

Milan, 16/12/51

My dear Lederberg,

Your experience of mine as a correspondent will, I hope, help you to understand that if I have not replied earlier to your letters, and ^{to} the sending of your most exciting CSRS paper has not been a consequence of my being dead in the meanwhile, or of my having dropped bacterial genetics. Neither of these things is true, and, on the contrary, K 12 is still consuming most of my time, even if the later discoveries in your laboratory may have made of it a rather obsolete object for research. I have also had, from the editors, a copy of Microbial Genetics, and was glad to see among ^{the} major papers our letter to Nature. I have ~~been~~ doing a great deal of work on the same subject - two papers on it are nearly ready - essentially with a view to confirm ^{the} experiments reported in the letter to Nature, and to test ^{the} spontaneous origin of mutations in the case of chloromycetin resistance by methods other than the fluctuation test - or complementary to it. The result with which I was most pleased was the creation of a second step resistant by recombination, on drug free media, ^{between} ~~at~~ two independent and non-allelic first steps. A puzzling result, on the other hand, was that no two allelic independent first steps were obtained; although ^{the number of} the number of those tested was not great, it seems likely that ^{the number of} genes involved in such resistances is rather high. Also, interactions may be rather complex; however, I have left a detailed analysis of this or similar cases for times when the mating and the genetic system of coli will be more fully elucidated. I have spent a rather long time on this, as I happened to be interested in ~~the~~ biometrical genetics - in spite of its unpopularity between geneticists - since a long time. Similar results were obtained with terramycin resistance; ~~the~~ which could be easily expected especially in view of the fact that chloromycetin ~~the~~ resistant organisms are easily resistant to terramycin, and viceversa.

Since some months I am back to work on maps. Outcrosses of 58-161 and W 677 (or related strains) ~~had~~ to W 826 and W 836 had shown segregations which, although not easily understood, were in favor of a chromosome mutation having been induced either in B-M- or T-L-B₁*. By the way, although I have often been using, after your paper, the B-M- designation, I have never found a trace of the biotinless gene; it must have back mutated early in my strains.

Out of the many methods which I have tried to obtain strain

* now probably in the letter

