SG17.1 is a homozygous viable strain with *P-GAL4* insertion in the X-Chromosome. At 0 hr pupal brain (Figure 1a), reporter gene expression is seen in cells which are more or less uniformly scattered in the entire brain and Ventral ganglion. The number of cells with expression is slightly higher on the dorsal side of the ventral ganglion. During 6, 14, and 24 hr (images not shown), a similar pattern as that of the zero hr stage is seen with a reduction in the intensity and the number of cells expressing the reporter gene. At 43 hr stage (Figure 1b) expression gets confined predominantly to a pair of cell clusters at the suboesophageal ganglion (SOG) (Truman, 1990), whereas it is withdrawn from rest of the brain regions. This expression in the SOG cell clusters at 43 hr seems to be transient, as it is not seen in the later stages. Interestingly, in the 65 hr pupal brain, the global scattered expression pattern is reiterated (image not shown). At later stages, transient expression in isolated cells in the antennal lobe, superior medial Protocerebrum (Ref: Flybrain) and the SOG is seen. Figures are shown for 96hr (Figure 1c) and pharates (Figure 1d).

Earlier studies on the adults of this particular strain have shown reporter gene expression in the components of the gustatory system, including maxillary palp and taste nerve, third segment of the antenna, olfactory region, and Calyx in the brain (Shyamala and Chopra, 1999). The present finding as to its expression during pupal stages implies a developmental role to the native gene at the site of *P-GAL4* insertion.

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The *mus309* mutations, defective in DNA double-strand break repair, mobilize the *gypsy* element in *Drosophila melanogaster*.

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In *Drosophila melanogaster* the mutagen-sensitive *mus309* locus on the third chromosome right arm (86F4) encodes, in a manner similar to its orthologues in other organisms, the mammalian *BLM* locus included, a RecQ helicase (Boyd *et al.*, 1981, 1987; Ellis *et al*., 1995; Karow *et al*., 1997; Moghaghegh *et al*., 2001; Wu *et al*., 2001) and, accordingly, is involved in DNA double-strand break repair (reviewed in Brabant *et al*., 2000; Heyer *et al*., 2003; Heyer, 2004).

The *mus 309* mutation is also known to be defective in double-strand DNA break repair after P element excision (Beall and Rio, 1996; McVey *et al*., 2004).

The ct^6 mutation of *Drosophila melanogaster* (*ct*, cut 1 – 20.0) for its part is caused by an insertion of the mobile *gypsy* element (Lindsley and Zimm, 1992).

My intention was to construct two *w* ct^6*; mus309/Tb* stocks (*w*, white 1 – 1.5; *Tb*, Tubby 3 – 90.6) carrying either the $mus309^{D2}$ or the $mus309^{D3}$ allele, both of which are sterile in females and semi-sterile in males (Boyd *et al*., 1981; Kusano *et al*., 2001).

Both the *mus309* alleles used carry mutational changes that could potentially impair or abolish at least the helicase function of the MUS309 protein. In the *mus309*^{D2} allele, there is a stop codon between the sequence motifs encoding the third and fourth helicase motif of the protein. The *mus309*^{D3} allele for its part has a glutamic acid to lysine substitution in the conserved helicase II motif, as well as another amino acid substitution close to the C terminus (Kusano *et al*., 2001).

While constructing the stocks, I first constructed several lines carrying the *w ct*^{6} constitution in their X chromosomes and either the *mus309/Tb* or the *+/Tb* constitution in their 3rd chromosomes. These could not be distinguished on the basis of the morphological phenotype, because the *mus309* mutations do not have any morphological effect. The lines were submitted to natural selection in order to follow whether the Tubby balancer was maintained in them and consequently were carrying the *mus309* mutation, too.

The lines were checked for the first time after five generations of selection. It was observed that in five of the eight original *D2* lines which had maintained the Tubby phenotype the cut phenotype had reverted, but in one line which also had maintained the Tubby phenotype the cut phenotype had not reverted. This line was preserved as a stock. Similarly, in one of the eight original *D3* lines the cut phenotype had reverted and also the white phenotype had reverted. Subsequently, a double-revertant stock was constructed from this line.

Thus, it is highly likely that the *mus309* mutations, defective, *e.g*., in P element excision, had mobilized the *gypsy* element residing in the *ct*^{6} allele of the cut locus, thus causing its reversion and also the reversion of the *w* mutation.

Both the *D3* and the *D2* stocks mentioned have since been stable now for approximately 20 generations.

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Five mammalian carcinogens are active in a short-term tumorogenicity assay in *Drosophila*.

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Earlier we showed that mammalian carcinogens cause tumors in *wts*-heterozygous flies. We developed the only test capable to demonstrate tumor formation in *Drosophila* by carcinogenic compounds. The clone formation is based on heterozygosity loss in the imaginal disc cells as a result