courtship song (Burnet *et al.*, 1977), and male competitive mating success and female choice (Markow, 1987).

This newly-constructed al^1; th^1 stock is available from the Long Laboratory at the University of California – Irvine.

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Novel X ray induced chromosome aberrations in *Drosophila melanogaster.*

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Introduction

We have made use of the new collections of P element insertion lines (Salzberg *et al.*, 1997; Rorth, 1996) in order to induce deletions in chromosomal regions of the *Drosophila* genome not previously uncovered by deficiencies.

Material and Methods

To obtain deficiencies in specified regions of the X and 3rd chromosomes of *Drosophila*, we used stocks with P-element insertions at the following sites; 16E3-5, 16F1-4, 18D1-4, 76B5-11, 76D, 76F, 83A5-9, 83B and 98C. We used the following stocks from the European Drosophila Stock Center in Umeå, Sweden (H) and the Bloomington Drosophila Stock Center (P):

#H1 *y[1] w[1]; P{w[+mC]=lacW}l(3)S000101[S000101]/TM3, Sb[1] Ser[1]*
(*In situ* hybridization site: 83B)

#H11 *y[1] w[1]; P{w[+mC]=lacW}l(3)S000606[S000606]/TM3, Sb[1] Ser[1]*
(*In situ* hybridization site: 76D)

#H52 *y[1] w[1]; P{w[+mC]=lacW}l(3)S003108[S003108]/TM3, Sb[1] Ser[1]*
(*In situ* hybridization site: 98C)

#H1403 *y[1] w[1]; P{w[+mC]=lacW}l(3)S083207[S083207]/TM3, Sb[1] Ser[1]*
(*In situ* hybridization site: 76B5-11)

#H1437 *y[1] w[1]; P{w[+mC]=lacW}l(3)S085401[S085401]/TM3, Sb[1] Ser[1]*
(*In situ* hybridization site: 76D)

#H1573 *y[1] w[1]; P{w[+mC]=lacW}l(3)S096511[S096511]/TM3, Sb[1] Ser[1]*
(*In situ* hybridization site: 83A5-9)

#H2144 *y[1] w[1]; P{w[+mC]=lacW}l(3)S133801[S133801]/TM3, Sb[1] Ser[1]*
(*In situ* hybridization site: 76F)

#H2145 *y[1] w[1]; P{w[+mC]=lacW}l(3)S133804[S133804]/TM3, Sb[1] Ser[1]*

(*In situ* hybridization site: 76F)
#P141 *w[1118] P{w[+mC]=EP}EP970* (Insertion site: 016F01-04)
#P1443 *w[1118] P{w[+mC]=EP}EP1322* (Insertion site: 016E03-05)
#P1458 *w[1118] P{w[+mC]=EP}EP1455* (Insertion site: 018D01-04).

Three-days old males were γ-irradiated with doses of 2,000 or 2,500 rad and immediately crossed with virgin females (a) from the same stock, if the P-element was inserted on the 3rd chromosome (Figure 1A) or (b) from a *y w ct m f* stock, if the P-element was located on the X chromosome (Figure 1B).

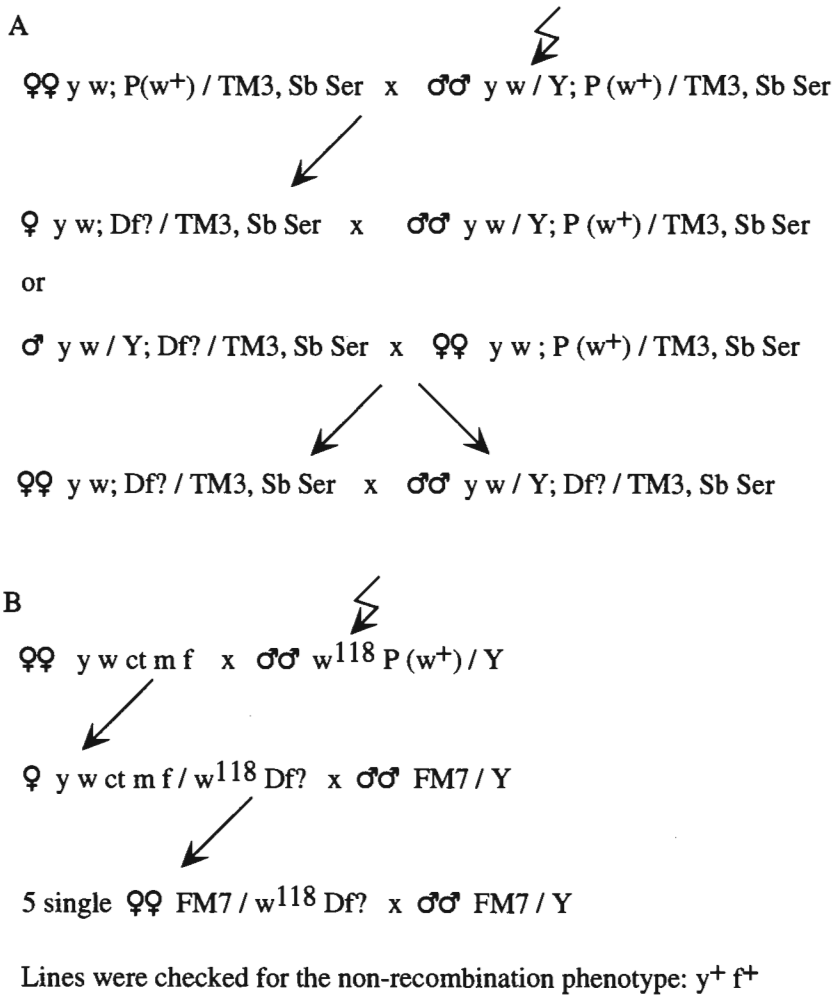

Figure 1. Crossing schemes for isolation of A) 3rd chromosome and B) X-chromosome deficiencies.

Using the schemes delineated in Figure 1, 23 white-eyed lines originating from the stocks with P-elements on the 3rd chromosome and 14 white-eyed lines originating from stocks with P-elements on the X chromosome were established. To check these lines for deficiencies, cytological preparations of salivary gland chromosomes were made and stained with Giemsa (according to Ashburner, 1989).

Results

We were able to map chromosomal aberrations in a total of 15 white-eyed lines (Table 1). Fifteen lines, 8 originating from the 3rd chromosome and 7 from the X chromosome insertion lines, appeared to have normal polytene chromosomes. In the remaining 7 lines the chromosomes were impossible to analyze due to multiple aberrations.

Table 1.

Aberration	Cytology
1. In(3L)RR1	In(3L)76F; het(3)
2. In(3L)RR3	In(3L)76F; het(3)
3. In(3L)RR10	In(3L)69F; het(3)
4. In(3L)RR17	In(3L)76D; 74AB
5. Df(3L)RR20	Df(3L)76D4; 76E
6. Tp(3L; het(?))RR29	Tp(3L; het(?))66E; 71F-72A; het(?)
7. In(3L)RR32 + T(3R; 4)RR32	In(3L)76F; het(3) + T(3R; 4)92F; 102E-F
8. In(3R)RR33 + T(1; 3R)RR33	In(3R)83A; het(3) + T(1; 3R)5D; 100A
9. Df(3R)RR38	Df(3R)98C; 98F1
10. T(1; 3R)RR39	T(1; 3R)6C; 100C
11. In(3L)RR41	In(3R)83A; het(3)
12. In(3L)RR44	In(3R)83B; het(3)
13. Df(1)RR62	Df(1)3C; 3D
14. In(1)RR63	In(1)16F; het(1)
15. Df(1)RR79	Df(1)16C; 16F

het(1) and het(3) – heterochromatin of chromosomes 1 and 3, respectively
het(?) - heterochromatin of undetermined nature

There was a considerable variation in the occurrence of white-eyed offspring between the different P-insertion lines. Loss of the *white* marker among the offspring in two lines with P insertions on X, #P141 and #P1458, differed from 0.030% (12 in 40,000 offspring) to 0.079% (11 in 14,000 offspring), which is statistically significant ($P = 0.008$).

Discussion

In four of the mapped lines the aberrations were localized to regions other than the site of the *white* marker P-insertion; In(3L)RR10, Tp(3L;het(3))RR29, T(1;3R)RR39 and Df(1)RR62. This indicates that multiple events have occurred, which is also evident from the characterization of the other lines.

60% of the aberrations were found to have one breakpoint in centric heterochromatin. This seems to be a high figure compared to the report by Mukhina *et al.* (1981) and might be due to a preference of P-elements to localize close to possible heterochromatic nuclear domains.

Some chromosomal regions seem to be refractory to X-ray induced mutations, *e.g.*, chromosomal region 76B, where strain P1403 has a P-element insertion. In this strain we scored over 60,000 offspring and found no white-eyed individuals. Since the insert is very close to the *M(3)76A* locus, induced excisions of and aberrations in this gene might cause haplo-insufficiency. Alternatively, this line actually contains more than one P-element, and a single *white* marker deletion is not possible to score.

In conclusion, P-element insertions with readily visible markers can be used to screen for chromosomal aberrations in specific regions, but deletions constitute only a small fraction of the chromosomal rearrangements recovered. We also conclude that two novel deficiencies were isolated in regions that are not uncovered in the currently available deficiency kits; 16C;16F and 98C;98F1. All stocks are available from the European *Drosophila* Stock Center in Umeå.

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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.