[13] Morgan, D. T., *J. Hered.*, **34**, 194–198 (1943).
[14] Lorz, A. P., *Bot. Rev.*, **13**, 597–624 (1947).
[15] Kaufmann, B. P., *Ibid.*, **14**, 57–126 (1948).
[16] Duncan, R. E., *J. Hered.* (in press) (1950).
[17] Witkus, E. R., *Am. J. Bot.*, **32**, 326–330 (1945).
[18] Brown, S. W., *Ibid.*, **36**, 703–716 (1949).
[19] Huskins, G. L., and Steinitz, L. M., *J. Hered.*, **39**, 66–77 (1948).
[20] Jacobi, W., *Zeitschr. mikr.-anat. Forsch.*, **38**, 161–240 (1935).
[21] Huskins, G. L., *Am. Nat.*, **81**, 401–434 (1947).

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*ALLELISM OF SECOND CHROMOSOME LETHALS IN D. MELANOGASTER**

BY BRUCE WALLACE

THE BIOLOGICAL LABORATORY, COLD SPRING HARBOR, N. Y.

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Our knowledge of the genetic composition of populations, other than that of human blood group genes, is based primarily on lethal chromosomes from populations of Drosophila. Paradoxically, because of the advanced genetic techniques in Drosophila, our information for species of this genus consists of *chromosomal* frequencies although the dynamics of population genetics depends upon *gene* frequencies. It would be possible to analyze Drosophila populations for specific gene loci but it has proved more profitable to evaluate the easily collected lethal chromosome data by estimating the number of loci on a given chromosome at which lethal alleles may exist. This estimation, which can be made by determining the frequency of allelism between lethals of independent origin, has been made by Wright[1, 2] for the third chromosome of *D. pseudoobscura* (285–289 loci) and by Ives[3] for the second chromosome of *D. melanogaster* (495 loci). In connection with experimental populations which are exposed to continuous gamma irradiation, it has been necessary to determine the number of loci which are capable of mutating to lethality under the influence of these radiations.

Flies of the Oregon-R strain of *D. melanogaster* carrying lethal-free second chromosomes were placed in a population cage and were allowed to oviposit on food in small plastic cups throughout the day (8 hrs.) or overnight (16 hrs.). (See Wallace[4] for a detailed description of the cages and the cups.) At the end of each egg-collecting period, a fresh cup was exposed to the parental flies, and the old cup, with its eggs, was placed in a cage which encircled a 500-mg. radium bomb. To keep the developing flies of each cup separate from the rest, a thin-walled plastic tube was inserted into the food of each cup and was plugged at its free end with cotton.

The flies developing under these conditions were exposed throughout their developmental stages and for part of their adult life to a constant dose of gamma rays of approximately 5 r per hour. After 18 days (426 hrs. or 2175 r, average), males were removed and mated individually in vials with CyL/Pm females. (The CyL chromosome carries the dominant genes Curley and Lobe and two inversions which suppress most of the crossing-over.) Several CyL/+ F1 males of each culture were mated singly in vials with CyL/Pm females, and the CyL/+ F2 males and females of each culture were inbred in vials to determine the lethal mutation rate. If a lethal had been induced on a treated chromosome, this was noticed by the absence of wild-type flies in the F3. Each suspected lethal was confirmed by mating for an additional generation in a regular culture bottle. After confirmation, 125 lethals which had arisen in different original males and, therefore, were independent in origin, were subcultured in four bottles in order to obtain flies for the allelism tests. With the exception of the latter tests which were kept at room temperature (22° ±) all phases of the experiment were carried out at 25°C.

To determine the frequency of induced mutations, 3772 second chromosomes derived from 336 treated males were analyzed; 456 were lethal. The frequency of lethal chromosomes, then was 12.09 ± 0.53%. The frequency of lethal genes, calculated by means of the Poisson distribution, was 12.89 ± 0.60%.

The tests for the frequency of allelism were made by intercrossing CyL/+ flies from 100 lethal cultures of independent origin. (The remainder of the 125 cultures originally chosen either produced too few flies for the required matings or gave rise to small numbers of wild-type flies which could have obscured the test.) Fourteen of the 4950 $\left(\frac{100 \times 99}{2}\right)$ matings failed to produce wild-type flies. The probability, then, that one tested chromosome was allelic to another is 0.28%. (The limits of the 95% confidence interval of this frequency are 0.16% and 0.48%).

The minimum number of loci, n, capable of mutating to lethality is the inverse of the probability that one lethal gene is allelic to another. This number can be calculated from the frequency of lethal chromosomes (a = 12.09 ± 0.53%), the frequency of lethal genes (b = 12.89 ± 0.60%) and the frequency of allelism between lethal chromosomes (c = 0.28% with limits 0.16% and 0.48%): $n = b^2/ca^2$. The most probable minimum number calculated from our data is 400. By substituting the most divergent values of a, b and c into the equation, the limits of the minimum number may be estimated as 234 and 718.

The distribution of allelic lethals among the 100 chromosomes which were tested gives a quasi-independent confirmation of the above estimate of the minimum number of loci. In one case chromosome A was allelic to both

chromosomes *B* and *C*, but *B* and *C* were non-allelic, therefore, it is known that the tests for allelism involved lethal genes at 101 loci. Among these 101 lethals there were 73 which occurred only once and 14 which occurred twice. For any given number of potentially lethal loci it is possible to predict the distribution of singles, pairs and triplets among 101 independently chosen lethals by means of the Poisson series. If $n = 234$, there should be 65.6 singles, 14.2 pairs and 2.0 triplets; if $n = 400$, these values should be 78.4, 9.9 and 0.8; if $n = 718$, 87.7, 6.2 and 0.3. The expected distribution when $n = 718$ is significantly different from the observed distribution; it is probably that the minimum number of lethal loci lies nearer the calculated 400.

It is of interest to note the similarity between the number of loci capable of mutating to lethality under the influence of gamma rays (400 with limits of 234 and 718) and the number capable of mutating spontaneously (495 with limits of 285 and 705). This agreement indicates that these loci are so situated that an ionizing particle is likely to affect only one at a time. Since nearly 2000 bands have been recorded in the second chromosome of *D. melanogaster* in salivary preparations and since induced lethals have frequently proved to be small deficiencies (Slizynski[5]), it seems likely that loci mutating frequently to lethality may be separated from one another by material relatively inert in this respect.

In conclusion, several points should be emphasized. The 400 loci calculated above represent a minimum number of loci which can mutate to lethality. The maximum number could be substantially larger but, nevertheless, remain undetected because of great differences in the mutation rates of different loci. It should be noted, too, that observed allelism does not prove identity; overlapping deficiencies may act as allelic lethals. These considerations have been pointed out by Wright[1] in his analysis of Dobzhansky's data.

Summary.—Through an analysis of the frequency of allelism of 100 second chromosome lethals induced in *D. melanogaster* by chronic gamma-ray treatment, it has been estimated that the minimum number of loci capable of mutating to lethality under these conditions is 400 (234–718).

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[1] Dobzhansky, Th., and Wright, S., "Genetics of Natural Populations. V. Relations between Mutation Rate and Accumulation of Lethals in Populations of *Drosophila pseudoobscura*," *Genetics*, 26, 23–51 (1941).

[2] Wright, S., Dobzhansky, Th., and Hovanitz, W., "Genetics of Natural Populations. VII. The Allelism of Lethals in the Third Chromosome of *Drosophila pseudoobscura*," *Ibid.*, 27, 363–394 (1942).

[3] Ives, P. T., "The Genetic Structure of American Populations of *Drosophila melanogaster*", *Ibid.*, **30**, 167–196 (1945).

[4] Wallace, B., "A New Population Cage," *Drosophila Information Service*, **23**, 108–109 (1949).

[5] Slizynski, B. M., "Salivary Chromosome Studies of Lethals in *Drosophila melanogaster*," *Genetics*, **23**, 283–290 (1938).

COHOMOLOGY THEORY OF ABELIAN GROUPS AND HOMOTOPY THEORY II

BY SAMUEL EILENBERG AND SAUNDERS MACLANE*

DEPARTMENTS OF MATHEMATICS, COLUMBIA UNIVERSITY AND THE UNIVERSITY OF CHICAGO

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1. Generic Homology Groups.—The cohomology groups of a group Π are the groups of the cell complex $K(\Pi, 1)$ which has as its cells in each dimension $q \geqq 1$ the q-tuples $[x_1, \ldots, x_q]$ of elements $x_i \in \Pi$, with $\partial[x] = 0$ and

$$\partial[x_1, \ldots, x_q] = [x_2, \ldots, x_q] + \sum_{i=1}^{q-1} (-1)^i [x_1, \ldots, x_i x_{i+1}, \ldots, x_q] + (-1)^q [x_1, \ldots, x_{q-1}]. \quad (1)$$

This boundary formula does not use inverses of the letters x_i, and no letter x_i is repeated in any one cell of the boundary. These two properties may be conveniently expressed in the complex $K(F, 1)$ constructed from the free group F with a denumerable set of free generators $g_1, g_2, \ldots$. Call an element of F *generic* if it is a product $x = 1 g_{i_1} g_{i_2} \ldots g_{i_k}$ of $k \geqq 0$ distinct generators, and call two generic elements x and y *disjoint* if they involve no generator in common. A cell $[x_1, \ldots, x_q]$ is generic if the entries $x_1, \ldots, x_q$ are generic and pairwise disjoint. Then formula (1) shows that the boundary of any generic cell is a linear combination (with integral coefficients) of generic cells. Consequently the generic cells span a subcomplex $K(F^*, 1)$ of $K(F, 1)$.

The usual proof[1] that the cohomology groups of a free group F are zero in all dimensions greater than 1 gives the integral homology groups of this "generic" complex $K(F^*, 1)$ as

$$H_q(K(F^*, 1)) = 0, \qquad q > 1 \quad (2)$$

$$H_1(K(F^*, 1)) = F_a, \quad (3)$$

where F_a is the free abelian group on a denumerable set of generators g_1',