Gareu

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DEPARTMENT OF GENETICS
COLD SPRING HARBOR, LONG ISLAND, N. Y.

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February 22, 1955

Dear Lu,

No the manuscript I promised did not get lost in the mail. It and the parents are suffering from a prolonged labor. You remember that the most critical experiment for our conclusions involved growing UV phage on UV cells and plating infective centers:

Our interpretation was based first of all on the assumption that the live cells were capable of repairing most of the UV damage to the phage (hence its high UV resistance) and that UV to the cells damaged this repair mechanism. The initial slope of the curve then measures the decreased efficiency of repair by those cells whose repair mechanism had received some damages, and the point at which the slope reverts to normal measures the fraction of cells whose repair mechanism still functions normally. If you plot the fraction of cells with undamaged repair mechanisms as a function of UV dose to the cells, the curve is almost identical to the UV inactivation curve for phage plated on live cells — which we interpreted to ~~show~~ show that

The radiosensitive volumes of the two are the same. But unfortunately there is another uninteresting interpretation to these double-UV curves which we have not been able to eliminate. The latent period for UV phage on UV cells is greatly prolonged — longer than for UV phage on live cells or live phage on UV cells. Also when you put unexpected UV cells in broth they retain their ability to produce an infective center with live phage only for about 1 hour and then there is a drop off to about 10% infective centers if you wait at 2 hours. So is UV really hitting a "repair mechanism" in the cell, or simply setting up some unbalanced growth (such as Cohen finds with his thymine mutant) which eventually eliminates the infective center if the latent period becomes too prolonged?

Now our theory goes even one step further. It not only assumes some cellular repair mechanism, but goes on to say that the repair is genetic (instead of "physiological") due to exchange between homologous regions of phage and cell chromosomes. Well we don't really have any critical data to support genetic vs. physiological repair, although we are working on a few leads such as host induced modifications.

If you have any suggestions for either of these problems please rush them along. But I am afraid we can not think about publishing for the present.

Your last letter has left me confused about the state of the job you talked about. Since the day of reckoning is drawing nigh when Polio will throw me out into the cruel working world (next August to

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be exact) I am anxious to make final plans as soon as possible. Your offer sounded good, so I would prefer to hold off on other possibilities until I hear further from you.

My best regards to everyone — the lab and Zella.

Alan

P.S. Don't let this letter discourage you about the meeting you were planning on. I still have some meaningful data to talk about.