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Complementation tests using *P*-induced mutations place *adipose* in 55B.

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Recent recombination data (Doane, 1999) placed the *adipose* (*adp*) locus in *Drosophila melanogaster* at ~0.6 cM centromere-distal to the region on the genetic map of chromosome 2*R* that contains the genes *stauffen* (*stau*) and *Polycomblike* (*Pcl*). These data also located *adp* approximately 1 map unit proximal to the female sterility locus renamed *maternal metaphase arrest* (*mama*; synonyms, *fs(2)adp* or adp^{fs} and *fs(2)lto5DF6*). The position of *adp* on the polytene chromosome map remained uncertain, however, because of discrepancies among the reported breakpoints for some of the chromosomal deficiencies used to locate it cytologically (summarized by Doane, 1999).

The smallest deletion known to uncover the recessive mutant phenotypes of both *adp* and *mama* mutations in deficiency heterozygotes is associated with the chromosomal inversion $In(2R)Pcl^{11}$ (Doane, 1994, 1999). This Pcl^{11} deletion lies at the centromere-proximal breakpoint of the inversion (Doane, 1994) and hence in 55A4 (see FlyBase 2000). The Pcl^{11} deletion fails to complement the lethal *P* element induced mutations $stau^{ry9}$ and Pcl^{P1}, but does complement a mutant allele of *three-rows* (Doane, 1999). The latter gene is now assigned to 54F4-55A1 (FlyBase 2000). Therefore, the proximal breakpoint for the Pcl^{11} deletion is fairly well defined genetically and cytologically, but its distal limit remains to be determined. Nevertheless, publication of *P* element-disrupted vital sites in this part of the genome (Spradling *et al.*, 1999), coupled with complementation data presented below, have helped to define the distal limit of the Pcl^{11} deletion. They also are consistent with *adp* being located within 55B, as had been estimated previously by the Berkeley *Drosophila* Genome Project.

Table 1 gives the results of a complementation analysis that tested the gene mutations listed in the second column over the Pcl^{11} deletion. (All of these mutations complement mutant alleles of *adp* or *mama*.) The first five strains were obtained from the Bloomington *Drosophila* Stock Center; each contained a lethal allele induced by a single *P* element insertion. Results for $stau^{ry9}$ were previously reported (Doane, 1999). The *fj* mutant (presumably fj^{1}) has been in my laboratory for many years, and was derived from a stock formerly maintained at Yale University. The rest of the lethal stocks were part of the Tearle collection maintained by the European *Drosophila* Stock Centre of Umea, Sweden. They were received in April of 1999 and included alleles of the genes *l(2)PC4-B* through *l(2)PC4-Q*, as listed, plus $l(2)PC4\text{-}A^{139}$. Upon arrival, all of the Tearle mutants were tested over *Df(2)PC4* because they originally had been isolated over that deficiency. However, the stock presumed to contain $l(2)PC4\text{-}A^{139}$ complemented *Df(2)PC4* in my hands, so it was not tested further. The remaining Tearle lethals were crossed *inter se* to verify they represented separate genes before subjecting them to complementation tests over the Pcl^{11} deletion.

Table 1. Complementation Analysis of Mutations Tested Over the *Pcl*^{11} Deletion.

Gene	MutationTested	Polytene Site	Phenotype of *Pcl*^{11} Heterozygotes[1]
stauffen (*stau*)	*stau*^{ry9}	55B5	Lethal
Polycomblike (*Pcl*)	*l(2)s1859*	55B5-55B6	Lethal
polyA-binding protein (*pAbp*)	*l(2)k10109*	55B5-55B6	Lethal
Heat shock-factor (*Hsf*)	*l(2)03091*	55B5-55B6	Lethal
lola like (*lolal*)	*l(2)k02512*	55B5-55B10	Lethal
four-jointed (*fj*)	*fj*^{1}	55C1-55C2	Wild type
lethal(2)PC4-B	*l(2)PC4-B*^{110}	54F6-55B12	Lethal
lethal(2)PC4-D	*l(2)PC4-D*^{202}	55A1-55F1-2?	Wild type
lethal(2)PC4-E	*l(2)PC4-E*^{119}	55A1-55F1-2?	Wild type
lethal(2)PC4-F	*l(2)PC4-F*^{198}	55A1-55F1-2?	Wild type
lethal(2)PC4-G	*l(2)PC4-G*^{223}	55A1-55F1-2?	Wild type
lethal(2)PC4-H	*l(2)PC4-H*^{236}	55A1-55F1-2?	Lethal
lethal(2)PC4-K	*l(2)PC4-K*^{363}	55A1-55F1-2?	Wild type
lethal(2)PC4-M	*l(2)PC4-M*^{420}	55A1-55F1-2?	Wild type
lethal(2)PC4-P	*l(2)PC4-P*^{41}	55A1-55F1-2?	Wild type
lethal(2)PC4-Q	*l(2)PC4-Q*^{267}	55A1-55F1-2?	Wild type

[1]Each test was repeated at least two or three times by crossing strains in column 2 to a *SM1, Cy/Df(2R)Pcl*^{11} stock and scoring their progeny. All flies were reared at 25° C. on a standard cornmeal-molasses-brewers yeast diet. After emerging from pupal cases, F_1 flies were aged 6-8 days before being classified and counted. Mutant phenotypes which were scored included lethality, female sterility and appropriate visible traits that included relative amount of lipid stores in fat bodies. Progeny counts ranged from ~75 to over 100 flies for vial cultures and several hundred for bottle crosses.

The updated location (Spradling *et al.*, 1999; FlyBase 2000) for each gene on the polytene chromosome map of 2R is provided in column 3 of Table 1. The region containing *fj* was estimated to be 55C1-2 (FlyBase 2000), but the others were determined by deletion analysis or *in situ* hybridization studies. Although the breakpoints of *Df(2R)PC4* at 55A1 and 55F1-2, define the overall chromosomal region containing lethal genes in the Tearle collection, assignment to subregions within this span remain unclear for the most part (*c.f.* Tearle, 1996, and FlyBase 2000).

The last column in Table 1 gives the results of the complementation analysis. All of the *P*-induced lethals, including those for *stau*, *Pcl*, *Hsf*, *pAbp*, and *lolal*, failed to complement the lethality of *Pcl*^{11}. Thus, in addition to *adp* and *mama*, these genes appear to be located within the boundaries of the deletion associated with *In(2R)Pcl*^{11}. By contrast, *fj*^{1}/*Pcl*^{11} heterozygotes displayed a wild phenotype, implying that *fj* is positioned beyond the distal boundary of the *Pcl*^{11} deletion. It follows that the right end of the *Pcl*^{11} deletion is proximal to 55C1-2, if the estimated location for *fj* is correct. This would place *adp*, as well as *mama,* between 55B5-6 and 55C1-2.

Only two of the Tearle lethals in Table 1 were uncovered by the *Pcl*^{11} deletion, namely *l(2)PC4-B*^{110} and *l(2)PC4-H*^{236}. This suggests that the genes *l(2)PC4-B* and *l(2)PC4-H* lie between 55A4 and 55C1-2, the proposed limits of this deletion. The remaining Tearle lethals were viable over *Pcl*^{11} and therefore appear to be located outside of the region between 55A4 and 55C1-2.

Now that DNA sequencing of the *Drosophila* genome has been completed (see Science, Vol. 287, issue for March 24, 2000), the major task of identifying the function(s) for every gene identified solely by its molecular characteristics has begun. Determination of the sequential order of known genes on the polytene chromosome map can greatly assist in this task. The sequential order for some of the genes addressed in this study, namely *stau - Pcl - pAbp - adp - mama - fj*, has been determined (Doane, 1999, and unpublished data), but the position of other genes in Table 1 relative to them is

still unknown. Ultimately the order for all of these genes must be superimposed upon the transcription units defined by sequencing the genome in this region.

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Mutation Notes — Other Species

Mapping of two visible mutations in *D. pseudoobscura bogotana.*

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In 1961, a subspecies of *D. pseudoobscura* was discovered in an isolated area of Colombia. Females of this subspecies, when crossed to mainland or "USA" males, produce sterile hybrid male progeny. Previously, we possessed visible markers only in the USA subspecies of *D. pseudoobscura.* Here I present data describing and mapping two visible markers in the Bogota subspecies: *white* (*w*) and *winglet* (*wgl*).

The white-eye marker arose spontaneously in the wildtype Bogota-ER stock and was found to be X-linked recessive. In the first experiment, Bog *w* females were crossed to males from a USA *w* stock. The production of sterile male progeny confirmed that Bog *w* is indeed a Bogota strain and not the result of contamination from USA. Also, the appearance of all white-eyed daughters proved that Bog *w* is allelic to USA *w*. By homology, we thus place the Bog *w* locus at 1-80.5.

The *winglet* marker arose spontaneously in a male from the Toro #1 Bogota stock, a wildtype line that was kindly provided by M. Noor. The wings of *winglet* males and females are very small, dusky in color, and often held away from the body. Subsequent crosses showed that this trait is X-linked, recessive, and causes a slight reduction in viability.

In the first of two mapping crosses, Bog *wgl* virgin females were crossed to Bog *w* males, and F1 females were crossed to their *wgl* brothers. Data from 1277 male F2 progeny showed that *w* and *wgl* recombine at a frequency of 9.7%.

In a second cross, Bog *wgl* virgin females were crossed to the multiply-marked USA stock carrying *ct* (1-22.5), *sd* (1-43.0), *y* (1-74.5), and *se* (1-156.5) (see Orr, 1995). Virgin F1 females were backcrossed to the multiply-marked stock. Progeny were scored relative to the *y* and *wgl* markers only. In 1110 backcross males, *wgl* and *y* recombined at a frequency of 17.1%. Taking these results together, we place the *wgl* locus to the right of *w* at 1-90.2.

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