Kaufmann, Berwind P., University of Michigan, Ann Arbor. Participation of chromosome 4 in end-to-end association with other chromosomes in salivary-gland cells of D. melanogaster.

The frequencies of adhesion of the tip of chromosome 4 with the tips of other chromosomes have been determined in two X-ray-induced reciprocal translocations. One involves 2L and 4, with breakage points in 21A and 101E; the other involves 2R and 4, with breakage points in 60E and 102C.

When the homologues that form the left or right limb of chromosome 2 are completely synapsed in the F1 translocation heterozygotes (resulting from crosses between irradiated males and unirradiated females), it is apparent that 2L/4 is slightly longer than the normal 2L pairing partner of maternal origin, and that 2R/4 is longer than the 2R partner. Because of the positional advantage afforded chromosome 4 when it is removed from its normal chromocentral location, it is possible to compare the end-to-end pairing potential of this small chromosome with that of the X and the limbs of the longer autosomes.

Quantitative studies undertaken some years ago disclosed nonrandom, stock-specific patterns of end-to-end association of X, 2L, 2R, 3L, and 3R. In the Oregon-R wild-type stock used in the studies reported here, the tip of X participates with a higher frequency than the tip of any other chromosome in establishing end-to-end adhesions, whereas the tips of 3L, 3R, 2R, and 2L, and 4 participate with successively lower frequencies in such activity. Uniformity of the pattern was manifested in four 200+ cell samples; a X2 test afforded a P value of .75, indicating that the small observed deviations could be attributed to sampling errors.

When the tip of chromosome 4 is positioned so that it becomes part of the "pairing pool," it accounts for 42% of the end-to-end contacts in the 2L/4 translocation and 33% in the 2R/4 rearrangement. These departures from the wild-type pattern (in which the tip of 4 is rarely involved) are attained primarily by restriction of pairing of the tips of 3L, 3R, 2R, 2L, and 4, but not of X. The complete data, together with a statistical analysis and evaluation, will be published elsewhere.

One other observation that merits comment in this preliminary note is the interaction of the 21A and 101E loci, since it leads to marked puffing of 21A and B (involving heterochromatin?). The phenomenon is most strikingly manifested in those cases where synopsis fails and the homologue of paternal origin (2L/4) shows a large puff, whereas the homologue of maternal origin (2L) remains unpuffed.

This work was supported by USPHS Research Grant, GM-10499.

Pelecanos, M., Dept. of General Biology, University of Thessaloniki, Greece. An early oögonial stage highly sensitive to the larval feeding of diethyl sulphate.

The mutagenic activity of the monofunctional alkylating agent diethyl sulphate has been previously studied in several stages of Drosophila oogenesis (Pelecanos and Alderson 1964, Pelecanos 1966). However, previous data did not allow an answer to whether or not the early larval (0-24 hours) oögonia are more responsive than the later ones. This is mainly due to the fact that the sensitivity of the oögonial stages was measured as the rate of induced sex-linked recessive lethal mutations; consequently, the identity of lethals arising in a brood progeny of a female, was not tested and the extent of bunching was not known. Moreover, since the oögonial stages undergo many mitotic divisions during the course of their development, bunches (clusters) of identical mutations may play a predominant role in the determination of the mutational pattern of female larvae. In order to assess the extent of identical lethals in both the early and the late oögonial stages, we have studied the rate of recessive lethal mutations induced in the second chromosome. Treatment with diethyl sulphate is as previously described (Pelecanos and Alderson 1964). Two three-day broods were studied by individually mating each treated Oregon-K female to two Cy L4/Pm males. The duration of the several treatments is as follows: (1) 0-12 hrs treatment : newly hatched larvae treated for the first 12 hours of larval life. (2) 0-24 hrs : newly hatched larvae treated for the first 24 hours of larval life. (3) 12-24 hrs : 12-h-old larvae treated until the 24th h. of larval life. (4) 24-48 hrs : 24-h-old larvae treated until the 48th h. and (5) 48-72 hrs : 48-h-old larvae treated until the 72nd hour of larval life. We have not thought necessary to set up also a 72-96 hrs treatment.