

Columbia University
in the City of New York

DEPARTMENT OF ZOOLOGY

August 20, 1943

Dr. R. A. Fisher
The Galton Laboratory
Rothamsted Experimental Station
Harpenden, Hertfordshire

Dear Fisher

Congratulations on your election to the Balfour professorship. I conclude from your acceptance that provisions for research and instruction have been made. Both had run down badly. I was always sorry that Punnett didn't want students (or seemed not to) for the other schools on which genetics depends were well developed, and the schools which genetics can help, like agriculture, were ready, too. Only genetics, through which the contributions of the one might pass to the other was apathetic. I hope this will change now for Cambridge is in many ways an ideal place for genetics. We wish you all success.

I think perhaps we've not been talking about the same things in respect of genes "which enhance normal development". Those structural and functional conditions would seem to me more "normal" which improved the chance of survival and increased fertility etc. True, this might sometimes be a diminution, sometimes an increase in the part or function, but in respect of important structures or functions this could not "fluctuate constantly". In the illustration I used, as long as the bird's tail serves an important function in copulation, minus fluctuations should be penalized, plus fluctuations should be selected. Genes tending to produce the latter would "enhance normal development".

I wasn't disturbed by Gruneberg's treatment of umbrous, since I think it quite likely that the umbrous "gene" of Mather and North is one of several which darken agoutis and yellows. Genes with such effects might be called "umbrous" or "sable" - I have always preferred to call them simply "darkeners".

I probably have a collection of them in the very dark yellow line which I derived from backcrosses with an agouti line. I'd like to send you some of these as soon as transport permits. I probably shan't have opportunity to analyse this complex.

As for Roberts' pallid mutation, I don't know of any outside of the United States. Since it arose from wild-type far from any source of imported mice, I suppose it is unique.

Sincerely, as ever


L. C. Dunn

LCD:AT

P.S. In regard to "modifying" action of darkening or "murbrous" genes, an experience of mine & Eusebi's is interesting. Among wild type agoutis we found a family with only about $\frac{2}{3}$ of the normal amount of melanin (by weight) yet the visible color was normal (our Genetics Vol 36, p 47). We suspected a mutation to lesser amount of melanin which in this ^{case} did not act as a "modifier" since the wild type had (if you wish to express it so) more than enough melanin to ensure normal color. L.C.D.