Khovanova, E.M. Research Institute of Medical Radiology, Academy of Medical Sciences, Obninsk, U.S.S.R. Somatic mosaicism in D. melanogaster x D. simulans hybrids.

The frequency of somatic mosaics among D. melanogaster x D. simulans hybrids was studied in two series of experiments. In the first series (qq y/y D. melanogaster x dd +/Y D. simulans) 158 hybrid females out of 1460 had mosaic spots (1 or 2 yellow macrochaetae) which corresponds to 108 per 1000. In the

second series (qq y, Muller-5/y, Muller-5 D. melanogaster x 🔗 +/Y D. simulans) mosaic spots were observed in 345 out of 2479 hybrid females, i.e. 139 per 1000. This result was quite unexpected, because in our preceding experiments with D. melanogaster heterozygous inversions markedly (by about an order of magnitude) decreased the frequency of somatic mosaics, namely 59 out of 8214 y/+ D. melanogaster females, or 7.2 per 1000, had yellow spots, as compared to only 3 out of 5100, or 0.6 per 1000 y, Muller-5/+ D. melanogaster females. A short inversion dl-49, being heterozygous, exerted only a small effect almost indistinguishable from the results of y/+D, melanogaster series (23 out of 4121, i.e. 5.6 per 1000 y, In d1-49/+ D. melanogaster females). Mosaic spots in D. melanogaster x D. simulans hybrids may be due to somatic crossing-over, point mutations, deletions, or elimination of X-chromosome, bearing normal allele in yellow locus. It may be suggested that the role of somatic crossing-over in the origin of mosaic spots in hybrids is considerably smaller than in heterozygous D. melanogaster females, in which case most of the spots result from somatic crossing-over, and an inversion in one of the homologous X-chromosomes sharply decreases the frequency of mosaics. It is probable that more important role in the origin of somatic mosaicism in D. melanogaster x D. simulans hybrids is played by elimination of one of the X-chromosomes. The research of the mechanisms of somatic mosaicism in D. melanogaster x D. simulans hybrids is in progress now.

Alexandrov, I.D. Research Institute of Medical Radiology Academy of Medical Sciences of U.S.S.R., Obninsk, U.S.S.R. A preliminary analysis of negative complementation in white locus of D. melanogaster.

In a previous note (of DIS, this issue) the data on quantitatively different antimorphic action of two pseudo-allelic w mutants were presented. This difference was observed in different heterozygotes and that (the first fact) was why it was considered to be fundamentally interallelic. To test if this difference is connected with the site of w mutants on the

genetic map of white locus, the further experiments included more w mutants as well as deletions of the whole w⁺ locus. The w¹, w^{10gA} (both suppress z), w^{13gA}, w^{15gA}, w^{57gA}, w^{59gA} w^{60gA}, w^{69gA} (all gamma-ray induced and do not suppress z) mutants and the deletions w²⁵⁸⁻⁴⁵ and w were used. The w is a recombinant combining the left end of In(1)w^{mJ} with the right end of In(1)w^{mJ}. The w⁺ allele from D-32 stock was used throughout.

For quantitative determination of red eye pigments in qq heterozygous for w^{+32} and any one of the mutations the spectrophotometric method was applied, the details of which may be found in a previous note. The quantities of red pigments are also expressed here as the extinction (E) per 10 heads extracted per 1 ml of 30% AEA.

The results of these analyses are listed in Table below. It may be seen that these data confirm the existence of difference in antimorphic action of $w^{10}g^A$ and $w^{69}g^A$ mutants. They are found to be the extreme items of the series of w mutants studied. It is important that all white mutants have pronounced antimorphic action in contrast to the action of deletions. So, QQ heterozygous for any of two deletions and QQ homozygous for w^+ have practically the same quantities of red pigment. This is the second fact which shows that the decrease in red pigment content in QQ heterozygous for any of the w mutants studied is a result of the action of the w mutants themselves. This effect of the w mutants may be considered as a negative complementation at the phenotypic level. As seen, the negative complementation permits w mutants of the left or the right sections of the white locus to be distinguished more clearly from each other. It is important that w^1 and $w^{15}g^A$ positioned in different sections of the locus have the same degree of negative complementation. This fact shows that w mutants with functionally the same effects may occur in both the left and the right sections of the white locus and, thus, no drastic effect of $w^{69}g^A$ may be the peculiarity of the left section of the locus. The characteristics of negative complementation in the white locus and the possibil-