Recently, Gersh (Genetics 56:309-319) described a characteristic loss of bristles evidenced in w²⁵⁸-⁴⁵ males covered by the variegated duplication w⁺⁴⁵. 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 w²⁵⁸-⁴⁵. Cytogenetic studies in this laboratory indicate that bands 3B3 and 3B4, as well as 3C1 and 3C2, are deleted in w²⁵⁸-⁴⁵; thus the bristle effect cannot be securely attributed to 3C1, rather than to 3B3 or 4.

By use of a new deficiency resembling w²⁵⁸-⁴⁵ and a new duplication resembling w⁻⁴⁵, 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 3B1 to 3C2, inclusive, with an inversion whose right breakpoint is in the proximal heterochromatin. This deficiency can be covered by Dp w⁻⁴⁵ and expresses a bristle syndrome similar to that described above for w²⁵⁸-⁴⁵; Dp w⁻⁴⁵ males. The effect is more extreme in XO males. Both w⁻⁶⁴d and w²⁵⁸-⁴⁵ are also covered by Dp(1;3) w⁻⁶⁵g, a section from 3B1-3D6 inserted in the proximal heterochromatin of 3L. Again, the bristle syndrome is evidenced in covered males.

Dp(1;3)N²₆₄⁻₆₅a, similar to Dp w⁻⁶⁵g, but not including 3B1-2, fails to cover either deficiency. A new duplication, Dp(1;4) w⁻⁶⁵g, (see Report of New Mutants) extends to the right of w only through the rest locus (3C4), resembling in that respect Dp w⁻⁴⁵, 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 w²⁵⁸-⁴⁵. 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⁻⁶⁵₄⁻₆₅₃R males, deficient for 3C2-3, show none of the defects when covered by Dp w⁻⁶⁵g.

Dp w⁻⁶⁵g may not, in fact, fully cover the deficient areas in w⁻⁶⁴d and w²⁵⁸-⁴⁵; 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⁻⁶⁵g, thereby allowing the extreme phenotype of homzygous deficient to be expressed. Alternatively, Dp w⁻⁶⁵g 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⁻⁶⁵g were at 3B1, or further to the left, it should cover w⁻⁶⁴d and w²⁵⁸-⁴⁵ as well as Dp w⁻⁶⁵g covers them. The fact that Dp N²₆₄⁻₆₅a covers neither w²⁵⁸-⁴⁵ nor w⁻⁶⁴d suggests that Dp N²₆₄⁻₆₅a 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 3B3-2 and 3C1; Bridges' standard map shows only 2. If this is accepted, the most plausible location for the bristle anomalies is 3B3; Dp N²₆₄⁻₆₅a, then, would extend only to 3B4 (on the standard map).

Mgalinetz, 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 ³²P 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.