

May 2, 1958

Dr. Jack Schultz
The Institute for Cancer Research
7701 Burholme Avenue
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Dear Jack:

Thank you for your note and ms. of March 27. I apologize for having taken so long to answer but have been just swamped with doings at Gatlinburg and here.

As to the ms., of course it is not foolish, though I am not certain that chromosomal changes can yet be excluded for the phenomena in somatic cells to which you allude.

The idea of selective stimulation and proliferation of antibody-forming cells is becoming quite respectable, though Coons feels it is exaggerated.

At the Gatlinburg meeting there was quite a lot of discussion mainly instigated by Ephrussi on "local states" of genetic material, and we evolved a scheme rather different from the conventional chromosomal vs. cytoplasmic heredity. I suggested instead "nucleic" and "epinucleic" for information which (presumably) recorded in the nucleotide sequence of a nucleic acid or elsewhere respectively. It is reasonably certain that in the cytoplasm we have both nucleic and epinucleic factors (e.g. kappa versus, possibly, the serotype system) and the same probably holds in the nucleus, e.g., mutant genes versus local states. I myself am rather doubtful that local states, puffs, etc. are going to turn out to have developed new, specific base sequences, but quien sabe?

I am enclosing a ms. that would be pertinent to the present discussion, if we could put firm reliance on the evidence for mutual exclusion which I do not believe we should as yet.

Yours sincerely,

Joshua Lederberg
Professor of Medical Genetics

JL/ew
encl.

P. S. A specific point: page 5: Did Algire show that the antigens were indif-
fensible or that homograft destruction required access to the graft by host cells,
or both?

page 8: At Gatlinburg, Medawar withdrew the claim that the homograft antigen is
DNA-protein. It now seems to be mucoprotein, as one would have guessed.

Schultz)