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February 11, 1953

Dear Dr. Hayes:

Your letter of the 25th reaches me at a time when I am ,in fact, precoccupied with another matter, so that I will not be able to study it until I return to Madison shortly.

May I comment briefly on the plans for the CSH symposum? It would not be either profitable or feasible for us to prepare a critique of your "empirical and provocative" paper, so you assume a free hand and need not worry (as you would not in any case) about an unkind discussion. On the other hand, the points that you ask permission to quote will be reviewed to the extent that they will be ready for publication in our own brief paper, which will have to do with the untangling of transduction and lysogenization, especially in E. coli. It would be better on the whole, I think, if your references to our work were confined to the published data, as these are all that I am sufficiently certain of now.

Re your last questions, thus my views have been pretty well stated in that recent paper in Genetics. Until we know a good deal more about both F+ and the mechanics of recombination in K-12, the statement of F+ being possibly a genetic vector may have unsuspected meanings. As far as I can see now, we have evidence only of whole cells. As to the transmission of defective genotypes, the most pertinent evidence comes from diploids, and the only defects we have ever seen have involved the Mal-S segment. We did not for example find Mtl v Lac-, except for types that proved to be homozygous diploid Lac-/Lac-.On the other hand, we have occasionally gotten complete diploids (even with respect to Mal-S). I will not say that defective gametes can never occur, but complete ones certainly do. All this is old hat now, and most of it in the 1951 CSH. As I have said before, however, I will be surprised if the final answer does not prove us somehow both right in some way that is not yet obvious.

I do not have the references here, but understand you have been interested in the XII somatic antigen of Salmonella. If you still have them, would you be kind enough to send me reprints? This antigen, or some fraction, seems to be the receptor for our transducing phage PLT22.

Yours sincerely,

Joshua Lederberg