stant along stream lines but constant throughout the entire flow. But as the point $Q' \to \infty$ along the rear shock the value of $N$ will approach the value $N_1$ of this quantity in the free stream. Hence $N = N_1$ and we again have the contradiction obtained in § 4.

*6. Natural Boundary Conditions.*—Removing the above restrictions let us suppose that the flow behind the rear shock is rotational and thus has the same general character as the flow between the two shock lines in figure 1. Now it is natural to assume that as we approach infinity behind the rear shock in the direction of the free stream the flow quantities, i.e., the pressure, density, and velocity, will approach the values of these quantities in the free stream. But this means that $N \to N_1$ and since $N$ is constant along stream lines it follows that $N$ will have the constant value $N_1$ everywhere behind the rear shock. Hence the consideration in § 4 again applies and leads to a contradiction. We have commented briefly on this state of affairs in § 1.

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[1] See Brown, W. F., and Thomas, T. Y., "Limiting Behavior of Pressure Derivatives Behind Shocks in Supersonic Gas Flow," to appear in *J. Rat. Mech. Analysis*, 1954, §1. Also earlier papers by Thomas, T. Y., for example, "First Approximation of Pressure Distribution on Curved Profiles at Supersonic Speeds," these PROCEEDINGS, **35**, 617–627 (1949).

[2] See, for example, Thomas, T. Y., "The Fundamental Hydrodynamical Equations and Shock Conditions for Gases," *Math. Mag.*, **22**, 187 (1949). It can be inferred from the discussion here that $N = N_1$ implies $\rho_2 = \rho_1$ which contradicts the assumption that we have a compression shock.

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# *GENETIC CHANGES IN AMERICAN POPULATIONS OF DROSOPHILA MELANOGASTER*

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In earlier reports[1, 2] data were presented showing the frequency of genetically lethal second chromosomes in natural populations of *Drosophila melanogaster* in a few widely separated areas in the United States. The frequency of lethal chromosomes was so high in these areas, and the amount of allelism among the lethals was so low in the three areas in which samples of flies were taken directly from nature, that it seemed most probable that breeding units within populations of this species average considerably larger than in those of other species of Drosophila which had been reported at that time.

In the earlier studies with *D. melanogaster*, in all areas except Florida, samples were taken only in the early fall, when the natural population was close to peak size. Beginning in 1946 samples were taken from the local Massachusetts area at three times in the season: The first in June or July as soon as a large enough sample (about 100 flies minimum) could be collected within a week at one site to make the analysis worth while; the second about a month later; and the last collection as

late in the season as possible before the first killing frost in September or October. Beginning in 1950 a number of other areas were sampled in the early fall, and the Florida site was sampled in the spring of 1951. In most of these samples allelism tests were also completed with the strictly lethal chromosomes.

The only change in method from the earlier work consisted of the substitution of a new marker stock, known as *Sifter* to Drosophila geneticists, to facilitate making the test matings. Concurrent tests with *Bl/Cy* and *Sifter* in several different seasons showed no significant difference in lethal frequency (including semi-lethals) in the two methods of analysis.

Table 1 shows the frequency of lethals in different geographic areas, all collections from each area being summed.[3] Each collection site was in a rural or suburban area. None was within a large city area. It can be seen that the lethal frequency was higher in the three southernmost areas, Virginia, Texas, and Florida, than in the other more northerly localities. Lethals include all cases with less than 17% of homozygotes, as in the 1945 report. A comparison with the similar data of table 2 of that report shows that the lethal frequency is about 15% lower in these later

TABLE 1

FREQUENCY OF LETHALS IN DIFFERENT AREAS OF THE UNITED STATES

| DATE | AREA | TESTS | LE | % |
|---|---|---|---|---|
| 1947–1952 | Amherst, Mass. | 3163 | 1105 | 34.9 |
| 1950 | New York State | 527 | 170 | 32.3 |
| 1952 | Cannonsburg, Pa. | 117 | 33 | 28.2 |
| 1951 | Wooster, Ohio | 166 | 60 | 36.2 |
| 1952 | Lincoln, Neb. | 133 | 34 | 25.6 |
| 1951 | Pullman, Wash. | 138 | 54 | 39.1 |
| 1950–1952 | Blacksburg, Va. | 805 | 346 | 43.0 |
| 1952 | Austin, Texas | 98 | 41 | 41.8 |
| 1951 | Winter Park, Fla. | 131 | 67 | 51.1 |
| Totals | | 5278 | 1910 | 36.2 |

samples from each of the three areas represented in both tables, Massachusetts, Florida, and Ohio. (In Ohio the two collection sites were in neighboring small city areas.) The other areas in the present study are also lower than would have been expected from the earlier study.

Tables 2 and 3 show in detail the change that has taken place in the two areas most critically sampled, Massachusetts and Florida. Items 1 and 2 of table 2 are from the 1945 report. Item 3 is from the 1947 report but has been corrected for the amount of allelism by removing those allelism tests involving lethal chromosomes derived from collected females. All samples in table 2 were collected in September or October, and the allelism data are for lethal chromosomes derived from collected males. In table 3 the 1940, 1942 data are from the 1945 report. In the 1951 Florida sample, females and males were separated soon enough after collection so that no increase in frequency of alleles was observed among lethal chromosomes derived from the females of the sample. In this case, therefore, the allelism data include tests of lethal chromosomes derived from both males and females.

Table 2 shows that apparently the major change in both lethal frequency and amount of allelism appeared first in 1947. The frequency dropped 10% from the

previous year and the amount of allelism approximately doubled compared to the average of the 1938–1946 period. The clearest statistical difference, however, is between the averages of the two groups of years, items 5 and 12. For the frequency of lethals, $P$ is less than .0001, and for the amount of allelism it is approximately .0013, as determined by chi-square. The variation within each group of years is not significant, since the value of $P$ is above .10 for each of the four comparisons.

Inspection of the data of table 2 shows, however, that there is actually considerable variation in lethal frequency within each group of years, the statistical significance of which may be obscured by the necessarily limited amount of data which could be collected each year. Particularly is this true in the 1947–1952 period where it looks as though the lethal frequency dropped secondarily in 1949 and came

TABLE 2

FREQUENCY OF LETHALS AND ALLELES IN DIFFERENT SEASONS IN SOUTH AMHERST, MASS.

| ITEM | YEAR | TESTS | LETHALS | % | CROSSTESTS | ALLELES | % |
|---|---|---|---|---|---|---|---|
| 1 | 1938 | 151 | 68 | 45.0 | 1176 | 5 | 0.43 |
| 2 | 1941 | 108 | 64 | 59.3 | None | | |
| 3 | 1945 | 190 | 87 | 45.8 | 666 | 3 | 0.45 |
| 4 | 1946 | 79 | 39 | 49.3 | 465 | 1 | 0.22 |
| 5 | Total 1–4 | 528 | 258 | 48.8 | 2307 | 9 | 0.39 |
| 6 | 1947 | 257 | 101 | 39.3 | 1039 | 9 | 0.87 |
| 7 | 1948 | 162 | 60 | 37.0 | 1219 | 11 | 0.90 |
| 8 | 1949 | 198 | 60 | 30.3 | 1223 | 11 | 0.90 |
| 9 | 1950 | 163 | 52 | 31.9 | 528 | 4 | 0.76 |
| 10 | 1951 | 182 | 68 | 37.4 | 1377 | 17 | 1.24 |
| 11 | 1952 | 190 | 72 | 37.9 | 820 | 5 | 0.61 |
| 12 | Total 6–11 | 1152 | 413 | 35.9 | 6206 | 57 | 0.92 |

TABLE 3

THE FREQUENCY OF LETHALS AND ALLELES AT DIFFERENT TIMES IN WINTER PARK, FLORIDA

| YEARS | TESTS | LETHALS | % | CROSSTESTS | ALLELES | % |
|---|---|---|---|---|---|---|
| 1940, 1942 | 337 | 220 | 65.3 | 2281 | 10 | 0.44 |
| 1951 | 131 | 67 | 51.1 | 1326 | 5 | 0.38 |

back up to its 1947–1948 level in 1951–1952. The value of $P$ for the six years as a group is .3; for the comparison of the three pairs of years (paired chronologically to give two degrees of freedom) it is .07; and for the comparison of 1949–1950, first with 1947–1948, and then with 1951–1952 it is .04 and .06, respectively. The $P$ values are large enough so that the possibility of this variation being purely random cannot be considered unlikely. At the same time they are small enough to render more acceptable the alternative interpretation that there was a U-shaped decrease and increase in lethal frequency in the 1947–1952 period. There is no suggestion of any related inverse change in the amount of allelism observed in this period, perhaps because the data are not large enough to demonstrate comparatively small changes in this category. It is probably only coincidental that this apparent cycle of change occurred at exactly two-year intervals in this period of time.

The data of table 3 show that the drop in lethal frequency ($P$ is .0007) in the

Florida areas was not accompanied by a rise in the observed amount of allelism among lethals. In 1951 the lethal frequency and the amount of allelism in the Florida area each resembles closely the corresponding average in the Massachusetts area in the 1938–1946 period.

Some additional information on the Massachusetts population is given in table 4. These data show for the 1947–1952 period the average lethal frequency in the first, second, and last collections and the average amount of allelism in the first and last collections. The data are for five of the six seasons; in 1949 it was possible to make collections only in August and October. Inspection of the data suggests that in the time between the first and second collections, when the population was expanding rapidly, there was an increase in lethal frequency, but that in the rest of the season there was no change. It also suggests that there was the same amount of allelism among lethals at both phases of population development, expansion and peak.

In five of the six seasons in which the first collection came before August (including 1946) there was an increase in lethal frequency in the second collection; 1952 was the exceptional year. The increase was not statistically significant in any one season. Considering only the chi-square $P$ value of .08, the average increase in table 4 is on the borderline of significance statistically. If the increase is real (the

TABLE 4

THE 1947–1952 AVERAGE INTRA-SEASONAL FREQUENCIES OF LETHALS AND ALLELES IN SOUTH AMHERST, MASS.

| SAMPLE | TESTS | LETHALS | % | CROSSTESTS | ALLELES | % |
|---|---|---|---|---|---|---|
| First | 726 | 233 | 32.1 | 2628 | 29 | 1.10 |
| Second | 550 | 202 | 36.7 | | | |
| Last | 954 | 353 | 37.0 | 4983 | 46 | 0.92 |

author feels that more data are needed to determine that fact) it can probably be satisfactorily accounted for by two or three generations of accumulation of new mutations.[4] The increase is not as large as the average increase in Russian populations.[5] Goldschmidt has reported no increase during the expansion phase of Jerusalem populations.[6] If new mutations are the cause in the present case, one must assume an earlier elimination of lethals in the comparatively dormant season, or in the earlier part of the breeding season than is represented in the first collections, due either to inbreeding or selection. There are no data on the amount of allelism at actual breeding sites in the local area.

The complete picture is not given with respect to allelism in the simple averages of table 4. In some seasons it was possible to test for allelism within and between three or more collections per season. The details of this study will appear elsewhere. They will show that each season probably differs from the others in the nature of the breeding units which send flies to the collection site. The picture is much more dynamic and complex than the averages given here suggest. The present data do show, nevertheless, that in this group of seasons the average amount of allelism among the lethal chromosomes was approximately the same in a potential gene pool at the time when the population was in its stage of rapid expansion as it was a little later on when the peak of population size had been reached.

It is apparent from the data of tables 1, 2, and 3 that there was a significant

change in the genetic structure of natural populations of *D. melanogaster* in America during the past decade. One simple interpretation is that there has been a decrease in population size, in the number of pairs of individuals which constitute the average breeding unit. This would cause an increase in the amount of inbreeding, which would in turn be reflected in an increase in allelism and a decrease in the frequency of lethal chromosomes. That description fits the data of the local area. Another possibility is an increased selection against lethal heterozygotes, which would lower the lethal frequency without necessarily changing the amount of allelism noticeably, such as appears to be the case in the Florida data of table 3. There is no conclusive evidence concerning these interpretations.

It is possible that in the most southerly regions *D. melanogaster* is suffering from competition with its closely related species, *D. simulans*. With collection conditions as alike as it was possible to make them, the sample of 1942 from Florida contained an estimated 15% of simulans in about 300 males, while in the 1951 sample of 990 flies there was 86% simulans, with no difference in percentage of simulans in males and females. The small sample from Austin, Texas, in 1952 had 40% simulans in the 116 males of the collection. *D. simulans* was very infrequent in the collections from other areas. It has appeared locally only in one or two males per season in late summer collections.

The causative agent for the presumed decrease in size in the northerly populations of *D. melanogaster* during the 1940's is not known. The change in climate to a warmer and drier summer, generally, has not been sufficient locally to interfere noticeably with the presumed food source of the species, decaying fruits, vegetables, and garbage, at least so far as can be seen by casual observation. The introduction of new fungicide and insecticide sprays in orchards and buildings may be a factor. So far as can be determined there was no change in spraying program in 1947 as compared to 1946 but several new sprays were introduced in subsequent seasons. These might have contributed to the apparent population drop in 1949 and 1950. Whatever the cause the species seems to have adapted itself to the situation and may have improved its position in 1951 and 1952.

It seems obvious that experiments with large continuous cage populations may suggest the nature of the influence of certain environmental agents on the genetic factors of natural populations, beginning with some of the genetic factors already under investigation.[7-10] At the same time it should perhaps be emphasized that in a natural population the combination of important variables and effective agents is probably so complex that only very general applications can be made of the findings from studies of artificial cage populations. It is not possible to "experiment" with such variables and agents and at the same time maintain truly natural populations. Much remains to be done of value, however, at the descriptive genetic level in the study of natural populations of *Drosophila melanogaster*.

[1] Ives, P. T., *Genetics*, **30**, 167–196 (1945).

[2] Ives, P. T., *Evolution*, **1**, 42–47 (1947).

[3] The author is indebted to the following persons for collecting flies: Dr. Mary Alexander, Dr. R. P. Levine, Dr. M. Levitan, Dr. D. D. Miller, Dr. R. Moree, Mrs. H. H. Plough, Mr. M. Seiger, and Dr. W. P. Spencer. He is also grateful to Dr. B. Wallace for the use of his unpublished data from three samples of 449 chromosomes which are included in the New York State sample after the individual tests had been reclassified by the standard for lethals used in the other samples of the present study.

[4] Ives, P. T., *Evolution*, **4**, 236–252 (1950).
[5] Dubinin, N. P., *Genetics*, **31**, 21–38 (1946).
[6] Goldschmidt, Elizabeth, *Drosophila Information Service*, **26**, 102–103 (1952).
[7] Wallace, B., *Evolution*, **4**, 172–174 (1950).
[8] Wallace, B., and King, J. C., these PROCEEDINGS, **38**, 706–715 (1952).
[9] Wallace, B., and Madden, Carol, *Genetics*, **38**, 456–470 (1953).
[10] Levine, R. P., and Ives, P. T., these PROCEEDINGS, **39**, 817–823 (1953).

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## *PSEUDO-ALLELISM AT THE VERMILION LOCUS IN DROSOPHILA MELANOGASTER*

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A number of cases have been reported in Drosophila[1–6] and Aspergillus,[7, 8] and apparent cases in Neurospora[9, 10] and cotton,[11] in which recombination occurs between mutants which otherwise classify as allelic by the usual phenotypic and genetic criteria. Such mutants have been designated as pseudo-alleles. In addition to recombination, pseudo-alleles manifest a differential phenotype depending upon whether the mutants are compounded in coupling or in repulsion. Thus in a case of pseudo-alleles which can be designated as $m_1$ and $m_2$, both of which are recessive to wild-type, the phenotype of individuals of the genotype $m_1 + / + m_2$ is mutant, while that of individuals $m_1 m_2 / + +$ is wild-type. Alternative explanations have been proposed to account for this situation. On the one hand, it has been reasoned that pseudo-alleles represent separate genes whose loci and functions are distinct. The phenotypic differences alluded to are then interpreted as being the consequence of position effect.[1, 2, 4] Alternatively, it has been argued that pseudo-alleles are components of a larger "physiological" gene, whose functions are indistinguishable but whose component parts are separable by crossing-over.[8, 12] The phenotypic differences associated with pseudo-alleles are therefore those expected according to the usual rules of dominance.

These alternative interpretations imply conflicting definitions of the gene. On the one hand the gene as defined in terms of linkage relations, mechanism of action and mutability is one and the same. On the other the limits determined for the gene in terms of linkage do not necessarily coincide with the limits of the gene in terms of function.

It is the purpose of this report to discuss a case of pseudo-allelism at the vermilion eye color ($v$) locus in *D. melanogaster* and to determine what bearing it has on the interpretation of pseudo-allelism and the gene.

*Phenotypic Differentiation of v Mutants.*—In an earlier discussion of several sex-linked, recessive $v$ mutants of *D. melanogaster*, it was noted that while they are essentially phenotypically indistinguishable, it is possible to separate them into two classes on the basis of their phenotypes in the presence of a non-allelic suppressor mutant.[13] Certain of the $v$ mutants (collectively designated as $v^s$) are suppressed, while others (collectively designated as $v^u$) are unsuppressed. It was