October 7, 1953

Dear Dr. Rizet:

We have been following your work on Podospora with the greatest interest. I hope you will continue to favor me with reprints dealing with it.

Our labo. group recently held a seminar, during which some questions came up that I ask to bring to your attention. I would not exclude the possibility that they have been dealt with in your two reviews (Rev. Cytol. Biol. Veg. 1949 and 1952), and I hope that, if so, you will forgive our overlooking this.

In connection with the formation ~~of spores~~ typically dikaryotic +/-, should any serious attention be given to the possibility that we have, in this case, a regular (atypical) reduction of the centromere at the first division. Lindegren once suggested that a small para-centric inversion might so interfere with regular synapsis as to lead to such a precocious reduction. [Such an inversion might also prevent crossing-over between the centromere and the marker]. This hypothesis is, a priori, no less attractive than the assumption of a regular, single crossover near the centromere. The only criterion I can visualize at the instant would be the behavior of any marker on the "sex"-chromosome which did show regular first-division reduction, showing that the centromere (or some point at least) must do the same.

We were especially interested in the barrage results [e.g. in view of possible connections with the infective F+ factor in E. coli:: I hope Professor Ephrussi will have forwarded reprints to you addressed under cover to him]. If I understand your conclusion, it is that the s^S produced from crosses of S x s obtain from the passage of some "plasmid" from S to s. However, you note that the result is the same regardless of the sexual polarity (with respect to ascogonia/spermatia) of the cross, while the results of s^S x s are affected by this polarity. I note however that you emphasize that it is the issue of the Ss heterozygote whichmay show the s^S type, so perhaps I have oversimplified your conception. It appeared to me that the induced reversion effect of s on s^S would be much more readily compatible with a slightly different scheme, your views on which [if not already given] would be of considerable interest here:

Let us assume that it is s (rather than S) which carries a plasmid ø, and that ø is in a sense essentially inviable in the presence of the S gene. The sS genotype would then differ from the original s in completely [or in view of occasional spontaneous reversions] almost completely] lacking ø. This might be comparable to the relationship of kappa not to K but to other "sensitivity genes" in Paramedium. Alternatively, S might carry an alternative plasmid $ which competes against ø in a S- genotype, but this is a needless multiplication pf particles. To explain spontaneous reversion, one must assume either a de novo initiation of ø from another source, or its persistence at a very low level. Induced reversion would be simply the "infection" of s(lacking ø)with ø. One could then state that barrage results from the confrontation ofhyphae carrying ø and S respectively.

Yours sincerely,

Joshua Lederberg