NON-RANDOM GENE DISTRIBUTION AMONG TOMATO CHROMOSOMES*

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It was pointed out in 1956 by Rick and Butler that the genes of the cultivated tomato, Lycopersicon esculentum Mill., do not seem to be randomly distributed among the 12 chromosomes. A satisfactory test for randomness could not be made at that time because certain chromosomes had been favored historically by having had a longer opportunity for detection of new linkages, and by having accumulated several marker genes apiece with a consequently greater length tested for new linkages. Notwithstanding these obstacles to the application of an exact probability treatment, they concluded "...it seems inevitable that this deviation from random distribution is real."

A better opportunity for testing random distribution has been afforded by a sample of 18 spontaneous seedling mutations accumulated here in the past six years. These were acquired from various sources in many different background genotypes. The only limitation imposed upon the sample is that of seedling expression—a relatively severe restriction for the reason that it favors mutations affecting pigmentation, stature, leaf shape, and disposition of hairs. Although it cannot be presently disproved that this restriction might affect chromosomal distribution of the genes concerned, such as effect is unlikely for the following considerations. First, as indicated below, the concentration of this sample on chromosome 2 is matched by the same tendency of mature-plant characters. Second, in other organisms that are better known genetically the genes affecting a particular organ seem to be distributed in the same fashion as the total known genotype.

Descriptions and linkage data have been published for all but two mutants, La and wu, of this sample; references are indicated in Table 1. Each mutant was systematically screened for linkages by crosses with a series of marker genes, one per chromosome except that Wo was used as an additional marker for chromosome 2 in the tests with ff and Mi for the location of yv on chromosome 6. Linkages were

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* Benzer, S., these PROCEEDINGS, 41, 344 (1955).
* Benzer, S., and E. Freese, these PROCEEDINGS, 44, 112 (1958).
* Freese, E., these PROCEEDINGS, 45, 622 (1959).
* Garen, A., personal communication.
* Uchida, H., personal communication.
* Tessman, I., Virology (in press).
* Hershey, A. D., and R. Rotman, Genetics, 34, 44 (1949).
detected by standard F2 tests, mostly in repulsion phase. For nearly all combinations a minimum of 400 plants was scored, thereby screening a linkage distance of about 40 units on either side of the marker gene. Tests were usually discontinued as soon as a significant indication of linkage was encountered.

### TABLE 1

**SUMMARY OF LINKAGE TESTS**

<table>
<thead>
<tr>
<th>Mutant</th>
<th>Reference</th>
<th>Chromosome and Tester Gene</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cu</td>
<td></td>
<td>1 2 3 4 57 6 7 8 9 10 V ?</td>
</tr>
<tr>
<td>d1*</td>
<td></td>
<td>y d1 E L x * x x x x x x x x x x 2</td>
</tr>
<tr>
<td>d1*</td>
<td></td>
<td>L x x x x x x x x x x x x x x x x 2</td>
</tr>
<tr>
<td>dv</td>
<td></td>
<td>x L L x x x x x x x x x x x x x x 2</td>
</tr>
<tr>
<td>ff</td>
<td></td>
<td>L x x x x L+ x x x x x x x x 2</td>
</tr>
<tr>
<td>Me</td>
<td></td>
<td>L x x x x x x x x x x x x x x 2</td>
</tr>
<tr>
<td>Wo*</td>
<td></td>
<td>L x x x x x x x x x x x x x x 2</td>
</tr>
<tr>
<td>rv</td>
<td></td>
<td>x x x x x x x x x x x x x x x x 2</td>
</tr>
<tr>
<td>sf</td>
<td></td>
<td>x x x x x L x x x x x x x x 2</td>
</tr>
<tr>
<td>ye</td>
<td></td>
<td>x x x x x L+ x x x x x x x x 2</td>
</tr>
<tr>
<td>ff</td>
<td></td>
<td>x x x x x x x x x x x x x x 2</td>
</tr>
<tr>
<td>Fw</td>
<td></td>
<td>x x x x x x L x x x x x x x 2</td>
</tr>
<tr>
<td>dl</td>
<td></td>
<td>x x x x x x L x x x x x x 2</td>
</tr>
<tr>
<td>ah</td>
<td></td>
<td>x x x x x x x x x x x x x 2</td>
</tr>
<tr>
<td>ag</td>
<td></td>
<td>x x x x x x x x x x x x 2</td>
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<tr>
<td>gh</td>
<td></td>
<td>x x x x x x x x x x x x 2</td>
</tr>
<tr>
<td>ht</td>
<td></td>
<td>x x x x x x x x x x x x 2</td>
</tr>
<tr>
<td>La</td>
<td></td>
<td>x x x x x x x x x x x x x x x x 2</td>
</tr>
<tr>
<td>w4</td>
<td></td>
<td>x x x x x x x x x x x x x x x x 2</td>
</tr>
</tbody>
</table>

* x signifies no indication of linkage. L signifies positive indication of linkage.

† Linkage of ff detected with Wo.

† Linkage of ye detected with Mi.

The results, summarized in Table 1, reveal the following distribution of the 18 mutants: chromosome 2: Cu, d1* dv, ff, Me, Wo*; 5: rv, sf; 6: ye; 7: tf, Fw; 8: dl; 9: ah; 10: ag; group V (chromosome 11 or 12): gh; no linkage: ht, La, w4.

A problem in the application of a probability treatment is posed by the three unlocated genes. For present purposes they are included in the analysis since they were tested against nearly all the markers. It is further assumed that neither is located on any of the tester chromosomes, thereby avoiding any bias in favor of chromosome 2. The probability that as many as 6 genes would be distributed to any single chromosome is given by the sum of the first 13 terms of the expansion of the binomial \((p + q)^4\), where \(p\), the probability of a gene residing on a specific chromosome, is \(\frac{1}{12}\). The value obtained, 0.011, reveals a significant deviation from random distribution. If the data for ff (for which the discovery of a locus on chromosome 2 was favored by the use of 2 markers) are omitted, the probability calculated from the first 12 terms of \((p + q)^4\), 0.039, is still significant.

A non-random distribution of tomato genes has been suggested by other investigations. In a sample of 10 male-sterile mutants of a single horticultural variety, Pratt4 assigned 2 to chromosome 2, three to group V, one each to chromosomes 1, 3, and 8, and two were not located. The deviation from randomness was not significant in this small sample, but the tendency is revealed of an accumulation on the already heavily laden chromosome 2. Currence,7 testing the association between earliness and four markers on chromosome 2, found that most of the hereditary seasonal difference between parents could be ascribed to this chromosome. While the other chromosomes, which were not tested, doubtless exerted a genetic influence that was cancelled out, chromosome 2 wielded an astoundingly large share of genetic influence upon earliness.
A probability treatment of all located tomato genes is a vastly more difficult task for aforementioned reasons and because the chromosome map is based upon trisomic as well as ordinary linkage tests. The following data are therefore presented for what they might be worth without statistical tests. In the latest revised chromosome map of the tomato (including the 15 genes of the present sample), 22 of the total of 87 placed genes lie on chromosome 2. Group V has 14 and chromosome 10 has 11. Thus, 25 per cent of the total lie on chromosome 2 and 54 per cent lie on the three chromosomes, 2, 10, and V.

Taking the distributions of the total and of the three restricted samples into account, the conclusion is unavoidable that mutant genes of the tomato seem to concentrate on certain chromosomes.

The following causes of the observed deviation might be considered: (1) The selected markers might not detect linkages on their respective chromosomes with equal likelihood. It would be a strange coincidence if the markers were equivalent in this respect. Their screening ability largely hinges upon their position. A few tomato chromosomes have been sufficiently explored to permit selection of favorably located markers (8, 10, V). The marker for chromosome 2, di, is less favorably situated, for, according to the latest revised map, only 2 genes have been detected in 11 units to its left, but 13 are known in the 66 units to its right. Position of the marker gene would therefore not tend to discriminate in favor of this chromosome except for the use of Wo to detect the linkage of ff.

(2) Tomato linkage groups probably differ in their total lengths. Visible pachytene lengths vary from 22.5 to 52.0 μ, but information concerning length of linkage maps is still very inadequate. Total pachytene length or achromatic length is weakly correlated with map length. By definition, chromosome 2 has the second longest pachytene length and in map length (77 units) is third longest. On the other hand, the longest linkage group must reside on one of the shortest chromosomes (No. 10) according to trisomic tests. Greater physical length might therefore account for at least part of the observed disproportion.

(3) Tomato genes might be disproportionately distributed regardless of chromosome length. The present linkage map for chromosome 2 offers support for this hypothesis, for at least four genes (Wo, Me, Cu, and mas) are tightly clustered near the middle of the group. Noteworthy is the presence of three dominant genes in this cluster, only 12 of the 87 mapped tomato genes being dominant.

Disproportionate gene distribution is nothing new in genetics. The high concentration of genes on the left end of the X chromosome of Drosophila melanogaster has been known for a long time. Another example is furnished by chromosome 9 of maize, the great majority of whose thirty-odd genes are concentrated in the short arm, comprising only one-third the total cytological length.9

Although the evidence for non-random gene distribution is abundant, convincing explanations are less readily found. For the tomato in particular, the question might be framed: "Why is one-fourth of the mutant genes concentrated on one of the 12 chromosomes?" The fact that chromosome 2 bears the only nucleolar organizer of the tomato set seems to have no particular bearing on this problem for accumulation of genes has not been found on the nucleolar chromosomes of maize,10 barley,11 or peas.12

The consistent grouping of duplicate genes in maize has been proposed by
Rhoades\(^9\) as evidence of duplication of chromosomal segments, which would result in apparent unequal gene distribution. Within such groups recessive mutations would be masked by duplicates in some other part of the genome. For the tomato, to the contrary, the following evidence argues against the presence of extensive duplications: (1) intolerance of chromosomal unbalance; (2) sharply defined monogenic segregations and rarity of duplicate interactions; (3) failure of chromosome pairing in haploids.\(^1\)

The concentration of genes in specific regions might reflect a tendency toward increased mutability. Equally plausible might be selection for the grouping of genes (or reduction of crossingover between them) that function well together. Although the known crossover rates are too high to preserve large blocks together indefinitely, the tendency for genes to concentrate in specific regions, as in the vicinity of \(W_0\) on chromosome 2, might be advantageous. Mather\(^13\) has called attention to the opposing selective values of linkage to preserve intact favorable groups of genes and recombination to increase variation. It is possible that, to compensate for the relatively high chromosome number of the tomato, the unequal distribution has evolved as a means of preserving balanced gene groups.

Whatever the cause or causes of this unusual condition, it has serious consequences upon the use of the tomato in genetic and breeding investigations. If the observed non-random distribution represents the status of the total germ plasm—a conclusion supported by Currence's\(^7\) data—it would have a net effect of reducing chromosome number and increase in the probability of linkages. The transfer of monogenic characters from one genotype to another should ordinarily involve no serious difficulty, as modern experience in tomato breeding testifies. But with quantitative characters the problem of unfavorable linkages is intensified. The integration of several earliness genes in Currence's\(^7\) example from one parent into the genotype of another without shifting many adjacent genes would present a formidable problem.

If the picture presented here is typical of spontaneous mutation, it would be of interest to learn how artificially induced mutations are distributed. Toward this end, work is now in progress to test the linkage relations of a series of new x-ray-induced tomato mutants produced by Stubbe.\(^14\)

**Summary.**—In a sample of 18 spontaneous seedling mutants of the tomato, 6 were found to be located on chromosome 2. According to a binomial test, this distribution is non-random. For reasons presented the deviation does not likely result from the use of favorably located marker genes or from the presence of duplicated segments but probably reflects greater physical length and especially higher genetic activity of chromosome 2.

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ON THE RADICAL OF A POSITIVE SEMIRING

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W. Slowikowski and W. Zawadowski\(^4\) defined a positive semiring \(\mathfrak{S}\) to be a commutative semiring with zero and identity in which \(1 + x\) has an inverse, for every \(x \in \mathfrak{S}\). They\(^4\) defined the radical of a positive semiring \(\mathfrak{S}\) to be the intersection of all maximal ideals of \(\mathfrak{S}\). In his review of this paper of these authors, E. Hewitt\(^3\) raised the following query: “It would be interesting to know the relation between the radical \(\text{Rad} \mathfrak{S}\) and the Jacobson radical \(A\) introduced by Bourne.”\(^1\) We are now able to give the following answer:

In our paper on the semiradical of a semiring, Zassenhaus and I proved that the semiradical \(\sigma(S)\) of a semiring \(S\) is the intersection of its semimaximal semimodular left ideals.\(^2\) We recall the definition of a semimodular left ideal. A closed left ideal \(L\) of a semiring \(S\) is called semimodular if there exist elements \(s_1, s_2\) in the \(S\) such that \(s_1 + u \neq s_2 + u(L + \sigma(S))\) for \(u \in S\) and if for any element \(x\) of \(S\) there is an element \(y\) of \(S\) such that \(x + xs_2 + y \equiv xs_1 + y(L)^2\).

It is clear that if \(S\) possesses the elements 0 and 1, as is the case with a positive semiring, then every ideal is semimodular. We simply let \(s_2 = 0\) and \(s_1 = 1\) and semimaximality is merely maximality. Thus the radical \(\text{rad} \mathfrak{S}\) is the semiradical \(\sigma(\mathfrak{S})\).

\(^1\) Bourne, S., “The Jacobson Radical of a Semiring,” these PROCEEDINGS, 37, p. 166 (1951).