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 García-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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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 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). F1 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 (PS), 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 (ciD a dominant mutant, lethal in homozygotes, located on chromosome 4), homozygous FF, were crossed with females of wild type homozygous SS. F1 males of phenotype ciD 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, recessive mutant located on chromosome 2), homozygous FF, were crossed with females of wild type homozygous SS. F1 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.