DISTRIBUTION OF GENETIC TYPES OF TRANSDUCING LAMBDA PHAGES[1]

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THE transduction of the galactose genes of *Escherichia coli* by bacteriophage lambda occurs via the incorporation into the phage genome of a specific, connected region of the host genome (MORSE, LEDERBERG and LEDERBERG 1956). When it incorporates the galactose genes, the phage also loses some of its own genetic material and becomes "defective"—i.e., unable to form plaques (CAMPBELL 1957; ARBER 1958). Strains carrying such a "transducing phage" (λdg) produce under appropriate circumstances lysates which transduce the galactose markers at high frequency. As the essential features of high frequency transduction (HFT) have become better understood, it has become increasingly clear that the most important outstanding questions are those concerning the mechanism of formation of λdg. These can be answered only by the study of low frequency transduction (LFT).

Low frequency transduction is observed with lysates prepared from *gal*$^+$ cells lysogenic for normal (non-transducing) λ. In practice, one generally induces the culture to lyse with small doses of ultraviolet irradiation, but transduction can be performed even by particles produced from spontaneous lysis (MORSE 1962). Lysates produced by infection of non-lysogenic cells do not transduce. The formation of λdg therefore requires some kind of interaction between the λ prophage and the galactose genes which are closely linked to it.

When does this interaction take place? In principle, λdg might arise either (1) as a prophage in a lysogenic cell, whose descendants will then liberate λdg when they lyse or (2) at the time that the prophage is induced or activated and begins to multiply vegetatively. If λdg does arise as a prophage, the LFT lysates obtained from parallel cultures grown up from small inocula should not contain equal numbers of transducing particles. There should instead be a clonal distribution of λdg among cultures, just as there is for spontaneous mutants (LURIA and DELBRÜCK 1943). Some clonality is indeed apparent from the data of MORSE (1962) and of FRASER (1962). However, FRASER has shown that some and perhaps all of the observed clonality is due to clones of cells which produce transducing phage at a high rate. These cells do not themselves carry λdg prophage, because each clone produces a mixture of different kinds of λdg.

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A more sensitive test for the occurrence of λ*dg* prophage is to examine the distribution among parallel lysates of different *types* of λ*dg*. Each λ*dg* of independent origin has a characteristic buoyant density in a CsCl gradient (WEIGLE, MESELSON and PAIGEN 1959), and can be distinguished genetically by the exact extent of the region of the phage genome which it has lost on becoming a transducing phage. Both properties are stable, hereditary characters of λ*dg* as it occurs in HFT lysates. If λ*dg* prophages are formed during vegetative growth of the donor culture, each type of λ*dg* should be distributed clonally among parallel cultures. In those cultures where one type arose very early and is consequently very numerous, another may have arisen late and be rare. One expects, therefore, that the ratio of one type to another should be very different from one lysate to another, if the lysates were derived from independent single cell isolates.

WEIGLE (1961) has observed fluctuations in the density distribution curves from different LFT lysates, which might be ascribable to such clonality. However, at least part of these differences are due to changes in the relative values of the "peak" and "hump" fractions described by FRASER (1962), which apparently arise by two different mechanisms.

It therefore seemed of interest to examine genetically the λ*dg* derived from different lysates, to see whether significant differences did exist between separate LFT cultures.

MATERIALS AND METHODS

The general methods for the study of galactose transduction with λ, the composition of the media, and the origin of the bacterial strains and phage mutants used have been previously described (CAMPBELL 1959, 1961). We shall recall only those points especially pertinent to the present paper.

Bacterial strains: Three substrains of *Escherichia coli* were employed. Strain C600 is gal^+ and was used for making the donor cultures. Strain W3350 is gal^-. It and a streptomycin-resistant mutant isolated from it were used as recipients. Strain W3350 is a double mutant, gal_1 gal_2, and these two mutations lie in different cistrons, affecting the formation of two different enzymes. Our experimental procedure therefore selects for λ*dg* particles carrying both of these cistrons intact. Other types of λ*dg* are not known, but might in principle occur.

As donors in our transduction experiments, we used doubly lysogenic strains carrying the two prophages λ imm^{λ} and λ imm^{434}, which differ in their immunity specificity (KAISER and JACOB 1957). Our reason for employing such strains was that FRASER (1962 and personal communication) had found that doubly lysogenic strains of this type showed more variation than singly lysogenic strains did in the number of λ*dg* found in parallel lysates. As mentioned above, she later showed that this clonality was explainable on grounds other than the presence of preformed transducing phages in the donor culture, so that in retrospect there is probably no real advantage to our having used a doubly lysogenic donor. At any rate, two double lysogens were isolated from a mixed infection of strain C600 with the two phages λ imm^{λ} and λ imm^{434}. In this infec-

tion, each cell of C600 was infected with an average of 12 particles of λ *imm*$^{\lambda}$ and nine of λ *imm*^{434}. From each such strain, a derivative unable to adsorb λ was isolated by streaking against a virulent mutant of λ and isolating a resistant colony. These two strains (A and B) were used for the experiments of Tables 1 and 2.

Transductions: In order to facilitate the performance of a large number of transductions, certain minor modifications of previous procedures were introduced. Lysates were made as follows: A petri dish containing 1.6 ml of tryptone broth and 0.2 ml 0.01 M $MgSO_4$ was warmed to 37°, and 0.2 ml of an overnight standing culture of lysogenic cells was added. After an additional hour at 37°, the culture was irradiated with a General Electric Germicidal Lamp for 25 seconds, giving a total dose of about 400 erg/mm^2. Following three hours subsequent incubation at 37°, chloroform was added to kill any surviving bacteria, and the lysate was used for transduction.

In low frequency transduction, 0.2 ml lysate was plated with 0.1 ml of a culture of W3350 *gal*$^-$ *str-R* cells (at a density of about 10^9 cells/ml) on an eosin-methylene blue galactose plate containing 100 μg/ml streptomycin. The streptomycin eliminates any donor bacteria not killed by the chloroform. For high frequency transduction, the recipient cells were grown to saturation, centrifuged, and resuspended at ⅓ their growth density in 0.01 M $MgSO_4$. 0.01–0.02 ml of lysate was added to 1 ml cell suspension, and after 20 minutes at 37°, 0.1 ml was plated together with some anti-λ serum on eosin-methylene blue agar. Streptomycin was not added in this case because the donors were already streptomycin-resistant. Surviving donors were never a problem in high frequency transduction.

From the number of colonies seen on these plates, one can calculate, using the appropriate correction factors (CAMPBELL 1957; ARBER 1958) that a typical LFT lysate from strain A contained about 2×10^3 λ*dg* per ml, and from strain B about 2×10^4. Typical HFT lysates derived from strain A had a λ*dg* concentration of about 5×10^6 per ml.

Scoring of λdg *types by crossing with phage mutants:* Each λ*dg* is characterized by the position of the left-hand endpoint of the deleted region of phage genes (CAMPBELL 1959). This position can be determined by crossing the λ*dg* with each of a collection of suppressor sensitive (*sus*) mutants. These mutants fail to form plaques on strain W3350. Since λ*dg* does not form plaques either, the combination can generate plaque-forming recombinants only if the λ*dg* carries the wild-type allele of the *sus* mutant used—i.e., if that mutational site is outside of the deleted region. To do such a cross, one places on a background layer of W3350 a drop of lysate from a *sus* mutant, together with a drop of culture carrying the λ*dg* as prophage. Then one induces with ultraviolet and looks for the formation of plaques following induction.

In order to perform the test, one must have a culture which is lysogenic *only* for λ*dg* prophage and does not carry a normal prophage as well. To obtain such cultures, one must transduce under conditions of very low multiplicity (CAMPBELL 1957). This is easy to do with HFT lysates, but with LFT lysates the fre-

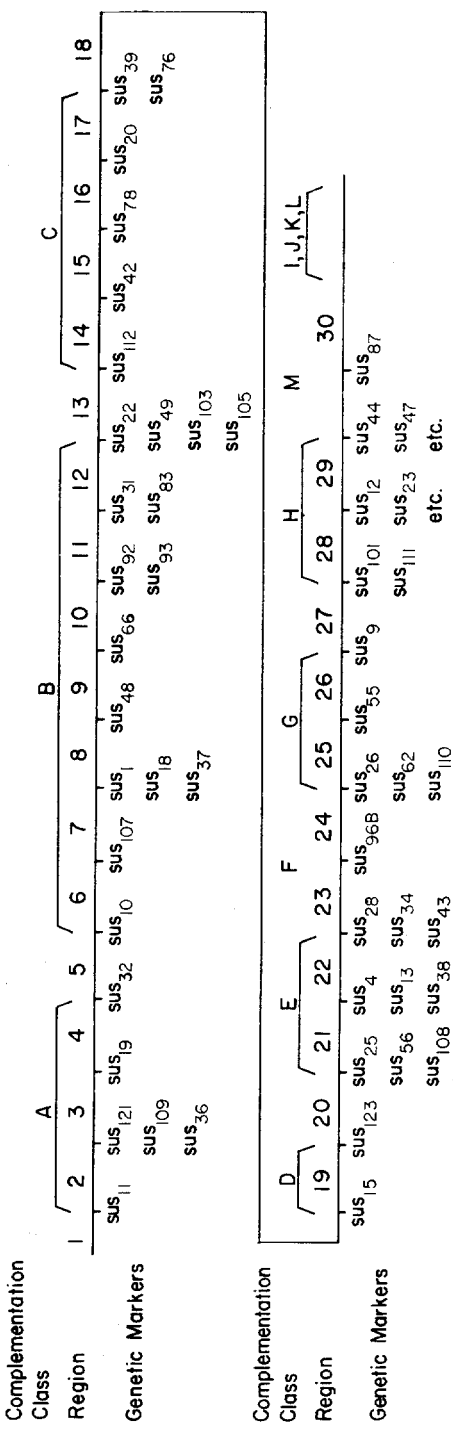

FIGURE 1.—Genetic map of the left-hand end of the λ chromosome, based on observed deletion endpoints of λ*dg*. The λ*dg* deletion may terminate in any one of regions 1 to 30. All genes to the left of that region are present in λ*dg*, and all those to the right are absent.

quency of transduction is too low to make it feasible. Therefore, almost all the products of LFT transduction carry an active, non-transducing λ in addition to λ*dg*. In order to study the type of λ*dg* carried by each such strain, one must prepare a HFT lysate from it, transduce a suitable recipient at low multiplicity, and examine the defective lysogenic transductants from each. A few transductants (usually four) made with each such HFT were tested, and those which proved to be defective lysogens were examined genetically by crossing them with *sus* mutants. Since all the λ*dg* in a HFT lysate are identical, each HFT lysate is represented by a single entry in Tables 1, 2, and 4.

RESULTS

Genetic map of λ: Figure 1 shows the genetic map of the left-hand region of phage λ, based on all the λ*dg*'s tested in this paper. For simplicity, not all the markers of class H are shown. Each λ*dg* terminates at some point along the linear map, which is thereby subdivided into 30 different regions. Each λ*dg* isolate is characterized by the region into which its endpoint falls. All the genetic sites to the left of this endpoint are present in the λ*dg*, all those to the right are absent. Obviously, two λ*dg*'s which are identical by this criterion might turn out to be different if still more genetic markers were available.

Figure 1 disagrees with our previous map (CAMPBELL 1961) at two places. Since deletion mapping will not tolerate exceptions, we must explain the discrepancies. The first case (sus_{37} and sus_{48} in Class B) was a tabulational error. The previous data agreed with the order found in the present study, but a mistake had been made in transcribing them. In the second case, the marker sus_{38} of Class E had been misplaced. This lysate contained rather many spontaneous reversions, so that scoring had been difficult.

Throughout this and previous work, we have kept in stock each type of λ*dg* which dissects the map in a new place. Whenever an apparent contradiction with previous data is found, all the critical strains are re-examined. This has always resulted, as it did with λsus_{38} in finding some mistake in the scoring, either of the old or of the new results, never in discovering a violation of the linearity of the linkage map.

The present data extend the map further to the right than was previously possible, and show that sus_{87}, which is the one mutant comprising Class M, lies to the left of classes I, J, K, and L, which remain unmapped. sus_{87} has not been separated from mutants of class H such as sus_{44}, but presumably it lies to their right.

Distribution of types of λdg *of independent origin:* Before examining the composition of particular LFT lysates, we first looked at the distribution of types of λ*dg* of *completely* independent origin. A culture of the donor bacteria was streaked out, and different colonies were grown up and induced. One transductant produced by infection of the W3350 *str-R* strain with each such LFT lysate was purified, grown, induced and used as a donor for high frequency transduction. The resulting defective transductants were tested for the extent of the deletion they carried by crossing each with a series of *sus* mutants. In

TABLE 1

Distribution of transducing phage from Strain A

Experiment	Region 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	Total
41	**4**	0	2	1	1	0	0	0	3	1	0	1	**12**	1	0	1	0	**7**	0	0	0	0	**33**	**4**	0	1	0	1	1	0	74
48	**6**	3	0	2	1	0	2	0	0	1	1	0	**9**	0	1	2	1	**4**	2	1	0	1	**21**	**4**	0	1	0	2	0	0	66
Total	**10**	3	2	3	2	0	2	0	3	2	1	1	**21**	1	1	3	1	**11**	2	1	0	1	**54**	**8**	0	2	0	3	1	0	140

The distribution of λ*dg* types among the major classes (shown in boldface type) is not significantly different between the two experiments ($\chi^2=5.40$, $P=0.38$).

TABLE 2

Distribution of transducing phage from Strain B

Experiment	Region 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	Total
42	2	0	0	0	1	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	**10**	0	0	0	0	0	0	0	15
49	2	0	0	1	0	0	0	0	0	0	0	0	1	1	1	0	0	0	1	1	0	0	**1**	0	0	0	0	0	1	0	10
Total	4	0	0	1	1	0	1	0	0	0	0	0	2	1	1	0	0	0	1	1	0	0	**11**	0	0	0	0	0	1	0	25

The distribution of λ*dg* types between Class 23 and the minor classes is significantly different between the two experiments ($\chi^2=7.9$, $P<0.01$).

this way, we obtain a distribution of types of transducing phage which cannot be biased by clonality, because each HFT lysate is made from a different LFT lysate, which was prepared from the descendants of a different *gal*+ cell. The results, shown in Tables 1 and 2, therefore give us a control distribution for the fluctuation test performed later (Table 4).

In each of the four experiments (41, 42, 48, 49), we started with 100 individual colonies of the donor culture. The complete experiment was not done with all of these for the following reasons: (1) In some cases, the colony picked no longer was doubly lysogenic. These colonies were discarded. (2) Approximately one third of the transductants in low frequency transduction are haploid rather than heterogenotic for the *gal*+ character. Since we picked only one transductant from LFT, if it did not produce an HFT lysate the clone was discarded.

The breakdown of numbers of colonies discarded for various reasons is given in Table 3. We see that the two donor strains A and B differ enormously in the stability of the doubly lysogenic condition. That this was a property of the strain and not just of the colony used for the original restreaking, is indicated by experiments 48 and 49, where we used a doubly lysogenic *subcolony* of strains A or B respectively.

The data of Tables 1 and 2 cannot be directly analyzed by the method of χ-square because of the large number of classes of infrequent occurrence. We have therefore lumped the rare classes together into a single class for the purposes of calculation. Each of the columns used as a separate class is shown in boldfaced type. The remainder, shown in ordinary type, were considered rare classes and were lumped. It can be seen that there was no significant difference between experiments 41 and 48, but a highly significant difference between experiments 42 and 49.

Strain B thus showed some fluctuations, but not at the stage where we might have expected to find them. Inasmuch as each transductant is here derived from a separate donor, it is impossible to attribute the difference between experiments 42 and 49 to the existence of clones of λ*dg* prophage in the donor culture. One must rather postulate classes of cells with a hereditary tendency

TABLE 3

Summary of all colonies tested in experiments 41–49

Type of colony	Experiment			
	41	48	42	49
Tested in tables 1 or 2	74	66	15	10
Singly lysogenic for λ*imm*λ	1	5	1	33
Singly lysogenic for λ*imm*^{434}	6	6	78	56
Non lysogenic	0	1	0	0
Lost through accident	0	1	0	1
Haploid transductant from LFT	19	21	6	0
Total	100	100	100	100

TABLE 4

Distribution of transducing phage among LFT lysates (Experiment 83)

Lysate	Region 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	Total
I	**2**	0	1	**5**	**1**	0	0	0	0	0	0	0	**8**	0	1	2	0	**6**	**0**	1	0	0	**6**	**0**	0	1	0	0	0	1	35
II	**0**	0	0	**9**	**0**	1	0	0	0	0	0	0	**2**	0	2	2	0	**3**	**2**	1	0	0	**7**	**0**	0	1	0	0	0	0	30
III	**5**	0	1	**7**	**3**	1	2	0	0	0	0	0	**6**	1	0	0	0	**3**	**2**	0	0	0	**5**	**1**	0	1	0	0	0	0	38
IV	4	2	1	**5**	**2**	0	0	0	1	0	0	0	**7**	0	0	1	0	**3**	**0**	0	0	0	**11**	**1**	0	1	0	0	1	0	40
V	**1**	0	0	4	**2**	0	0	0	0	0	0	0	**6**	1	1	1	0	4	**2**	2	0	0	**5**	**3**	1	1	0	0	0	0	34
VI	**3**	0	1	**0**	**4**	0	2	0	0	0	0	0	**1**	0	0	0	0	4	**2**	1	0	0	**8**	**1**	0	1	0	0	0	0	28
VII	**0**	0	1	**1**	**1**	0	1	0	0	1	0	0	**3**	0	1	0	0	**1**	**0**	0	0	0	**2**	**2**	0	0	0	0	0	0	14
VIII	**3**	0	1	**2**	**0**	0	3	0	0	0	0	0	**5**	2	0	0	0	**1**	**0**	0	0	0	**1**	**1**	0	1	0	0	0	0	20
IX	**2**	0	0	**3**	**1**	0	0	0	1	0	0	0	**5**	1	1	2	0	**8**	**2**	1	0	0	**7**	**2**	0	0	2	0	0	0	38
X	**8**	0	0	**3**	**3**	0	0	1	0	0	0	0	**3**	0	0	0	0	**5**	**0**	1	2	0	**6**	**0**	0	0	0	0	0	0	32
Total	**28**	2	6	**39**	**17**	2	8	1	2	1	0	0	**46**	5	6	8	0	**38**	**10**	7	2	0	**58**	**11**	1	7	2	0	1	1	309

The distribution of λdg types among the major classes (shown in boldface type) is not significantly different among the ten lysates ($\chi^2=83.2$, $P=0.17$).

to produce a particular type of transducing phage from active phage. As Strain B was highly unstable, it seemed possible that some sort of rearrangement of the prophages might have intervened which would affect the types of λ*dg* most readily formed. At any rate, Strain A clearly seemed more suitable for the further experiments we wished to perform.

Distribution of types within separate LFT lysates: In order to detect any clones of λ*dg* in the donor culture, the following scheme was devised: A colony of Strain A was restreaked, and ten doubly lysogenic subcolonies picked. Each of these was grown up and induced to give a LFT lysate. Each lysate was used to transduce the *gal*$^-$ *str-R* recipient, and 50 transductants coming from each lysate were purified by restreaking. Of the transductants thus produced, some were heterogenotes and others were haploids. Each of the heterogenotes was induced, and the resulting HFT lysate was used in a further transduction to produce defective lysogens which could be tested genetically.

We thus have ten donor cultures (I–X) for LFT, and for each a number less than 50 of λ*dg* produced from it. The results are presented in Table 4. As before, for statistical analysis we lump all classes shown in ordinary type. This gives a table with ten rows and nine columns, and hence 72 degrees of freedom. If we test the hypothesis that the ten groups are random samples from the same pool, we find $\chi^2 = 83.2$ and $P = 0.17$. There is thus no significant evidence for clonality.

If we compare the total data of Table 4 with those of Table 1, we find (using the six classes of Table 1) that $\chi^2 = 24.5$, $P < 0.01$. So there is a significant difference between the behavior of Strain A in the two experiments.

Assortment of immunity markers: In presenting Tables 1, 2, and 4, we have not subdivided the data according to whether the λ*dg* tested was λ *imm*$^\lambda$, or *imm*^{434}. It is of some interest to ask how these markers do assort.

In experiments 41 to 49 (but not Experiment 83), we tested the immunity specificity also of the products of low frequency transduction. Almost all of these (156/165) were immune to both λ *imm*$^\lambda$ and λ *imm*^{434}. Since almost all the transductants are produced by simultaneous infection with a λ*dg* and an active λ, one expects that these will sometimes have been of opposite immunity types, but the very high proportion observed probably indicates that there was a great deal of superinfection on the plates.

In all experiments we have tested the immunity specificity of the defective heterogenotes produced in high frequency transduction. These have one immunity or the other, never both. Within any one experiment, the distribution of endpoints among those λ*dg* which are *imm*$^\lambda$ has not differed significantly from those which are *imm*^{434}, which justifies our pooling the data in the table. This does not necessarily mean that both prophages can produce λ*dg*, but may simply reflect recombination between the immunity markers and the *gal* genes during phage multiplication.

There have been, however, large differences between experiments in the proportion of the two immunity types found. The data are given in Table 5. The total number of transductants is greater than the total number of λ*dg*'s

TABLE 5

Total number of transductants of different immunity types

Experiment		Strain	Immunity type of transductants $imm\lambda$	imm^{434}
41		A	62	109
48		A	36	112
Total		A	98	221
42		B	9	11
49		B	0	21
Total		B	9	32
83 Lysate	I	A	22	58
	II	A	21	63
	III	A	18	98
	IV	A	19	92
	V	A	13	60
	VI	A	18	42
	VII	A	2	17
	VIII	A	5	33
	IX	A	59	49
	X	A	60	35
Total		A	237	547

tabulated in Tables 1, 2, and 4, because sometimes as many as four transductants derived from the same λdg were tested. The proportions of the immunity markers varied widely among the lysates of Experiment 83, and also between the two experiments with Strain B.

DISCUSSION

The nonrandom distribution of mutants appearing in a series of parallel cultures was demonstrated by LURIA and DELBRÜCK (1943). A more precise mathematical treatment of the theoretical distribution was developed by LEA and COULSON (1949). Instead of a series of n parallel cultures in which a single type of mutant is counted, one can look at a single tube and enumerate each of n different types of mutants. The expected distributions are identical if the mutation rate to each different mutant type is the same. If the mutation rates are different, the distribution is modified in the same way as if one were dealing with a series of parallel cultures of unequal size.

The mathematical derivation of the distribution presupposes that (1) a mutant can arise with equal probability at any time during the growth of the culture, (2) all the progeny of a mutant are mutants, and (3) there is no selective differential between the mutant and the parent. In principle, it could thus apply to the number of individuals carrying a particular type of transducing phage, were it true that transducing phage arises as prophage.

Our experimental result is a negative one. No evidence was found for a varia-

tion in the proportion of types among parallel cultures. We cannot of course assert from this that clones of λ*dg* prophage do not exist. It is relevant, however, to inquire how large are the fluctuations we might expect to see under the conditions of our experiment.

LEA and COULSON have shown that, in the limiting form of the theoretical distribution, the quantity

$$\frac{11.6}{\frac{r}{m} - \ln m + 4.5} - 2.02 \qquad (1)$$

is distributed approximately normally with unit variance, where r equals the number of mutants in a particular culture, and m equals the mean number of mutations which have occurred per culture.

We wish to calculate whether, under the conditions of our experiment, the variation of r is sufficiently great to cause noticeable departures from equality in the proportions of different mutant types. The calculation is not strongly influenced by the exact value of m, only by its order of magnitude.

The proportion of λ*dg* particles in a LFT lysate is in the range 10^{-5} to 10^{-4} (FRASER 1962). Since each cell which yields λ*dg* produces an equal number of active phage (approximately), the proportion of such cells would be twice this. Let us therefore assume that the fraction of cells which carry λ*dg* is 10^{-4}. To accumulate such a fraction in a population which has grown up from a small inoculum to, say, 10^9 cells, we must have a mutation rate of about 10^{-5} per cell per generation. The value of m would then be 10^4. Since we are dividing the data into nine classes, the mean number of mutations *to each type* would be about 10^3.

LEA and COULSON'S approximation (1) is quite good for the middle of the distribution, from about the tenth to the 90th percentile, and becomes less reliable at the ends. If we calculate these two percentiles, we get a range into which 80 percent of the values of r/m will fall. This corresponds to $(r/m) - \ln m = -0.9$ and $(r/m) - \ln m = 11.2$, which, for $m = 10^3$, becomes $r/m = 6.0$ and $r/m = 18.1$—i.e., even restricting ourselves to the middle 80 percent of the distribution, the number of a particular type of mutant can vary three-fold. The distribution is very skewed, so that much higher values of r/m are still not too uncommon.

It therefore would be quite possible to see the fluctuations caused by a clonal distribution in an experiment such as the one we have performed. Unfortunately, with only ten cultures to examine, it is also quite possible that no large fluctuations happened to occur. The performance of still larger experiments seems quite uncalled for, however, in view of our finding that a cell line can change the distribution of types it produces, as between experiments 42 and 49 with Strain B, or Tables 1 and 4 with Strain A. This implies that, even had fluctuations been observed, we would have been unjustified in attributing them to clones of λ*dg* prophages in the donor culture. Their absence would suggest that λ*dg* does not arise as a prophage, but rather at the time of or subsequent to the induction of the donor strain.

The variation in the distribution of λ*dg* types produced by a given strain is an interesting phenomenon in its own right. It is actually entirely ascribable to a

change in the frequency of the most common type of λ*dg*—those whose endpoint is in Region 23. If this class is omitted, the remaining differences are not significant; this may just mean that changes in the frequency of the most common type are most likely to be detectable.

In the case of Strain B, the change in λ*dg* distribution was accompanied by a significant alteration in the proportion of the two immunity types, but this is not true for Strain A. By contrast, the various lysates of Table 4 are highly disparate in the proportion of λ*dg* of various immunity types, although the endpoint distribution is unchanged. There seem, therefore, to be several hereditary states for the prophages in a doubly lysogenic strain such as this one. The fact that several cases have cropped up in an experiment of this size suggests that the transitions between states must be rather rapid.

It is also possible that the difference between two groups of data such as those of experiments 42 and 49 is attributable to environmental factors rather than to any hereditary difference between the original colony of Strain B and the subcolony used for Experiment 49. We do not favor this interpretation. We think that environmental variations are more likely to influence the absolute number of variants than the distribution of types among them, and hence, that fluctuations in the proportions of different types are more significant than those of absolute numbers. However, this is purely a matter of opinion at present.

SUMMARY

The distribution of different types of λ*dg* transducing phages has been determined in one manner which would, and another manner which would not, be affected by the existence of clones of cells carrying λ*dg* which had arisen in the prophage state. The results show no evidence of clonality, but indicate that the distribution pattern given by a particular strain can change.

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