

My dear Bill,

I have received, and not answered for a while, your letter of the 27th November. I was very pleased to hear that the various amendments have proved possible. O.K. for the various alterations you proposed; the one on ~~the~~ table<sup>2</sup> is exactly as it looked, I think, on the table I sent ~~to the Editor~~ with the manuscript which was not understood by the printer. Re your questions : (1) on page 94, (#8) refers to strain No. 8 of my collection. It is obviously of no significance to the reader, except that it is the strain on which I spent the last three months and may have to refer to this point <sup>in papers</sup> later. Would the symbol # be understandable to readers as meaning "strain No." ? I thought it were a customary symbol, at least in the American literature. Substituting <sup>with</sup> the longer paraphrase would mean altering all the successive lines. There would be no great harm in deleting (8) altogether, if (#8) makes no sense to most readers. (2) Reference for Davis : B.D. Davis, Studies on nutritionally deficient bacterial mutants isolated by means of penicillin, Experientia, 6:41-50 (1950). (3) Reference to my paper, 1952 : Genetic analysis of drug-resistance, Bull. World Hlth Org. 6:185-206. Did ~~he~~ send you an offprint ? if not, one is enclosed.

Work: I have lost the last month looking for a presumptive linkage of my  $F^R$  (F-refractory) with methionine. A preliminary test had indicated the possibility of linkage; i.e., crossing #8 which is M- with  $TLB_1-S^R$  on minimal +st+methionine, most recombinants are M- and they seemed to be ~~mainly~~ all  $F^R$  (at the boundary of significance). I have therefore enlarged the data conspicuously.

ly, and now seen that linkage with M- does not exist. F<sup>r</sup> is still therefore independent of all markers tested, though I must wait for some more data coming on M-, and the next step I shall try is resistance to colicine E, as this also seems independent of the other markers, and perhaps independent of F+ polarity, according to data published by Fredericq, <sup>coupled with mine</sup> Another waste of time has been the analysis of a new F-agent, which seems to be different from the F of K-12 in that it can infect my F<sup>r</sup>. I have no conclusive evidence that this is true. Recombination with the new F-agent happens at a too low rate to be of real use. I am sorry, because I hoped to test whether a different F-agent would determine a different pattern of segregation, i.e. a different probability that, given ~~of the F+~~ markers reaching the F- cell. I have still a hope that the low fertility ~~this~~ be due to instability of the F-agent.

How is your work going? Yours ever