tion mean and variance in fecundity are given by $1 + \Sigma P_{ij(j-1)} \varepsilon_\mu$ and $1 + \Sigma P_{ij(j-1)} \varepsilon_\sigma^2$ respectively. An individual female's offspring number would be randomly sampled from the appropriate distribution having mean $1 + n_{A_2}\varepsilon_\mu$ and variance $1 + n_{A_2}\varepsilon_\sigma^2$, where n_{A_2} is the total number of A_2 alleles at all 10 loci. (For each mating, maternal genotype determined offspring number, i.e. litter size.)

A program to simulate a population of size $N = 100$ was run with $P_{i_1} = 0.25$, $P_{i_3} = 0.25$ initially and heritability of fecundity was estimated over either 50 generations or until fixation, whichever occurred first. The table shows representative values. It includes Crow's[10] index of opportunity for selection, I, and Nei and Murata's[11] estimator of effective population number, $N_e = N/(1/\text{mean} + I(1 + 3h^2))$. We have also shown the time in generations, T, for all 10 loci to be fixed, the values for these variables giving an indication of the effects of the distribution of fecundity on certain aspects of evolutionary change.

The critical results, however, are the heritabilities, estimated by daughter-dam regression (fecundity being determined by maternal genotype). For low values of ε_μ, whatever the distribution, h^2 does not differ significantly from zero, even though the population is undergoing rapid genetical change towards the elimination of all genetical variability. (In all cases shown except that with $\varepsilon_\mu = 0.1$, $\varepsilon_\sigma^2 = 0.08$, selection acted to increase mean fecundity; this will be reported in detail elsewhere[12].) It appears, from the results shown and others[12], that heritability increases as the variance increases relative to the mean. The apparent opportunity for selection indicated by I and the apparent heritability indicated by h^2 both give little aid in predicting time to fixation for genes affecting both mean and variance of family size. It seems possible that the success of artifical selection for fecundity, despite its low heritability[13], may reflect the effects displayed here of genes acting upon the dispersion of fecundity as well as its location.

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Robertsonian translocations in Mus musculus from Sicily[1]

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Summary. The karyotypes of 6 mice from different places in Sicily have been determined. 3 of them had abnormal chromosome numbers of 2n = 26, 2n = 27 and 2n = 29, caused by Robertsonian translocations of one acrocentric chromosome to another resulting in metacentric chromosomes, The newly described metacentric chromosomes are Rb(4.3)1Sic, Rb(15.2)2Sic, Rb(12.6)3Sic, Rb(13.5)4Sic, Rb(14.10)5Sic, Rb(17.8)6Sic and Rb(16.9)7Sic.

As in other mammalian species, mainly rodents, a Robertsonian karyotype diversity has been observed in the house mouse, Mus musculus[3-6]. So far mice with karyotypes deviant from that of the normal house mouse, 2n = 40, have been found in different valleys of the Alps[4] and the Apennines[5,6]. The metacentric chromosomes were almost always found to be homozyguous. Within distinct mouse populations, the karyotype was usually constant. Mice from Sicily up to now seemed to have a normal karyotype[4]. We have now found several house mice from feral populations in Sicily having metacentric chromosomes.

Material and methods. From the following locations in Sicily, wild house mice were obtained: 2 from Pioppo, 1 from Monte Lepre, 2 from Toretta and 2 from Misilmeri, all in the surroundings of Palermo. 1 mouse was karyotyped directly by Dr J. Olert and shown to have 11 metacentric chromosomes. The remaining 5 animals were bred with normal laboratory mice and inter se. Their karyotype was deduced by examining that of the F1 animals and in 1 case of the parental Sicilian mouse itself. Mitotic metaphase plates were obtained after Colchicin or Colcemid treatment from bone marrow cells or PHA-stimulated spleen cells[7]. The chromosomes were stained either directly with Orcein or, after Trypsin treatment, with Giemsa[8]. Meiotic metaphase I figures were obtained by the method of Evans et al.[9].

Results and discussion. 3 of the Sicilian mice had a karyotype of 2n = 40 acrocentric chromosomes, 1 male had 2n = 29, 1 female 2n = 27 and another male 2n = 26 chromosomes. The animal with 29 chromosomes, 11 of which are metacentric, was not studied further. The female with 2n = 27 chromosomes was karyotyped directly and shown to have 13 metacentrics. Of 4 F1 animals from matings with normal males, 1 had 7 and 3 had 6 metacentric chromosomes. We concluded that this Sicilian

1 We thank Dr J. Olert, Pathologisches Institut der Universität Ulm, for karyotyping 1 mouse from Sicily.
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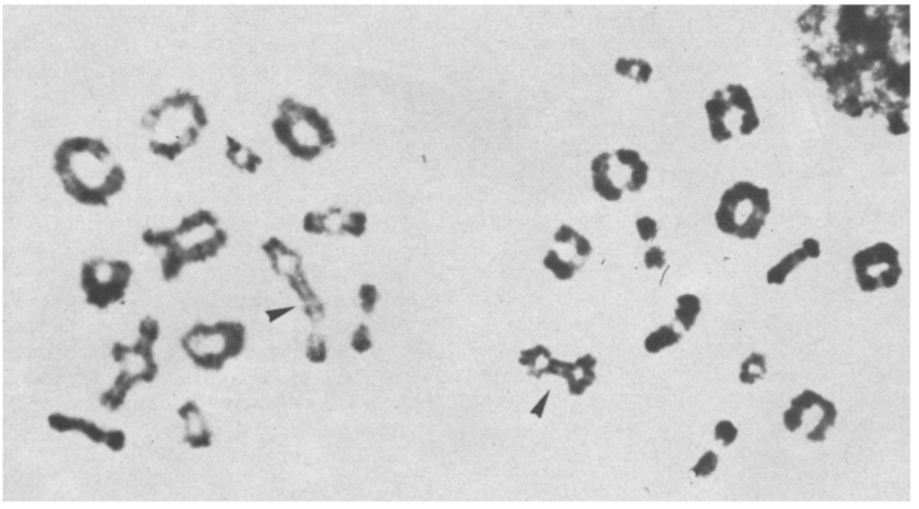

Fig. 1. Meiotic metaphase I figures of F1 animals obtained by mating the 2n = 27 and the 2n = 26 mice. The F1 mice have 2n = 27 chromosomes, which in meiosis give 6 bivalents, 6 ringbivalents and 1 trivalent (arrow).

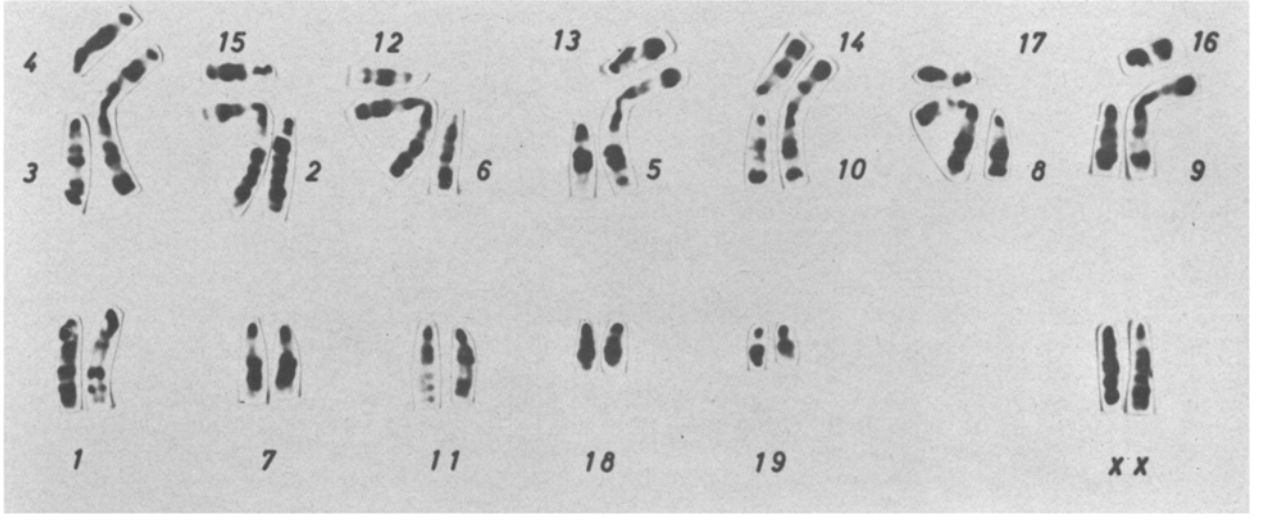

Fig. 2. Mitotic metaphase of an F1 animal obtained by mating the Sicilan mouse with 2n = 26 chromosoms with a normal mouse, stained with the Trypsin-Giemsa technique. The composition of the metacentric chromosomes is shown. The same chromosomes are found in the F1 animal with 7 metacentrics that we derived from the female with 2n = 27 chromosomes.

mouse has 6 pairs of metacentrics plus 1 unpaired one. The karyotype of the male with 2n = 26 chromosomes was determined by examining 5 F1 animals obtained from matings with normal females. All these F1 animals had 7 metacentrics. This shows that the parental male has 7 pairs of metacentric chromosomes. This result was confirmed by the fact that 2 F1 inter se offspring animals of these Sicilian mice had 13 metacentrics[10]. We looked at meiotic metaphase I figures of the F1 inter se animals in order to assay the homology of the parental metacentrics. Since we are establishing a breeding colony, we could not afford to analyse more than 2 F1 males, which unfortunately had only 13 metacentrics. Figure 1 shows that 6 of the metacentrics are homologuous by means of meiotic pairing, i.e. we found 6 ringbivalents. The 7th metacentric chromosome, for which the 2n = 27 female is heterozyguous was identified by analysis of Trypsin-Giemsa stained karyotypes of the same F1 animals. It is Rb(14.10)5Sic.

To assay the composition of the metacentric chromosomes, we looked at Trypsin-Giemsa stained karyotypes of F1 animals produced by mating the Sicilian mice with normal mice[6]. Following the nomenclature of the Committee on Standardized Genetic Nomenclature for mice[11,12] we assigned the following compositions (figure 2): Rb(4.3)1Sic, Rb(15.2)2Sic, Rb(12.6)3Sic, Rb(13.5)4Sic, Rb(14.10)5Sic, Rb(17.8)6Sic and Rb(16.9)7Sic. 6 of these chromosomes are entirely new, while Rb(17.8)6Sic has the same composition as Rb1IeM[6].

We are currently establishing a breeding colony to maintain these chromosomes, but until this is done we will not know whether these chromosomes affect fertility with normal mice[6]. However, it seems strange to us that 2 out of 3 animals had a high but odd number of metacentric chromosomes, implying heterozygosity. These animals seem therefore not to come from an isolated population, as is proposed for the other populations with high numbers of metacentrics.

10 After acceptance of the manuscript the male Sicilian mouse was karyotyped directly. It has 14 metacentric chromosomes.

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