

observed. However, individualization may sometimes occur also in vitro, since we found a damaged cyst in the coiling stage which contained unconnected spermatozoa. An "individualization cone" as seen in vitro (Tokuyasu et al. 1972) and in vitro (Cross and Shellenbarger 1979) on *D. melanogaster* was not detected. In our preparations coiling occurs even in cysts which are not so much elongated as is expected from in vivo investigation.

The following preliminary conclusions can be drawn from our observations. (1) The elongation of the spermatids is independent of an intact cyst. (2) The elongation of the cyst is more a consequence of the elongation growth of spermatids. (3) Individualization seems to occur only in an intact cyst. (4) Individualization and coiling of spermatids may also occur in completely elongated cysts, or spermatids, respectively.

References: Cross, D.P. and D.L. Shellenbarger 1979, *J. Embryol. exp. Morph.* 53:345; Fowler, G.L. and R. Johannisson 1976, in: *Invertebrate Tissue Culture* (E. Karstak and K. Maramorusch, eds.) p. 161, Academic Press; Fowler, G.L. and J. Uhlmann 1974, *DIS* 51:81; Tokuyasu, K. et al. 1972, *Z. Zellforsch.* 127:492.

Lohs-Schardin, M. Biologisches Institut I, Freiburg, West Germany. A new allele of *Ubx* causing a strong phenotype.

The allele *Ultrabithorax*⁷⁸ was discovered in a line selected for an embryonic mutation. Recombination experiments demonstrate that the mutation is located in the bithorax region (3-58.8). Flies heterozygous for *Ubx*⁷⁸ are fertile

and show the typical *Ubx*-phenotype (Lindsley and Grell 1968) which is characterized by enlarged haltere. The homozygous condition is lethal at late embryonic stages or shortly after hatching. In the homozygous state, the *Ubx*⁷⁸ allele has a more extreme phenotype than any other *Ubx* allele described so far. It resembles the phenotype of larvae homozygous for *Df(3)P9*, the deficiency of the entire bithorax complex (Lewis 1978). On all segments posterior to the mesothorax the homozygous larvae show morphological structures which are characteristic for the mesothorax (Fig. 1). These characteristics include the thorax-type ventral row of fine denticles with some stronger denticles in the posterior segments. Keilin's organs, "black" sense organs (Lohs-Schardin et al. 1980) and a separate section of the tracheal trunk are found on all segments, but the 8th abdominal segment. However, the phenotype differs from larvae homozygous for *Df(3)P9* at the posterior end where the telson appears normal.



Fig. 1. The most posterior series (7th and 8th segments and telson) of a homozygous *Ubx*⁷⁸ larva. The abdominal segments show the thin thoracic-type denticle rows, but some denticles of the 8th segment are slightly stronger. The 7th segment carries two "black" sense organs (Lohs-Schardin et al. 1980) and Keilin's organs.

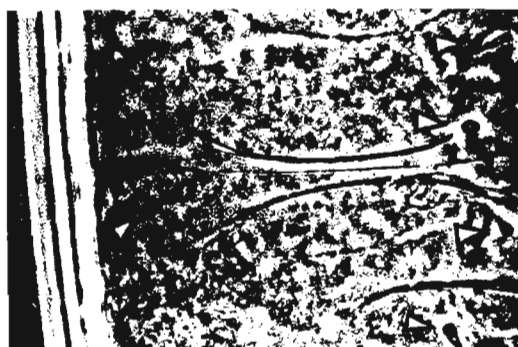


Fig. 2. A series of abdominal segments from a homozygous *Pc3;Ubx*⁷⁸ larva. The rudimentary spiracles of four segments and the thin thoracic-type denticles of one of these segments are visible.

Trans-combination between *Ubx*⁷⁸ and other mutations of the bithorax region [*bx3*, *pbx*, *bx3pbx*, *Cbx* (Lindsley and Grell 1968)] produce flies with extreme phenotypes; in this respect *Ubx*⁷⁸ is comparable to some other *Ubx* alleles (*Ubx*¹³⁰, *Ubx*⁸⁰) and to deficiencies of the whole bithorax complex [*Df(3)P115*, *Df(3)P9* (Morata 1975)].

In larvae homozygous for Pc^3 (3-48) and Ubx^{78} each segment posterior to the mesothorax is influenced by both mutations. Ubx^{78} exerts its effect on the ventral denticle rows which are thorax-like but at the same time Ubx^{78} enhances the expression of Pc^3 : most segments develop rudimentary posterior spiracles with "Filzkörper" and carry posterior sense organs which decrease in size towards the anterior segments (Fig. 2) while Pc^3 carries these structures only on the posterior 4 segments. This effect is even stronger than in larvae homozygous for Pc^3 combined with 3 doses of the bithorax complex (Lewis 1978).

When exposed to ether vapors at the cellular blastoderm stage, Ubx^{78} produces in 75% of the treated embryos bithorax phenocopies as compared to 25% phenocopies in the sib controls. The response to ether is known to be higher in embryos carrying bithorax mutations associated with break-points in the bithorax region [Ubx^{80} , Ubx^{130} , $Df(3)P9$ (Capdevilla and Garcia-Bellido 1978)]. Cytological analysis of salivary gland chromosomes with the genetical constitution $Ubx^{78}/Df(3)P9$ [$Dp(3)P115$ translocated] and $Ubx^{78}/+$ failed to reveal any deficiency in the bithorax region linked to Ubx^{78} .

The allele Ubx^{78} resembles the deficiency of the entire complex by its homozygous phenotype. However in combination with Pc^3 it shows effects ascribed to increased doses of the bithorax complex. Yet the yield of ether phenocopies is increased as in bithorax mutants known to carry a break-point within the bithorax region.

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References: Capdevilla, M.P. and A. Garcia-Bellido 1978, Roux's Archives 185:105-126; Lewis, E.B. 1978, Nature 276:565-570; Lindsley, D.C. and E.H. Grell 1968, Genetic Variations of *D. melanogaster*, Publs. Carnegie Inst.; Lohs-Schardin, M., C. Cremer and C. Nüsslein-Volhard 1980, Dev. Biol. 33:239-255; Morata, G. 1975, JEEM 34:19-31.

Loukas, M. Agricultural College of Athens, Greece. A new esterase locus in *D. melanogaster*.

We found that *D. melanogaster* possess the corresponding locus to Est-9 of *D. subobscura*. This esterase is detected only in the adults and only when 1-leucyl- β -naphthylamide is used as a substrate together with α -naphthyl acetate.

(For the technique used for detecting the enzyme, see Loukas and Krimbas 1975.) It is located on the fly's head and migrates in the gel as fast as Est-9 of *D. subobscura*.

In order to locate this gene we performed the following crosses (in all cases we refer to the same Fast and Slow alleles): For chromosome 3: Males of the "curled" strain (*cu* a recessive mutant located on chromosome 3), homozygous for the Slow allele (SS), were crossed with females of wild type homozygous for the Fast allele (FF). F_1 males were then crossed with females of the curled strain. Half of the wild and half of the curled progeny of this backcross were heterozygous (FS), while the other half were homozygous (SS). So, the esterase gene is not located on chromosome 3. For chromosome 4: Males of the "cubitus interruptus-Dominant" strain (*ci^D* a dominant mutant, lethal in homozygotes, located on chromosome 4), homozygous FF, were crossed with females of wild type homozygous SS. F_1 males of phenotype *ci^D* were then backcrossed with the females of wild type. Half of the wild and half of the mutant progeny were heterozygous, while the other half were homozygous (SS). So, the esterase gene is not located on chromosome 4. For chromosome 2: Males of the "orange" strain (*or*, a recessive mutant located on chromosome 2), homozygous FF, were crossed with females of wild type homozygous SS. F_1 males were then crossed with females of the orange strain. All the wild type progeny were heterozygous and all the orange ones homozygous FF. So, the esterase gene is located on chromosome 2.

Taking into consideration all the similarities between this esterase gene and the Est-9 of *D. subobscura* (similar biochemical properties of the enzymes and probably similar physiological role) as well as the fact that Est-9 is located on chromosome E of *D. subobscura* which is homologous to 2R of *D. melanogaster* (Krimbas and Loukas 1980), we suggest that these esterase loci are homologous.

References: Krimbas, C.B. and M. Loukas 1980, DIS 55:55; Loukas, M. and C.B. Krimbas 1975, Genetics 80:331-347.