February 27, 1968

Dear Neil,

Your letter of February 6 reached me the day before I left Cold Spring Harbor for Austin, Texas, to give a set of lectures. I returned from Texas a week later with a blooming cold and much fatigue. Both conditions are now relieved. They account, however, for the long delay in replying to your letter.

I was delighted to receive an outline of your recent progress and very much pleased with the results. One aspect—the placement of the controlling element associated with \( w^c \)—resembles that of Judd with \( w^{zm} \) and its derivatives. In both cases, the "controlling element" lies within a common region of the white locus. I noted this initially in reviewing Judd's abstract that appeared in the July, 1967, issue of Genetics. In a recent talk with Judd at Austin, I learned that in his case, he is finding something strange at the white locus in salivary chromosome examinations and he, also, is not able to interpret its exact nature. From this conversation I gathered that you know of his recent results and therefore, I need not recount them.

The similarity of placement of the "controlling element" in the \( w^0 \) and \( w^{zm} \) cases conforms with the cases in which where different known systems have taken over control of gene action at the \( wx \) locus. The tests of this were conducted by Oliver Nelson. He is now writing up the results of a large number of tests aimed at placement of sites of change within the \( wx \) locus that are responsible for mutant expressions. Most of the mutants were "spontaneous" in origin. A few were produced by X-rays. Both multisite and single site mutants are represented among these selections. In addition, five cases of change in gene action induced by the presence of known controlling elements were included in the site mapping tests. One multisite (deficiency) mutant covers a segment within the locus that is close to its middle. The controlling element in each of the five cases lies within this region of the locus. Three of the five
cases are independent inceptions of control of action of the \textit{Wx} gene by the \textit{Ac} system. A fourth is a stable mutant derived from one of these. The fifth is an instance of control by the \textit{Spm} system. When homozygous none of the five cases gives rise to a wild-type allele. Also, the stable mutant when combined with the mutable allele from which it arose gives no wild-type alleles. Otherwise, each combination gives rise to a few wild-type alleles. It has been possible to order the sites within the controlling element region that distinguish one allele from another. There is no evidence that the different alleles of the \textit{c.e.} alter crossover frequencies within the locus when combined with alleles derived from spontaneous mutation.

Nelson plans to send the manuscript to me sometime this coming spring. I will then have more precise information. Just now, I have only the detailed information that was available from tests conducted up to April, 1966. These are the data that I showed to you this past June. Some of the more recent information given above comes from a telephone conversation that I had with Nelson several weeks ago.

When I have the needed details, I will send you a summary. I am sure Nelson will allow you to refer to his yet unpublished work provided you send him a copy of your statement in order that no misrepresentation appear in it. Peter Peterson has an abstract in the July, 1967, Genetics in which he refers to Nelson's results. His statements, however, were incorrect and misleading. This was disturbing to Nelson (and to me). Peterson did not check with Nelson before submitting the abstract.

Again, many thanks for keeping me informed. It is much appreciated. Also, give my very best to Katie.

As always,