Estonian Journal of Ecology, 2009, **58**, 3, 181–191 doi: 10.3176/eco.2009.3.03

Can human hunters substitute for large carnivores? An examination based on disease in ungulate populations

Yukichika Kawata

Division of Food Hygiene, Department of Animal and Food Hygiene, Obihiro University of Agriculture and Veterinary Medicine, Inada-cho, Obihiro, Hokkaido 080-8555, Japan; ykawata@obihiro.ac.jp

Received 17 December 2008, revised 26 May 2009

Abstract. It is pointed out that large carnivores such as wolves prey on weak individuals in ungulate populations, which results in a healthier local ungulate population. However, this hypothesis has not been fully evaluated. The purpose of the paper is to examine whether human hunting can improve the health status of local ungulate populations. To accomplish this, we focus on disease and build an epidemiological Susceptible–Infected–Recovered model. On the basis of numerical simulations of our model, we demonstrated that although human hunting helps prevent the spread of disease, it does not help in the selective removal of infected individuals in a population.

Key words: predator–prey, hunting, substitution of predation, SIR model.

INTRODUCTION

Human hunters often regard large carnivores such as wolves and lynxes as their competitors in the hunting of ungulates and other game animals. Due to this perception and other reasons, large carnivores have been persecuted throughout their geographic range, which has occasionally resulted in the extinction of a species at the regional or national level. For example, according to the International Union for Conservation of Nature and Natural Resources (IUCN) 2008 Red List of Threatened Species (http://www.iucnredlist.org/details/3746) wolf (*Canis lupus*) has become regionally extinct in Austria, Belgium, Ireland, Japan, Luxembourg, the Netherlands, Switzerland, and the United Kingdom, and is possibly extinct in Bangladesh as well. The present range of wolves is much more restricted than it used to be.

At least two criticisms can be levelled at this stern reality of persecution. First, there must be some positive or beneficial aspects of large carnivores towards ungulates, and we have yet to recognize the value of these aspects. Some instances can easily be adduced. For example – as far as the author is aware since this line of argument is not often presented – large carnivores and other wild carnivores consume carcasses of wild animals; this consumptive behaviour may be effective

in preventing diseases from spreading provided the diseases are not infectious to large carnivores. Associated with this, as an increasing number of studies have pointed out recently, wolves attack weak individuals such as the sick, the infirm, the physically handicapped, and/or juveniles. Such predation by wolves contributes to the maintenance of a healthy ungulate population (Schaller, 1972; Moore, 2002; Packer et al., 2003 for the case of predators in general; Skonhoft, 2006: 832; Kawata, 2007: 58). In addition, predation by large carnivores can regulate prey populations and this can lead to a reduction in agricultural and forestry damage caused by ungulates.

Second, given these beneficial aspects of carnivores and the effectiveness of their hunting, which contribute to the maintenance of healthy ungulate populations as mentioned above, it is doubtful whether human hunters can substitute the ecological role played by large carnivores. On the one hand, large carnivores – at least, wolves – can easily detect weak individuals that are more susceptible to attack. On the other hand, human hunters often chance upon healthy individuals in an ungulate population and kill them; moreover, some have a tendency to preferentially hunt big and/or strong individuals. This leads us to the question whether human hunters can contribute to improving the physical condition of local ungulate populations as large carnivores do.

In this paper, I pose the following two questions: (1) Can human hunters prevent the spread of disease among ungulates? and (2) Can human hunters help eradicate disease among individuals in an ungulate population? In what follows, I build a mathematical model and use numerical simulations to examine the above two questions.

MATERIALS AND METHODS

Mathematical model

A basic model employed in epidemiology is the Susceptible–Infected–Recovered (SIR) model, which can be traced back to Kermack & McKendrick (1927), or more directly to Anderson & May (1979), May & Anderson (1979), Anderson (1991). Many models have since been derived from or are modified versions of the SIR model. In what follows, to simplify the model and numerical simulations, we ignore the aspect 'Recovered'.

Suppose that we manage a local population of an ungulate species and that each individual in the population can be classified as either susceptible to or infected with a disease agent. Susceptible individuals are those for whom the possibility of being infected exists, and some of the infected individuals will get sick and die. Hereinafter, we denote the number of susceptible and infected individuals as S and I, respectively.

We further suppose the following. Both susceptible and infected individuals have the same reproductive ability, and the birth rate is a decreasing density-dependent function of the total population N, which is the sum total of S and I. Then, the birth rate can be described as $b(N) = b_1 - b_2 N$. If a foetus is infectible, then the

term $b(N)N$, which represents the number of newborn individuals, should be included in the equation of susceptible individuals. Suppose that the death rate is an increasing function of population density and is described as $d(N) = d_1 + d_2 N$ and $d(N)N = d(N)S + d(N)I$. The rate of increase in the number of infected individuals is proportional to the product of the number of susceptible individuals and infected individuals. Let the transmission rate be $\alpha (0 \le \alpha \le 1)$; the increase rate is given by αSI. Suppose that the harvest by hunters is proportional to the population size and described as $H = \beta S + \beta I$, where β $(0 \le \beta \le 1)$ denotes the harvest rate. Finally, suppose that μ $(0 \le \mu \le 1)$ is the incidence rate: this represents the proportion of individuals in the population showing symptoms of a disease.

Next, the dynamics of susceptible and infected individuals can be described by the following differential equations (see also Fig. 1).

$$\frac{dS}{dt} = b(N)N - d(N)S - \alpha SI - \beta S, \tag{1}$$

$$\frac{dI}{dt} = \alpha SI - d(N)I - \mu I - \beta I. \tag{2}$$

By summing up these differential equations, we obtain the following equation, which describes the dynamics of the entire population.

$$\frac{dN}{dt} = b(N)N - d(N)N - \mu I - \beta N = r\left[1 - \frac{N}{K}\right]N - \mu I - \beta N, \tag{3}$$

where $r = b_1 - d_1$ $(0 < r)$ and $K = \dfrac{r}{b_2 + d_2}$ $(0 < K)$, which refer to the intrinsic growth rate and the carrying capacity, respectively.

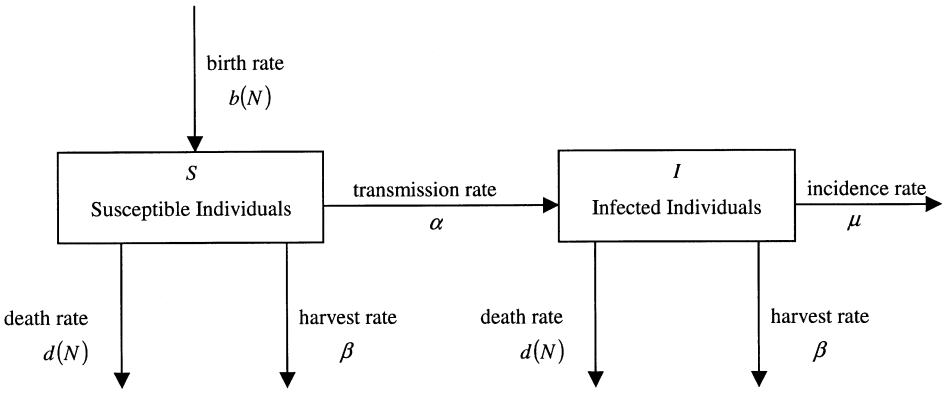

Fig. 1. Flow diagram of disease transmission dynamics in a population using a susceptible (S)–Infected (I) disease spread model.

Steady state condition

In this analysis, we are interested in the steady state or sustainable situation. Therefore, in what follows we examine the steady state conditions for these cases: with hunting and without hunting, and with disease and without disease. Firstly, we build a full model where both hunting and disease are included.

In the steady state, there is no increment in the population size since I, S, and N take the same value over time. In other words, $dN/dt = 0$, $dS/dt = 0$, and $dI/dt = 0$. From the first two conditions, we obtain the following equations.

$$I = \frac{\Psi}{\mu}, \tag{4}$$

$$S = \frac{b(N)N}{\Omega + \alpha I}, \tag{5}$$

where $\Psi = r\left[1 - \frac{N}{K}\right]N - \beta N$ and $\Omega = d(N) + \beta$. From the last condition $dI/dt = 0$, because $I \neq 0$, it follows that

$$S = \frac{\Omega + \mu}{\alpha}. \tag{6}$$

From eqs (4) to (6), we obtain the following equation.

$$\Omega^2 + \left[\mu + \frac{\alpha\Psi}{\mu}\right]\Omega + \alpha[\Psi - b(N)N] = 0. \tag{7}$$

Population size N, which satisfies Eq. (7), is the steady state population size, hereafter denoted as N^*. Since Eq. (7) is the third-degree equation of N, N^* can be searched numerically using the Solver function in Microsoft Excel. When N^* is detected numerically, S^* and I^* can be calculated using eqs (6) and (4), respectively.

Next, we examine the case where this ungulate population is completely free from disease. In this case, α is set to zero, and Eq. (3) is reduced to

$$\frac{dS}{dt} = \frac{dN}{dt} = r\left[1 - \frac{N}{K}\right]N - \beta N. \tag{8}$$

In the steady state, we obtain the following condition.

$$N^* = \frac{K[r-\beta]}{r}. \tag{9}$$

Equation (9) implies that if the intrinsic growth rate is higher than the harvest rate, N^* takes a positive value, and if otherwise, $N^* = 0$.

Parameter setting and numerical simulation procedure

As a base case, we set the parameter values as follows: $b_1 = 5$, $b_2 = 0.002$, $d_1 = 4.9$, $d_2 = 0.002$, $\alpha = 0.8$, $\beta = 0.05$, and $\mu = 0.08$. Then, it follows that $r = 0.1$ and $K = 25$ (Table 1). These parameter values are similar to those for the Hokkaido sika deer *Cervus nippon yesoensis* in Kawata (2006), where r and K are set at 0.15/year and 25/km^2, respectively. Since our model employs numerical simulation, we also conduct a sensitivity analysis to confirm the robustness of our results and determine the qualitative change.

We calculate the steady state population sizes S^* and I^* using the Solver function in Microsoft Excel. Firstly, we search for N^* that satisfies Eq. (7), and then calculate S^* and I^* using eqs (6) and (4), respectively. The following constraints should be satisfied: $S^* \geq 0$, $I^* \geq 0$, and $S^* + I^* = N^*$. Basically, if at least one of these constraints is not satisfied, the initial value of N is changed. This is because of the possibility that we obtained a local non-steady solution, although there also exists a steady state solution. However, because we calculate steady state solutions numerically, some margin of error may be observed between N^*, which is calculated with Eq. (7), and the sum of $S^* + I^*$, which are calculated with eqs (6) and (4). In such a case, we adopt the latter value.

Table 1. Parameter settings for the base case and cases with changed parameters

		Base	Case 1	Case 2	Case 3
			Changed parameters		
			α, β	μ, α, β	r, α, β
r		0.1			0.2
K		25			
	b_1	5			
	b_2	0.002			0.004
	d_1	4.9			4.8
	d_2	0.002			0.004
α		0.8	0–0.9	0–0.9	0–0.9
β		0.05	0–0.1	0–0.1	0–0.1
μ		0.08		0.04	

Note 1: β is set such that $\beta \leq r$. Otherwise, N^* is less than 0, as is easily derived from Eq. (9). *Note 2*: The blank columns denote that the values are the same as those in the base case.

RESULTS

When the parameter values are set according to the base case, the steady state population sizes S^*, I^*, and N^* are 6.31, 1.78, and 8.09, respectively. In Fig. 2, we present the steady state population sizes where the values of the transmission rate α are set 0.8 and 0.4, and the harvest rate β is changed from 0 to 0.1. Moreover, in Fig. 2, $N^*(\alpha = 0)$ is the case where all individuals in the population are free from infection. As β increases, the steady state population size N diminishes, and N becomes zero when $\beta = r$, as is suggested by Eq. (9).

When $\alpha = 0.4$, $S^*(\alpha = 0.4)$, $I^*(\alpha = 0.4)$, and $N^*(\alpha = 0.4)$ are the steady state population sizes of S^*, I^*, and N^*, respectively. As β increases, $I^*(\alpha = 0.4)$ decreases but $S^*(\alpha = 0.4)$ is almost stable (decreases only slightly), and therefore, $N^*(\alpha = 0.4)$ decreases. When $\beta = 0.0497$, it follows that $I^*(\alpha = 0.4) = 0$ and $N^*(\alpha = 0.4) = N^*(\alpha = 0)$. This means that the population size of $I^*(\alpha = 0.4)$ becomes zero and $N^*(\alpha = 0.4)$ coincides with $N^*(\alpha = 0)$ when $\beta = 0.0497$. For $\beta > 0.0497$, the line of $N^*(\alpha = 0.4)$ coincides with that of $N^*(\alpha = 0)$. These results remain the same for the case when $\alpha = 0.8$. As is easily seen from Fig. 2, as α increases, N^* decreases, but as β increases, the difference between $N^*(\alpha = 0)$ and others such as $N^*(\alpha = 0.4)$ diminishes.

Next, we show two cases where (1) the incidence rate μ is halved ($\mu = 0.04$) and (2) the intrinsic growth rate r is doubled ($r = 0.2$). Figure 3 presents the results obtained when μ is halved. As compared with the base case, the steady state population sizes N^* and I^* increase, whereas S^* is almost stable (decreases only slightly) provided $I^* > 0$. Further, when $I^* = 0$, S^* and N^* coincide with the values of the case where $\alpha = 0$.

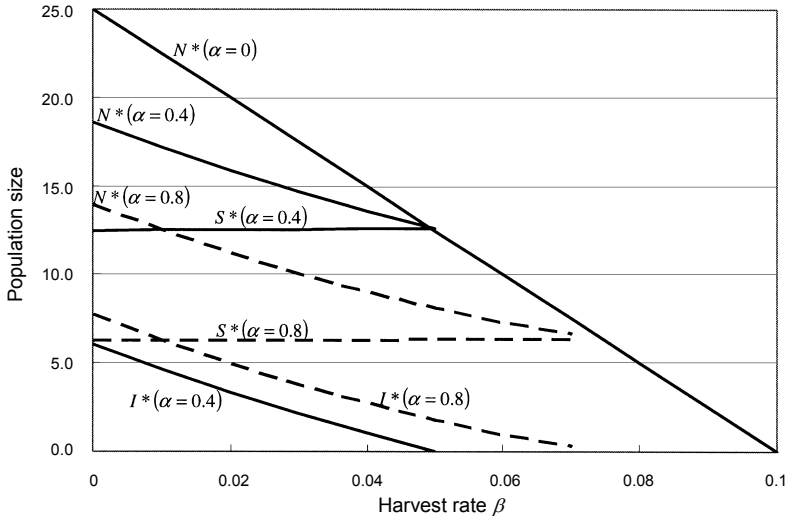

Fig. 2. Harvest rate and steady state population size ($r = 0.1$, $K = 25$, $\mu = 0.08$).

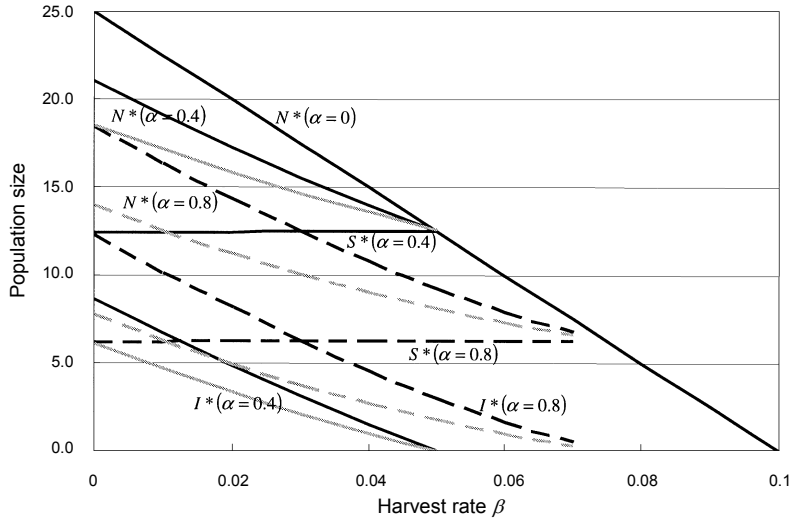

Fig. 3. Harvest rate and steady state population size ($r = 0.1$, $K = 25$, $\mu = 0.04$). *Note*: The grey lines are $N^*(\alpha = 0.4)$, $N^*(\alpha = 0.8)$, $I^*(\alpha = 0.4)$, and $I^*(\alpha = 0.8)$ from Fig. 2. Since the trajectory of $N^*(\alpha = 0)$ is the same, and $S^*(\alpha = 0.4)$ and $S^*(\alpha = 0.8)$ are almost the same for the cases where $\mu = 0.08$ and 0.04, they are not depicted with the grey lines in this figure.

The results shown in Fig. 4 pertain to the case where r is doubled. As compared with the base case, the steady state population sizes N^* and I^*

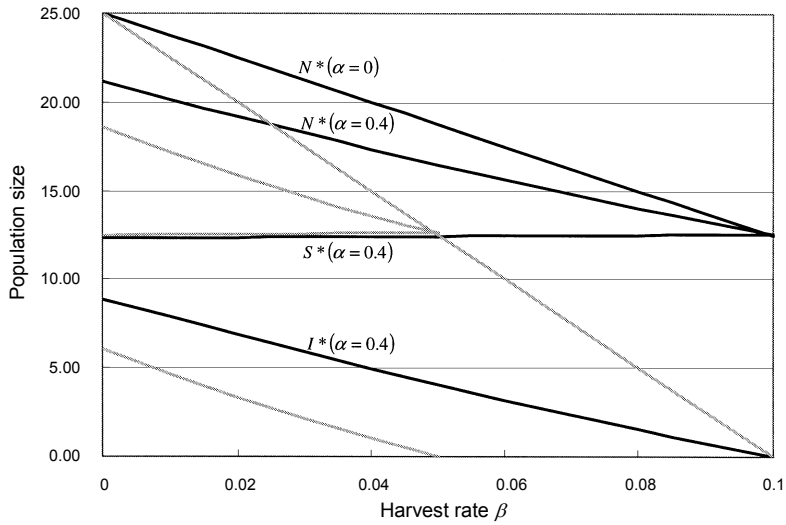

Fig. 4. Harvest rate and steady state population size ($r = 0.2$, $K = 25$, $\mu = 0.08$). *Note*: The grey lines are $N^*(\alpha = 0)$, $N^*(\alpha = 0.4)$, $S^*(\alpha = 0.4)$, and $I^*(\alpha = 0.4)$ from Fig. 2.

increase, whereas S^* is almost stable (decreases only slightly). Moreover, because r is doubled, the value of β when $N^*(\alpha = 0) = 0$ is also doubled, and the slope of $N^*(\alpha = 0)$ becomes gradual.

DISCUSSION

Firstly, we explain the meaning of steady state in greater detail. Let $S^* = 10$ head/km^2, $I^* = 4$ head/km^2, and $\beta = 0.05$. Then, the sustainable total harvest is $H^* = 0.05 \times (10 + 4)$ head/km^2 = 0.7 head/km^2, while those of susceptible and infected individuals are 0.5 and 0.2 head/km^2, respectively. Therefore, as long as 0.5 and 0.2 head/km^2 are hunted every year, $S^* = 10$ head/km^2 and $I^* = 4$ head/km^2 are maintained in the subsequent years. This scenario is referred to as the steady state.

Now, we examine the implication of our results. For some value of β, we have the following prediction (called prediction I). Once we reach some steady state, we may maintain S^* and I^* provided we randomly hunt ungulates. This is because β is the same for S and I. In some cases, there is a possibility that the population size may deviate from S^* and I^*, but this can be easily modified.

We also have the following prediction (called prediction II). As Fig. 2 suggests, the steady state population size of S^* is almost stable (increases only slightly) as the hunting rate β increases. Therefore, as long as we neglect the disposal and hunting costs of infected individuals, the choice between a large and a small value of β is almost a matter of indifference to hunters.

Next, we examine the case where the value of β is increased from one year to the next. If random hunting is continued under this situation, S^* and I^* in one year are not satisfied, and S' and I' are attained in the following year, where $S' < S^*$ and $I' > I^*$. Therefore, it is necessary to adjust S' and I' according to the new levels of S^* and I^* under the new value of β. We refer to the above as prediction III.

Let us now introduce the disposal and hunting costs of infected individuals. As the value of β increases, the hunting cost may increase drastically because it is quite difficult to catch the remaining few individuals. On the other hand, the disposal cost decreases because the amount of hunting decreases (prediction IV). Figure 5 depicts these hunting costs, including the sum of the costs (denoted as total cost) and the total revenue, which is generated from the hunting of susceptible individuals. The optimum β is obtained at the point where $dTR/d\beta = dTC/d\beta$ or MR = MC. In other words, hunters select β such that TR(β) – TC(β) is maximized.

Finally, we examine the two questions posed in this paper. First, we deal with Question 1. From prediction III, we can state that it is reasonable for hunters to continue hunting at the rate β^*. In addition, from prediction I, we can gather that it is relatively easy to maintain an optimum population level. Therefore, S^* and I^* are maintained such that they correspond to this β^*, and the spread of the disease is prevented. We now proceed to Question 2. The targeted removal of

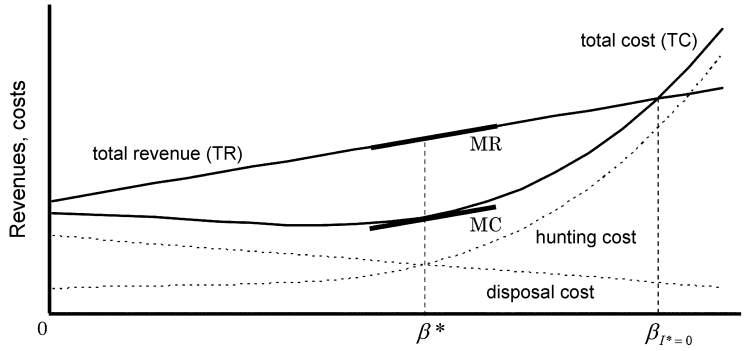

Fig. 5. Determination of the optimum harvest rate.

infected individuals by human hunters is not realistic. While increasing the value of β, infected individuals must be hunted selectively or intentionally, as prediction III suggests. However, as prediction IV suggests, hunting costs may drastically increase as the value of β increases. Moreover, at $\beta_{I^*=0}$, it may often be the case that TR < TC is maintained. In other words, only when TR is greater than TC at $\beta_{I^*=0}$ will all of the infected individuals be removed.

As the above discussions are based on theoretical parameter values, we performed a sensibility analysis. In what follows, we examine the results of the sensibility analysis. As Fig. 2 suggests, when the transmission rate α increases, the steady state population size decreases. Figure 3 suggests that as the incidence rate μ decreases, the steady state population size increases. Figure 4 suggests that as the intrinsic growth rate r increases, the steady state population size increases. These are consistent with intuition. Furthermore, as these figures suggest, the shape of S^* and I^* are retained, which implies that the above discussion and conclusions remain valid when the parameter values are changed.

CONCLUSIONS

It is pointed out that large carnivores, particularly wolves, attack weak prey, i.e. the sick, the infirm, the physically handicapped, and/or juveniles. However, given the extensive hunting of large carnivores and the lack of attention paid to these beneficial aspects, the current population sizes of large carnivores have diminished considerably in many areas. Moreover, in some regions they have become extinct. Although the reintroduction of large carnivores is occasionally considered and executed, it is not always feasible. Our study suggests that to some extent, human hunting can act as a substitute for predation by large carnivores.

However, the replacement of large carnivores by human hunters for the purpose of predation has some limitations. Although human hunters can prevent

the spread of disease, they may be unable to remove infected individuals completely because of cost issues, based on our results presented above. In addition, Choisy & Rohani (2006) suggest that harvesting may increase the severity of the wildlife disease epidemic. On the other hand, typically, large carnivores selectively hunt such infected individuals. At best, human hunters can approximate the role played by large carnivores. Therefore, greater attention should be paid to the ecological role and beneficial aspects of large carnivores.

In this paper, several issues remain to be resolved. To name a few, our results should be compared with the situations where large carnivores do exist. For a comparison between hunting by humans and predation by large carnivores, a modified model is required, and we leave this examination for future research. In addition, we had set the parameter values artificially, based on a former study. Accumulation of more empirical data is essential for confirming and enhancing the accuracy of our conclusion.

ACKNOWLEDGEMENTS

The author expresses heartfelt gratitude to two referees for the valuable comments and suggestions of many references, most of which have been incorporated in this paper, owing to which the quality of the manuscript has improved substantially.

REFERENCES

Anderson, R. M. 1991. Discussion: the Kermack–McKendrick epidemic threshold theorem. *Bull. Math. Biol.*, **53**(1–2), 3–32.

Anderson, R. M. & May, R. M. 1979. Population biology of infectious diseases: Part I. *Nature*, **280**, 361–367.

Choisy, M. & Rohani, P. 2006. Harvesting can increase severity of wildlife disease epidemics. *Proc. Roy. Soc. Biol. Sci.*, **273**, 2025–2034.

Kawata, Y. 2006. Economic resource or mammalian pest?: A reconsideration of the management of wild deer. *Jpn. J. Rural Econ.*, **8**, 12–25.

Kawata, Y. 2007. An economic analysis of the influence of different attitudes toward game animals: emphasizing the significance of large carnivores. *Balt. J. Econ.*, **6**(2), 57–78.

Kermack, W. O. & McKendrick, A. G. 1927. A contribution to the mathematical theory of epidemics. *Proc. Roy. Soc. London, Ser. A*, **115**, 700–721. (Reprinted in *Bull. Math. Biol.*, 1991, **53**, 33–55.)

May, R. M. & Anderson, R. M. 1979. Population biology of infectious diseases: Part II. *Nature*, **280**, 455–461.

Moore, J. 2002. *Parasites and the Behaviour of Animals*. Oxford University Press.

Packer, C., Holt, R. D., Hudson, P. J., Lafferty, K. D. & Dobson, A. P. 2003. Keeping the herds healthy and alert: implications of predator control for infectious disease. *Ecol. Lett.*, **6**(9), 797–802.

Schaller, G. B. 1972. *The Serengeti Lion: A Study of Predator–Prey Relations*. University of Chicago Press.

Skonhoft, A. 2006. The costs and benefits of animal predation: an analysis of Scandinavian wolf re-colonization. *Ecol. Econ.*, **58**(4), 830–841.

Kas jahimehed suudavad suurkiskjaid asendada? Sõraliste populatsiooni haigusel põhinev uuring

Yukichika Kawata

On oletatud, et suurkiskjate, näiteks hundi saagiks langevad sõraliste populatsiooni nõrgemad isendid, mille tulemusena paraneb populatsiooni tervislik seisund. Siiski pole see hüpotees piisavalt kontrollitud. Artikli eesmärgiks on uurida, kas inimeste peetav jaht parandab sõraliste populatsiooni tervislikku seisundit. Selleks on keskendutud haigusele ja koostatud tundlikkuse-haigestumise-paranemise mudel, mille abil on näidatud, et kuigi jahipidamine takistab haiguse levikut, ei aita see haigeid isendeid populatsioonist eraldada.