



WISSENSCHAFTLICHE KURZMITTEILUNG

Host chromosomal evolution and parasites of the house mouse *Mus musculus domesticus* in Scotland

By LAURENCE RESSOUCHE, GUILA GANEM, J.-M. DEROTHE, J. B. SEARLE, F. RENAUD,
and CATHERINE MOULIA

*Departement de Biologie Evolution, Environment, Université Montpellier II, Montpellier, France and
Department of Zoology, University of Oxford, Oxford, U. K.*

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Parasitological studies in the hybrid zone between *Mus musculus musculus* and *M. m. domesticus* (sensu AUFRAY et al. 1990 and BOURSOT et al. 1993) showed that the resistance to oxyuroids (*Aspiculuris tetraptera* Schulz, 1924 and *Syphacia obvelata* Rudolphi, 1802) is determined by different genetic systems in each subspecific genome (SAGE et al. 1986; Moulia et al. 1991, 1995). These studies underlined that pinworms may constitute good markers to distinguish between recently evolved groups such as chromosomal races.

This study focuses on chromosomal races of the house mouse occurring in northern Scotland and in the Orkney archipelago (Fig. 1). Previous cytogenetic analyses in northern Scotland (SEARLE 1991; SEARLE et al. 1993) have demonstrated a staggered hybrid zone between a chromosomal race homozygous for metacentrics Rb(4.10), Rb(9.12), Rb(6.13), and Rb(11.14) ($2n = 32$) and the standard race ($2n = 40$). In the center of this zone most individuals are homozygous for two metacentrics Rb(4.10) and Rb(9.12) ($2n = 36$) (Fig. 1). As this 36-chromosome karyotype is common over a large area (100 km²), it has been suggested that these individuals may constitute a distinct third chromosomal race (SEARLE et al. 1993). Two alternative scenarios may explain the distribution of these three races. One is that the 36-chromosome form originally occupied the county of Caithness, within which the 32-chromosome race arose in the North-eastern part of this range, as a result of the fixation of two new metacentrics. The second scenario proposes that the 36-chromosome race arose by “zonal riation” (SEARLE 1991, 1993) following a secondary contact between the 32-chromosome and the standard races.

Chromosomal races are also found on three islands of the Orkney archipelago (ADOLPH and KLEIN 1981; BERRY et al. 1992): Westray ($2n = 36$; homozygous for Rb(4.12) and Rb(6.14), Eday ($2n = 34$; homozygous for Rb(3.14), Rb(4.10) and Rb(9.12)) and Faray ($2n = 34$ –36; homozygous for Rb(3.14) and Rb(9.12), polymorphic for Rb(4.10) (Fig. 1). Genetic investigations suggest that the various Scottish chromosomal races (mainland and islands) are probably closely related and share a common origin (NASH et al. 1983).

Here we report the first attempt to distinguish between chromosomal races of the house mouse (*M. m. domesticus*) using a parasitological approach.

One hundred and sixty-six house mice from the three mainland races were collected during two field trips (24 March–10 April and 15–23 September 1992; Fig. 1; for further details see GANEM et al. 1996). Eighty seven animals from four island populations (Papa-Westray, Sanday, Eday, Westray; Fig. 1) were trapped during late February–April 1992.

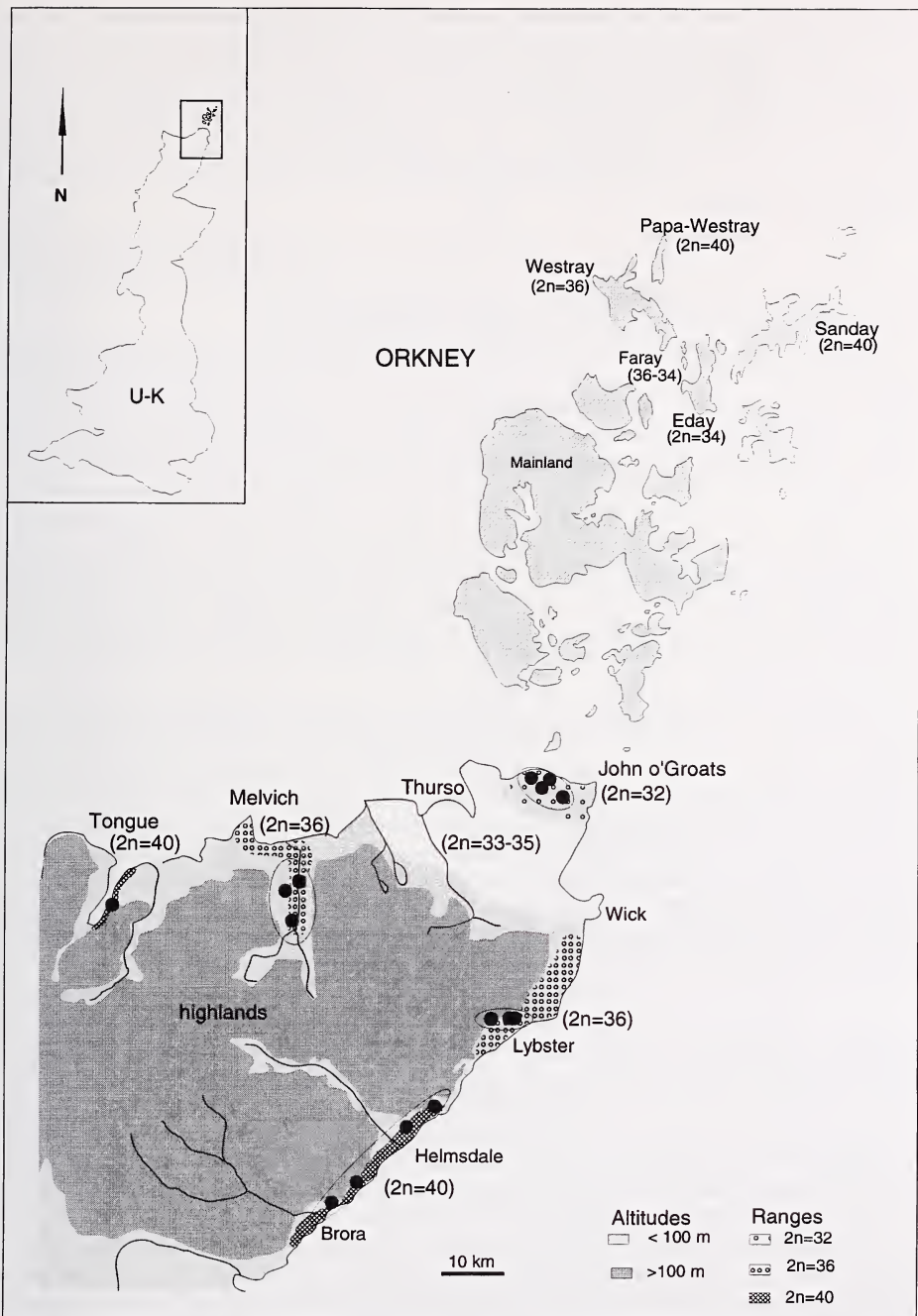


Fig. 1. Geographic location of the different chromosomal zones described in this study. The mice from mainland Scotland used for the parasitological analysis came from the sites marked by closed circles. Pooled localities are indicated and their mean diploid provided. Mice with reduced chromosome numbers are almost completely limited to Caithness, which forms a boundary with Sutherland along a line approximately between Melvich and Helmsdale.

Only adults were kept in the laboratory to study behaviour (GANEM and SEARLE 1996 a, b) and parasitology (this study). They were maintained in male-female pairs for 3–4 months in the same animal room and under standardised conditions. Because pregnancy may modify the immune and parasitological state, females that became pregnant were excluded from the parasitological study. When mice were killed, their karyotypes were prepared. The intestines of each mouse were dissected and the nematodes isolated under a binocular microscope. Parasite loads were estimated by counting all oxyuroids without distinguishing species.

Data on parasite loads were log-transformed for homoscedasticity (Bartlett's test: $\chi^2 = 2.26$, d. f. = 5, $p < 0.01$). An analysis of variance was applied to the transformed data and a Student-Newman-Keuls (SNK) test was used as a posthoc test for multiple comparisons between samples (SCHERRER 1984; SOKAL and ROHLF 1995).

Mainland and island mice were divided into three groups according to their karyotype (mainland: 2 n = 32, 2 n = 36, 2 n = 40; islands: 2 n = 34, 2 n = 36, 2 n = 40). Differences between karyotypes from mainland and island samples were tested separately. A three-way Anova with karyotype, season, and sex as main factors showed a significant effect of karyotype on the mainland ($F_{2,154} = 10.5$, $p = 0.0001$): mice of the 36-race displayed the lowest parasite load and mice from the 32 and 40-races the highest (SNK post-hoc tests: 36–40: $p < 0.01$; 36–32: $p < 0.01$; 40–32: $p > 0.05$; Fig. 2 a). Similarly, within the island sample, mice of the 36-race showed the lowest parasite load ($F_{2,81} = 11.6$; $p = 0.0001$; 36 i–40 i: $p < 0.05$; 36 i–34 i: $p < 0.01$; 34 i–40 i: $p > 0.05$; Fig. 2 b). However, a sex effect was detected in the island sample ($F_{1,81} = 10.4$, $p = 0.002$) due to a lower parasite load in the standard-type females than in the males ($t = 2.46$, d. f. = 35, $p = 0.02$; Fig. 2 b). Nevertheless, on average, both mainland and island standard race mice show similar susceptibilities ($t = 0.02$, d. f. = 115, $p > 0.05$).

The long period of laboratory standardisation is believed to minimise environmental effects (i. e. site, season) on the parasitological state of each individual. Indeed, even if housed in different cages, mice are easily contaminated by pinworm eggs from infested individuals occurring in the same room. Moreover, the oxyuroids have a direct cycle with pre-patent periods no longer than three weeks. Therefore, during the 3–4 month period in the laboratory, each mouse could have been challenged to several reexposures to these parasites (SCOTT and GIBBS 1986). The fact that there was no seasonal variation between the mainland samples ($F_{1,154} = 2.1$, $p > 0.05$) strongly supports this proposal. Thus, our results reflect to a large extent the occurrence of variation in resistance to pinworms in the different chromosomal races of the house mouse in northern Scotland.

Only one race (the island standard-type) displays sex differences in its infestation level due to a higher parasite load in the males. Such a phenomenon has been reported, although in a different context, for other wild rodent populations (BEHNKE 1975). In the present study, the sample size may not have been sufficient to detect such microvariation within the other races. However, given that male-enhanced susceptibility can depend on the immunomodulating effect of testosterone (ALEXANDER and STIMSON 1988; ZUK 1990), an additional hypothesis may be that male mice in the two island standard-race populations have higher basal levels of testosterone than those in the other populations. A more detailed study should clarify this point.

The most striking result of this study concerns the resistance of the 36-chromosome races when compared to the standard and 32- or 34-races. Among the two scenarios explaining the origin of the 36-chromosome mainland race, the parasitological results are consistent with a hybrid origin. The reduced susceptibility to pinworms of this race would be related to "outbreeding vigour" due to the increased polymorphism generated by divergence of the parental races. These results contrast with the enhanced susceptibility of hybrids between *M. m. domesticus* and *M. m. musculus* (MOULIA et al. 1991, 1993), but do not contradict it. In this case, the apparent "outbreeding depression" would result from

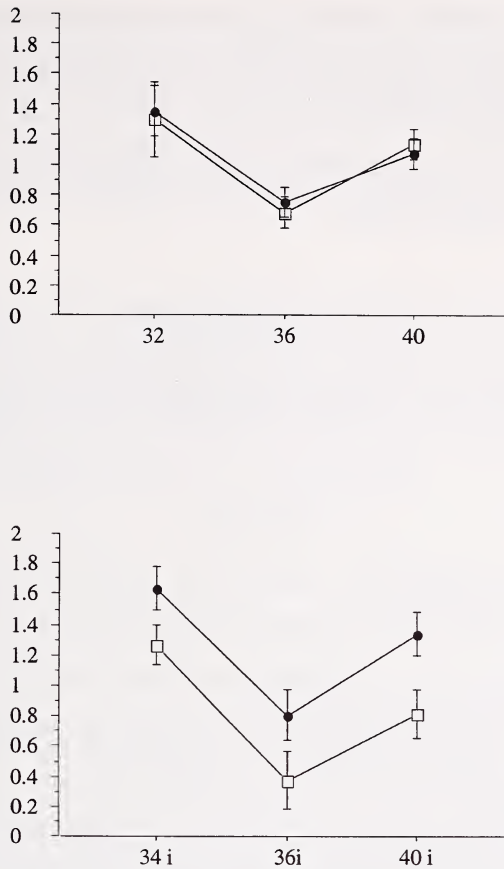


Fig. 2. Mean (\pm s. e.) parasite load (P.L.) of each sample of chromosomal race. 2a: mainland Scotland: male (●) and female (□) P.L. are shown independently (non-significant difference). 32, 36, 40 refer to 32-, 36- and 40-chromosome races respectively. 2b: Orkney's islands: male (●) and female (□) P.L. are shown independently (significantly different, see text); 34i, 36i et 40i refer to 34-, 36- and 40-chromosome island races.

the accumulation of incompatible alleles in the two subspecies (ORR 1995). Additional support for the hybrid origin hypothesis is provided by the intermediate pattern in behavioural divergence shown by the mainland 36-chromosome race when compared to the other two mainland races (GANEM and SEARLE 1996 a, b).

The alternative hypothesis, i. e. the 32-race arose within the 36-race, is much less likely since it would imply that the 32-chromosome mice developed a higher susceptibility than the putative parental mice and yet still managed to prosper and expand their range.

The similarities in parasite resistance between both the mainland and the island 36-races is puzzling. Indeed, the two races occupy different geographic areas and share only one metacentric, Rb(9.12), which is also present in the other chromosomal races analysed in this study. Even if the various Scottish chromosomal races share a common origin (NASH et al. 1983; DAVIS 1983), the mainland and island 36-chromosome mice have most likely experienced different histories, suggesting that parasite resistance in these two races most likely evolved through different evolutionary processes. One possible explanation lies in the founding of the initial Westray 36-chromosome population by resistant individ-

uals. Alternatively, resistance to pinworms in this island chromosomal race may have arisen through a hybridisation event as in the mainland 36-race. To determine the origin of resistance in these mice will require extending the parasitological analysis to other island populations of the archipelago and clarifying the genetic relationships of these mice using molecular markers.

Although, these preliminary results need to be confirmed by controlled infestations in laboratory, they do suggest that the parasitological approach may open new perspectives in the field of chromosomal evolution.

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References

- ADOLPH, S.; KLEIN, J. (1981): Robertsonian variation in *Mus musculus* from central Europe, Spain, and Scotland. *J. Hered.* **72**, 219–221.
- ALEXANDER, J.; STIMSON, W. H. (1988): Sex hormones and the course of parasitic infection. *Parasitol. Today* **4**, 189–193.
- AUFFRAY, J.-C.; MARSHALL, J. T.; THALER, L.; BONHOMME, F. (1990): Focus on the nomenclature of european species of *Mus*. *Mouse Genome* **88**, 7–8.
- BEHNKE, J. M. (1975): *Aspiculuris tetraptera* in wild *Mus musculus*. The prevalence of infection in male and female mice. *J. Helminthol.* **49**, 85–90.
- BERRY, R. J.; BERRY, A. J.; ANDERSON, T. J. C.; SCRIVEN, P. (1992): The house mice of Faray, Orkney. *J. Zool. (London)* **228**, 233–246.
- BOURSOT, P.; AUFFRAY, J.-C.; BRITTON-DAVIDIAN, J.; BONHOMME, F. (1993): The evolution of house mice. *Annu. Rev. Ecol. Syst.* **24**, 119–152.
- DAVIS, S. J. M. (1983): Morphometric variation of populations of house mouse *Mus domesticus* in Britain and Faroe. *J. Zool. (London)* **199**, 521–534.
- GANEM, G.; SEARLE, J. B. (1996 a): Behavioural discrimination among chromosomal races of the house mouse (*Mus musculus domesticus*). *J. Evol. Biol.* **9**, 817–831.
- GANEM, G.; SEARLE, J. B. (1996 b): Corticosterone and interchromosomal race discrimination in the house mouse. *Hormones and Behavior* **30**, 69–73.
- GANEM, G.; ALIBERT, P.; SEARLE, J. B. (1996): An ecological comparison between standard and chromosomally divergent house mice in northern Scotland. *Z. Säugetierkunde* **61**, 176–188.
- MOULIA, C.; AUSSSEL, J. P.; BONHOMME, F.; BOURSOT, P.; NIELSEN, J. T.; RENAUD, F. (1991): Wormy mice in a hybrid zone, a genetic control of susceptibility to parasite infection. *J. Evol. Biol.* **4**, 679–687.
- MOULIA, C.; LE BRUN, N.; DALLAS, J.; ORTH, A.; RENAUD, F. (1993): Experimental evidence of genetic determinism in high susceptibility to intestinal pinworm infection in mice, a hybrid zone model. *Parasitology* **106**, 387–393.
- MOULIA, C.; LE BRUN, N.; LOUBES, C.; MARIN, R.; RENAUD, F. (1995): Hybrid vigour against parasites in interspecific crosses between two mice species. *Heredity* **74**, 48–52.
- NASH, H. R.; BROOKER, P. C.; DAVIS, J. M. (1983): The Robertsonian translocation house-mouse populations of north east Scotland, a study of their origin and evolution. *Heredity* **50**, 303–310.
- ORR, H. A. (1995): The population genetics of speciation: the evolution of hybrid incompatibilities. *Genetics* **139**, 1805–1813.
- SAGE, R. D.; HEYNEMAN, D.; LIM, K. C.; WILSON, A. C. (1986): Wormy mice in a hybrid zone. *Nature* **324**, 60–63.
- SCHERRER, B. (1984): Biostatistiques. Chicoutimi: Gaëtan Morin.
- SCOTT, M. E.; GIBBS, H. C. (1986): Long-term population dynamics of pinworms (*Syphacia obvelata* and *Aspiculuris tetraptera*) in mice. *J. Parasitol.* **72**, 652–662.

- SEARLE, J. B. (1991): A hybrid zone comprising staggered chromosomal clines in the house mouse (*Mus musculus domesticus*). *Proc. R. Soc. Lond. B.* **246**, 47–52.
- SEARLE, J. B. (1993): Chromosomal hybrid zones in eutherian mammals. In: *Hybrid zones and the evolutionary process*. Ed. by R. G. HARRISON, New York: Oxford Univ. Press. Pp. 309–353.
- SEARLE, J. B.; NARAIN NAVARRO, Y.; GANEM, G. (1993): Further studies of a staggered hybrid zone in *Mus musculus domesticus* (the house mouse). *Heredity* **71**, 61–71.
- SOKAL, R. R.; ROHLF, F. J. (1995): *Biometry*, 3rd ed. New York: Freeman.
- ZUK, M. (1990): Reproductive strategies and disease susceptibility, an evolutionary viewpoint. *Parasitol. Today* **18**, 109–115.

Authors addresses: LAURENCE RESSOUCHE, J.-M. DERO THE, F. RENAUD, and CATHERINE MOULIA, Laboratoire de Parasitologie Comparée, UMR 5555 CNRS-UP-UM II, département de Biologie, Evolution, Environnement, Université Montpellier II, pl. E. Bataillon, C. C. 105, F-34095 Montpellier cedex 5, France; GUILA GANEM, Laboratoire de Génétique et Environnement, ISEM, UMR 5554 CNRS-UM II, département de Biologie, Evolution, Environnement, Université Montpellier II, pl. E. Bataillon, C. C. 65, F-34095 Montpellier cedex 5, France; J. B. SEARLE, Department of Biology, York University, PO Box No 373, York YO1 5YW, U. K.

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