Mather, Wharton B. University of Queensland, Australia. D. rubida inversion polymorphism.

It has been shown previously (Mather, 1964) that certain inversions vary significantly in frequency at different times of the year at Port Moresby, New Guinea. It is the purpose of this report to measure inversion frequency in a different geographical region at Bulolo, New Guinea and to compare the inversion frequency patterns in these two populations.

Material was collected from fermenting banana baits at Bulolo in August, 1963 and February, 1964.

The material was analyzed by mating males and despermed females from the wild against a standard strain and scoring seven larvae from each mating against a photographic map (Mather, 1961). Salivary chromosomes were prepared by the method given in Strickberger (1962).

The results are set out in Table 1. It should be noted that when comparisons were made between August and February only certain inversions showed significant differences in frequency. These are indicated.

Particular attention should be drawn to the following results:

1. In chromosome II L inversion A has a significantly higher frequency in August than February in both males and females.

2. In chromosome II R inversion A has a significantly higher frequency in August than in February in females but not in males.

Table 1

Chromosome II	August 1963 (percent)		February 1964 (percent)	
	♂	♀	♂	♀
Standard	0	0	0	1.0
LA	14.4	15.7	5.2	8.1
RA	35.6	44.3	31.0	26.3
B	44.9	48.1	41.4	46.9
C	0.4	1.4	0	0
D	99.6	98.6	100.0	98.6
Chromosome III				
Standard	27.1	28.6	27.9	27.6
A		0.9		
B		0.9		
D	53.4	53.5	45.8	49.0
E	49.6	54.5	54.8	54.3
H	12.3	12.2	13.4	13.3
I	21.6	14.6	15.2	16.2
J	0	0	0.3	0
Flies scored	121	106	146	103

N. B. Arrows indicate differences significant at the 5% level.

When the range for inversion frequency at Bisianumu, Port Moresby, (Mather, 1964) is compared with that at Bulolo (Table 2) it is found that for all inversions there are different non-overlapping ranges. Bulolo is higher for II LA, II RA, B, D, III D, E, H, I, and J and lower for II RC, and III A and B. Perhaps the most outstanding feature of the Bulolo population is that it is virtually homozygous for the complex inversion II RD.

Thus, on cytological grounds the Bulolo population may be designated a different race from the Port Moresby population. Sexual isolation tests are to be carried out between the two populations.

Table 2

Chromosome II	Port Moresby Range %	Bulolo Range %
Standard	23.5 - 47.2	0.0 - 1.0
LA	0 - 2.9	5.2 - 15.7
RA	5.4 - 10.1	26.3 - 44.3
B	12.7 - 38.2	41.4 - 48.1
C	23.6 - 34.1	0 - 1.4
D	25.9 - 46.1	98.6 - 100
F	0 - 1.0	-

Table 2.--continued

Chromosome III	Port Moresby Range %	Bulolo Range %
Standard	52.9 - 72.2	27.1 - 28.6
A	2.3 - 5.6	0 - 0.9
B	1.0 - 6.7	0 - 0.9
D	4.4 - 9.8	45.8 - 53.5
E	22.2 - 41.3	49.6 - 54.8
H	0 - 1.5	12.2 - 13.4
I	0 - 1.1	14.6 - 21.6
J	-	0 - 0.3
Flies scored	361	476

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References: Mather, W. B., 1961. Chromosomal polymorphism in Drosophila rubida Mather Genetics, 46, 797.

Mather, W. B., 1964. Temporal variation in Drosophila rubida inversion polymorphism. Heredity, 19, 331.

Strickberger, M. W., 1962. Experiments in genetics with Drosophila. John Wiley.

McIntire, Sarah A. and Thomas Gregg. Miami University, Oxford, Ohio. Pteridines and the white locus in D. melanogaster.

It has been shown by Green (Heredity, 1959) and Judd (Genetics, 1964) that the white locus in D. melanogaster is genetically complex in that non-complementary mutations exist in at least five different sites that are separable by crossing over. It has also been shown (Gregg unpublished) that, to some extent, different alleles at the white locus control the presence of different pteridine compounds associated with red pigment in the wild type eye. Since the pteridines are a more direct reflection of the action of the genes that control their presence than the phenotype of the eye itself, it was felt that a study of the pteridines present in heads of mutant individuals carrying different white alleles might clarify the nature of the genetic complexity of the locus.

Pteridine accumulation patterns for thirty-seven white alleles were determined, using chromatographic methods similar to those of Throckmorton (Univ. Texas Publ. 6205), but using a paper in the shape of an Erlenmeyer flask silhouette (Harrison, Hayes, and Chua, Ohio Jour. Sci. in press) for better separation. The results are shown in the table below.

It appears to be impossible to explain the ten patterns in terms of a single genetic block, which would indicate that the locus is polycistronic in nature. But, if the white locus is polycistronic and if the established recombination sites represent different cistrons, it is surprising to find mutations at different sites producing the same pattern of pteridine accumulation. This particular observation is more readily explained if one assumes that the white locus is a single cistron. It is also difficult to explain the results by assuming that there are several cistrons present, any one of which could contain more than one of the established recombination sites. For instance, if pattern II is controlled by a cistron containing sites 1, 2, and 4, then it should also contain 3, but alleles at 3 produce at least two other patterns, III and IV, but not pattern II. The same is true for pattern IV, in that this pattern is produced by alleles at sites 2, 3, and 5, while alleles at site 4 produce other patterns.

However, the difficulties of explaining the lack of correspondence between the recombination sites and the pteridine patterns, assuming a polycistronic locus, appear to be considerably less troublesome than explaining the ten different patterns on the basis of a locus containing a single cistron. Therefore, in spite of the lack of correspondence, the existence of ten distinct patterns appears to be strong evidence for the polycistronic nature of the white locus.