Lefevre, G. San Fernando Valley State College, Northridge, California. Mutant phenotypes immediately to the left of w. Recently, Gersh (Genetics 56:309-319) described a characteristic loss of bristles evidenced in w^{258-45} males covered by the variegated duplication w^{Vco}. Several head bristles are missing, including orbitals, ocellars, verticals, and postverticals; thoracic bristles as well are occasionally absent. This syndrome was ascribed to a locus in salivary chromosome band 3C1, deficiency for which is lethal and which is deleted in w^{258-45}. Cytogenetic studies in this laboratory indicate that bands 3B3 and 3B4, as well as 3C1 and 3C2, are deleted in w^{258-45}; thus the bristle effect cannot be securely attributed to 3C1, rather than to 3B3 or 4.

By use of a new deficiency resembling w^{258-45} and a new duplication resembling w^{Vco}, further information is now available on mutant phenotypes just to the left of w. The mutant $In(1)w^{-64d}$ (see Report of New Mutants) combines a deficiency from 3B3 to 3C2, inclusive, with an inversion whose right breakpoint is in the proximal heterochromatin. This deficiency can be covered by Dp w^{Vco} and expresses a bristle syndrome similar to that described above for w^{258-45}; Dp w^{Vco} males. The effect is more extreme in XO males. Both w^{-64d} and w^{258-45} are also covered by $Dp(1;3)w^{m49a7}$, a section from 3B1-3D6 inserted in the proximal heterochromatin of 3L. Again, the bristle syndrome is evidenced in covered males. $Dp(1;3)N^{264-58a}$, similar to Dp w^{m49a7}, but not including 3B1-2, fails to cover either deficiency. A new duplication, $Dp(1;4)w^{m65g}$, (see Report of New Mutants) extends to the right of w only through the rst locus (3C4), resembling in that respect Dp w^{Vco}; its extent to the left of w is uncertain, but it is quite short. Difficulty in visualizing this short duplication in the heterochromatin of the fourth chromosome has made it impossible so far to determine its exact extent cytologically. However, it produces a low frequency of covered males with both w^{-64d} and w^{258-45}. These males exhibit in exaggerated form the bristle abnormalities mentioned above. The head is virtually devoid of bristles, and the thorax is sparsely bristled. Moreover, the eyes are small and rough, the antennae somewhat abnormal with reduced aristae, and the wings are usually blistered and misshapen. Despite these abnormalities, some males are fertile. The effect is not due to the presence of the duplication, since w^{m4L}-rst^{3R} males, deficient for 3C2-3, show none of the defects when covered by Dp w^{m65g}.

Dp w^{m65g} may not, in fact, fully cover the deficient areas in w^{-64d} and w^{258-45}; the "covered" males may be truly deficient for a short region between their left breakpoints (which follow 3B2) and the left end of Dp w^{m65g}, thereby allowing the extreme phenotype of homozygous deficient to be expressed. Alternatively, Dp w^{m65g} may have its left breakpoint near 3B2 or 3, thus giving rise to extreme variegation for the area just to the right of 3B2. If the left end of w^{m65g} were at 3B1, or further to the left, it should cover w^{-64d} and w^{258-45} as well as Dp w^{m49a7} covers them. The fact that Dp $N^{264-58a}$ covers neither w^{258-45} nor w^{-64d} suggests that Dp $N^{264-58a}$ does not extend as far to the left as its description suggests, i.e., to 3B3. Alternatively, the bristle anomalies described above reside in 3B3 or 3B4 rather than in 3C1.

A final fact to take into consideration in attributing phenotypes to specific bands to the left of the 3C region is that many salivary preparations, in which the 3B region is well stretched and stained, clearly show 3 delicate bands between 3B1-2 and 3C1; Bridges' standard map shows only 2. If this is accepted, the most plausible location for the bristle anomalies is 3B3; Dp $N^{264-58a}$, then, would extend only to 3B4 (on the standard map).

Mglinetz, V.A. Institute of Medical Radiology, Obninsk, USSR. Elimination of radiation induced chromosome aberration in experimental populations of Drosophila melanogaster.

Behaviour of chromosome aberration originating in experimental numerically stable populations of D. melanogaster was studied. The aberrations were induced by (i) single 5000 r gamma-irradiation, (ii) fractionated gamma-irradiation by 1000 r monthly fractions, and (iii) chronic irradiation from P^{32} radioisotope source supplied to the food. It was found, that chromosome translocations induced by ionizing irradiation were eliminated within the first generations, while among 66 induced inversions only 7 remained in populations for 2 to 5 generations. None of the inversions became stable in the populations.