J. evol. biol. 10 (1997) 77–85
1010–061X/96/010077–09 $1.50 + 0.20/0

Journal of Evolutionary Biology

Parent–offspring regression suggests heritable susceptibility to ectoparasites in a natural population of kittiwake *Rissa tridactyla*

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Key words: Host-parasite interactions; heritability; *Ixodes uriae*; *Rissa tridactyla*; susceptibility.

Abstract

Little information is available on the genetic variability of host susceptibility to parasites in natural populations despite its importance for the understanding of the evolution of host-parasite interactions. A long-term demographic and epidemiologic survey of a seabird population allowed us to investigate the potential correlation between parent and offspring ectoparasite load, while controlling for various environmental factors. In particular, parasite loads were measured for all individuals (i.e., parents and offspring) when they were nestlings and the effect of the year and breeding cliff were taken into account. The positive correlation found between parent and offspring parasite loads suggests a heritable susceptibility to ectoparasitism by ticks in this host population and that this character has the potential to respond to natural selection.

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Introduction

Host susceptibility or resistance to parasites has important consequences for the evolution of parasite virulence (May and Anderson, 1983; 1990; Bull, 1994; Read, 1994), for host sexual selection (Hamilton and Zuk, 1982; Clayton, 1991), host sexual reproduction (Hamilton, 1980), the population dynamics of host and parasite populations (Anderson and May, 1978; Anderson, 1988), and for the development of approaches to disease control. Genetic variation in resistance is required for the hosts to be able to adapt to the rapidly evolving parasites. In particular, Hamilton and Zuk's (1982) good gene model of sexual selection, which suggests that females choose males on the basis of criteria that reveal the capacity of males to cope with parasites present in the host population, relies on the assumption that host-parasite coevolution maintains heritable parasite resistance.

Evidence from laboratory studies suggests that some of the observed phenotypic variance in host susceptibility to parasites may be attributed to genetic factors (Madhavi and Anderson, 1985; Wakelin and Blackwell, 1988; Carton and Nappi, 1991; Young et al., 1995). A considerable number of these studies dealt with rodents–cestodes associations (e.g., Wassom et al., 1986; Gregory et al., 1990). On the other hand, little information exists on genetic variation of susceptibility to parasites within natural populations (Lively, 1989; Grosholz, 1994; Henter and Via, 1995), and notably concerning vertebrate hosts (Møller, 1990a). The aim of the present study was, therefore, to test if the within-population variability in susceptibility to parasites reflects genetic variation in a natural population of the bird *Rissa tridactyla* parasitized by the ectoparasite *Ixodes uriae*.

Material and methods

The kittiwake is a pelagic seabird that lives in northern Palearctic and Nearctic, and that comes to breed on sea cliffs in spring and summer. Females lay one brood per year, ranging from one to three eggs. The male and female participate equally in egg brooding and chick rearing. The mean age at first reproduction is 4 years, and the high survival rate allows several reproductive attempts during the individual lifespan, which can reach 15 years or more (Coulson and Thomas, 1985; Danchin and Monnat, 1992).

Ixodes uriae is a haematophagous ectoparasite. Its life-cycle is spread over several years depending on the seasonal presence of its potential hosts (Eveleigh and Threlfall, 1974, Steele et al., 1990) In the western Palearctic it parasitizes most seabirds species, principally puffins *Fratercula arctica*, guillemots *Uria aalge*, and kittiwakes (Mehl and Traavik, 1983). Each tick development stage (larva, nymph and adult female) takes only one blood meal, which lasts several days (Eveleigh and Threlfall, 1974). Between these blood meals, *Ixodes uriae* go back to the cliff substratum, where it moults to the next stage or lays its eggs. Therefore the tick spends almost all its life in the nesting substratum and there is no actual vertical transmission between parents and offspring. Direct pathogenic effects of *I. uriae* on

kittiwake chicks have been observed in cases of hyperinfestations (Chastel et al., 1987). Moreover, this tick is the vector of different microparasites, such as arboviruses (Chastel, 1988) and the Lyme disease agent, *Borrelia burgdorferi* (Olsén et al., 1993). The impact of *I. uriae* on kittiwake population dynamics is suspected through its effect on reproductive success (Boulinier and Danchin, 1996). Experimental evidence of deleterious effects of haematophagous ectoparasites on their host exit for different bird-ectoparasite systems (e.g., Møller, 1990b; Chapman and Georges, 1991).

The study has been conducted on quasi-monospecific kittiwake colonies in Cap Sizun, West Brittany, France (48° 5′ N, 4° 36′ W). Between 1983 and 1993, the number of ticks (*I. uriae*) was recorded on nestlings at the time of banding (age ranging from 6 to 35 days). An average of 426 nestlings per year was searched for ticks (range: 178–830). This was done through visual examination and palpation. This method allows the detection mainly of nymphs and adults (Danchin, 1992) and does not provide an absolute estimate of the nestling tick load; however, this estimate can be considered as a reliable measure to compare parasite loads between individuals. In fact, in 1994 we checked a sample of individuals twice in order to determine the temporal repeatability of the parasite load. This was done considering one chick per nest, and a minimum of seven days between the two records. The measure was highly repeatable (Spearman rank correlation, $n = 28$, $r = 0.67$, $p < 0.001$). Nestlings were individually colour-banded, allowing life-long identification. Their wing length was recorded in order to estimate their age through a linear regression (Monnat and Pasquet, unpublished). The breeding sites were mapped allowing the localisation of every breeding attempt. Colonies were sub-divided in cliffs delimited by topographic criteria. The number of nests on a given cliff ranged from 1 to 121. Reproduction was monitored each year. The sex of reproducing birds was determined by observing sexual displays.

We used parent (F_0)–offspring (F_1) regression to estimate the heritability of ectoparasite load. Resemblance between parents and offspring may arise from a number of environmental effects which would inflate the estimate of genetic variation. In order to reduce the potential contribution of environmental variation to the resemblance between parents and offspring, we statistically factored out spatial and temporal variability in both parents and offspring parasite load. Moreover we factored out the potential effect of nestling age on parent and offspring parasite load. Parasite load was measured for both parent and offspring when they were nestlings. For the parents, we performed an ANCOVA on log-transformed parasite load ($\log[x + 1]$) with year and breeding cliff as factors and with nestling age and squared nestling age as covariates. Similarly, for the offspring we performed an ANCOVA on log-transformed parasite load with year and breeding cliff as factors and with nestling age and squared nestling age as covariates. We used the residuals of these models to estimate parent-offspring regression. For each parent, the mean of its offspring was used. We used age and squared nestling age as covariates because a quadratic relationship between nestling parasite load and nestling age was found on a larger and independent data set (Boulinier and Danchin, 1996). We also tested if parental contribution on offspring phenotype differed between fathers and

Table 1. ANCOVA on the parental log-transformed parasite load in relation to the year, breeding cliff, nestling age, and squared nestling age.

Sources	df	Type III SS	*F* value	*p*
Year	6	5.23	2.29	0.040
Breeding cliff	16	5.03	0.83	0.653
Nestling age	1	0.13	0.34	0.563
Squared nestling age	1	0.01	0.02	0.901
Error	104	39.53		

mothers. To investigate this issue, we used an ANCOVA on log-transformed offspring parasite load with parent sex as a factor and log-transformed parent residual parasite load as a covariate. As the slope between father parasite load and offspring parasite load did not differ from the slope between mother parasite load and offspring parasite load (ANCOVA: $p > 0.10$ for the parent sex–offspring parasite load interaction), we randomly selected one parent to compute the parent-offspring regression when data were available for both. Heritability of susceptibility to parasite load was estimated as twice the slope of the parent-offspring regression (Falconer, 1989). All analyses were performed using SAS (SAS Institute, 1990).

Results

Tick load of the F_0 individuals was measured during 7 consecutive years (1983–1989) and in 17 different cliffs. Only temporal variation was a significant predictor of F_0 parasite load, whereas spatial variation as well as the nestling age when parasite load was measured were not correlated with F_0 parasite load (Tab. 1). Tick load of the F_1 individuals was also measured during 7 consecutive years (1987–1993) and in 13 cliffs. The F_1 parasite load significantly differed among years and cliffs, but the age and squared age were not significantly associated to parasite load (Tab. 2).

The residuals of offspring parasite load were positively correlated with the residuals of parent parasite load (Fig. 1; slope $\pm$ se $= 0.360 \pm 0.116$, $n = 129$, $p = 0.002$). The corresponding heritability ($\pm$ se) of the parasite load was $h^2 = 0.720 \pm 0.232$.

Table 2. ANCOVA on the offspring log-transformed parasite load in relation to the year, breeding cliff, nestling age and squared nestling age.

Sources	df	Type III SS	*F* value	*p*
Year	6	40.07	7.77	0.001
Breeding cliff	12	32.47	3.15	0.003
Nestling age	1	1.81	2.10	0.149
Squared nestling age	1	1.49	1.73	0.190
Error	301	321.30		

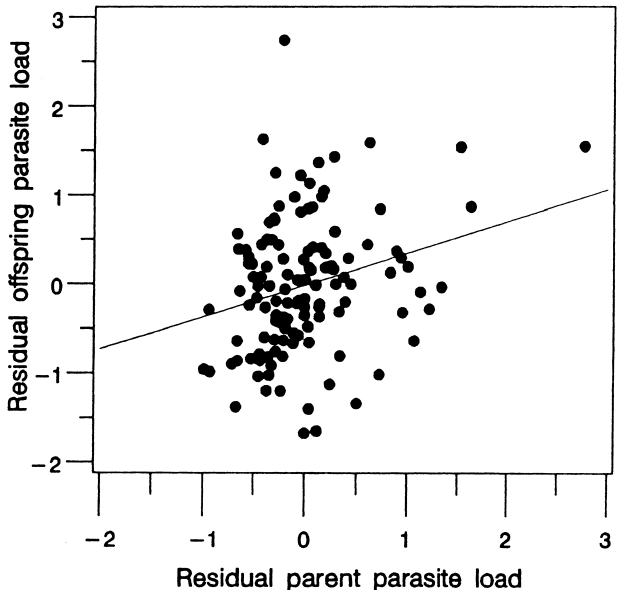

Fig. 1. Regression of the residuals of offspring (F_1) parasite load on the residuals of parent (F_0) parasite load. The figure shows one outlier point and one potentially influential point to the right. Even when removing the potentially influential point, the regression is still significant ($p = 0.017$, $n = 128$).

Discussion

We found a positive correlation between parent and offspring parasite load after correcting for environmental factors, suggesting that heritable variation in susceptibility to ectoparasites exists in this population of kittiwakes.

Estimates of genetic variance for natural populations are often biased because of the possible non-controlled environmental factors that may lead to resemblance among relatives. Statistically factoring out the temporal (year effect) and the spatial (breeding cliff effect) variations, as well as the nestling age, should have reduced the role of non-genetic factors in the parent-regression. Other levels of environmental variation could, however, substantially account for the observed correlation. For instance, ticks are heterogenously distributed within cliffs (Boulinier et al., 1996; Boulinier et al., unpublished). Moreover, genetic heterogeneity of the parasite in its hability to parasitize hosts may exist in natural tick populations. In these cases, if host individuals show high natal philopatry (breed at the same place where they were born), and if local tick infestation is positively autocorrelated in time, then we should expect positive correlation between parent and offspring parasite load. Several elements, however, weaken this hypothesis. First, despite a classic natal philopatry at the scale of the colony, among the colour-banded birds that recruited during the course of the study, only a low proportion bred in the same cliff where

they were born (14.5%, $n = 807$ individuals), and non recruited in their birth nest. Second, local tick numbers were found positively correlated between following years but not after a lag of few years (Boulinier et al., unpublished), thus this should not have biased the parent-offspring relationship since kittiwakes breed for the first time only on average when they are four year-old (Danchin and Monnat, 1992).

We have mostly considered the role of physical environmental variation as a possible source of resemblance among relatives; another source of variation could be host body condition. If nestlings in good body condition (e.g., with high feeding rate) could allocate more resources to antiparasite immune defences, then they could become healthy adults and produce healthy nestlings with low parasite load. This hypothesis can be tested by correlating nestling parasite load and an index of body condition (i.e., wing length regressed against body mass; Barret and Runde, 1980). Nestling body mass was recorded only since 1991, and therefore we could not include it in the ANCOVA model. However, we did not find a statistically significant correlation between body condition and parasite load from 1991 to 1994 (Pearson's correlation coefficient $r = -0.047$, $n = 960$, $p = 0.146$), suggesting that the possible non-genetic effect through host condition does not provide an alternative explanation for the correlation between parent–offspring parasite load (there was no significant effects of the year and cliff, which were thus removed from the analysis). Potential assortative mating with respect to susceptibility to parasitism might have increased the estimate of heritability, and would need to be investigated specifically. Finally, parent–offspring regression may provide unreliable estimates of heritability if the studied trait is measured at different ages, because ontogeny may affect the expression of a character. However, this cannot apply in the present case because parasite load was measured when both parents and offspring were nestlings.

Another problem when investigating offspring–parent regression in natural populations may arise from uncertainty of paternity. Although adoptions occurred, brood parasitism was never observed in the Cap Sizun kittiwake colonies (E. Danchin, J-Y. Monnat, pers. obs.) Extra-pair copulations also occur (T. Boulinier, E. Danchin, J-Y. Monnat, pers. obs.), but at unknown frequencies. Extra-pair copulations have been recorded at higher rates in social species than in solitary ones (Møller and Birkhead, 1993), but their effects as well as those of adoptions would have been to lower the slope of the parent–offspring regression. Thus, we feel confident that the observed relationship reflects some degree of genetic variation in susceptibility to tick parasitism in this population of kittiwakes.

Various resistance or susceptibility mechanisms could lead to the observed correlation. Immunity to ticks in vertebrate hosts has been shown in natural and unnatural host-parasite associations (Randolph, 1979, 1994; see Rechav, 1992 for a review). As shown from laboratories investigations, the main effects of acquired immunity can be to reduce the proportion of ticks that attach to the host and to reduce the proportion of the attached parasites that engorge. Any reduction of the mean length of the blood meal will reduce the nestling parasite load at any time. Detailed knowledge of the immunity mechanism is available for several tick-host

systems (Rechav, 1992). In particular, the tick blood meal can contain specific immunoglobulins that can bind to tick proteins (e.g., Wang and Nuttall, 1994). This happens when a tick feeds on a host which has acquired immunity from previous exposure, but could also happen if specific maternal immunoglobulins were transmitted through the egg as it is known for other avian host-parasite systems (e.g., Smith et al., 1994). If it was mostly such passive immunity transfer that generates differential parasite loads among nestlings, this would lead to a positive mother-offspring regression, but not to a father-offspring one. As the interaction between the parental sex and parasite load was not significant (ANCOVA, residual parent parasite load: $F_{1,124} = 10.14$, $p = 0.0018$; parental sex: $F_{1,124} = 0.01$, $p = 0.9160$; interaction between parental sex and residual parent parasite load: $F_{1,124} = 2.54$; $p = 0.1137$), it is likely that the mechanism involved is not limited to such maternal effects.

Estimates of heritability for morphological (e.g., Gebhardt-Henrich and van Noorwijk, 1991; Norris, 1993), physiological (e.g., Garland, 1988; Sorci et al., 1995), behavioural (e.g., Bakker and Pomiankowski, 1995) and life history traits (e.g., van Noordwijk, 1987) exist for natural populations of vertebrates, but are still scarce for susceptibility to parasitism. Our results add to those reported by Møller (1990a) showing the presence of genetic resistance to the haematophagous parasite *Ornithonyssus bursa* in the barn swallow *Hirundo rustica*.

In conclusion, the susceptibility to tick infestation in kittiwakes appears to be heritable in a natural population. This result adds evidence that host susceptiblility to parasites is partly genetically based, and suggests that this character has the potential to respond to natural selection.

Acknowledgements

We thank T. Garland and A. P. Møller for helpful comments on a previous draft of the manuscript. The S.E.P.N.B. (Société pour l'Etude et la Protection de la Nature en Bretagne) allowed us to work on the Cap Sizun Nature Reserve. Financial support was provided by the S.R.E.T.I.E., Ministère de l'Environnement. T.B. and G.S. were recipients of fellowships from the Ministère de la Recherche. T.B. was also recipient of a fellowship from the Ecole Vétérinaire d'Alfort, Ministère de l'Agriculture.

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Received 13 February 1996:
accepted 14 May 1996.