Luria

Second Revision

Proposal on Nomenclature

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March 14, 1946

A. Virus Strains

From the original PC stock of Bronfenbrenner, Kalmanson isolated from a single plaque his PC. This will be called T2K. From this Luria and Delbrück isolated in 1942 from a single plaque their gamma. This will be called T2L. T2 without qualification shall mean T2L.

From the original Bronfenbrenner PC Hershey isolated by picking a single plaque his "P9H", characterized by the fact that it is less well adsorbed. This will be called T2H.

These three strains are presumably mutants of one stock. They are differentiated by host range.

Hershey has one other stock, serologically related but of independent origin. This will be called T 16.

These four strains are differentiated by host range. Hershey has isolated the following indicator strains:

B/2H
B/2K, 2H
B/2L, 2H
B/6, 2K
B/2L, 2K, 2H

The last mutant of B is the one most commonly found.

B. "r" Mutants

All these "wild-type" strains show <u>lysis inhibition</u> in the adsorption tube. From all of them can be isolated a mutant without lysis inhibition (or with much less lysis inhibition). These mutants without lysis inhibition also give bigger plaques, or at least plaques with a clearer halo. These mutants will be designated as "r" (for "rapid lysis in the adsorption tube"). Thus T2Kr, T2Lr, etc. If it is desired to indicate specifically that one is referring to a wild type stock reasonably free of the "r" mutant, one should write $T2K+^{r}$, $T2L+^{r}$, etc.

No appreciable difference has been found in one-step growth experiments between "+r" and "r" strains.

No serological difference between 2 L, 2K, 2H has been found.

T4, T6, T 16 are different from the above (Hershey).

T4 differs from T6 (Delbrück).

There has never been found a host range difference on mutants of B between any $+^r$ and its corresponding "r". However, the efficiency of plating (e.o.p.) may differ for a "$+^r$" strain and its corresponding "r" mutant. Luria has found a host range difference between T2$+^r$ and T2r, on two strains of dysentery, V 75 and V Weil.

Spontaneously only the mutation from "$+^r$" to "r" seems to occur, i.e., one can pick the "r" mutant from almost all seemingly pure wild-type stocks, while the "r" stocks seem to stay pure. (Induced mutations occur in both directions).

C. Host Range Mutants

By plating a virus strain on a bacterial strain resistant to it an "h" mutant is picked up. This resistant strain may be either a mutant of the original host, or an independent bacterial strain. Both T1 and the even viruses give two types of "h" mutants, namely,

h^t (t for turbid), forming turbid plaques on the new host, and also having an e.o.p. on the new host only about 20% of the e.o.p. on B.

h^c (c for clear), forming clear plaques on the new host, and having an e.o.p. on the new host close to their e.o.p. on B.

(The distinction between plaques of h^c and h^t can only be made on layer plates, not on spread plates).

The h^c and h^t mutants are fairly stable. T2H on B/2H throws the two types with about equal frequency. T2K and T1 throw h^t much more frequently than h^c.

(Luria feels doubtful, from his results, as to the validity of the distinction between h^c and h^t mutants).

In some cases it may be necessary to indicate on which host the h mutant was isolated. This should be done by adding the name of the host in brackets, thus, for instance:

T2Hn^{t} (B/2, 2H)

The wild type should again be designated, if necessary, by "$_{+}{}^{h}$".

The "r" locus and "h" locus mutate independently.

Whether n^{c} and h^{t} represent independent mutations has not yet been decided.