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Rue d'Ulm, 26
TÉL. : ODÉON 45-75

Dear Dr. Lederberg:

I was very glad to receive your interesting reprints, namely your ~~inkx~~ extensive survey on bacterial genetics, but very unfortunately, I lost it in a train yesterday. If you have some extra ones, could you be so kind as to send me one more. I have in my laboratory a young fellow from Rio de Janeiro who works on bacterial mutations and intends to introduce this new field in his country next year. For this purpose, he collects the litterature on the subject and would be grateful to you if you could send him some of your reprints. His name is Dr.C.A.Elias. Thank you very much in advance.

This letter gives me the opportunity of telling you a little about my present two works, which may be of some interest to you.
1) Since 2 years, I tried to see if there is any relation between carcinogenicity of some water-soluble derivatives and their mutagenicity on bacteria. I finally came to the conclusion that so many factors are involved in both phenomena that the only significant results may involve chemicals of the same series and very close to each others, some of them being carcinogens and the others not. In doing so, we may expect that factors such as solubility, penetration, stability, steric ~~in~~ hindrance, etc are not too different within the same series, and that the differences in activity lie in the biochemical reaction itself.
A first result was obtained with:
1,2,5,6-dibenzanthracene-α,β ~~sodium~~ endosuccinate
anthracene-α,β endosuccinate
The former is carcinogenic and mutagenic; the latter is not K nor μ, although more toxic on the bacteria.
A second result, more significant, was recently obtained with carbamates:

		K	μ
ethyl carbamate:		++++	++++
isopropyl	-	++	++
propyl	-	+	+
butyl	-	0	0

Of course, both activities are not mathematically defined, and one could discuss the validity of my ++, but I can say that both activities run parallel in this series.

2) As regards the cancer problem, I have been very inclined to follow the so-called plasmagene story, or, at least the idea that some genetic change in a cytoplasmic constituent might be at the origin of a cancer cell (this change being capable of modifying the nutritional requirements of the cell.)
A path of attack seemed to check if carcinogenic agents could

induce mutations in cytoplasmic bodies, and first in viruses But no clear cut induced mutations had never been obtained as yet. Since last august, I tried to induce a mutation in a bacteriophage by irradiating ~~with UV~~ infected bacteria at the time of phage multiplication. Such a mutation has been obtained and the variation with the dose appears at present. As expected from my preceding work on the multiplication of that virus (T2), the phenomenon varies according to the time of the latent period, since multiplication does not proceed the same throughout this period. As you may expect, quantitative determinations are pretty hard to get with accuracy, because many things interfere. But it encourages me to handle now a method for inducing mutations in a virus.

My present purpose is to see if such mutations could not be induced by a pre-treatment of the bacteria, before infection.

Excuse me for that "bavardage". Hoping the best to you

very sincerely

Raymond Latarjet

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