Falk, R. The Hebrew University, Jerusalem, Israel. On the structure of the R(1)2, y f chromosome.

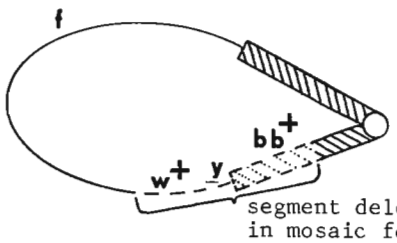
The exact origin of the ring R(1)2 is not known. It is assumed that it originated through a translocation between the two arms of an attached-X chromosome, CR(1)RM (Schultz & Catcheside 1939). Recently Vinikka et al. (1971) reconfirmed Schultz & Catcheside's observations that R(1)2 was duplicated for the bands 20A onward of the polytenic chromosome map. Schalet & Singer (1971) placed lethal l(1)A7 at band 20A1-2. This would mean that all essential loci proximal to l(1)A7 are represented twice in R(1)2.

The frequency of fragments that encompass the proximal loci of the X-chromosome induced in the R(1)2, y f is higher than that induced in a rod X-chromosome (Falk 1973). Dr. A. Schalet suggested to me that the higher frequency of such events induced in the ring than in the rod could have resulted from the fact that the proximal segment was present in duplicate in the ring chromosome.

In a previous study (Falk 1970) we induced recessive lethals in the proximal segment of the R(1)2, y f (obtained from Dr. I.I. Oster in 1965). The proportion of induced lethals that were located in this segment in the ring was not lower than that obtained in the rod X-chromosome. Furthermore, many of these lethals were found, by allelism tests, to be functional deletions of essential genes proximal to the locus of l(1)A7. This would suggest that in the ring studied by us no duplication for the most proximal essential loci existed. In a cytological analysis of the salivary gland chromosomes of R(1)2, y f/+ larvae no duplication for the bands of segment 20 was found (see Figure). Thus, the R(1)2, y f chromosome in our

laboratory is different from that described by Vinikka et al. and, in contrast to the R(1)2 present in the Leiden laboratory (personal communication), the R(1)2, y f in our laboratory is only slightly subvital for homozygous females and XO males. The variation in the structure of the R(1)2 chromosome in various laboratories could be due to mislabelling in the past (mixing R(1)1 with R(1)2 ?), but stems more probably from "evolutionary" diversifying changes that have been taking place in different laboratories which started with similar R(1)2 chromosomes (Schalet, personal communication).

Another clue to the structure of R(1)2, y f was recently obtained by Miss M. Yacoby from a cross of irradiated R(1)2, y f/Y^{bb-} males to $XY^{L}.Y^{S}$ (108-9), y^{2} $su(w^{a})w^{a}$ $Y^{L}.Y^{S}/In(1)sc^{4L}$ sc^{8R} + S, y sc^{4} sc^{8} w^{a} B females. A mosaic female was found, both her eyes were Bar (intermediate), one eye was completely apricot, the other was wild-type, except in a small upper section; the abdomen on one side showed an extreme bobbed effect. There were no indications of the fly being gynandromorphic (although one wing was slightly shorter than the other). The female died without leaving offspring. Upon dissection she was found to have female genitalia and gonads on both sides. We propose that this female, who started as a $In(1)sc^{4L}$ sc^{8R} + S, y



sc^4 sc^8 w^a B/R(1)2, y f zygote was obtained as a result of a chromatid deletion in the ring X-chromosome, including both the w^+ and the adjacent bb^+ loci. In other words, we propose that the single bb^+ segment present in the R(1)2, y f is located in the heterochromatic segment next to the "distal" euchromatic genes, and that the heterochromatic segment on the other side of the centromere, that is connected to the "proximal" genes, is void of bb^+ and probably originated from the long arm of the Y-chromosome. According to this interpretation the R(1)2, y f is an inverted closed X-chromosome (see scheme).

References: Falk, R. 1970, Mutation Res. 10:53-60; _____ 1973, Chromosomes Today 4:(in press); Schalet, A. and K. Singer 1971, DIS 46:131-132; Schultz, J. and D.G. Catcheside 1939, J. Genet. 35:315-320; Vinikka, Y., A. Hannah-Alava and P. Arajärvi 1971, Chromosoma 36:34-45.

Andrews, P.W. and D.B. Roberts. University of Oxford, England. A screen of a number of fluoro-compounds as possible selective agents for biochemical mutants in Drosophila melanogaster.

The current interest in genetic control in eukaryotes has made it desirable to select large number of mutants affecting the synthesis of a single protein. In Drosophila, Grell (1968) has used the sensitivity of Adh^- mutants to alcohol to select for such mutants, Glassman (1965) has used the sensitivity of Xdh^- mutants to purines to select for these mutants and more recently Sofer (1972) has selected for Adh^- flies using the sensitivity of the wild type, possessing the enzyme, to pent-1-ene-3-ol. We have investigated the possibility of using the resistance to a number of fluorocompounds, themselves non-toxic, as indicators of the absence of enzymes involved in their metabolism to toxic fluorocitrate (see Table 1).

Table 1

Fluorocompound	Enzyme mutants examined for resistance
2-Fluoroethanol*	Alcohol dehydrogenase minus - Adh^{nl}
4-Fluroobutanol*	Aldehyde oxidase minus - $Aldox^{nl}$ x
Ethyl α Fluorobutyrate*	Aliesterase minus x - strains carrying bw st sv^n & bw st ss.
5-Fluorotryptophan+	Tryptophan pyrolase minus - V^{36f}
6-Fluorotryptophan+	

* Obtained from K & K Laboratories
\+ Obtained from Sigma
x We are grateful to Professor Ursprung for sending us $Aldox^{nl}$ and to Dr. Ogita for the two Ali^- strains

Hypochlorite sterilised dechorionated eggs of either Oregon-R, Canton-S or mutant stocks (Table 1) were transferred to sterilised yeast/agar medium with, or without, the compound being tested. In some experiments the numbers of individuals hatching, pupating and ecloding were recorded (Table 2) but with two of the mutants, only qualitative observations were made ($Aldox^{nl}$ and Ali^-).

2-fluoroethanol and 4-fluorobutanol proved toxic to both wild type strains and to Adh^{nl}. There were suggestions that the latter were slightly more resistant, especially in the egg. In the cases where development was observed the larvae were generally very sluggish and development time was greatly increased. The mutant $Aldox^{nl}$ was similarly affected by these compounds.

The fluoroderivatives of tryptophan did not affect any stock over the concentration range used, up to 200 µg/ml. Ethyl fluorobutyrate (up to 2mg/ml) was slightly toxic to both Oregon-