2. The adjacent region of $le_2 \sim \alpha$ is affected differently by genotypic differences thus suggesting that specific and independent genes control each region.

Acknowledgment.—We thank Miss Rachel Ronen for technical assistance.

5. References

CATCHESIDE, D. G. 1968. The control of genetic recombination in *Neurospora crassa*. *Replication and Recombination of Genetic Material*, ed. W. J. Peacock and R. D. Brock. Canberra, Australia.

CLARK, A. J. 1967. The beginning of genetic analysis of recombination proficiency. *J. Cell. Physiol.*, *70*, Suppl. 1, 165-180.

HAWTHORNE, D. C., AND MORTIMER, R. K. 1968. Genetic mapping of nonsense suppressors in yeast. *Genetics*, *60*, 735-742.

RILEY, R., AND MILLER, T. E. 1966. The differential sensitivity of desynaptic and normal genotypes of barley to X-rays. *Mutation Res.*, *3*, 355-359.

SIMCHEN, G., AND STAMBERG, J. 1969*a*. Fine and coarse controls of genetic recombination. *Nature*, *222*, 329-332.

SIMCHEN, G., AND STAMBERG, J. 1969*b*. Genetic control of recombination in *Schizophyllum commune*: Specific and independent regulation of adjacent and non-adjacent chromosomal regions. *Heredity*, *24*, 379-381.

STAMBERG, J. 1968. Two independent gene systems controlling recombination in *Schizophyllum commune*. *Molec. Gen. Genet.*, *102*, 221-228.

THE FIFTH CHROMOSOME HISTOCOMPATIBILITY TYPES OF MOUSE STRAINS Hg/Hu AND C3Hf/A

PETER HULL

Biology Department, Strathclyde University

Received 18.viii.70

1. Introduction

THERE is some indication, in the case of both rats and mice, that maternal-foetal incompatibility may lead to the preferential survival of offspring having histocompatibility antigens different from those of their mother (Hull, 1969; Palm, 1970). Since there is evidence that maternal-foetal " autoincompatibility " of this type is associated with differences between offspring and their mother at the *H-3/H-13* chromosome V region (Hull, 1969), which also includes the *a* locus (Snell *et al.*, 1967), it was decided to type the two strains CeHf/HeHa and Hg/Hu to see if they showed chromosome V histocompatibility differences: these two strains gave rise to the stock in which incompatibility between female parent and offspring of the same genotype at the $+/a^t$ locus was originally seen (Hull, 1964).

2. Experimental material and methods of interpretation of results

Reciprocal F_1 tests were used to type Hg/Hu* and C3Hf with strains B10LP and C57BL/Sn, a pair of coisogenic resistant strains bred by Dr Snell, with skin grafts as the test transplants. A piece of skin 1·5 cm. in diameter was taken from donor animals of the same sex as the recipient and grafted on to the right flank. The graft was attached by five or six stitches, dusted with antibiotic powder and covered with a plaster of paris bandage. After

* It is almost certain that strain Hg/Hu is the result of a cross between two strains both of C57BL derivation (K.P. Hummel, personal communication.)

10-14 days the bandage was removed and the graft examined at 3-day intervals for signs of rejection. This appeared to take place in one of two distinct ways. In some cases the grafts healed on, then from 15 to 40 days after grafting parts of their tissue died: the final result was that the whole graft was replaced by a small scar. In other cases the grafts looked like the surrounding host skin (except for their hair colour): this stage usually lasted for several weeks, but gradually the hair became thinner and was lost and the graft itself became paler and smaller. These cases were classified as slow rejects. Mice whose grafts survived longer than 100 days with full hair cover, etc., were regarded as accepting.

TABLE 1

Results of skin grafting tests

Test	Mice	Recipient	Immunised	Donor	Mean days to rejection
A	12	B10LP × C3Hf/A	No	C57	36·7 (+2 accepted, 200 days)
B	9	B10LP × C3Hf/A	Yes	C57	21·1
C	14	C57 × C3Hf/A	No	B10LP	All accepted
D	10	B10LP × Hg/Hg	No	C57	All accepted
E	12	B10LP × Hg/Hu	Yes	C57	8 accepted, 4 slowly rejected
F	7	C57 × Hg/Hu	No	B10LP	35·6
G	19	C57 × Hg/Hu	Yes	B10LP	22·8 (+1 accepted)
H	5	Hg/Hu	No	Hg/Hu	4 accepted, 1 rejected
I	8	Hg/Hu × C57	No	Hg/Hu × C57	5 accepted, 3 slowly rejected

Using this test, it would be expected, for example that the F_1 hybrids between the tested strain (Hg/Hu or C3Hf) and C57 would reject the skin from B10LP except if the tested strain provided alleles of the histocompatibility loci found in B10LP but not in C57. It was known that certain group V histocompatibility barriers would result in the rejection of skin grafts, whether or not the recipients were pre-immunised with donor tissue, whilst others required pre-immunisation to show rejection (Snell and Bunker, 1964; Snell *et al.*, 1967). Certain of the F_1 tests were duplicated using unimmunised recipients as well as recipients which had been given three doses of about $0{\cdot}5 \times 10^6$ thymus cells in tissue culture medium at weekly intervals before grafting. All donor and recipient animals were at least 12 weeks old. The thymus cells were obtained from 3 to 4-week-old animals of the same sex as the recipient. Since C3Hf/HeHa, the substrain originally used (Hull, 1964), was unfortunately not available C3Hf/HeA was used in the F_1 tests. These two substrains had undergone at least 50 generations of brother-sister mating since separation, at which time C3Hf was already highly inbred. The results of the F_1 tests along with those of reciprocal transplants between *a* and a^t individuals from strain Hg/Hu or between Hg/Hu × C57 individuals are given in table 1. The strain Hg/Hu had been brother-sister inbred for 39 generations in 1964 and has now been inbred for more than 50, but heterozygosity had been maintained at the *a* locus by always mating together *aa* and aa^t individuals. Heterozygosity had also been maintained at the *hr* (Hairless) locus in the same way (Staats, 1964). Since there is known to be slightly over 10 per cent. crossing-over between the *H-3* locus and the *a* locus, while the *H-13* locus is between *a* and *H-3* and shows slightly more than 1 per cent. crossing-over with the former (Snell *et al.*, 1967), it appeared to

be of interest to determine the probability of homozygosity at two such selectively neutral loci linked to a locus kept heretozygous in this way, given n generations of brother-sister mating.

It is possible to write recurrence equations for the 11 different types of mating allowed under this system. If these types are present in the frequencies shown in table 2, where $J+K+M+N+P+Q+R+S+T+V+W = 1$,

TABLE 2

Possible types of brother × sister mating

Mating type	Initial frequency	Mating type	Initial frequency
$\frac{aHa}{aHa} \times \frac{aHa}{a^tHa}$	J	$\frac{aHa}{aHa} \times \frac{aHb}{a^tHc}$	R
$\frac{aHa}{aHb} \times \frac{aHa}{a^tHb}$	K	$\frac{aHa}{aHb} \times \frac{aHc}{a^tHc}$	S
$\frac{aHa}{aHa} \times \frac{aHb}{a^tHb}$	M	$\frac{aHa}{aHb} \times \frac{aHa}{a^tHc}$	T
$\frac{aHa}{aHa} \times \frac{aHb}{a^tHa}$	N	$\frac{aHa}{aHb} \times \frac{aHc}{a^tHa}$	V
$\frac{aHa}{aHa} \times \frac{aHa}{a^tHb}$	P	$\frac{aHa}{aHb} \times \frac{aHc}{a^tHd}$	W
$\frac{aHb}{aHa} \times \frac{aHa}{a^tHa}$	Q		

and a and a^t are the two alleles at the locus kept heterozygous and *Ha*, *Hb*, *Hc* and *Hd* are any four alleles at a linked histocompatibility locus, s is the cross-over frequency between the two loci and $l+s = 1$, then the frequencies of mating types after one generation of inbreeding (J′, etc.) will be:

$$\begin{bmatrix} J' \\ K' \\ M' \\ N' \\ P' \\ Q' \\ R' \\ S' \\ T' \\ V' \\ W' \end{bmatrix} = \begin{bmatrix} 1 & \frac{ls}{2} & 0 & ls & ls & \frac{1}{4} & 0 & 0 & \frac{ls}{4} & \frac{ls}{4} & 0 \\ 0 & \frac{1}{4} & 1 & ls & ls & \frac{1}{4} & 2ls & \frac{1}{2} & \frac{3ls}{4} & \frac{3ls}{4} & ls \\ 0 & \frac{l^2+s^2}{4} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{ls}{2} & 0 & 0 & 0 & \frac{1}{4} & 0 & 0 & \frac{ls}{4} & \frac{ls}{4} & 0 \\ 0 & \frac{l^2+s^2}{4} & 0 & s^2 & l^2 & 0 & 0 & 0 & \frac{l^2}{4} & \frac{s^2}{4} & 0 \\ 0 & \frac{1}{4} & 0 & l^2 & s^2 & \frac{1}{4} & 0 & 0 & \frac{s}{4} & \frac{l}{4} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{l^2}{4} & \frac{s^2}{4} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{s^2}{4} & \frac{l^2}{4} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & l^2+s^2 & 0 & \frac{2l^2+s^2}{4} & \frac{2s^2+l^2}{4} & \frac{l^2+s^2}{2} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{1}{2} & \frac{2ls+s^2}{4} & \frac{l^2+2ls}{4} & ls \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{l^2+s^2}{2} \end{bmatrix} \begin{bmatrix} J \\ K \\ M \\ N \\ P \\ Q \\ R \\ S \\ T \\ V \\ W \end{bmatrix}$$

It is possible to substitute in these recurrence equations generation after generation using a computer, assuming an initial cross of $aa \times a^t a^t$, an intercross and subsequent back-crosses of $aa \times aa^t$ brothers and sisters. This was

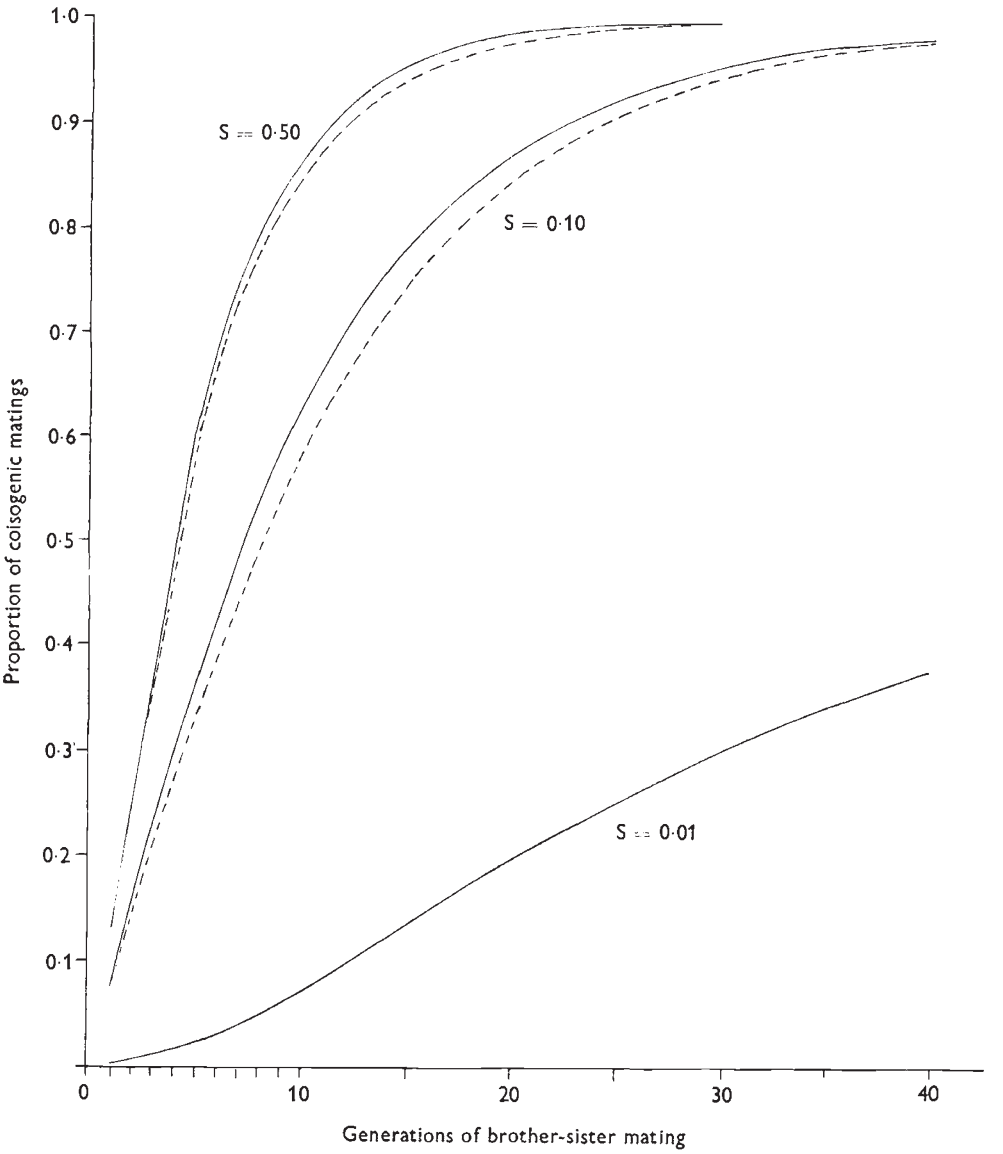

FIG. 1.—Expected proportions of coisogenic matings produced by brother-sister inbreeding with forced heterozygosity, with three intensities of linkage: s = 0·5, 0·1, or 0·01; with (dotted lines) or without (solid lines) autoincompatibility.

done for 50 generations, assuming s = 0·5 (no linkage), s = 0·10, s = 0·01 and s = 0·0. The probability of a " coisogenic mating " (*i.e.* that of a brother and sister picked at random being homozygous for the same allele at the histocompatibility locus) after *n* generations of brother-sister mating is given in fig. 1 (solid lines).

3. Discussion

It would appear, from tests A, B and C, that C3Hf/A is *H-3b* as are other substrains of C3H (Graff and Snell, 1969), while from the results of tests D, E, F and G it would seem that Hg/Hu cannot supply *H-3b* but may give *H-3a*. The results of tests H and I indicate that Hg/Hu is not entirely of one histocompatibility type, despite 50 generations of inbreeding. These tests were done by transplanting skin reciprocally between *aa* and aa^t individuals with normal fur, in order to maximise the probability of obtaining rejection if any group V histocompatibility loci had been held heterozygous together with the *a* locus: it will be seen from fig. 1 that residual heterozygosity would be more likely after 40 or 50 generations at the *H-13* locus than at the *H-3*. Heterozygosity at an unknown histocompatibility locus closely linked to *hr* (group III) is also possible. The results of test E might be interpreted as indicating that while Hg/Hu provides *H-3a* to all F_1 individuals, preventing the rejection of C57 skin, in only some cases is the weak histocompatibility allele possessed by C57 (possibly *H-13a*) provided by Hg/Hu: the remaining, supposed *H-13b*/*H-13x*, individuals might be expected to reject C57 skin slowly after pre-immunisation with C57 tissue. Alternatively it might be that the *H-3* allele provided by Hg/Hu partially complements the *H-3b* from B10LP so that rejection of *H-3a* tissue is slow or in some cases absent even after pre-immunisation.

It is assumed in the treatment of inbreeding used above (solid lines, fig. 1) that the loci concerned are selectively neutral. If there is autoincompatibility between homozygous females and their offspring of the same histocompatibility type, to the extent that, for example, a cross of *Ha*/*Ha* female with *Ha*/*Hb* male gives 0·60 *Ha*/*Hb* to 0·40 *Ha*/*Ha* offspring (which seems to be a maximum value, from the data of Hull, 1966, and Hull, 1969), then the recurrence equations may be rewritten as a computer programme as before, giving the results shown in fig. 1 (dotted lines). It will be seen that the expected difference between the effects of inbreeding with or without this degree of autoincompatibility are small. Under these circumstances it appears less likely that the slow rejections seen in test E are due to the fact that Hg/Hu still contains both *H-3a* and *H-3x* (despite the continued inbreeding), and more likely that they are due to some other locus more closely linked to the *a* or *hr* locus.

Thus there appear to be Vth chromosome histocompatibility differences between strains C3Hf/A and Hg/Hu, and the expected effects of these in combination would not be in disagreement with the hypothesis that those obtained with the $a^t/+$ system (Hull, 1964) were in fact due not to the *a* locus itself but to a closely linked histocompatibility locus. It may be that strain C3Hf/A is *H-3b* and Hg/Hu is *H-3a*.

It would be interesting to know if the *aa* mouse, obtained by Dr Hummel from Dr Gates in 1948 to found Hg/Hu, was related to C57. The question of whether Hg/Hu is homozygous at the *H-3* locus could be decided by testing it with coisogenic resistant strains differing only by a single histocompatibility locus developed by Dr Snell (Snell *et al.*, 1967).

4. Summary

1. Results of skin grafting tests indicate that strains C3Hf/A and Hg/Hu differ at the Vth chromosomal histocompatibility locus, *H-3*.

2. Residual heretozygosity exists within strain Hg/Hu, probably due to histocompatibility loci closely linked to the *a* or *hr* loci which were kept heterozygous during inbreeding.

Acknowledgments.—My thanks are due to Dr L. M. Boot, Dr K. P. Hummel and Dr G. D. Snell for supplying the strains of mice used in these tests.

5. References

GRAFF, R. J., AND SNELL, G. D. 1969. Histocompatibility genes of mice IX. *Transplantation, 8*, 861-875.

HULL, P. 1964. Partial incompatibility not affecting total litter size in the mouse. *Genetics, 50*, 563-570.

HULL, P. 1969. Maternal-foetal incompatibility associated with the *H-3* locus in the mouse. *Heredity, 24*, 203-209.

PALM, J. 1970. Maternal-foetal interactions and histocompatibility polymorphism. *Transplantation Proceedings, 2*, 162-173.

SNELL, G. D., AND BUNKER, H. P. 1964. Histocompatibility genes of mice IV. *Transplantation, 2*, 743-751.

SNELL, G. D., CUDKOWICZ, G., AND BUNKER, H. P. 1967. Histocompatibility genes of mice VII. *Transplantation, 5*, 492-503.

STAATS, J. 1964. Standardised nomenclature for inbred strains of mice. *Cancer Research, 24*, 147-168.

EVOLUTIONARY GENETICS RESEARCH REPORTS

JOHN R. G. TURNER

Department of Biology, University of York, York YO1 5DD

Received 3.x.70

DATA gathered in studies of micro-evolution, relating to the genetic composition of natural populations at a particular time, differ from other biological data in that they are essentially unique and unrepeatable. But, except in a few journals, it is often difficult to publish such data, as it is bulky and irrelevant in itself to the understanding of a research paper. The danger is therefore real that such information will not survive the life of its discoverer. The loss to future scientists could be serious.

The new series *Evolutionary Genetics Research Reports* is designed to preserve data of this kind. Each report consists of tables of data on the population genetics of a particular organism, with a small amount of explanatory text.

The reports are deposited with the National Lending Library for Science and Technology at Boston Spa, Yorkshire, and copies may be had by application to them in the usual way.

Unless an author decides to have copies prepared for his own distribution, only a few of each report will be prepared. Lists of titles will be issued from time to time, but the normal method of access will be by a full citation of the report in the published paper which discusses the data. Reports are also listed in *British Research and Development Reports*, and the *NLL Announcement Bulletin* (from 1971). Five reports have so far been prepared, dealing with lepidoptera and snails.

Scientists wishing to preserve their data in this way should write for further details to the editor, at the address above.