Shannon, M.P.[1], T.C. Kaufman[2], M.W. Shen[1], and B.H. Judd[1] [1]University of Texas, Austin, Texas. [2]University of British Columbia, Vancouver, Canada. Lethality patterns of zw mutants in D. melanogaster.

Judd, Shen and Kaufman (in press) have derived 116 lethal and semi-lethal recessive point mutations that map between z (3A3) and w (3C2) on the X chromosome of Drosophila melanogaster. These zw (zeste-white) mutations have been arranged into 12 complementation groups which correspond on a one to one basis to the 12 salivary gland chromomeres in this region. The genetic sequence from z to w of the complementation groups (designated by number in the order of discovery) is zw1, zw8, zw4, zw10, zw2, zw3, zw6, zw12, zw7, zw5, zw11, zw9. By use of mutant males marked with the gene y, we have determined the lethality patterns of 42 mutants representing all zw complementation groups. (The lethality pattern of a mutant includes both its effective lethal phase (see Hadorn, 1955) and observations on its rate of growth and longevity.)

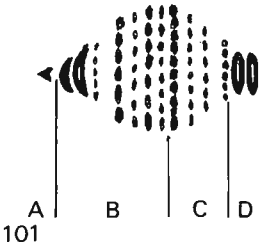
Mutants within a given complementation group have similar lethality patterns, as would be expected for members of an allelic series. The zw mutants are primarily post-embryonic in time of death, with the highest concentration of mortality occurring in the larval period (cf. Oster, 1952, 1954; Rizki, 1952; Seto, 1954). One group (zw2) is characterized by late embryonic-early larval "boundary lethality" (Hadorn, 1951). Three groups (zw6, zw12, zw5) show monophasic first instar lethality, and one group (zw7) shows diphasic first and second instar lethality. The remaining groups exhibit varying degrees of polyphasic lethality. Semi-lethal mutants show polyphasic lethality, even if lethal mutants within the same complementation group are monophasic in time of death. Our observations generally support the concept of the phase specificity of lethal factors (Hadorn, 1948, 1951, 1955); i.e., critical (lethal) phases are interspersed with relatively insensitive periods when deaths rarely occur. The zw mutants resemble other lethal Drosophila mutants in that they usually live for a considerable time after development has ceased (cf. Hadorn, 1955).

Phenogenetic studies (Kaufman, 1970) reveal that members of each zw complementation group also have similar morphological and cellular autonomy characteristics. We infer that the mutations in any one complementation group are quite specific in action, apparently affecting the same developmental processes.

References: Hadorn, E. 1948 Symp. Soc. Exp. Biol. Cambridge 2:177-195; Hadorn, E. 1951 Adv. Genetics 4:53-85; Hadorn, E. 1955 Developmental Genetics and Lethal Factors, Wiley, New York (1961 English translation from the German); Judd, B.H., M.W. Shen and T.C. Kaufman in press, Genetics; Kaufman, T.C. 1970 Ph.D. dissertation, Univ. of Texas at Austin; Oster, I.I. 1952 Heredity (London) 6:403-407; Oster, I.I. 1954 Nature 173:594-595; Rizki, M.T.M. 1952 J. Exp. Zool. 121:327-350; Seto, F. 1954 J. Exp. Zool. 126:17-32.

Roberts, P.A. Oregon State University, Corvallis, Oregon. A revised map of basal 4R.

We have reported elsewhere (Genetics 61:s50; ms. in preparation) that the behavior of T(3;4)10, recovered as one of a series of translocations producing ci position effect (Genetics, in press), strongly suggests that the centromere of 4 has not been correctly localized on the older map. The thin, rarely seen arm to the left of 101D is usually referred to as the "left arm" of 4 and the regularly visible remainder of 4, the "right arm" (Lindsley and Grell 1968). In T(3;4)10, the entire 101A-D region of the old map has been translocated to a point near the tip of 3R.



Metaphase views of the translocation show that the centromere of 3 is in its normal position. For this and other reasons (discussed more fully elsewhere), it seems unlikely that any one of the dark bands in 101D is the centromere of 4. The extra thickness of basal 4R, as we prefer to call 101A-D of the old map appears to be due to a position effect on DNA replication in this region in T(3;4)10. It provides a clear view of the pattern of bands in this region.

The revised map of this region (Figure 1) has fewer faint bands but is approximately the same length as the previous map. The revised map begins with subdivision 101B; we have left 101A for the unmapped 4L, centromere, and extreme proximal part of 4R. These parts of 4, attached to the other element of the reciprocal translocation, are not distinguishable, as yet, from the rest of the heterochromatin.