TABLE 2

Genotype of mother	Phenotype of offspring al Cy sp	al Cy+	+ + sp	+ + +
al In(2L)Cy +/+ In (2R) Cy sp	9	11248	13507	9
al Ins(2L+2R) Cy sp/ + + +	10901	10	12	14786

Table 2. Crossing over between the two Cy inversions in ♀♀ crossed to al sp/al sp ♂♂.

Further investigations involving combinations of other inversions, are in progress.

References: Lüning, K. B. 1952. Acta Zoologica 33:193-207.
Ramel, C. and Valentin, J. 1965. Hereditas 54:307-313.

Lefevre, G. San Fernando Valley State College, Northridge, California. Tests for deficiencies in the vicinity of the w locus.

Routinely in this laboratory, as well as in others, mutants suspected of being deficient for genetic material in the vicinity of the w locus are tested by crossing to w^{258-45}, a short deficiency extending from 3B3 through 3C2 (not just 3C1, as previously described by Bridges and Brehme in the "Mutants of Drosophila") and to w^{m4L}-rst^{3R} (w-), deficient for 3C2-3. Both of these known deficiencies can be "covered" by appropriate duplications: w^{258-45} by Dp w^{Vco} and Dp w^{m49a7} ; w^{m4L}-rst^{3R} by Dp w^{+51b7}, Dp w^{m49a7}, Dp $N^{264-58a}$ and Dp(w-ec)^{64d}, among others. The failure of nonduplication-bearing w^{258-45} / mutant females to survive implies that a deficiency exists in the mutant chromosome just to the left of the w locus; similarly, the failure of w^{m4L}-rst^{3R} / mutant females to survive points to a deficiency located just to the right of the w locus. If neither type of female survives, a deficiency surrounds the w locus.

It is now apparent that these tests may lead to erroneous conclusions regarding the extent of deficiencies near w, especially with regard to the use of w^{m4L}-rst^{3R}. In point of fact, males deficient for 3C2-3C6, phenotypically white, roughest, and "vertical" (absence of one or more vertical bristles, and attributed by Gersh (Genetics 51:477-480) to bands 3C5-6) occasionally survive. Males deficient for 3C2-4 survive more readily, expressing white and roughest phenotypes. Males deficient only for 3C2-3 survive in appreciable numbers, though late hatching, and appear white-eyed. (Mutants with the characteristics just described were obtained by M. M. Green from his mutable w^{c} stock and cytologically analyzed in this laboratory.) One may also recall that rst^{2} males, deficient for 3C4-6, are far from lethal and can be obtained readily without duplication.

In light of the apparent nonlethality of these deficiencies, the problem of interest becomes to explain the lethality of w^{m4L}-rst^{3R}, deficient only for 3C2-3. The answer must lie in the fact that w^{m4L}-rst^{3R} is inverted, with the right breakpoint in the proximal heterochromatin. The consequent position effect on rst, together with the deficiency for 3C2-3 (perhaps augmented by a position effect on 3C1), combine to produce lethality (synthetic lethal?) even though each of the components alone, i.e., In(1)w^{m4}, In(1)rst^{3}, and Df 3C2-3, is viable. Evidence that the typical lethality of w^{m4L}-rst^{3R} involves position effect stems from the observation that, when raised at elevated temperatures (29°C), some males survive, white-eyed and roughest. High temperature, like an extra Y chromosome, suppresses variegation. In any event, it is now apparent that deficiencies lethal in combination with w^{m4L}-rst^{3R} must possess a more extensive loss than just 3C2-3. The system will not detect loss of 3C3 alone, for example; nor as yet can a phenotype be ascribed to the loss of 3C3. This band can not contain a lethal locus, as supposed by Lefevre and Wilkins (Genetics 53: 175-187); but its absence in the recombinant chromosome w^{mJL}-rst^{3R}, in conjunction with the position effect on 3C4, is sufficient to produce lethality in XY males raised at normal temperatures.

It should also be pointed out that lethality of a deficiency in combination with w^{258-45} does not require that the lethal effect be attributed to the loss of 3C1. The lethality could be in the right portion of the 3B region (3B3 or 4); deficiency for 3C1 may not, by itself, be lethal. However, mutants having precisely the required cytological characteristics are not as yet available to test the viability of deficiency for 3C1 alone.

As a final note on the analysis of deficiencies near w, coverage by a specific duplication does not guarantee that the deficiency is shorter than the duplication. For example, Dp w^{Vco}, extending from 2C1-3C4, permits the ready survival of males deficient for all of the 3C material from 3C1 to 3C6, since deficiency of 3C5-6 alone is not lethal.