

August 1, 1955

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Dear Dr. Demerec:

I have only just now had an opportunity to see your paper in the Proc. of the 9th Int. Congress of Genetics. A small error crept into your account that I think you would want to know about, for the record. It concerns the genetic control of phage resistance.

In my 1947 Genetics paper, I mentioned at least three T1-resistant phenotypes. As I believe is clearly implied there, these are distinctly non-allelic. In particular, the "/1" types have always been definitely at different loci from the "/1,5" types. There may be more than three loci, but there are at least three, which show several percent of recombination among them. Unfortunately, in K-12 (as ~~in~~ spontaneous mutants) we never did find any coincidental auxotrophs: I would be willing to bet that these are non-allelic to the others. However, my wife has recently found that  $Lp_2^r$  is the same mutation as  $Mal_1^-$  (this is resistance to lambda-2 phage.) I am looking forward to reading Bryson's comment on the same topic.

With regard ~~to~~ to pleiotropy at the S locus, I must of course agree as to the allelism of the  $S^r$  and  $S^d$  mutants that have been intercrossed so far. I am less convinced that the other effects are at the same locus, which requires a backcross test that has not, I believe, so far been reported.  $S^d$  mutants, especially are subject to considerable selective pressure while they are being isolated and carried, and I suspect they accumulate many additional mutations in the course of handling.

To return to the T1-resistance, one apparent case of pseudoallelism turned up there, and was the basis of discovery of a nondisjunctional heterozygote, as reported in my 1949 (PNAS) paper. I have not studied " $V_1^P \times V_1^R$ " very extensively, but have not found any recombinants in some hundreds of tests. The heterozygous compound was, however, sensitive to T1, so that there is no cis-trans position effect in this case.

Yours sincerely,

Joshua Lederberg