combinations range from pre-larval lethality through hanging up as wandering third instars to late pupal lethality.

*l(3)82Fi:* alleles 1 and 2 induced with X rays, allele 3 with ENU. *T(2;3)82Fi*$^{2}$, 57A10-B1;82F10-83A1. Pre-larval lethal.

*l(3)82Fj:* one allele induced with X rays, pre-pupal lethal. *Ab(het;3R)82Fj*$^{1}$, het;83A1+.

*l(3)82Fk:* one allele induced with ENU, leaky late pupal/eclosion lethal.

**Other mutations recovered from X rays:**

*Df(3L)ru-22,* 61F8;62A3-5. Detected because the *Df(3R)3-4* chromosome used carried *ru*$^{1}$ although this wasn't indicated on its label.

*In(3LR)Sai*$^{1}$, 69D2-6;84E12-F3. Dominant outheld wings, recessive lethal allele of the *mirr* complementation group = *mirr*$^{Sai1}$.

*Sai*$^{2}$, dominant outheld wings; no cytological defect, maps genetically to 3-37.9 relative to *h* and *th.* Recessive lethal allele of the *mirr* complementation group = *mirr*$^{Sai2}$.

*Sai*$^{1}$, *Sai*$^{2}$, *D*$^{1}$, *D*$^{3}$, and *mirr*$^{DH-1}$ (homozygous viable hypomorphic *mirr* allele) fail to complement each other; *Sai*$^{1}$ is the strongest allele, then *D*$^{1}$ = *Sai*$^{2}$, then *D*$^{3}$. *D*$^{3}$/*mirr*$^{DH-1}$ is nearly completely viable, though with mild head defects and missing bristles.

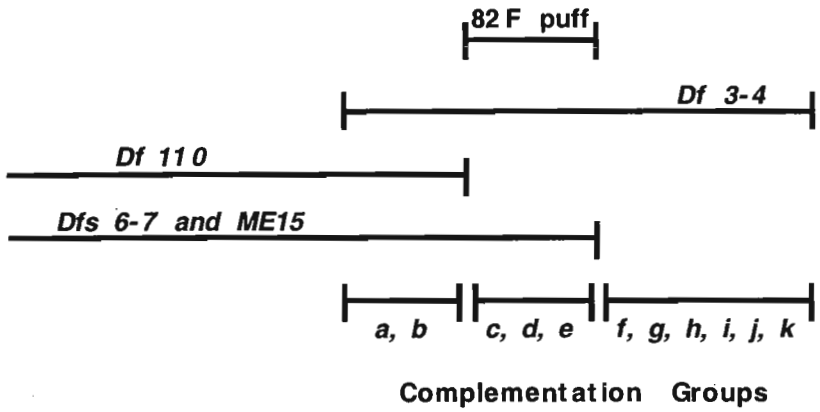


Figure 1.

Other mutations recovered from ENU: saw several, kept only a scarlet (= *st*$^{33}$), again detected because the *Df(3R)3-4* chromosome carried a *st* allele that wasn't indicated on its label.

Thirty-five mutations across 11 complementation groups = 3.2 hits per gene on average; although the distribution of numbers of hits per gene observed is very far off that expected from the Poisson distribution, that distribution predicts that the number of lethally- or visibly-mutable genes missed is 0.5.

## New lethal mutations in the 97B1-10 to 97D13 region of the *Drosophila melanogaster* 3rd chromosome.

**Ambrose R. Kidd III, Dhea Tolla, and Michael Bender.** Department of Genetics, University of Georgia, Athens, GA 30602.

In F2 EMS screens for mutations in the *dPC2* gene, we recovered sixteen lethal mutations and one visible mutation over *Df(3R)Tl-X* and *Df(3R)ro80b*. Together, the deficiencies cover the 97B1-10 to 97D13 region and overlap in the 97D1-2 region (Anderson *et al.*, 1985; Knibb *et al.*, 1993). Nine lethal mutations and the visible mutation fail to complement both deficiencies and thus map to the 97D1-2 region that includes the *dPC2* gene. These mutations are described elsewhere (D.T., A.R.K., and M.B., manuscript in preparation). Three (*dt6, dt12, dt14*) of the remaining 7 mutations recovered in our screens fail to complement *Df(3R)Tl-X* but complement *Df(3R)ro80b* and therefore are located beween 97B1-10 and 97D1 (Figure 1). The *dt6, dt12,* and *dt14* mutations fail to complement one another and also fail to complement *l(3)673*, a previously identified lethal in the region (K. Anderson, unpublished). These mutations have recently been shown to be allelic to

*scribble* (Bilder and Perrimon, personal communication). Four mutations recovered in our screens fail to

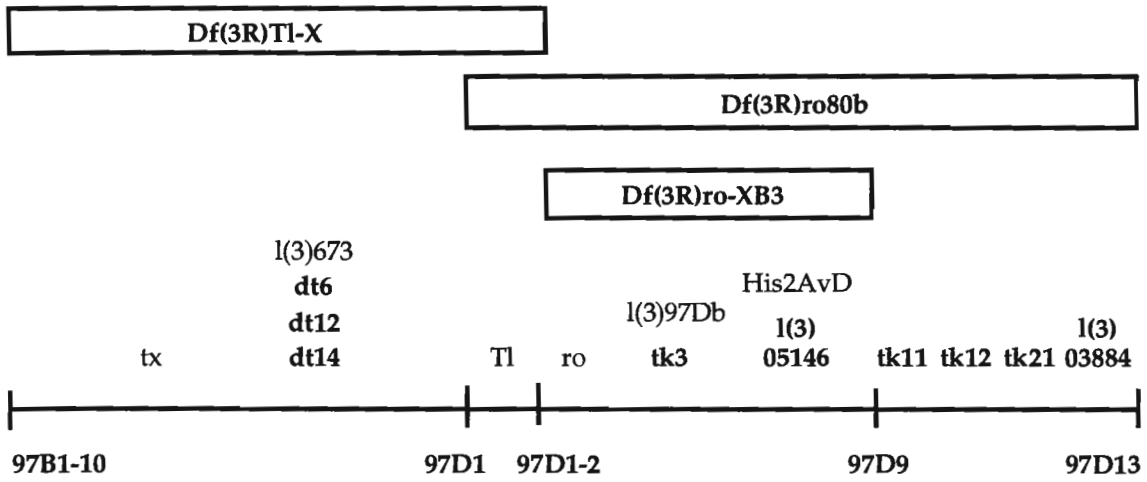


Figure 1. New lethal mutations in the 97B1-10 to 97D13 interval. The seven lethal EMS-induced mutations reported here and two lethal P element insertions are shown in bold type. Three previously identified lethal mutations (K. Anderson, unpublished; van Daal and Elgin, 1992; Knibb *et al.*, 1993) are shown in light type. Three other mutations (*taxi*, *tx*; *Toll*, *Tl*; and *rough*, *ro*) are also shown in light type for reference. The extents of *Df(3R)Tl-X*, *Df(3R)ro80b* and *Df(3R)ro-XB3* are shown by open bars at the top. Cytological positions determined from deficiency endpoints are indicated below the vertical hatch marks.

complement *Df(3R)ro80b* but complement *Df(3R)Tl-X* and therefore are located between 97D1-2 and 97D13 (Figure 1). One of these (*tk3*) fails to complement *Df(3R)ro-XB3*, a deficiency removing 97D2-9 (Knibb *et al.*, 1993). The *tk3* mutation fails to complement *l(3)97Db* (Knibb *et al.*, 1993). The other three mutations (*tk11, tk12, tk21*) complement *Df(3R)ro-XB3* and therefore are located between 97D9 and 97D13. The *tk11*, *tk12* and *tk21* mutations complement one another, defining three separate genes in this region (Figure 1).

Four lethal P-element insertions have been mapped within or near the 97B1-10 to 97D13 region by *in situ* hybridization [*l(3)neo59*, 97C/D (Cooley *et al.*, 1988), *l(3)03077*, 97C1-2; *l(3)05146*, 97D3-6; *l(3)03884*, 97D6-9 (Spradling *et al.*, 1995)]. The *l(3)03884* mutation complements *Df(3R)Tl-X* and *Df(3R)ro-XB3* but fails to complement *Df(3R)ro80b* and therefore maps to the 97D9 to 97D13 region (Figure 1). The *l(3)03884* mutation complements *tk11*, *tk12* and *tk21*, indicating that it defines a separate gene in this region. The *l(3)05146* mutation complements *Df(3R)Tl-X* but fails to complement *Df(3R)ro80b* and *Df(3R)ro-XB3* and therefore maps to the 97D1-2 to 97D9 region (Figure 1). The *l(3)05146* mutation fails to complement *His2AvD*, a lethal mutation that maps to this region (van Daal and Elgin, 1992). The other two P element insertion mutations complement *Df(3R)Tl-X* and *Df(3R)ro80b* and therefore map outside the 97B1-10 to 97D13 region defined by *Df(3R)Tl-X* and *Df(3R)ro80b* or carry lethal mutations unlinked to the P element insertion mapped by in situ hybridization.

Acknowledgments: We thank K. Anderson, B. Wakimoto, D. Seikhaus, and K. Matthews and the Bloomington Stock Center for providing stocks. Work in our laboratory is supported by a grant from the NIH (#GM53681) to M.B. D.T. was supported by a Howard Hughes summer undergraduate research fellowship and a Barry M. Goldwater Scholarship.

References: Anderson, K.V., G. Jurgens, and C. Nusslein-Volhard 1985, Cell 42: 779-789; Cooley, L., R. Kelly, and A. Spradling 1988, Science 239: 1121-1128; Knibb, W.R., R.G. Tearle, A. Elizur, and R. Saint 1993, Mol. Gen. Genet. 239: 109-114; Spradling, A.C., D. Stern, I. Kiss, J. Roote, T. Laverty, and G. M. Rubin 1995, Proc. Natl. Acad. Sci. USA 92: 10824-30; van Daal, A., and S.C.R. Elgin 1992, Mol. Biol. Cell 3: 593-602.