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Retroviruses and sexual size dimorphism in domestic cats (*Felis catus* L.)

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Hochberg and co-workers have predicted that an increase in host adult mortality due to parasites is balanced by an earlier age at first reproduction. In polygynous species we hypothesize that such a pattern would lead to diverging selection pressure on body size between sexes and increased sexual size dimorphism. In polygynous mammals, male body size is considered to be an important factor for reproductive success. Thus, under the pressure of a virulent infection, males should be selected for rapid growth and/or higher body size to be able to compete successfully as soon as possible with opponents. In contrast, under the same selection pressure, females should be selected for lighter adult body size or rapid growth to reach sexual maturity earlier. We investigated this hypothesis in the domestic cat *Felis catus*. Orange cats have greater body size dimorphism than non-orange cats. Orange females are lighter than non-orange females, and orange males are heavier than non-orange males. Here, we report the extent to which orange and non-orange individuals differ in infection prevalence for two retroviruses, feline immunodeficiency virus (FIV) and feline leukaemia virus (FeLV). FIV is thought to be transmitted almost exclusively through aggressive contacts between individuals, whereas FeLV transmission occurs mainly through social contacts. The pattern of infection of both diseases is consistent with the higher aggressiveness of orange cats. In both sexes, orange cats are significantly more infected by FIV, and tend to be less infected by FeLV than other cats. The pattern of infection is also consistent with an earlier age at first reproduction in orange than in non-orange cats, at least for females. These results suggest that microparasitism may have played an important role in the evolution of sexual size dimorphism of domestic cats.

Keywords: sexual size dimorphism; FIV; FeLV; epidemiology; life history traits; orange genotype

1. INTRODUCTION

In many mammals, males are larger than females (Ralls 1976; Andersson 1994). The primary focus of studies of sexual size dimorphism has been to identify its selective causes (Hedrick & Temeles 1989; Shine 1989). The most commonly invoked selective cause is sexual selection, where competition among males for access to receptive females, and active female choice for certain male phenotypes are the main mechanisms (Hedrick & Temeles 1989; Andersson 1994). Dimorphism may also arise from various forms of natural selection, such as resource competition between the sexes and antipredator defence (Shine 1989; Hedrick & Temeles 1989). Here, we examine the hypothesis that microparasitism may also play a role in evolution of sexual size dimorphism.

Hochberg *et al*. (1992) predicted that hosts should evolve towards early reproduction under a selection pressure imposed by a virulent parasite on adults, because a shorter time to reproduction may be necessary to offset the negative impact of parasite infection on adult mortality. In mammals, where the dominant mating system is polygyny (Kleiman 1977; Pusey 1987), strong competition between males occurs for access to oestrus females. Several studies have reported that body size is an important factor determining the outcome of dyadic conflicts, with the heaviest males having the advantage (red deer, *Cervus elaphus*: Clutton-Brock *et al*. 1982; elephant seals, *Mirounga angustirostris*: Cox & Leboeuf 1977; domestic cat, *Felis catus*: Yamane *et al*. 1996). Early reproduction in males could be achieved by increasing their growth rate to be able to fight successfully and thus gain more copulations earlier. Therefore, we might expect that natural selection would favour males with the highest growth rates (Clutton-Brock *et al*. 1982; Clutton-Brock 1991). In females, early reproduction potentially increases the number of reproductive events during a lifetime (Festa-Bianchet *et al*. 1995). In contrast with males, earlier reproduction in females can be achieved from selection for

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reduced adult size, as individuals reach sexual maturity earlier (Taylor 1965, 1968; Skogland 1989; Martin *et al*. 1994). If a virulent parasite provokes significant mortality among adults, a selection pressure to reproduce as early as possible may exist. By increasing male body mass and decreasing female body mass for a given age, parasite-mediated selection could therefore cause sexual size dimorphism to be reinforced in polygynous mammals.

To test this hypothesis within a population, one needs to consider genotypes which differ in their probability to be infected by parasites. The domestic cat is a convenient organism because genetic markers associated with different life history traits are known (Pontier *et al*. 1995). Males are on average 20% heavier than females (Pontier *et al*. 1995). Body weight in this species is an important factor determining fighting ability, dominance rank and mating success of males (Liberg 1981; Yamane *et al*. 1996). The domestic cat also shows a marked polymorphism in coat colour with known genetic determinism (Robinson 1977). Artificial selection by humans does not modify coat colour gene frequencies (Robinson 1987). We have shown that sexual dimorphism is 32% among orange cats, whereas it is only 16% in non-orange cats (Pontier *et al*. 1995). Furthermore, orange males are heavier than non-orange males, whereas orange females are lighter than non-orange females for all age classes (Jones & Horton 1984; Pontier *et al*. 1995). In addition, orange cats are strongly suspected to be more aggressive than non-orange cats (Pontier *et al*. 1995). Thus male–male competition may explain why males are heavier than females, but does not explain the difference in dimorphism between colour phenotypes. Parasitic infection may have played a role in the divergent degree of dimorphism between orange and non-orange phenotypes. Behavioural differences among individuals mean that some susceptible individuals are more likely to acquire infection according to their behaviour and to pathogen characteristics (Loehle 1995).

In this paper we investigated the hypothesis that host sexual size dimorphism can evolve in response to parasite-driven selection pressure. We tested if prevalence infection was correlated to the genotype of cats, and if infected hosts showed any of the possible patterns predicted by Hochberg *et al*. (1992). We studied two retroviruses: feline immunodeficiency virus (FIV) and feline leukaemia virus (FeLV). Both viruses are ancient (Benveniste *et al*. 1975), and commonly affect domestic cats worldwide (Pedersen 1988). These viruses attack the immune system, and clinical signs include fever, anaemia, lymphadenopathy and weight loss (Braley 1994). FIV infection (Pedersen *et al*. 1987) induces a long asymptomatic stage (lasting months to years) followed by an AIDS-like syndrome (Sparger 1993). Recovery from infection is not possible but not all infected cats develop the disease (Zenger 1992). FeLV is associated with malignant tumors and immunosuppression (Jarrett 1985). Most infected cats recover from infection, develop an immunity, and do not become excreting carriers, whereas others become persistently viraemic. After a variable asymptomatic stage, persistently viraemic cats develop an FeLV-related disease and die (Hardy 1993). FIV is thought to be transmitted by saliva almost exclusively through bites (Ueland & Nesse 1992; O'Neil *et al*. 1996), i.e. during aggressive contacts between cats. FeLV transmission occurs during any direct contact, occasionally through bites, but especially by licking, grooming, sharing feeding places (Jarrett 1985), i.e. during social behaviours, or from mother to foetus (Hoover *et al*. 1983). Thus, if orange cats are more aggressive and less social than non-orange cats (Pontier *et al*. 1995), orange cats should be more frequently infected by FIV and less by FeLV than non-orange cats. Moreover, because of their transmission modes, these viruses should shape host life history in different ways. As mainly adult hosts encounter FIV, orange cats should follow the response predicted by Hochberg *et al*. (1992): low age at first reproduction and high sexual size dimorphism, relative to non-orange cats. In contrast, cats of all age classes could encounter FeLV. However the influence of FeLV should be higher on kittens than on adults as intrinsic susceptibility decreases with age (Grant *et al*. 1980). Thus the influence of FeLV, if any, on sexual dimorphism should be opposite to that of FIV: high age at first reproduction and low sexual size dimorphism. FeLV influence should be more important in non-orange cats which are more social. Thus, whatever the virus considered (FIV or FeLV), we expect a higher sexual size dimorphism in orange cats compared to non-orange cats. In the light of these predictions, we analysed the risk factors of FIV and FeLV in natural populations of domestic cats.

2. MATERIAL AND METHODS

(a) *Data set*

Three rural cat populations in France have been monitored: Aimargues (AI) and Saint-Just Chaleyssin (STJ) since 1982, Barisey-la-Côte (BA) since 1990. The number of individuals varied from 60 (BA) to 300 (STJ) and remained stable throughout the study period (Pontier 1993; Pontier *et al*. 1995). All cat populations had similar demography: a female-biased sex ratio, low density (120–250 cats km^{-2}), and a polygynous mating system (Pontier & Natoli 1996), characteristics of the rural environment (Liberg 1981; Liberg & Sandell 1988; Pontier *et al*. 1995). Age, sex and genotypic structure were stable over years in each population (Pontier 1993; Fromont *et al*. 1996). Although all cats were owned, less than 2% were regularly seen by a veterinarian. Four cats were vaccinated against FeLV. Ten cats were not allowed to roam and about 8% were neutered. No difference between genotypes existed according to these covariates. For these reasons, these covariates were not considered.

Since 1991, individuals have been sampled each year for our epidemiological study in each population. Cats were either caught directly in the owner's house, or trapped in baited cages. Sample representativeness was evaluated each year by comparing sex, genotype and age distributions with those of the whole population for that year (Fromont *et al*. 1996).

Cats were anaesthetized with a mixture of ketamin chlorhydrate (Imalgène 1000, Rhône Mérieux, 15 $mg\,kg^{-1}$) and acepromazine (Vétranquil 0.5%, Sanofi, 0.5 $mg\,kg^{-1}$) by intramuscular injection. Blood samples were taken from the jugular vein. The ELISA method (Cite-combo, Idexx) was used to detect the presence of FeLV group-specific antigen (Lutz *et al*. 1983) and of FIV-specific antibodies which generally reveal virus carriers (Sparger 1993). All positive sera for FIV were confirmed by Western blot analysis (Lutz *et al*. 1988). FeLV and FIV were scored as present or absent for each sampled cat. Sex, age and coat colours genotype for individual cats were recorded. We considered four age classes, <1, 1–2, 2–3, and 3+-years-old (Liberg 1981), and two genotypes for both the sexes. The orange

allele is sex-linked, and its particular mode of transmission allows distinction between heterozygote and homozygote females (Robinson 1977). We distinguished orange (X_OY) and non-orange (X_+Y) males. The frequency of the orange allele is around 0.18 within the populations of the present study (Pontier *et al*. 1995). Thus the number of X_OX_O females is very low. The unique X_OX_O female was pooled with XOX_+ females. The non-orange class comprised X_+X_+ females.

(b) *Risk factors*

Our objective was to determine the risk factors of FIV and FeLV, to test whether orange and non-orange cats differ in their probability of being infected by both diseases. FIV and FeLV prevalences did not differ significantly among years in any of the populations (Courchamp 1996; Fromont *et al*. 1996). Because of the low prevalence of each virus within the cat populations, the low frequency of orange allele, and the low sample size analysed each year in each cat population (about 40 cats), we pooled all yearly samples for all populations. For individuals sampled repeatedly, a single blood sample was randomly selected. The pooled sample comprised 402 individuals. As only one individual was infected by both viruses, we analysed separately the FIV and FeLV individuals. Forty-seven cats were infected by FIV and 32 by FeLV.

To analyse the association of age, sex or genotype with the probability of FIV or FeLV infection, we used logistic regression models (Agresti 1990). These models relate the logit of the probability for an individual cat of being infected to the predictor variables, age, sex or genotype, as well as their interactions. The full model including all interactions between the risk factors has a null deviance, and the selection of a model that both fitted the data and did not include unnecessary terms (principle of parsimony) was carried out using the Akaike information criterion (AIC: Burnham *et al*. 1995). To assess the robustness of our results, we also considered models with slightly (i.e. <1) larger AIC values. The test of a single hypothesis is done for generalized linear models using the likelihood ratio test (LRT), based on the difference in deviance between two nested models, which follows a chi-square distribution (Agresti 1990). As tests of the main hypotheses were one-sided (the alternative hypothesis is an increase (FIV) or a decrease (FeLV) in prevalence in orange cats), we used instead a *t*-test of the parameter values (Yates 1984). The goodness-of-fit of the models used for testing hypotheses was assessed through analysis of residuals as well as the residual deviance. Interpreting the selected statistical model was done using the parameter estimates, which are logarithms of the odds-ratio (Agresti 1990). We provide the odds-ratio, back transforming confidence intervals calculated on the original logit scale using a normal approximation (Agresti 1990). All statistical analyses were done with S-Plus (Venables & Ripley 1994).

3. RESULTS

The prevalences of FIV and FeLV in our sample were, respectively, 11.1% and 7.8%. Within orange and non-orange cats, respectively, the prevalence of FIV was 31.1% and 12.8% in males, 12.5% and 3.1% in females, and the prevalence of FeLV was 4.7% and 10.7% in males, 4.2% and 7.5% in females (table 1).

(a) *Risk factors of FIV*

Only three of 98 cats less than one year old were infected; all three were orange males, see table 1. We therefore did not consider this outlying age class in subsequent analyses. Two statistical models had similar AIC values (table 2), and both included the effects of sex and genotype. Parameter estimates for these effects were similar for both models (table 3), and showed an increase in prevalence for males relative to females (*t*-value$=3.69$, d.f.$=9$, $p=0.0025$; an additive effects of genotype and sex ('G+S') model was used), and for orange relative to non-orange individuals (*t*-value$=2.83$, d.f.$=9$, $p=0.0099$). Both male and female orange cats are thus more frequently infected than non-orange ones. The parameter estimates for age provided weak evidence for an increase in prevalence with age (LRT$=4.20$, d.f.$=2$, $p=0.122$).

Table 1. *Total number of cats sampled and the number of positive individuals for FIV antibodies and FeLV antigens*

(Individuals are grouped by sex: F, female, M, male, and genotype: NO, non-orange, O, orange, and age class in years.)

total FIV	FIV+	total FeLV	FeLV+	sex	genotype	age
32	0	32	3	F	NO	<1
46	0	46	3	M	NO	<1
14	0	14	1	F	O	<1
9	3	6	1	M	O	<1
30	1	31	5	F	NO	1–2
36	4	36	3	M	NO	1–2
12	2	12	0	F	O	1–2
9	2	9	0	M	O	1–2
43	0	43	3	F	NO	2–3
43	7	45	9	M	NO	2–3
23	5	23	2	F	O	2–3
23	7	23	0	M	O	2–3
54	4	53	1	F	NO	3
24	8	22	1	M	NO	3
23	2	23	0	F	O	3
4	2	5	1	M	O	3

(b) *Risk factors of FeLV*

The models selected included only the effects of genotype and age (see table 2). Parameter estimates for the effect of genotype were again consistent in both models (with and without the effect of age, see table 3). There was some evidence for an increase in prevalence for non-orange versus orange individuals (*t*-value$=1.67$, d.f.$=11$, $p=0.062$; an additive effects of age and genotype ('A+G') model was used). There was no clear age trend (LRT$=6.17$, d.f.$=3$, $p=0.103$, see table 3).

(c) *Age at first reproduction*

Age at first reproduction was known for 13 orange females and 34 non-orange females. As expected, orange females reproduced significantly earlier than non-orange females (mean$\pm$s.e.: 7.69 months±0.61, $n=13$ versus 9.15 ± 0.34, $n=34$, one-tailed $t_{45}=-2.17$, $p=0.02$). Two orange females reproduced in the year of their birth. Data were not available in males. However, we found that three orange males were infected by FIV when aged less than one year (see table 1). They lived in different houses and were not siblings.

Table 2. *Model selection for FIV and FeLV prevalence data*

(The two best models according to AIC are indicated in bold. A, age; S, sex; G, genotype. The symbol ':' denotes the interaction between two factors.)

model	deviance	number of parameters	AIC
(*a*) FIV			
full model	0	12	24
A+G+S+A : G+A : S+G : S	3.80	10	23.80
A+G+S+A : G+G : S	4.43	8	20.43
A+G+S+A : G	7.21	7	21.23
A+G+S	**9.55**	**5**	**19.55**
G+S	**13.35**	**3**	**19.35**
S	21.19	2	25.19
null model	34.43	1	36.43
(*b*) FeLV			
full model	0	16	32
A+G+S+A : G+A : S+G : S	4.68	13	30.68
A+G+S+A : S+G : S	9.58	10	29.58
A+G+S+G : S	11.64	7	25.64
A+G+S	13.18	6	25.18
A+G	**13.20**	**5**	**23.20**
G	**19.37**	**2**	**23.37**
null model	22.29	1	24.29

Table 3. *Parameter values (standard errors in parentheses) for the best (according to AIC) models selected for FIV and FeLV prevalence data*

(The parameter values are logarithm of odds ratio: they represent the difference (on the logit scale) between males and females (sex), orange and non-orange (genotype), and between age class i and age class 2 (FIV) or age class 1 (FeLV).)

	model 'A+G+S'	model 'G+S'
(*a*) FIV		
sex	**−1.500 (0.376)**	**−1.307 (0.354)**
genotype	**−1.031 (0.354)**	**−0.977 (0.345)**
age class 3	0.280 (0.449)	
age class 4	0.874 (0.476)	
(*b*) FeLV		
genotype	**0.842 (0.504)**	**0.788 (0.498)**
age class 2	0.142 (0.525)	
age class 3	0.372 (0.469)	
age class 4	−1.04 (0.694)	

4. DISCUSSION

(a) *Risk factors*

We found no significant difference in FeLV prevalence between sexes nor among age classes, which could follow from the mode of transmission of this virus. Our results confirm a higher susceptibility of males, compared to females, to FIV infection (for review, see, Courchamp & Pontier 1994). Because males fight more often than females (Liberg 1981) for territorial defence and access to resources (receptive females and/or food), they are more likely than females to be infected by FIV. Mature individuals (1 year old) are found infected more often than immature individuals because they fight more often than juveniles (Liberg 1981). The increase of prevalence with age is partly linked to the long period of seropositivity which causes the number of infected cats to increase with age through a cumulative effect (Courchamp & Pontier 1994). As expected for both sexes, orange individuals are more likely to be infected by FIV compared to non-orange individuals, and tend to be less frequently infected by FeLV.

(b) *Genetic variation in behaviour*

Possible hypotheses for explaining the pattern of infection observed in the two genotypes are: (1) individuals have different immunological susceptibility to parasites according to their genotype, (2) individuals exhibit different behavioural patterns, such as level of aggressivity (Pontier *et al*. 1995), and thus have different probabilities of acquiring infections. Such a genetic polymorphism for aggressive behaviour is known for other species of mammals (Keeler 1942; Lea 1943; van Oortmerssen & Bakker 1981; Pasitschniak-Arts & Bendell 1990; Benus *et al.* 1991). The association between behaviour and coat colour has also been shown in different mammal species (Keeler 1942; Lea 1943; Pasitschniak-Arts & Bendell 1990). FIV and FeLV partly have the same target cells and effects, in particular a decrease in the CD4+ T-lymphocyte number (Tompkins *et al*. 1991). Moreover a pre-existing FeLV infection enhances the multiplication and spread of FIV in dually infected cats (Pedersen *et al*. 1990). This suggests that the same categories of cells are implicated in infection by both viruses. While it is perhaps possible that the orange coat colour gene is closely linked to a membrane molecule serving as a receptor or a co-receptor for FIV, the contradictory effect of the orange gene on FIV (highest susceptibility) and FeLV (lowest susceptibility) indicates that the orange locus should be linked to two membrane molecules, one favouring the entry of FIV, the second disfavouring that of FeLV. It seems more probable that other factors than intrinsic susceptibility act on the risk of cats being infected with retroviruses. The difference in transmission of the two feline retroviruses is compatible with increased aggressivity in orange cats. While FIV is practically uniquely transmitted through bites, FeLV is largely transmitted through peaceful social interactions, such as grooming, which are reduced in orange cats. The fact that orange cats are more infected with FIV but less with FeLV supports, therefore, the second hypothesis: that infection probability is a function of the level of aggressivity, at least in males (Pontier *et al*. 1995).

Three hypotheses may account for the different levels of infection among orange compared to non-orange females: (1) a behaviour which enhances the number of sexual partners during the reproductive period among orange females, (2) female choice for orange partners, or (3) the more aggressive behaviour of orange females towards males and/or females. Concerning the first hypothesis, it has been suggested that males could transmit FIV to females during mating, when they bite the neck of females (Courchamp & Pontier 1994). We do not have data on the sexual behaviour of females, but it seems that in rural areas, females do not often change sexual partners

because males tend to monopolize females (Liberg 1981; Pontier & Natoli 1996; Yamane *et al*. 1996), thus the number of sexual partners is very low. There is no evidence of mate choice (hypothesis 2), either by direct study of reproductive behaviour (E. Natoli, E. De Vito and D. Pontier, unpublished data), or by genetic analysis (Pontier *et al*. 1995). The assumption of panmixia, based either on the orange allele for which all the genotypes can be identified phenotypically (Pontier *et al*. 1995), or on electrophoretic loci (O'Brien 1980), is fulfilled in most populations. We thus favour the hypothesis of a higher level of aggressivity among orange females (hypothesis 3).

(c) *Size dimorphism and parasitism*

High aggressivity potentially changes the rate and also the type of contacts between individuals (Loehle 1995). This, in turn, increases the probability of aggressive individuals being infected by a microparasite transmitted mainly through bites, such as FIV. FIV may therefore have induced a selection pressure on orange cats to evolve toward early reproduction.

We found that orange males are already infected when less than one year old. The difference in age at first infection between genotypes suggests that orange males interact with other males earlier than non-orange cats. Because of their aggressivity, orange males may be involved in fights more often and at a younger age than non-orange males. Orange males could be under selective pressure for the earliest age possible at first mating. To reproduce at an early age, orange males should have high growth rates during early life. This selection for early reproduction could account for the large body weight of orange males at a given age compared to non-orange males.

Orange females should respond to FIV either by (1) reproducing earlier to increase their number of reproductive occasions, and/or (2) increasing their litter size. Earlier maturity (1) can be achieved by increasing growth rate (scenario 1a), or decreasing adult body weight (scenario 1b), under the hypothesis that sexual maturity in this species is reached at a given percentage of adult body weight (Taylor 1965, 1968; Skogland 1989; Martin *et al*. 1994). Following scenario (2), orange females are expected either to invest more in reproduction (scenario 2a) or to increase their adult body weight (scenario 2b). For a given weight, the litter size of orange females is expected to be larger than that of non-orange females (scenario 2a). If offspring number is size-dependent in this species as is often found in rodents (Myers & Masters 1983; Yoccoz *et al*. 1993; Risch *et al*. 1995), heavier orange females are expected to be selected for (scenario 2b). Our first results supported scenario (1b) since the mean age at first reproduction in orange females, that are lighter than non-orange (Pontier *et al*. 1995), is significantly lower than in non-orange females. Unfortunately, data are not available to test if orange females, for a given body weight, differ in litter size from non-orange females (scenario 2a).

Our results strongly suggest that the larger sexual size dimorphism of orange cats compared to non-orange cats results from a divergent optimization of age at first reproduction in males and females in response to FIV, although FIV might not be the only pathogen or factor involved. We cannot discard alternative hypotheses, such as the higher prevalence of FIV in orange cats as a consequence of their earlier sexual maturity. A detailed knowledge of age at first reproduction for both males and females, offspring growth pattern, litter size, sex ratio of litters, and the survival of young (which may not be constant among different litter sizes) and adults, is needed for further insight into the life-history responses of domestic cats to FIV. The selection pressure imposed by FeLV is more difficult to observe because of its more complex transmission routes that involve cats of all age classes while age-resistance does decrease the susceptibility of adults to infection. Further studies investigating mortality in kittens are required. In addition, there are large differences among natural populations in domestic cat life-history patterns, depending upon the spatial distribution of resources (Liberg & Sandell 1988; Pontier 1993). Domestic cats which live in large dense multimale–multifemale social groups in urban areas are likely to be more affected by FeLV than the domestic cats living at low density in rural areas (this study). An epidemiological study of urban cats would probably give an insight into the role of FeLV in the evolution of life history traits in this species.

Our present work corroborates empirically the prediction of Hochberg *et al*. (1992), that increased adult mortality of hosts due to parasitism selects for earlier reproduction and/or higher fecundity early in life (see, also, Michalakis & Hochberg 1994). Our results may extend the argument of Hochberg *et al*. (1992) to take into account the relationship between mating system, sexual size dimorphism and demographic traits. The closest example of an experimental result yielding this kind of relationship between parasitism and sexual size dimorphism was presented by Potti & Merino (1996) in the flycatcher *Ficedula hypoleuca*. They found a gender effect of parasitism on tarsus growth. However, to distinguish parasitism influence on sexual size dimorphism from possible other selective forces is difficult. We have this opportunity in the domestic cat as we have genotypic markers associated with different life-history traits. Whether the associations found are due to the coat colour genes themselves through pleiotropic effects or to linked genes should be investigated in the future.

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