The six inserts running in succession on chromosome 3L of *sc147-14* (Figure 1c) might be resulting from crossing-over (site 66A in *Cy/Pm;D/Sb* is polymorphic and hence our explanation is still consistent). The region in question lies off In(3L)69D3-E1;70C13-D1.

Therefore, it would be good to know how the transposable elements are distributed in the balancing line and balanced lines. It would be then easy to foresee crossing-over effects on the polymorphism in derivatives and to estimate correctly the frequencies of TE transpositions in between-line crosses of that sort. This could be of especial importance when TEs alone are markers of genomic regions in experiments of populational genetics, for example, in studying correlations between the pattern of TE distributions and the profile of alteration of polygenic traits.

Our data, however, are insufficient to say by which exact mechanism crossing-over takes place here. For example, recombination frequency may be dependent on whether inserts, especially multiple ones, are present at the same sites of the homologous chromosomes of the lines being crossed. Constructions of that kind either make chromosomes easily breakable and repairable at the homologous regions of TEs or allow exchange at long terminal repeats of TEs (McGinnis and Beckendorf, 1983; Goldberg *et al.*, 1983; Davis *et al.*, 1987; Tsubota *et al.*, 1989).

Acknowledgments: This work was supported by a grant from the Russian State Program "Frontier in Genetics". We are thankful to Vladimir Filonenko for translating this manuscript from Russian into English.

References: Davis, P.S., *et al.*, 1987, Proc. Nat. Acad. Sci. 84:174-178; Furman, D.P., *et al.*, 1993, Genetika (Russ.) 29:6; Goldberg, M.L., *et al.*, 1983, Proc. Nat. Acad. Sci. USA 80:16; Lindsley, D.L. and G.G. Zimm 1990, Dros. Inf. Serv. 68; MacIntyre, R.J., and T.R.F. Wright 1966, Dros. Inf. Serv. 41:141; McGinnis, W., and S.K. Beckendorf 1983, Nucl. Acids Res. 11:3; Tsubota, S.I., *et al.*, 1989, Genetics 122:881-890.

Jarman, M.G., and J.I. Collett. School of Biological Sciences, University of Sussex, Brighton, Sussex, BN1 9QG, U.K. Siting the genes of *Dipeptidase-B (Dip-B),* several 'recessive-lethal' complementation groups and the breakpoints of chromosomal deficiencies within the region 87F12- 88B1 of *D. melanogaster.*

This analysis was undertaken to establish the chromosomal site of *Dip-B* and to assess the possibility of association of mutant alleles of the complementation groups of *l(3)88Aa* and *Ac*, isolated by Mortin *et al.* (1992), with the expression of the *Dip-B* gene. At the outset of this analysis the status of the mapping of *Dip-B* was as follows. Electrophoretic analysis of heterozygotes of electrophoretic variants of *Dip-B* in *D. pseudsoobscura* (Collett, unpublished) had indicated a homotetrameric structure and Onishi and Voelker (1981) had shown a map position within the region 87F12-88C1 of Df(3R)red-31. Subsequent aneuploid mapping of about 80% of the genome by Hall (1983) confirmed the position of *Dip-B* at this site and failed to reveal any other affecting *Dip-B* expression. Following the screen for recessive lethal mutations in the region of Df(3R)P52 (88A3-88B3), Mortin *et al.* (1992) assigned mutations to a number of complementation groups throughout the region including those of *l(3)88Aa* and *Ac*. Several mutant alleles of these two genes had shown some sign of affecting Dip-B activity in heterozygotes. Here, following definition of deficiency breakpoints, *Dip-B*, three previously identified genes and at least one new gene are assigned to sites throughout the region, and the identity of one of these genes found by both mutation and transposon insertion is established.

Compiled in Table 1 are the chromosomal deficiencies used here, the methods and sources of identification of their breakpoints and the results of complementation analysis to confirm, to define and to refine the positions of their breakpoints. All deficiencies and mutant alleles were maintained in stocks with either the 'balancer' TM3 or TM6B. Included among these are two, M36 and M49, which were thought to be small deficiencies by Mortin *et al.* (1992) on the basis of the complementation pattern. This expectation was confirmed here, as indicated below, and thus their inclusion in Table 1. The alleles designated M and Z (Figure 1) were isolated by Mortin *et al.* (1992) and supplied by M. Mortin. The allele *k43* of l(3)*88*Ab was supplied by A. Shearn and the transposon induced allele of *l(3)03477, j1D1*, was supplied by the *Drosophila* Genome Center (Baltimore).

Complementation was deemed to have failed when no unmarked flies were found among at least 100 progeny from more than one cross, or, in several cases, when results in smaller progenies were consistent with the progenies of similar crosses of flies with other deficiencies or with other alleles within a complementation group (Jarman, 1997). Among the M and Z mutants, each was complementation-tested in pairwise matings. *k43* was tested with each M allele, and similarly, the allele of *l(3)03477* was tested with both M and Z mutants. These alleles are listed in Figure 1 in complementation groups according to the results of these tests. Those listed are consistent with Mortin *et al.*'s (1992) assessment with the exception of the mutant M30. This was found to be in the region of Df(3R)P1. But, in addition, these analyses revealed allelism between the M mutants of *l(3)88Aa* and the P-transposon induced allele *j1D1*. Thus the

Table 1. Complementation of Deficiencies Demarcating Chromosomal Region 87F12 - 88B1

		Deficiencies, Df(3R):									
Df(3R):	Assignment of Breakpoints	126c	urd	red-31	$293^{\gamma 5}$	$293^{\gamma 7}$	red-P52	M49	M36	su(Hw)[7]	red-P1
126c	87E1-2; 87F11-12(c)[1]	*		+	+	+	+				
urd	87F1; 87F15(dfh)[2]		*	-		+					
red-31	87F12-14(c)[1]; 88C1-3(c)[1]			*	-	-	-				
$293^{\gamma 5}$	87E2; 88A5-6(m)[3,4,5]				*	-	-	-	+	+	+
$293^{\gamma 7}$	88A1(m,g)[4,5]; 88A4-6(m,g)[4,5]					*	-	-	+	+	+
red-P52	88A3-4(g)[3]; 88B3(m)[6]						*	-	-	-	-
M49	88A3-4(ca); 88A6-7(ca)							*	-		+
M36	88A5-6(ca); 88A8-10(ca)								*	+	+
su(Hw)[7]	88A9(c)[7]; 88B3(m)[9]									*	-
red-P1	88B1(dfh,m)[8,9]; 88D3-4(c)[2]										*

c, cytological observation of polytene chromosomes; dfh, complementation analysis of deficiency heterozygotes; g, *in situ* hybridised genic DNA excluded from/included in deficiency region; m, molecularly-defined breakpoint; ca, complementation analysis presented here.
1. W.M.Gelbart cited by Hall and Kankel (1976). 2. Lindsley and Zimm (1992). 3. Dalton *et al.* (1989). 4. R.Kelley, pers. comm.
5. synonym: fs293gamma7. 6. Parkhurst *et al.* (1988). 7. R.Coyne, pers. comm. 8. Hamilton *et al.* (1993). 9. Breen and Harte (1991). The sources of stocks carrying the Deficiencies were as follows: 126c and red-31 , Umea *Drosophila* Center; urd,red-P52, su(Hw)[7], red-P1 Bloomington *Drosophila* Stock Center; $293^{\gamma 5 \text{ and } 7}$, R.Kelley; M49 and M36, M. Mortin. '+' indicates complementation and '-' its failure.

formerly identified gene named *P03477* is one and the same as *l(3)88Aa* and should be named according to its earlier mutational identification.

The positioning of the 'recessive lethals' *88*Aa, *Ab, Ac* and *Ad* follows from the landmark positioning of the *P*-alleles of *l(3)88Aa* by *in situ* hybridisation to88A4-5 (Spradling *et al.,* 1995). The argument runs as follows. Since M36 and *88Ac* mutants complement the 293 Deficiencies, their positions are distal to 88A6. Since M49 fails to complement alleles at the locus 88A4-5 as well as M36, it must be a deficiency. Then, since M36 fails to complement both M49 and the *88Ac* mutants, it too must be a deficiency. Thus the loci of these two complementation groups are restricted to the regions indicated in Figure 1. However, the failure of M36 and M49 to complement also indicates at least one more 'recessive lethal' complementation group in the region of their overlap. Following Mortin *et al.*'s(1992) earlier surmise and naming, *88Ad* should be recognised to exist within the cytological region 88A6-8, and the locus of *88Ac* must be to its right and bounded distally by the chromosomal region of Df(3R)su(Hw)[7]. Finally, the position of the complementation group *l(3)88Ab* is defined by its inclusion in the region of Df(3R)P52 and its exclusion from the region of Df(3R)M49. These relationships are laid out in the map presented in Figure 1.

The assignment of the *Dip-B* gene to a chromosomal site was made by the visualisation of Dip B activity following electrophoretic separation of preparations of flies heterozygous for a null allele of *Dip-B* (in a stock of *raised* flies) and each of the first six deficiencies listed in Table 1. Using the method of separation and identification of Dip B activity (Collett, 1989), null and active alleles were identified in at least 4 samples of each heterozygous genotype consisting of three 4-10 day old adult females, maintained at 18°C. until sampling. The results in every sampling were consistent with the deficiency breakpoints indicated in Figure 1. Thus *Dip-B* is within the region of 87F12-15.

In establishing the demarcation of the chromosomal region 87F12-88B1 (Bridges, 1938) by a set of deficiency breakpoints at intervals throughout the region, the map positions of Dip B and four 'recessive lethal' genes have now been defined within chromosomal segments equivalent to lengths of DNA cloned in P1-plasmids. These deficiencies should continue to be useful in similarly defining the map sites of other genes, identified and not yet identified, throughout the region. All stocks carrying mutant alleles and the deficiencies used here (except Df(3R)M49, now lost) are, or are soon to be, available in a *Drosophila* stock Center.

References: Breen, T.R., and P.J. Harte 1991, Mech.Dev. 35:113-127; Bridges, C.B., 1935, J. Hered. 29:11-13; Collett, J.I., 1989, Insect Biochem. 19:535-547; Dalton, D., R.Chadwick and W. McGinnis 1989, Genes and Dev. 3:1940-1956; Hall, J.C., and D.R. Kankel 1976, Genetics 83:517-535; Hall, N.A., 1983, *A Genetic and Developmental Analysis of Peptidases in* Drosophila melanogaster, pp. 96-97, D.Phil. thesis, University of Sussex, U.K.; Heino, T.I., A.O. Saura and V.Sorsa 1994, Dros. Inf. Serv. 73:619-738; Hamilton, B.J., M.A. Mortin and A.L. Greenleaf 1993, Genetics 134:517; Hall, J.C. and D.R.Kankel 1976, Genetics 83:517-529. Jarman, M.G., 1997, *Characterisation of the*

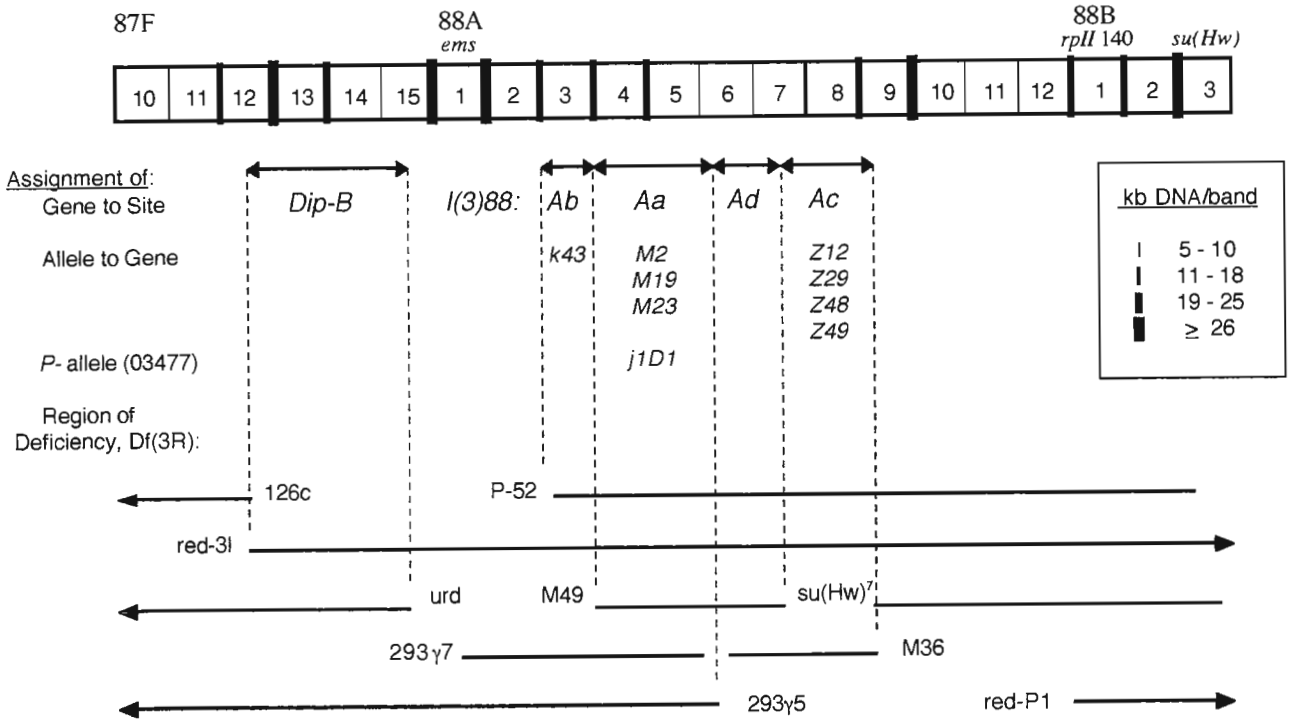

Figure 1. Sites of *Dipeptidase*-B and *l(3)88Aa-Ad* in the region 87F12-88B. The widths of the vertical demarcations of polytene bands represent the constituent amounts of DNA (Heino *et al.*, 1994), as indicated. The genes *ems, rpII140* and *su(Hw)*, assigned to single polytene bands (Flybase, 1997) and used in establishing the positions of deficiency breakpoints are also indicated. Heavy lines indicate the regions of deficiency (see Table 1 and text). Arrows indicate breakpoints in adjoining regions. The M and Z alleles and the *P* - allele *j1D1* of *l(3)03477*, located at 88A4-5 by *in situ* hybridisation (Spradling *et al.*,1995), were assigned by deficiency complementation analysis to the 'recessive lethal' genes *l(3)88Aa-Ac*. In addition, failure of the deficiencies M49 and M36 to complement indicates at least one further 'recessive lethal' gene site, designated l(3)88*Ad*. The vertical lines are guides to the boundaries of the regions of each gene as defined by deficiency breakpoints.

Contribution of the Dipetidases to the Life History of Drosophila, pp.30-41, D.Phil. thesis, University of Sussex, U.K.; Lindsley, D.L., and G.G. Zimm 1992, *The Genome of* Drosophila melanogaster, p. 876, Academic Press, New York; Mortin, M.A., R. Zuerner, S. Berger, and B.J. Hamilton 1992, Genetics 131:895-903; Onishi, S., and R.A. Voelker 1981, Biochem. Gen. 19:75-85; Parkhurst, S.M., D.A. Harrison, M.P. Remington, C. Spana, R.L. Kelley, R.S. Coyne, and V.G. Corces 1988, Genes and Dev. 2:1205-1215; Spradling, A.C., D.M.Stern, I. Kiss, J.Roote, T. Laverty, and G.M. Rubin 1995, Proc. Nat. Acad. Sci. 92:10824-10830.

Crowley, Thomas E. Department of Biological Sciences, Columbia University, code 2407, 1212 Amsterdam Ave., New York, NY. email: tc45@columbia.edu; phone, 212-854-4835; fax, 212-865-8246. Mutations near the *Trf* cluster cause a premeiotic defect in the *Drosophila* male germ line.

Abstract:

In situ hybridization, P transposase-mediated mutagenesis, and stage-specific markers are used to examine the regulation of expression and function of the *Trf* cluster during spermatogenesis in *Drosophila melanogaster*. The temporal regulation of the presence of the three mRNAs during sperm development is determined, and the effect of various mutations at the *Trf* site on the premeiotic stages is described. The molecular nature of each mutation is then established, and possible functions of *Trf* cluster gene products are discussed, in particular a potential role in cell cycle regulation is suggested.