originally subtelocentrics pair no. 1. Although fate of the deleted piece of the short arm is still uncertain, it might have been eliminated from the chromosome complement. Another explanation of the fate of the arm, however, is that the deleted arm translocated to the pair no. 3, and then the subtelocentric no. 3 chromosome developed. To determine whether the subtelocentric no. 3 has occurred by pericentric inversion or by translocation is difficult at present. Based on the measurement of length of chromosomes and the G-band analysis, the former event seems to be more likely than the latter.

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Heritability as an indicator of genetical variation in fecundity

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Summary. Heritability is well known to be a poor indicator of genetical variation in fitness. We show here that it is also an inadequate measure of genetical variation in fecundity.

Ewens and Thomson[3] have recently shown, for a general multilocus system, that the additive genetic variance in fitness is zero at equilibrium. They point out that this implies (from the definition of heritability) that the heritability of fitness in such a system will also be zero. Haldane[4] showed, for a single autosomal locus, that parent-offspring correlation in fitness (and hence its heritability) would be zero, but not the sib correlation. He emphasized the strong contrast between Darwinian fitness and other metric traits, where detectable additive genetical variation leads to nonzero heritability estimates. Sex-linkage is a complication[5], and systems not in equilibrium will depart somewhat from zero heritabilities in fitness, but nonetheless Haldane's results have considerable evolutionary interest. We report here results of a simulation study of the heritability of fecundity, showing that this is a trait where genetical variability in fecundity may be masked by the form of the distribution of fecundity.

Following Gillespie[6], we can consider the following model, for variation in fecundity determined by a single locus:

Genotype	A_1A_1	A_1A_2	A_2A_2
Mean	$1+\mu_1$	$1+\mu_2$	$1+\mu_3$
Variance	$1+\sigma^2_1$	$1+\sigma^2_2$	$1+\sigma^2_3$

In general, selection can act on both mean and variance[6], and stability is unlikely for large values of σ^2 in large populations[6,7]. In small populations, fixation is generally more rapid than with neutral alleles starting at the same frequency with the same variance effective population number.

That genetical variability in fecundity is determined by a single locus is most unlikely. We have accordingly considered by simulation a 10 locus elaboration of Gillespie's model, such that for each locus, $\mu_j = (j-1)\ \varepsilon_\mu$, $j = 1,2,3$, $\sigma^2 = (j-1)\ \varepsilon_{\sigma 2}$, $j = 1,2,3$. Varying the values of ε_μ and $\varepsilon_{\sigma 2}$ allows the distribution to vary from under-dispersed to over-dispersed, in this case from binomial through Poisson to negative binomial, these being appropriate to fecundity in various organisms[8,9]. If the frequencies of the 3 genotypes at the i^{th} locus are P_{ij}, $j = 1,2,3$, then popula-

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The relationship between the form of the distribution of family size and estimated heritability of family size

Distribution	ε_μ	ε_σ^2	Initial Mean	Variance	Initial I Variance/(mean)^2	h^2	T	N_e	n
Negative	0.1	0.2	2	3	0.75	0.00 ± 0.02	319.0 ± 45.2	80.0	6
binomial	0.2	0.4	3	5	0.56	0.09 ± 0.05	195.8 ± 13.9	96.3	5
	0.5	1.0	6	11	0.31	0.36 ± 0.07	141.3 ± 21.4	124.7	6
	1.0	2.0	11	21	0.17	0.33 ± 0.10	93.0 ± 10.6	220.9	5
Poisson	0.01	0.01	1.1	1.1	0.91	0.00 ± 0.03	615.7 ± 80.1	55.0	3
	0.05	0.05	1.5	1.5	0.67	0.09 ± 0.08	253.2 ± 25.3	66.1	4
	0.1	0.1	2	2	0.5	0.03 ± 0.01	325.0 ± 51.6	95.7	4
	1.0	1.0	11	11	0.09	0.60 ± 0.15	67.0 ± 10.8	289.5	4
Binomial	0.1	0.08	2	1.8	0.45	0.30 ± 0.16	68.2 ± 9.9	73.8	4
	0.2	0.1	3	2	0.22	0.00 ± 0.06	163.0 ± 18.0	180.0	4
	0.5	0.1	6	2	0.02	0.20 ± 0.06	286.2 ± 51.7	391.3	5
	1.0	0.1	11	2	0.02	0.00 ± 0.06	324.2 ± 42.3	930.8	5

For explanation of symbols see text. n, Number of replicates.

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

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