or flowers, were found to be very sensitive to alcohol, whereas species breeding in sweet, fermenting fruits were found to be more tolerant. This suggests that, if environmental ethanol is really a selective factor it is more likely to act upon larvae than on adults. If that is the case, ecological genetics studies should preferably be carried out on larvae rather than on adults.

Of course, a possibility remained that larval and adult tolerances were highly correlated, so that adult tolerance would be mainly a by-product of larval adaptation.

To test this last hypothesis, we have compared larval and adult ethanol sensitivies in European and Afrotropical strains of **D.melanogaster.** Because of the difficulty arising from ethanol evaporation, we tried to keep constant the alcohol concentration during whole development. To this end we worked out a powdered killed yeast medium (formula to be published elsewhere) which can be prepared with cold water. Every day the larvae were sieved out of the medium and transferred to a fresh one containing the appropriate ethanol concentration. The results obtained for the transferred larvae, for adults of the same strains, and also for larvae kept in the same medium without any transfer during their whole development are shown in Figure 1.

When the alcohol concentration during the development was kept constant (i.e., when larvae were transferred every day) the LC 50 values were quite low, being 2.5 and 4.1% ethanol for African and French flies, respectively. But when the larvae were kept in the same medium, from which the alcohol progressively evaporated, the LC 50 values were much higher (5.7 and 15.5%). Interestingly, these latter values were very close to those (7.1 and 17.0) found for adults of the same strains.

From these observations we can conclude that, at least in **D.melanogaster**, variations of larval and adult tolerance are highly correlated. As had been assumed previously (David & Bocquet 1975), the divergence between European and Afrotropical populations may be attributable to different amounts of alcohol in the resources.

References: David, J.R., P. Fouillet & M.F. Arens 1974, Arch. Zool. exp. gen. 115: 401-410; David, J.R. & C. Bocquet 1975, Nature 257: 588-590; David, J.R. & J. Van Herrewege 1983, Comp. Biochem. Physiol. 74: 283-288.

Van Zijll Langhout, B.W. and F.M.A. van Breugel. University of Leiden, Netherlands. Cytological localization of the Aldox gene of **Drosophila melanogaster** in the region 3R 89A1.2.

For successful microdissection of genes from salivary gland chromosomes, exact cytological localization of the gene in question is required. With the ultimate aim of cloning the well-studied and histochemically interesting Aldox gene, we tried to locate the gene as accurately as possible. So far Spillmann & Nothiger (1978) assigned the locus to the region 88F-89A1 to 89B1-4 on chromosome 3R. Dickson Burkhart (1984) recently isolated from a North Carolina population two Aldox-null alleles associated with inversion breakpoints close to the 89A bands. While studying $Aldox^{n1}$ heterozygotes with various wildtype 3R chromosomes, we discovered that the $Aldox^{n1}$ mutation, originally isolated from an Urbana-S wild type strain (Dickinson 1970), in fact might be a small one-band or intraband deficiency. F1 larvae from a cross Aldox sbd x wildtype (Leiden) consistently showed much less stainable material in 89A (Figure 1) on one of the two homologous chromosomes. More proximal and distal regions fitted exactly with the Bridges (1935) map. Our conclusion is that the Aldox gene must be located in the double band 89A1.2. Upon inspection of the paper of Ashburner (1967), we found a very similar situation of unequal banding on the two homologous chromosomes on at least one of his photographs (viz. Figure 14A) of the Oregon-R wildtype strain. This suggests, that this wildtype strain could have been polymorphic for the small Aldox deficiency we have described here.

References: Spillmann, E. & R. Nöthiger 1978, DIS 53:124; Dickson Burkhart, B. et al. 1984, Genetics 107:295-306; Dickinson, W.J. 1970, Genetics 66:487-496; Bridges, C.B. 1935, J. Heredity 26:60-64; Ashburner, M. 1967, Chromosoma 21:398-428.

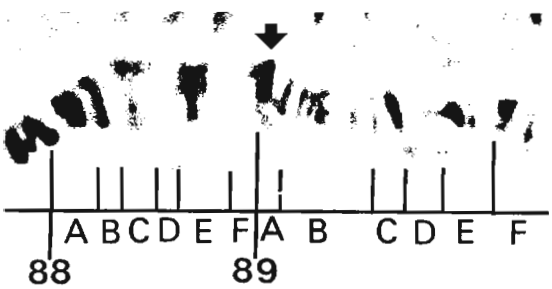

Figure 1. Part of chromosome 3R showing a deficiency for the Aldox locus in the lower part of band 89A.