 times higher fox territory density was necessary for rabies to spread in a single vector species as opposed to spread in the community.

At densities around the threshold, the rabies risk was high. But the risk declined with a further increase of vector density. A significant decline was not found for the intrinsic mean time to extinction t_m (Fig. 1D). Thus, an established disease had a large chance to persist for longer than 5 yr. However, the decline in persistence metric c_1 (Fig. 1G) demonstrated that there was a high risk of extinction shortly after the disease was introduced. In time series of disease dynamics, we found that this extinction risk was induced by strong outbreaks of the newly introduced disease into the large healthy populations. During the outbreak, rabies depleted its host populations. The resulting low numbers of susceptible animals impeded disease spread, which eventually led to random disease extinction. In contrast, if the virus survived in the small vector community, then it controlled further population growth. Hence, later large outbreaks became impossible. Instead, disease extinction was then driven by stochastic effects in these reduced populations. The chance of disease extinction was higher during the initial phase of disease establishment than after an epizootic had developed ($c_1<1$ and $t_m>100$ yr).

By increasing the territory density of vectors further, the chance of disease extinction (following the initial outbreak) declined because the vector populations recovered quickly and the period of low numbers of susceptible animals was short. At very high vector densities (particularly at high fox territory density), immediate extinction after an initial outbreak again became important (Fig. 1G).

Hibernation of raccoon dogs altered the disease risk (compare the columns in Fig. 1), expressed as shifts in the patterns of the risk contour lines. To clarify the effects of hibernation on the threshold density, characteristic points of the isolines for 50% risk of rabies persistence after 5 yr were extracted for all hibernation scenarios (Table 2). As expected, the risk of having disease in only the fox population was independent of raccoon dog hibernation. However, hibernation influenced the risk of rabies spread in an isolated raccoon dog population. If the entire raccoon dog population hibernated (Hib2 and 3), raccoon dog density had to be about 15–17% higher for a 50% risk of disease persistence after 5 yr. The case of Hib4 (incubation period not prolonged) was different: The virus did not persist in the raccoon dog population at realistic densities (Fig. 1C) because many infected animals died during hibernation without transmitting the disease.

We now compare the lowest joint density for different hibernation scenarios. Reduced rabies transmission into the hibernating raccoon dog population increased the total density by more than 10% (compare Hib2 and Hib3). The joint threshold density of Hib4 was 23% higher (Table 2) than the threshold density without hibernation (Hib0).

At densities higher than the threshold density, rabies persistence risk was reduced if the disease eradicated itself in an initial strong outbreak (c_1 small). This early self-eradication shifted to higher fox densities in the order of hibernation scenarios Hib0, 1, 2, 3. Thus, the stronger hibernation impeded transmission from or to raccoon dogs, the more foxes were required to cause self-eradication of the disease as a result of the initial outbreak.

If rabies incubation was not slowed down by hibernation (Hib4), then the risk of disease persistence was lower compared to the other cases (right column in Fig. 1). In this hibernation scenario, high risk of persistence was found only when fox density was sufficiently high and raccoon dog density was low (Fig. 1F). The raccoon dog population alone could no longer sustain the disease (because raccoon dogs succumbing to rabies during hibernation effectively reduced the pool of infectious animals). At medium fox densities, rabies could persist in the community for 5 yr. However, the low values of t_m indicated that the disease would not persist much longer. At high densities of both species, the disease self-eradicated very quickly because the initial outbreak depleted the vector populations and hibernation impeded transmission involving raccoon dogs.

Strength of a persistent disease

The extent of disease was evaluated if the virus survived for at least 75 yr in the system. Figure 2 depicts prevalence and relative population loss for the raccoon dog (rows 1 and 2) and the fox population (rows 3 and 4). Raccoon dog territory density varies along the *x*-axis. Fox territory density is indicated as gray shades. Each column in Figure 2 represents one hibernation scenario. Again we describe the influence of population densities for the scenario without raccoon dog hibernation (Hib0) before looking at the effects of hibernation.

The relationship between prevalence and vector densities was complex (Fig. 2A): For low raccoon dog density, the fox density impacted on raccoon dog prevalence strongly. Increasing fox density increased the prevalence in the raccoon dog population. However, the influence of fox density decreased with increasing raccoon dog density. If fox territory density was low, then an increase in raccoon dog density increased prevalence in the population. But if fox density was high, then raccoon dog prevalence *declined* with increasing raccoon dog density. At very high raccoon dog densities, fox density lost its impact completely, and prevalence in the raccoon dog strongly increased with increasing raccoon dog territory density. (Note: The black line below the main pattern displayed simulations where the fox population was so low it went extinct before the disease was introduced.)

A similar pattern was found for the disease-induced decline of the raccoon dog population (Fig. 2D). Extensive disease in low-density raccoon dog popula-

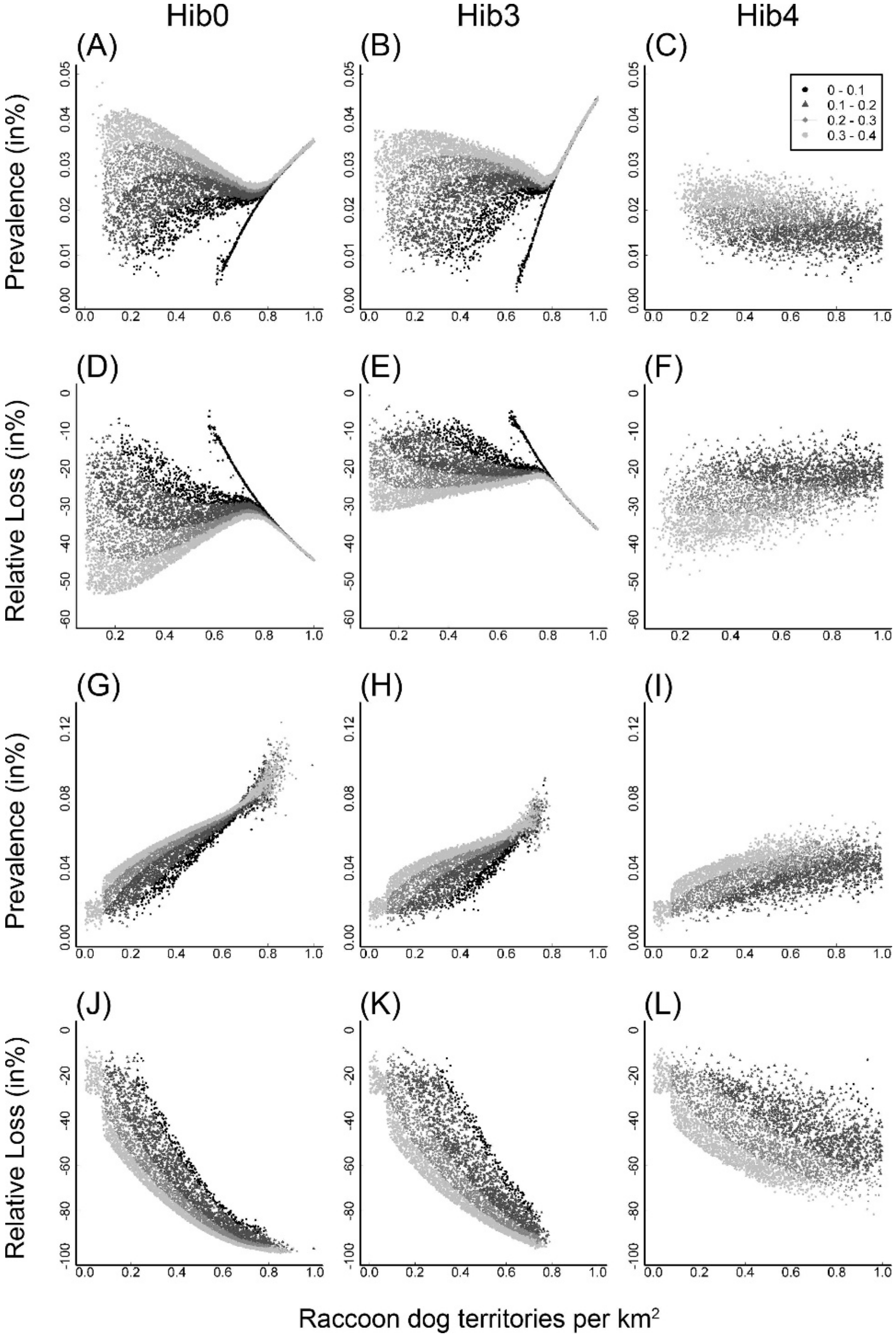

FIGURE 2. Extent of rabies in simulated populations of raccoon dogs (A–F) and foxes (G–L). Different shades and symbols represent different ranges of fox territory density (see legend in C).

tions (at high fox densities) could cause more than 50% population reduction. Thus, for low raccoon dog density foxes influenced disease in the raccoon dog population; however, the fox impact vanished at high densities of raccoon dogs.

The reason for the limited influence of foxes became clear when analyzing disease in the fox population. Rabies prevalence in the fox population (Fig. 2G) increased with increasing raccoon dog territory density. In contrast, an increase of fox territory density caused only a weak increase in prevalence. The impact of fox density declined with increasing raccoon dog density.

Increasing prevalence in foxes was accompanied by a sharp decline in the fox population (Fig. 2J). At high raccoon dog densities the fox population was dramatically reduced by at least 80% or even went extinct. The data revealed that the increase in fox prevalence was driven by the decline in population size as opposed to the number of infected animals. In fact, the number of infected foxes even declined with increasing raccoon dog territory density (not shown).

At high raccoon dog territory density the disease was driven from the raccoon dog population into the fox population. Rabies depressed the fox population more than the raccoon dog population (up to 100% for foxes compared to 55% for raccoon dogs). Because of the small remaining fox population, the number of rabid foxes was low and could not reinforce the disease in the raccoon dog population. If raccoon dog density was large enough (above the single species disease threshold of around 0.6 territories per km^2), then the disease could effectively be maintained in the raccoon dog population only, with spillover transmission causing major suppression of the fox population.

We now look at the impact of raccoon dog hibernation. If the incubation period was prolonged in hibernating raccoon dogs (Hib1–3), patterns in dependence of disease strength metrics on vector densities remained relatively unchanged (compare columns 1 and 2 in Fig. 2). In contrast, if the incubation period was not extended and rabid raccoon dogs died during hibernation without transmitting the virus (Hib4), then the epizootiology was very different (last column in Fig. 2). In this case, raccoon dog prevalence was generally lower than in other scenarios. It increased more with fox than with raccoon dog density (Fig. 2C). Prevalence in foxes was increased by an increase in density of both species. For Hib4, increasing raccoon dog density did not reduce the impact of fox density. This is not surprising, as the disease was not able to spread only in the raccoon dog population (see Fig. 1C). Thus, for this scenario only, foxes remained an important vector even at high raccoon dog densities.

Risk of rabies in Finland

Table 3 provides a summary of rabies risk and extent for the area of Finland. Values in Table 3 rest on a conservative estimate: We considered the full range of vector species densities given in Kauhala et al. (2006)—these are 0.38–0.77 adult raccoon dogs per km^2 and 0.35–0.44 adult foxes per km^2. We assumed a cross-species infection rate that predicted high risk of a persistent zoonosis for a joint vector density, which is central for the given range. Thus, values in Table 3 should be considered as upper limits.

The model predicted that rabies introduction to southern Finland would be likely to cause an epizootic. Even if hibernating raccoon dogs died of rabies (Hib4), which reduced disease risk compared to other hibernation scenarios, there was at least a 50% risk that rabies persisted for at least 5 yr. Other assumptions about hibernation made an epizootic more than 90% likely.

Prevalence in the fox population was predicted to range around 4%, and around 3% in the raccoon dog population. Nev-

TABLE 3. Potential rabies epizootics in southern Finland.[a]

	Persistence risk (%)		Average prevalence (%)				Average relative population loss (%)			
	Both species		Raccoon dog		Fox		Raccoon dog		Fox	
	5%	95%	5%	95%	5%	95%	5%	95%	5%	95%
Hib0	71	98	2	3	3	5	30	37	42	67
Hib1	87	98	3	3	3	5	28	34	43	68
Hib2	95	99	3	4	3	4	23	29	39	67
Hib3	82	97	2	3	2	4	16	21	31	59
Hib4	52	88	1	2	2	3	17	24	19	36

[a] Values indicate the 5% and 95% quantiles of all simulation results from parameter settings within the window of southern Finnish fox densities (range: 0.35–0.44 adult foxes per km^2) and raccoon dog densities (range: 0.38–0.77 adult raccoon dogs per km^2), according to Kauhala et al. (2006).

ertheless, because the raccoon dog population was larger, the number of rabid raccoon dogs could be up to twice as high as the number of rabid foxes. For both vector species, the model predicted severe population declines, but the fox population was depleted comparably more: A loss of half the fox population was possible, whereas the raccoon dog population would decline by 37% at most. Hibernation generally mitigated the strength of disease. For scenarios Hib3 and Hib4, the epizootic was less extensive.

DISCUSSION

Hibernation of raccoon dogs

Hibernation of raccoon dogs depends on climatic conditions. Thus, raccoon dog populations at different latitudes throughout Europe might or might not hibernate. In Germany, raccoon dogs are known to be active during winter, while the hibernation period of raccoon dogs in Poland lasts from December to February or March (Kauhala et al., 2007, and references therein). For subarctic areas in northeastern Europe, hibernation is common (Kauhala et al., 2007; Mustonen et al., 2007), but global warming is likely to alter this pattern (Kauhala et al., 2007). We accounted for the range of possible raccoon dog behaviors by simulating the model for five different assumptions on raccoon dog hibernation.

We confirmed that an enhanced incubation period was necessary for rabies to spread in a hibernating raccoon dog population. However, we found that in a vector community the disease could still spread even if the incubation period was not prolonged by hibernation (Hib4). In this case, the epizootic was less severe than in other hibernation scenarios. Thus, the virus benefited from a prolonged incubation during hibernation.

If infected hibernating raccoon dogs did not die from rabies during winter, hibernation only slightly altered the risk or extent of persistent disease. However, hibernation affected the risk of an initial outbreak. Raccoon dog hibernation shifted the joint threshold density upwards slightly. The chance of disease extinction due to quick self-eradication was shifted toward higher fox densities. Further research is necessary to reveal how the seasonal effect of hibernation affects the risk of disease.

The effects of hibernation on disease epizootiology also may have implications for Lyssavirus spread in hibernating bat communities. Hibernation appears to dampen the epizootic, and this may have evolutionary consequences for the virus.

Impact of minor outbreaks

We have demonstrated that initially extensive outbreaks might significantly reduce the risk of rabies persistence because the disease depletes its hosts and subsequently self-eradicates. Self-eradication is a known prediction of nonspatial rabies models with "global" disease transmission. Spatially explicit models more realistically predict a wavelike expansion of the disease: The virus disappears from the depleted population locally, but spreads further through areas with higher numbers of susceptible animals (Smith and Harris, 1991; Jeltsch et al., 1997). We therefore expected this finding to be relevant on small spatial scales.

Generally, it has been assumed that at population densities above the threshold density (defined by $R_0=1$) rabies would persist. However, this assumption neglects the temporal effect of disease in a previously healthy population. In the course of disease spread the population declined, which changed the conditions of disease spread. Thus, the system underwent a self-adapting transient process, during which the dynamics were different from the stable-state dynamics. Therefore, the chance of disease extinction could be higher than expected from an established disease. This suggests that disease eradication may become an end-point in high-density isolated populations.

Raccoon dogs as the main driver of disease

Except for scenario Hib4, the fox population suffered more from a rabies epizootic than the raccoon dog population. In particular, at high raccoon dog density the fox population was strongly depressed and could even be eradicated. Thus, with increasing raccoon dog density epizootics became increasingly independent of the fox population. The "host community" effectively turned into a single host species. The switching density can be identified from metrics of disease extent in Figure 2. Above the switching density, foxes did not influence the disease metrics. This switching density was around 0.8 raccoon dog territories per km^2 for hibernation scenarios Hib0–3. The transition from a host community to a single host species changed the disease dynamics. This became clear from the coefficient of variation (CV) of raccoon dog prevalence (not shown). This quantifier of fluctuations (for example, from epizootic cycles) dropped by more than 10% (and even higher if raccoon dogs hibernated). Thus, disease extent was more variable in the community than in the raccoon dog population.

With increasing raccoon dog density, the importance of the red fox changed from an essential vector to a spillover host unnecessary for disease propagation. In this situation, we observed that rabies frequently vanished from the fox population but was reintroduced from the raccoon dog population. Raccoon dogs drove the virus back into the fox population, thereby keeping the fox population small. Thus, rabies promoted apparent competition in the host community.

The maximum territory density of raccoon dogs was much higher than that of foxes. Thus, at higher fox density the fox population may suppress the raccoon dog population. However, this argument neglects the density-regulatory function of territory density. Instead, we suggest that productivity of a vector species determines dominance. High productivity lets a disease-depleted vector population recover quickly and supply susceptible animals that can become infected and transmit disease into another vector species. If that second species has lower productivity, it will not have recovered from the previous disease outbreak. The new disease pressure will hinder its recovery and the population stays small.

In fact, the productivity of raccoon dogs is much higher than fox productivity (Helle and Kauhala, 1995; Kauhala, 1996), but population growth is density-limited by the availability of food and territories—modeled by territory density as a carrying capacity. Hence, at low territory density, populations grew slower in the model. Therefore, foxes were important if raccoon dog densities were low. However, as soon as raccoon dog capacity and therefore reproduction became strong enough, raccoon dogs dominated foxes in the epizootiology.

Risk of rabies in Finland

Southern Finland is threatened by rabies introduction from neighboring countries. The low-density fox population would not permit rabies to spread on its own (Singer et al., 2008). However, it was predicted that the established community of foxes and raccoon dogs as two major hosts could sustain the virus (Singer et al., 2008). Here, we presented more detailed analysis of disease epizootiology in the two vector species.

The model results in Table 3 suggest that few intruding infectious animals result in a high risk of establishing rabies in Finland. The short outbreak of rabies in the late 1980s (Nyberg et al., 1992) suggested that the Finnish vector community was a potent rabies reservoir. According to our model results, such an outbreak would develop into a persistent epizootic in at least 50% of cases (Hib4) and the risk would be much higher for the other hibernation scenarios. Thus, it was necessary to conduct the antirabies vaccination campaign in Finland in the late 1980s to ensure disease elimination. In the context of recently increasing population densities, disease risk is now potentially higher (although early self-eradication could come into effect, as discussed above).

The model predicted a prevalence of around 3–4% in the vector community. A slightly higher prevalence could be expected for foxes than for raccoon dogs. Nevertheless, because of the higher raccoon dog density, more rabies cases in raccoon dogs would be expected than in foxes. This finding is in accordance with observations from the Finnish outbreak, with 48 verified raccoon dog cases but only 12 red fox cases recorded (Nyberg et al., 1992). Also, Estonia (a neighboring Baltic state, separated from Finland by the Gulf of Finland), has recorded higher numbers of rabid raccoon dogs than rabid foxes, although total numbers of cases have strongly declined recently due to vaccination. In contrast, in Latvia the number of fox cases is higher than the number of raccoon dog cases. According to our model, this would imply a lower disease-free raccoon dog density in Latvia than in Finland or Estonia. In Lithuania, the number of both fox and raccoon dog cases had strongly increased during recent years but dropped in 2007. At the peak of disease, more raccoon dog cases than fox cases were reported (WHO, 2008).

The model also predicted that the disease would regulate population sizes. For densities reported in Finland, the raccoon dog population would be expected to decline by 20–40% and the fox population by 30–70% (see Table 3). A 3–5-fold increase in fox populations in central and western Europe was observed after rabies eradication (although fox densities there were higher and the raccoon dog was mostly absent), and it was assumed that disease eradication was partly responsible for the growth in population size (Chautan et al., 2000).

Implications for disease control

This study showed that rabies can spread at much lower vector densities than previously assumed where a community of potential vectors exists. Thus, rabies epizootics may occur in regions that were considered “rabies-safe” after a change in the community of carnivores. For instance, the raccoon dog population is increasing rapidly in Germany—since the mid-1990s the hunting bag has increased exponentially (Drygala et al., 2007). The presence of raccoon dogs strongly enhances the risk of rabies recurrence in Europe. High raccoon dog densities would be predicted to substantially change the course of an epizootic, and they may become the major rabies vector. Higher numbers of rabid animals (thus an increased risk to livestock and humans) would be expected.

In light of the implications of raccoon dogs as a second vector of wildlife rabies, control strategies should be developed to cope with the enhanced threat. Fortunately, raccoon dogs become immunized by fox rabies vaccine baits, with very high rates of successful immunization reported (Cliquet et al., 2006). Nevertheless, rabies control will be much more difficult in a vector community. In systems where both foxes and raccoon dogs occur, vaccination campaigns should target both species with potentially high bait density, and should consider behavioral characteristics of the respective species. Therefore vaccinations should continue to be carried out twice each year: in late winter/spring when adult raccoon dogs become active in the breeding season and hungry after hibernation, and in autumn when juveniles disperse. Raccoon dog hibernation also has to be considered when designing spring vaccination campaigns, as these should take place after raccoon dogs become active (Singer et al., 2008). A second complication will be the high reproductive success of raccoon dogs and the quicker population recovery. Control schemes have to target the large number of susceptible juvenile raccoon dogs each year.

The ability to develop cost-effective control strategies depends on thorough knowledge of vector behavior and disease transmission. There is a lack of knowledge concerning territoriality and interaction of vector species (including potential spill-over species such as badgers). Closing these gaps will be essential for the long-term goal of a rabies-free Europe.

ACKNOWLEDGMENTS

G.C.S. and A.S. acknowledge support from the Department for the Environment, Food and Rural Affairs.

LITERATURE CITED

Anderson, R. M., H. C. Jackson, R. M. May, and A. D. M. Smith. 1981. Population dynamics of fox rabies in Europe. Nature 289: 765–771.

Botvinkin, A. D., T. A. Nikiforova, and G. N. Sidorov. 1985. Experimental rabies in hibernator rodents. Acta Virologica 29: 44–50.

Cerkasskij, V. L. 1980. The role of the raccoon dog in the epizootiology of rabies. Rabies Bulletin Europe 3: 11–14.

Chautan, M., D. Pontier, and M. Artois. 2000. Role of rabies in recent demographic changes in red fox (*Vulpes vulpes*) populations in Europe. Mammalia 64: 391–410.

Cliquet, F., A. L. Guiot, A. Munier, J. Bailly, C. E. Rupprecht, and J. Barrat. 2006. Safety and efficacy of the oral rabies vaccine SAG2 in raccoon dogs. Vaccine 24: 4386–4392.

Drygala, F., N. Stier, H. Zoller, K. Boegelsack, H. M. Mix, and M. Roth. 2007. Habitatnutzung des Marderhundes (*Nyctereutes procyonoides*) im Nordosten Deutschlands. Mammalian Biology. doi:10.1016/j.mambio.2007.09.005.

Eisinger, D., H. H. Thulke, T. Selhorst, and T. Müller. 2005. Emergency vaccination of rabies under limited resources—Combating or containing? BMC Infectious Diseases 5: 10.

Goszczynski, J. 1989. Population-dynamics of the red fox in central Poland. Acta Theriologica 34: 141–154.

———. 1999. Fox, raccoon dog and badger densities in north eastern Poland. Acta Theriologica 44: 413–420.

Grimm, V., and C. Wissel. 2004. The intrinsic mean time to extinction: A unifying approach to analysing persistence and viability of populations. Oikos 105: 501–511.

Helle, E., and K. Kauhala. 1991. Distribution history and present status of the raccoon dog in Finland. Holarctic Ecology 14: 278–286.

———, and ———. 1993. Age structure, mortality, and sex-ratio of the raccoon dog in Finland. Journal of Mammalogy 74: 936–942.

———, and ———. 1995. Reproduction in the raccoon dog in Finland. Journal of Mammalogy 76: 1036–1046.

Holmala, K., and K. Kauhala. 2006. Ecology of wildlife rabies in Europe. Mammal Review 36: 17–36.

Jeltsch, F., M. S. Müller, V. Grimm, C. Wissel, and R. Brandl. 1997. Pattern formation triggered by rare events: lessons from the spread of rabies. Proceedings of the Royal Society of London. Series B 264: 495–503.

Kantorovich, R. A. 1975. Ecology of the raccoon dog and its role in wildlife rabies. *In* Report of consultations on the WHO/FAO coordinated research program on wildlife rabies in Europe. World Health Organization, Geneva, Switzerland, pp. 19–21.

Kauhala, K. 1993. Growth, size, and fat reserves of the raccoon dog in Finland. Acta Theriologica 38: 139–150.

———. 1996. Reproductive strategies of the raccoon dog and the red fox in Finland. Acta Theriologica 41: 51–58.

———, AND E. HELLE. 1995. Population ecology of the raccoon dog in southern Finland—A synthesis. Wildlife Biology 1: 3–9.

———, AND K. HOLMALA. 2006. Contact rate and risk of rabies spread between medium-sized carnivores in southeast Finland. Annales Zoologici Fennici 43: 348–357.

———, ———, AND K. TASKINEN. 1993. Home-range of the raccoon dog (*Nyctereutes-Procyonoides*) in southern Finland. Journal of Zoology 231: 95–106.

———, P. LAUKKANEN, AND I. VON REGE. 1998. Summer food composition and food niche overlap of the raccoon dog, red fox and badger in Finland. Ecography 21: 457–463.

———, ———, W. LAMMERS, AND J. SCHREGEL. 2006. Home ranges and densities of medium-sized carnivores in southeast Finland with special reference to rabies spread. Acta Theriologica 51: 1–13.

———, ———, AND J. SCHREGEL. 2007. Seasonal activity patterns and movements of the raccoon dog, a vector of diseases and parasites, in southern Finland. Mammalian Biology 72: 342–353.

KIM, C. H., C. G. LEE, H. C. YOON, H. M. NAM, C. K. PARK, J. C. LEE, M. I. KANG, AND S. H. WEE. 2006. Rabies, an emerging disease in Korea. Journal of Veterinary Medicine Series B-Infectious Diseases and Veterinary Public Health 53: 111–115.

KING, A. A., A. R. FOOKS, , M. AUBERT, and A. WANDELER (eds.). 2004. Historical perspective of rabies in Europe and the Mediterranean Basin. Office International des Épizooties (World Organization of Animal Health), Paris, France, 359 pp.

LAVROV, N. P. 1971. I togi introduktsii enotovidnoj sobaki (Npg) vothel'nye oblasti SSSR. Trudy kafedry biologii MGZPI 29: 101–166. [In Russian.]

MATOUCH, O., J. VITASEK, Z. SEMERAD, AND M. MALENA. 2007. Rabies-free status of the Czech Republic after 15 years of oral vaccination. Revue Scientifique Et Technique Office International des Épizooties 26: 577–584.

MUSTONEN, A. M., J. ASIKAINEN, K. KAUHALA, T. PAAKKONEN, AND P. NIEMINEN. 2007. Seasonal rhythms of body temperature in the free-ranging raccoon dog (*Nyctereutes procyonoides*) with special emphasis on winter sleep. Chronobiology International 24: 1095–1107.

NASIMOVIC, A. A. and J. A. ISAKOV (eds.). 1985. Pesec, lisica, enotovidnaja sobaka: Razmescenie zapazov, ekologija, ispolzovanie i ohrana. Nauka, Moskva. [In Russian.]

NIIN, E., J. BARRAT, M. KRISTIAN, J. M. DEMERSON, AND F. CLIQUET. 2006. First oral vaccination of wildlife against rabies in Estonia. Developments in Biologicals 125: 145–147.

NOWAK, E. 1984. Verbreitungs- und Bestandsentwicklung des Marderhundes, *Nyctereutes procyonoides* (Gray, 1834) in Europa. Zeitschrift für Jagdwissenschaft 30: 137–154. [In German.]

NYBERG, M., K. KULONEN, E. NEUVONEN, C. EK-KOMMONEN, M. NUORGAM, AND B. WESTERLING. 1992. An epidemic of sylvatic rabies in Finland—Descriptive epidemiology and results of oral vaccination. Acta Veterinaria Scandinavica 33: 43–57.

OGNEV, S. I. 1962. Mammals of eastern Europe and northern Asia. Israel Program for Scientific Translations, Jerusalem, 283 pp.

POTZSCH, C. J., A. KLIEMT, D. KLOSS, R. SCHRODER, AND W. MULLER. 1992. Rabies in Europe—Trends and developments. Developments in Biologicals 125: 59–68.

R-DEVELOPMENT-CORE-TEAM. 2006. R: A language and environment for statistical computing, R Foundation for Statistical Computing, Vienna, Austria.

SINGER, A. 2006. Analysing effects of spatiotemporally distributed species interactions in *Maculinea* systems. PhD Thesis, Philipps-Universität, Marburg, Germany, 228 pp.

———, K. KAUHALA, K. HOLMALA, G. C. SMITH. 2008. Rabies risk in raccoon dogs and foxes. *In* Towards the elimination of rabies in Eurasia, B. Dodet, A. R. Fooks, T. Müller and N. Tordo, the Scientific & Technical Department of the Office International des Épizooties (eds.). Karger, Basel, France, pp. 213–222.

SMITH, G. C., AND S. HARRIS. 1991. Rabies in urban foxes (*Vulpes vulpes*) in Britain: the use of a spatial stochastic simulation model to examine the pattern of spread and evaluate the efficacy of different control régimes. Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences 334: 459–479.

———, AND D. WILKINSON. 2003. Modeling control of rabies outbreaks in red fox populations to evaluate culling, vaccination, and vaccination combined with fertility control. Journal of Wildlife Diseases 39: 278–286.

WESTERLING, B. 1991. Raivotauti Suomessa ja sen torjunta vuosina 1988–90. Suomen Riista 37: 93–100. In Finnish with English summary.

WLODEK, K., AND A. KRZYWINSKI. 1986. The biology and behaviour of the raccoon dog (*Nyctereutes procyonoides*) in Poland. Zeitschrift für Jagdwissenschaft 32: 203–215.

WORLD HEALTH ORGANIZATION (WHO). 2008. Rabies Bulletin Europe, http://www.who-rabies-bulletin.org. Accessed 6 August 2008.

Received for publication 8 August 2008.

APPENDIX 1:
RACCOON DOG AND FOX—RABIES MODEL

The stochastic rabies model simulated disease spread in northeastern Europe following a rabies outbreak (with the introduction of three infected animals to avoid immediate stochastic extinction). The simulated area comprised 5,400 km^2 to map initial disease spread. The rabies model incorporated population dynamics of the two vector species (raccoon dog and red fox) and intra- and interspecific rabies transmission. Population and disease processes were modeled identically for both vector species, although using species-specific parameter values. The model ran on 3-mo time steps to reflect available data and describe seasonality in population and epidemiologic processes. In each time step, the process order was: Births (in spring), infected animals can become rabid, natural mortality, intraspecific infection, interspecific infection (excluding cubs), death of rabid animals, and aging of juveniles to adults (in spring).

Vector population structure and population processes

In northeastern Europe, both species live in small family groups with one male and one female adult: in the model, a family group was represented only by females (sex ratio is generally 1:1). Two developmental stages were considered: a juvenile stage during their first year and an adult stage for older females.

Reproduction: Cubs were born healthy in spring, although healthy and infected (incubating) animals could give birth (no vertical transmission). The number of offspring per group (surviving until summer) was calculated from a Poisson distribution around a mean litter size for juvenile and adult animals.

Mortality: In each time step, animals died according to a density-independent seasonal mortality risk (reflecting main mortality from hunting and other causes). In late autumn and winter intraspecific competition for territory and food additionally led to density-dependent mortality. Thus winter mortality risk was split into a density-dependent and a density-independent term. The latter reflected the 50% death rate from fox hunting in Poland (Goszczynski, 1989):

$$p_{\mathrm{win}}^{\mathrm{mort}} = \min\left[0.5\cdot d_{\mathrm{win}}\cdot\left(1+\frac{N}{N_{\mathrm{sta}}}\right),1\right],$$

where d_{win} is mortality rate in winter (depending on the age class), N is the number of females in the stage class and N_{sta} is the number of territories according to population density found in the field (Kauhala et al., 2006); N_{sta} can be considered as a carrying capacity in the linear density dependence process.

Within-group rabies transmission

In the model, an infectious animal infected all family members due to the high within-group contact rates (Smith and Wilkinson, 2003; Eisinger et al., 2005; Kauhala and Holmala, 2006). In summer, young cubs remained with their parents and hardly contacted other animals, and thus cubs of rabid parents died without further disease transmission. The number of cubs dying from rabies in summer was drawn from a Poisson distribution around the mean number of cubs in rabid families.

Between-family-group transmission

In a time step, each healthy animal could be infected by conspecific rabid animals (binomial distribution). The transmission probability in the model depended on the number of rabid animals in a time step relative to the population size at which contact rates were estimated. Transmission probability assumed an infectious period of 3 days.

Cross-species infection

Close proximity between raccoon dogs and foxes has been observed (Kauhala and Holmala, 2006), although knowledge of cross-species transmission is unavailable. Thus, for simplicity, the number of animals infected across species was calculated by a Poisson distribution with mean number of infectious animals (from one rabies vector species) times number of susceptibles (from the other rabies vector species) times contact rate, with the maximum number of newly infected animals limited by the number of susceptibles. The cross transmission process was implemented symmetrically in both directions of transmission.

Cross-species transmission was adjusted to make rabies spread likely in the area of northeastern Europe, as raccoon dog and fox rabies is common in several Baltic states (World Health Organization, 2008).

Incubation

In each time step, an infected animal became infectious with a fixed probability.

TABLE A1. Model parameters.

Reproduction	
Fox	
Mean number of female offspring per adult female[a]	2.175[b]
Mean number of female offspring per juvenile female[a]	1.900[b]
Raccoon dog	
Mean number of female offspring per adult female[a]	4.610[c]
Mean number of female offspring per juvenile female[a]	2.75[c]
Mortality (per season)	
Fox	
Probability of adult death[d]	(0.110, 0.110, 0.110, 0.290)[b]
Probability of juvenile death[d]	(0.110, 0.480, 0.170, 0.430)[b]
Raccoon dog	
Probability of adult death[d]	(0.022, 0.022, 0.314, 0.286)[c,e]
Probability of juvenile death[d]	(0.022, 0.500, 0.415, 0.498)[c,e]
Population size	
Fox	
Stable population size in winter (territories per km^2)	0.2[f]
Raccoon dog	
Stable population size in winter (territories per km^2)	0.3[f]
Rabies	
Fox	
Incubation period within one season	0.95[g,h,i]
Adult to adult cross group infection rate (all seasons)	0.14[g]
All other adult/ juvenile cross group infection rates[d]	(0.14, 0.0, 0.14, 0.14)[g]
Density at contact rate estimation (families/km^2)	0.215[c,g]
Raccoon dog	
Incubation period within one season	0.95[j]
Distribution of cross group infection rates[k,l]	(0.27, 0.43, 0.24, 0.05, 0.01)[f]
Density at contact rate estimation (territories per km^2)	0.38[g]
Cross species	
Cross species infection rate[l]	0.0016

[a] Surviving spring season.

[b] Goszczynski (1989).

[c] Helle and Kauhala (1995).

[d] Applied to spring, summer, autumn, and winter, respectively.

[e] Helle and Kauhala (1993).

[f] Kauhala et al. (2006).

[g] Eisinger et al. (2005).

[h] Smith and Harris (1991).

[i] Suppo et al. (2000).

[j] As foxes.

[k] Probabilities to contact 0, 1, 2, 3, or 4 neighboring groups.

[l] Juveniles are not involved in transmission during summer. Winter transmission depends on hibernation scenario.

Parameterization

All parameter values are listed in Table A1. Birth and death rates of foxes (Goszczynski, 1989) and raccoon dogs (Helle and Kauhala, 1993, 1995) were derived from life tables. During the study on raccoon dogs in Finland, population size declined. In order to obtain a stable population (for the model), productivity of adult raccoon dogs was slightly adjusted. From the annual mortality rates, seasonal rates were derived, taking into account seasonal variation (Goszczynski, 1989; Helle and Kauhala, 1993). The model correctly reproduced ratios of adults to juveniles found in the study area.

Fox neighborhood contact rates were taken from a spatially explicit model (Eisinger et al., 2005), with mean field contact probabilities scaled according to the process described therein.

Contact rates between raccoon dog groups were derived from proximity counts of animals and territory overlap estimated in a radio-tracking study (Kauhala and Holmala, 2006). The calculations assume that individual animals contact their neighbors independently.

To estimate incubation probability within a three-mo period, data from three different modeling approaches (Smith and Harris, 1991; Suppo et al., 2000; Eisinger et al., 2005) were compared. The probability that an infected fox incubated rabies ranged from 0.8 to 1.0, but tended toward higher values. For raccoon dogs, such data were not available and we assumed the same risk as for foxes.

LITERATURE CITED IN APPENDIX

EISINGER, D., H. H. THULKE, T. MÜLLER, AND T. SELHORST. 2005. Emergency vaccination of rabies under limited resources—Combating or containing? BMC Infectious Diseases 5: 10.

GOSZCZYNSKI, J. 1989. Population dynamics of the red fox in central Poland. Acta Theriologica 34: 141–154.

HELLE, E., AND K. KAUHALA. 1993. Age structure, mortality, and sex-ratio of the raccoon dog in Finland. Journal of Mammalogy 74: 936–942.

———, AND ———. 1995. Reproduction in the raccoon dog in Finland. Journal of Mammalogy 76: 1036–1046.

KAUHALA, K., AND K. HOLMALA. 2006. Contact rate and risk of rabies spread between medium-sized carnivores in southeast Finland. Annales Zoologici Fennici 43: 348–357.

———, ———, W. LAMMERS, AND J. SCHREGEL. 2006. Home ranges and densities of medium-sized carnivores in south-east Finland with special reference to rabies spread. Acta Theriologica 51: 1–13.

SMITH, G. C., AND S. HARRIS. 1991. Rabies in urban foxes (*Vulpes vulpes*) in Britain: The use of a spatial stochastic simulation model to examine the pattern of spread and evaluate the efficacy of different control regimes. Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences 334: 459–479.

———, AND D. WILKINSON. 2003. Modeling control of rabies outbreaks in red fox populations to evaluate culling, vaccination, and vaccination combined with fertility control. Journal of Wildlife Diseases 39: 278–286.

SUPPO, C., J. M. NAULIN, M. LANGLAIS, AND M. ARTOIS. 2000. A modelling approach to vaccination and contraception programmes for rabies control in fox populations. Proceedings of the Royal Society of London. Series B 267: 1575–1582.

WORLD HEALTH ORGANIZATION. 2008. Rabies Bulletin Europe, http://www.who-rabies-bulletin.org. Accessed 6 August 2008.