1975, Exp.Gerontol. 10:333; Geiss, M. & H.Planel 1977, Gerontol. 23:325; Strehler, B. 1959, Quart.Rev.Biol. 34:117; ____ 1962, J.Gerontol. 17:347; ____ 1964, J.Gerontol. 18:83; Nothel, H. 1965; Strahlen-therapie 126:269; Lamb, M. 1964; J.Insect Physiol. 10:487; ____ 1965, Exp. Gerontol. 1:181; Sonnenblick, B. & G.Welch 1970, Int.J.Radiat.Biol. 18:423; Gartner, L. 1973b, Int.J.Radiat.Biol. 23:23.

TABLE 1. LIFE SPAN OF FEMALE IMAGOES

Dose (R)	N	Mean Life Span Days ± S.E.	Signif.	Maximum Life Span Days
0 R	47	90.5 ± 4.55	NS	138
4000 R	44	90.1 ± 4.24	NS	134
8000 R	37	84.3 ± 5.03		129

TABLE 2. LIFE SPAN OF MALE IMAGOES

Dose (R)	N	Mean Life Span Days ± S.E.	Signif.	Maximum Life Span Days
0 R	38	81.0 ± 6.24		138
4000 R	41	81.7 ± 5.84	NS	134
8000 R	33	77.8 ± 5.62	NS	127

TABLE 3. LIFE SPAN OF COMBINED POPULATION

Dose (R)	N	Mean Life Span Days ± S.E.	Signif.	Maximum Life Span Days
0 R	85	85.8 ± 3.75		138
4000 R	85	85.9 ± 3.60	NS	134
8000 R	70	81.1 ± 3.71	NS	129

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A method to screen for Df(3R)P9 homozygotes.

Drosophila melanogaster embryos that are homozygous for a deletion of the bithorax complex Df(3R)P9 die in late embryogenesis or early first instar (Lewis 1978). These animals have an abnormal pattern of ventral denticle belts indicating that all the segments from the anterior metathorax to the seventh abdominal segment have been transformed to mesothorax (Lewis 1978). This transformation is of great interest to both geneticists and developmental biologists. However, to date there has been no method to recognize the homozygous (Df(3R)P9 phenotype until after the cuticle has differentiated. We have observed that Df(3R)P9 embryos incubated at 18°C fail to complete germ band shortening (GBS). Thus this morphological criterion can be used to select for the homozygous mutant class.

We crossed flies of the genotype Df(3R)P9/Dp(3;3)P5;Sb inter se, and collected eggs on agar plates for 30 min or less at 25°C. We then transferred the eggs to 18°C and incubated them until the embryos had begun GBS (about 18 or 19 hr after collection). This corresponds to stage 10 (Bownes 1975) of embryonic development. They were then dechorionated on double-stick tape, covered with paraffin oil (Baker), and observed under a dissecting scope. A total of 258 embryos were observed. 199 (77%) of the embryos we observed developed normally, completing GBS, head involution and dorsal closure (Turner & Mahowald 1979). However 59 (23%) of the embryos never completed GBS, even though other developmental processes such as head involution were normal (Figure 1, Table 1). We separated those embryos with incomplete GBS from those with complete GBS and allowed both classes to continue embryogenesis at room temperature. It should be noted here that the two classes are indistinguishable prior to and during GBS, so we waited until after the normally developing embryos had completed GBS to separate out the incomplete GBS class.

Most of the complete GBS class continued to develop and hatched normally. We assumed that all of the larvae that hatched successfully were heterozygotes and phenotypically wild

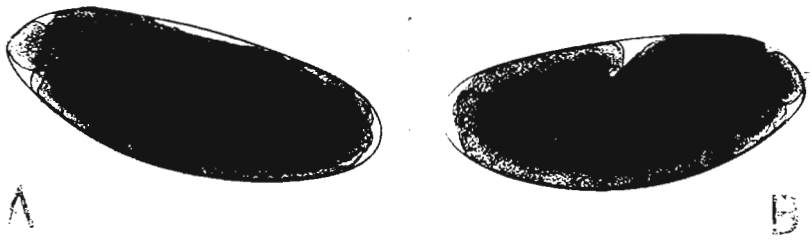

Figure 1. Morphology of Df(3R)P9 complete and incomplete GBS embryos. Df(3R)P9 embryos of the same age were dechorionated and observed by microscope. The majority of embryos (A) completed GBS, while approximately one quarter (B) exhibited incomplete GBS. (x 960)

Table 1. Assortment of embryos with respect to germ band shortening and ventral belt pattern.

Class	Ventral Belt Pattern	
	Wild Type	Df(3R)P9
Complete GBS	197	2
Incomplete GBS	4	55

type. Those that failed to hatch, as well as some of the hatched larvae, were mounted for phase contrast microscopy in lactic acid and ethanol (Lewis 1978), and their denticle belt pattern examined. Only 2 of the complete GBS class examined had the Df(3R)P9 phenotype; these may have been overlooked in the bulk screening procedure. We also mounted all of the incomplete GBS animals. All but 3 of these embryos failed to hatch. 55 out of 59 expressed the Df(3R)P9 phenotype; the other 4 animals had a wild type belt pattern. These could have been incorrectly identified during the screening procedure if they were younger than the other embryos.

This screening method should only be performed on embryos incubated at 18°C, since the results are not as clean when they have been incubated at 25°C. However, incomplete GBS has also been observed in another Df(3R)P9 stock that is marked with multiple wing hair. This indicates that the incomplete GBS phenotype may be used to screen for Df(3R)P9 homozygotes in all Df(3R)P9 stocks.

References: Bownes 1975, J.Embryol.Exp.Morph. 33:78-801; Lewis 1978, Nature 276:565-570; Turner & Mahowald 1979, Dev.Biol. 68:96-109.

Gerasimova, T.I.[1] and Yu.V.Ilyin[2] [1]Institute of Molecular Genetics, USSR Academy of Sciences, [2]Institute of Molecular Biology, USSR Academy of Sciences. The role of the mobile element mdg4 in the formation of unstable cut mutations in Drosophila melanogaster.

Earlier, an unstable ct^{MR2} allele was obtained in a cross of Oregon-R females and MRh12/Cy males under hybrid dysgenesis (Gerasimove 1981) (the MRh12/Cy genome contains multiple copies of the P-element). This mutation was characterized in the homozygous stock by a high frequency of reversions, the occurrence of new unstable visible and lethal mutations and super-unstable ct mutations, as well as the emergence of new mutations at other loci in the X-chromosome (Gerasimova 1981, 1982). In the present study, we look into the molecular nature of the mobile element integrated at the cut locus in the ct^{MR2} mutant. To this end, we have carried out in situ hybridization on crushed salivary-gland chromosomes of ct^{MR2} larvae, using the standard procedure (Ilyin et al. 1978). In our hybridization assays, we used plasmid DNA (labeled with ^{3}H and ^{125}I) containing the following elements: mdg1, mdg2, mdg3, mdg4 (Tchurikov et al. 1981), copia (Finnegan et al. 1978) obtained from D.Finnegan, fb elements obtained from S.Potter (Potter et al. 1980), and P-element from G.M.Rubin (Rubin & Spradling 1982). The cut locus is known to be located in the 7B region of the X-chromosome. Mdg4 is the only one of the above-listed elements that hybridizes with the 7B region in the ct^{MR2} mutant. This is a typical mdg which contains direct and inverted repeats at the edges. The total length of mdg4 is 7 kb. Its structure has been revealed earlier by Yu.V.Ilyin. In situ hybridization was performed with different subfragments of mdg4. The picture was the same: the label was invariably found in 7B. At the same time the original Oregon stock, whence ct^{MR2} was derived, does not contain mdg4 in the 7B region (or anywhere in the X-chromosome).