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CLEVELAND 6, OHIO

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SCHOOL OF MEDICINE
DEPARTMENT OF MICROBIOLOGY

November 9, 1964

Dr. Joshua Lederberg
Department of Genetics
Stanford University Medical School
Stanford, California

Dear Dr. Lederberg:

In my laboratory we have become interested in why K12 bacteria carrying lambda prophage do not support the growth of T4rII bacteriophage, whereas the absence of prophage allows growth of the T4rII. One question that we have asked is whether the difference between K12(lp⁺) and K12(lp⁻) is attributable to expression of a lambda gene or to inhibition of a gene on the bacterial chromosome. It is possible that a meaningful answer might be forth coming if we could obtain a strain of bacteria that has lambda on the host chromosome and an episome with those host genes that neighbor the lambda prophage site. Hirota and Sneath described several F^I particles with different markers. It would appear that episome F₈ or F₁₃ state I (high fertility for gal, try) would be logical episomes to use. Since the work was done in your laboratory I wonder if you have retained these strains and whether you could send them to me.

One preliminary answer I would need to know or examine is whether a strain marked lp⁺/F^Ilp⁻gal⁺ could be obtained and maintained long enough to test it as a host for T4rII or whether the lambda gets on the episome so rapidly that both host chromosome and episome contain lambda. If there is a reasonable time interval during which the episome expresses and does not contain the prophage, I would plan to use a K/4 as episome donor and F⁻(lp⁺) as acceptor. At varying times after mixing the two cultures I would infect with T4rII and look for progeny phage.

It is entirely possible that similar experiments have been done with negative results. If you have heard whether this is so I would appreciate this information.

Sincerely yours,



Lazarus Astrachan

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