Phenotypes Associated with Second Chromosome P Element Insertions in Drosophila melanogaster

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ABSTRACT In Drosophila melanogaster, P element transposition has been a productive means of insertional mutagenesis. Thousands of genes have been tagged with natural and engineered P element constructs. Nevertheless, chromosomes carrying P element insertions tend to have high levels of background mutations from P elements inserting and excising during transposition. Consequently, the phenotypes seen when P element-bearing chromosomes are homozygous are often not attributable to the P insertions themselves. In this study, 178 strains in the Bloomington Drosophila Stock Center collection with P insertions on the second chromosome were complementation tested against molecularly defined chromosomal deletions and previously characterized single-gene mutations to determine if recessive lethality or sterility is associated with the P insertions rather than background mutations. This information should prove valuable to geneticists using these strains for experimental studies of gene function.

During P element transposition, it is common for a P element to insert into a chromosome and immediately excise, leaving a mutational footprint, before inserting into the genome in its final position (Cooley et al. 1988, Kania et al. 1995, Deak et al. 1997, Salzberg et al. 1997, Spradling et al. 1999, Ashburner et al. 2005). These “hit and run” events often disrupt genes; consequently, the phenotypes seen when insertion-bearing chromosomes are made homozygous cannot necessarily be attributed to the disruption of genes where P elements are located. Proof that a phenotype is associated with a P element insertion usually comes from reverting the phenotype upon transposase-mediated excision of the P element or showing that the P insertion fails to complement a loss-of-function mutation in the gene where the P insertion is found.

Most P element insertions from early Drosophila transposition screens were given symbols that reflected the phenotypes seen when the chromosomes were made homozygous (Spradling et al. 1999). For example, P[lacW]fl(2)k01209k01209 was named as a recessive lethal on the second chromosome (“fl(2)”), while other insertions were named for phenotypes such as female sterility (fs) and male sterility (ms). The Berkeley Drosophila Genome Project put considerable effort into verifying the phenotypes of P insertions by complementation testing the insertions against chromosomal deletions, other P insertions, and previously characterized loss-of-function mutations (Spradling et al. 1999). In addition, many labs have investigated the phenotypes of particular insertions. Nevertheless, the purported phenotypes of hundreds of P insertions from early screens have never been confirmed, and insertions from more recent screens have generally been given phenotype-neutral symbols. In the study described here, we analyzed 178 second chromosome P insertions with potentially misleading symbols in the Bloomington Drosophila Stock Center collection to determine whether the insertions are responsible for recessive lethal or sterile phenotypes.

MATERIALS AND METHODS

Our approach was straightforward: we identified chromosomal deletions that encompass the insertion sites of P elements, made complementation crosses between deletion and P insertion stocks, and scored the appropriate progeny classes for lethality, female
Table 1  Insertions with no associated lethal or sterile phenotype

| Original Symbol | Previous Mapping | Complementing Deletions and Mutations | Phenotype<sup>b</sup> | New Insertion Symbol | New Allele<sup>c</sup> |
|-----------------|-----------------|---------------------------------------|----------------------|----------------------|----------------------|
| P{lacW}rackk15001 | in situ | Dr(2)BSC341 | v | P{P}rackk15001 | CR1403<sup>c</sup> |
| P{lacW}pakb15001 | seq | Dr(2)BSC160, Dr(2)BSC2247 | v | P{P}pakb15001 | CR1403<sup>c</sup> |
| P{hsneofs}(2)neo<sup>9</sup> | in situ | Dr(2)U144 | ff | P{hsneofs}(2)neo<sup>9</sup> | |
| P{hsneofs}(2)neo<sup>12</sup> | in situ | Dr(2)ED2247 | ff | P{hsneofs}(2)neo<sup>12</sup> | |
| P{CP38-Adh}mfs(2)1 | in situ | Dr(2)BED1473, Dr(2)BED1378 | mf, ff | P{CP38-Adh}mfs(2)1 | CR1403<sup>c</sup> |
| P{lacW}Wk(2)k16406 | seq | Dr(2)ED3181 | v, h | P{lacW}k16406 | |
| P{lacW}Wk(2)k16215 | seq | Dr(2)BRSC693 | v | P{lacW}k16215 | |
| P{lacW}Wk(2)k08407 | seq | Dr(2)RBC482 | v | P{lacW}k08407 | |
| P{lacW}Wk(2)k03110 | seq | Dr(2)RBC313 | v | P{lacW}k03110 | |
| P{lacW}Wk(2)k03111 | seq | Dr(2)RBC131 | v | P{lacW}k03111 | |
| P{lacW}Wk(2)k03112 | seq | Dr(2)RBC326 | v | P{lacW}k03112 | |
| P{lacW}Wk(2)k03609 | seq | Dr(2)Exel7142 | v | P{lacW}k03609 | |
| P{lacW}Wk(2)k03610 | seq | Dr(2)Exel145 | v | P{lacW}k03610 | |
| P{lacW}Wk(2)k03620 | seq | Dr(2)Exe17142 | v | P{lacW}k03620 | |
| P{lacW}Wk(2)k05420 | seq | Dr(2)Exe3385 | v | P{lacW}k05420 | |
| P{lacW}Wk(2)k05421 | seq | Dr(2)Exe4582 | v | P{lacW}k05421 | |
| P{lacW}Wk(2)k06420 | seq | Dr(2)Exel7142 | v | P{lacW}k06420 | |
| P{lacW}Wk(2)k07005 | seq | Dr(2)Exe17142 | v | P{lacW}k07005 | |
| P{lacW}Wk(2)k07015 | seq | Dr(2)Exe17142 | v | P{lacW}k07015 | |
| P{lacW}Wk(2)k07127 | seq | Dr(2)Exe17142 | v | P{lacW}k07127 | |
| P{lacW}Wk(2)k07142 | seq | Dr(2)Exe17142 | v | P{lacW}k07142 | |
| P{lacW}Wk(2)k08002 | seq | Dr(2)Exe17142 | v | P{lacW}k08002 | |
| P{lacW}Wk(2)k08003 | seq | Dr(2)Exe17142 | v | P{lacW}k08003 | |
| P{lacW}Wk(2)k08004 | seq | Dr(2)Exe17142 | v | P{lacW}k08004 | |
| P{lacW}Wk(2)k08005 | seq | Dr(2)Exe17142 | v | P{lacW}k08005 | |
| P{lacW}Wk(2)k08006 | seq | Dr(2)Exe17142 | v | P{lacW}k08006 | |
| P{lacW}Wk(2)k08115 | seq | Dr(2)Exe17142 | v | P{lacW}k08115 | |
| P{lacW}Wk(2)k09202 | seq | Dr(2)Exe17142 | v | P{lacW}k09202 | |
| P{lacW}Wk(2)k09221 | seq | Dr(2)Exe17142 | v | P{lacW}k09221 | |
| P{lacW}Wk(2)k09610 | seq | Dr(2)Exe17142 | v | P{lacW}k09610 | |
| P{lacW}Wk(2)k09854 | seq | Dr(2)Exe17142 | v | P{lacW}k09854 | |
| P{lacW}Wk(2)k09920 | seq | Dr(2)Exe17142 | v | P{lacW}k09920 | |
| P{lacW}Wk(2)k09922 | seq | Dr(2)Exe17142 | v | P{lacW}k09922 | |
| P{lacW}Wk(2)k09924 | seq | Dr(2)Exe17142 | v | P{lacW}k09924 | |
| P{lacW}Wk(2)k10003 | seq | Dr(2)Exe17142 | v | P{lacW}k10003 | |
| P{lacW}Wk(2)k10004 | seq | Dr(2)Exe17142 | v | P{lacW}k10004 | |
| P{lacW}Wk(2)k10113 | seq | Dr(2)Exe17142 | v | P{lacW}k10113 | |
| P{lacW}Wk(2)k10127 | seq | Dr(2)Exe17142 | v | P{lacW}k10127 | |
| P{lacW}Wk(2)k10217 | seq | Dr(2)Exe17142 | v | P{lacW}k10217 | |
| P{lacW}Wk(2)k10609 | seq | Dr(2)Exe17142 | v | P{lacW}k10609 | |
| P{lacW}Wk(2)k10815 | seq | Dr(2)Exe17142 | v | P{lacW}k10815 | |
| P{lacW}Wk(2)k11120 | seq | Dr(2)Exe17142 | v | P{lacW}k11120 | |
| P{lacW}Wk(2)k11206 | seq | Dr(2)Exe17142 | v | P{lacW}k11206 | |
| P{lacW}Wk(2)k11307 | seq | Dr(2)Exe17142 | v | P{lacW}k11307 | |
| P{lacW}Wk(2)k11311 | seq | Dr(2)Exe17142 | v | P{lacW}k11311 | |
| P{lacW}Wk(2)k11404 | seq | Dr(2)Exe17142 | v | P{lacW}k11404 | |
| P{lacW}Wk(2)k11405 | seq | Dr(2)Exe17142 | v | P{lacW}k11405 | |
| P{lacW}Wk(2)k13211 | seq | Dr(2)Exe17142 | v | P{lacW}k13211 | |
| P{lacW}Wk(2)k13617 | seq | Dr(2)Exe17142 | v | P{lacW}k13617 | |
| P{lacW}Wk(2)k14206 | seq | Dr(2)Exe17142 | v | P{lacW}k14206 | |
| P{lacW}Wk(2)k15617 | seq | Dr(2)Exe17142 | v | P{lacW}k15617 | |
| P{lacW}Wk(2)k16213 | seq | Dr(2)Exe17142 | v | P{lacW}k16213 | |
| P{lacW}Wk(2)k16406 | seq | Dr(2)Exe17142 | v | P{lacW}k16406 | |
| P{lacW}Wk(2)k16919 | seq | Dr(2)Exe17142 | v | P{lacW}k16919 | |
| P{ZP}Z03050 | seq | Dr(2)Exe17142 | v | P{ZP}Z03050 | |
| P{ZP}Z03497 | seq | Dr(2)Exe17142 | v | P{ZP}Z03497 | |
| P{ZP}Z03605 | seq | Dr(2)Exe17142 | v | P{ZP}Z03605 | |
| P{ZP}Z03686 | seq | Dr(2)Exe17142 | v | P{ZP}Z03686 | |

(continued)
sterility, or male sterility. Assessing P insertions that had previously been localized to a specific genomic sequence often required only a single deletion cross. P elements that had previously been localized by in situ hybridization to polytene chromosomes; seq, insertion mapped from sequence of insertion site. 

When an insertion lies in the region of gene overlap, FlyBase uses the symbol of one gene in the P insertion symbol, and lists associated alleles for the other genes.

Data availability
Changes to FlyBase entries have been coordinated with this report and will appear in a 2016 FlyBase update. Strains may be obtained from the Bloomington Drosophila Stock Center.

RESULTS AND DISCUSSION
Table 1 shows the results of complementation tests with 95 P element stocks that indicate the recessive phenotypes seen when insertion chromosomes are made homozygous do not map to the P insertions themselves. Based on these results, we updated most of the insertion symbols. If the P insertion was positioned between genes, then the symbol was updated to show the insertion as an allele implying no phenotypes. For example, P[PZ]l(2)10333 was changed to P[PZ]l(2)10491. We

### Table 1, continued

| Original Symbol | Previous Mapping | Complementing Deletions and Mutations | Phenotype | New Insertion Symbol | New Allele |
|-----------------|------------------|--------------------------------------|-----------|----------------------|------------|
| P[PZ]l(2)R20H1.220 | seq | Dr(2)BSC142 | v | P[PZ]dec14.220 | |
| P{lacW}l(2)2978 | seq | Dr(2)E6768, Dr(2)BSC203 | v | P{lacW}Wyst2978 | |
| P{lacW}K63 | seq | Dr(2)BSC290 | v | P{lacW}K63 | |
| P{lacW}l(2)1878 | seq | Dr(2)Exel1762, PCNA2735, PCNA32470, PCNA2735 | ff | P{lacW}PCNA1878 | |

**Table 1** shows the results of complementation tests with 95 P element stocks that indicate the recessive phenotypes seen when insertion chromosomes are made homozygous do not map to the P insertions themselves. Based on these results, we updated most of the insertion symbols. If the P insertion was positioned between genes, then the symbol was updated to show the insertion as an allele implying no phenotypes. For example, P[PZ]l(2)10333 was changed to P[PZ]l(2)10491. We
Table 2 Insertions causing lethal phenotypes

| Original Symbol | Noncomplementing Deletions and Mutations | Phenotype | Complementing Deletions | New Insertion Symbol |
|-----------------|----------------------------------------|-----------|------------------------|---------------------|
| P{lacW}lola<sup>409901</sup> | lola<sup>20642</sup>, lola<sup>ORC4</sup> | | | |
| P{lacW}{l(2)}k00705<sup>g00705</sup> | betaTub566<sup>YC0063</sup> | | | |
| P{lacW}{l(2)}k05911<sup>h05911</sup> | CG31728<sup>BD2493</sup>, Df(2R)BSC727, Df(2L)BSC768 | | | |
| P{PZ}{l(2)}b(2)02024<sup>b2024</sup> | Ef1alpha48D<sup>00275</sup>, Df(2R)BSC329, Df(2R)BSC79 | | | |
| P{lacW}{l(2)}k09848<sup>g09848</sup> | CG7845<sup>a00845</sup>, Df(2R)RED1482 | | | |
| P{PZ}{l(2)}b(2)01855<sup>b1855</sup> | ds(2)LAC071, Df(2L)Exel8003 | | | |
| P{lacW}{l(2)}k13905<sup>x13905</sup> | Df(2L)ugl<sup>-17C</sup> | | | |
| P{lacW}{l(2)}k14807<sup>y14807</sup> | Adfl<sup>11489</sup>, Df(2R)RDE1552 | | | |
| P{PZ}{l(2)}b(2)03563<sup>b03563</sup> | Df(2R)BSC668, Df(2R)RED2354 | | | |
| P{PZ}{l(2)}b(2)05287<sup>y05287</sup> | CG12050<sup>KG03759</sup>, Df(2L)BSC302, Df(2L)Exel7080 | | | |
| P{lacW}{l(2)}k10502<sup>h10502</sup> | Letm1<sup>M02224</sup>, Df(2R)RED4061 | pl | | P{lacW}Letm<sup>1x10502</sup> |
| P{lacW}LeuRS<sup>S0501</sup> | LeuRS<sup>03210</sup>, Df(2L)ucm-P2 | | | |
| P{lacW}Ranbp1<sup>sh977</sup> | Ranbp1<sup>b217</sup>, Df(2R)BSC99 | | | |
| P{lacW}Arpc4<sup>Sh1036</sup> | Arpc4<sup>a00819</sup>, Df(2L)Exel6015 | | | |
| P{lacW}Wbsh<sup>Sh181</sup> | Df(2L)BSC149, Df(2L)Exel8038 | | | |
| P{PZ}{l(2)}b(2)08770<sup>y08770</sup> | Ttd14<sup>VE01823</sup>, Ttd14<sup>KG03769</sup>, Df(2R)BSC335 | l/pl | | P{lacW}Ttd14<sup>d04808</sup> |
| P{PZ}{l(2)}b(2)08770<sup>y08770</sup> | Ttd14<sup>KG03769</sup> | | P{lacW}Ttd14<sup>d04808</sup> | |
| P{PZ}{l(2)}b(2)11068<sup>y110685</sup> | Df(2L)Exel6004, Df(2L)Exel108 | pl | | P{lacW}Ttd14<sup>d08770</sup> |
| P{PZ}{l(2)}b(2)10333<sup>l10333</sup> | Df(2L)Exel8038 | | | |

<sup>a</sup>Phenotype in complementation tests: l, lethal; pl, partially lethal; l/pl, lethal in combination with deletion and partially lethal in combination with other insertions.

<sup>b</sup>Results map lethality to lola in agreement with Bass et al. (2007), and not to region 94A as suggested by old sequence data.

<sup>c</sup>Unpublished results from the Gene Disruption Project showed failure to complement P{lacW}{l(2)}b(2)01068<sup>y010682</sup> (http://flybase.org/reports/FBrf0008159.html).

updated 47 symbols in this way. Since the lethality or sterility of these insertion chromosomes does not map to the insertion, and there is no way to know if it is the result of a single mutation or multiple mutations without further tests, the original gene and allele entries have been eliminated from FlyBase. For example, the l(2)10333 and l(2)k09610 allele entries have been eliminated. The gene and allele entries for noninsertion mutations were retained only when the phenotypes of the mutations have been characterized beyond simple lethality or sterility, or when the mutations are members of multiallele complementation groups. For example, P{lacW}bg88407 now represents the site of a nonlethal P insertion, and l(2)k08407<sup>y08407</sup> represents the linked noninsertion lethal mutation formerly confounded under P{lacW}{l(2)}k08407<sup>y08407</sup>. In this case, it is necessary to have an allele entry for the noninsertion l(2)k08407<sup>y08407</sup> mutation because there is presumably a single mutation causing the imaginal disc abnormalities described in Roch et al. (1998) for the homozygous chromosome.

Table 2 shows the results of complementation tests with 18 stocks mapping recessive lethality to the P insertions. In these cases, strong arguments can be made for the lethality arising from disruption of the gene associated with the P element insertion based on noncomplementation with deletions and other mutations in the gene, or from the position of the P insertion relative to genetic regions. For example, complementation tests with a deletion for the region of dachous (ds) and a ds point mutation showed that the lethality of P{PZ}l(2)101855<sup>y1855</sup> is attributable to ds disruption. This allowed the insertion symbol to be revised to P{PZ}d<sup>y1855</sup>.

Table 3 shows the results of complementation tests with 44 P element stocks that indicate lethality maps to the P insertion itself or to a closely linked site. Confidence that a P insertion is responsible for a phenotype and that the phenotype is not attributable to a hit-and-run mutation tightly linked to the P insertion increases as the size of the interval defined by noncomplementing deletions decreases. Although past experience has shown that noncomplementation with any deletion strongly predicts that phenotypes are caused by P insertions (Spradling et al. 1999), we cannot formally associate lethality with these insertions from our deletion crosses alone. For this reason, insertions within the transcribed regions of genes have been renamed to reflect their physical position, separate alleles have been named to represent the recessive lethality, and the respective FlyBase entries indicate their mapping to the same mutational event. For example, the insertions formerly called P{lacW}{l(2)}b(2)08601<sup>y08601</sup> and Lec4<sup>2</sup>, Df(2R)BSC335, the coincident or closely linked recessive lethal mutation is denoted l(2)k08601<sup>y08601</sup>, and FlyBase entries indicate the two genetic elements may be the same entity. We made 28 such changes (Table 3, top section). No symbol updates were needed for nine other insertions within the transcribed portions of genes (Table 3, middle section). The seven insertions that lie outside the transcribed portions of genes (Table 3, bottom section) retain their original insertion names, because it is standard practice for FlyBase not to represent such insertions with separate symbols for insertions and lethal loci.

Table 4 shows the results of complementation tests that allowed us to refine the mapping of 21 P insertions that had previously been mapped to polytene chromosome bands by in situ hybridization. Because we have no sequence localizations for these insertions, we cannot assign symbols reflecting their proximity to annotated genes. Consequently, they retain their original symbols indicating they are members of genetically defined, recessive lethal, or sterile complementation groups.

In summary, we have clarified the basis for phenotypic effects seen in 18 P element insertion stocks in the Bloomington Stock Center collection. This has allowed us to give the stocks
Table 3 Insertions with coincident or closely linked lethal mutations

| Original Symbol | Noncomplementing Deletions and Mutations | Phenotypea | New Insertion Symbol | New Allele Symbolsb | New Lethal Allele |
|-----------------|------------------------------------------|------------|----------------------|---------------------|------------------|
| P{lacW}l(2)SH0499 | Df(2)Rl(2)BSC334, Df(2)Rl(2)Jexel7153 | v-l | P{lacW}WEd(9)SH0499 | CG42518SH0499 | (2)SH0499SH0499 |
| P{lacW}l(2)AJ0016 | Df(2)Rl(2)BSC271 | | P{lacW}l(2)CG8078K00116 | CG3378Al2206 | (2)AJ0016AJ0016 |
| P{lacW}l(2)k02206 | Df(2)Rl(2)BSC402 | | P{lacW}l(2)CG3378S2206 | CG3378Al2206 | (2)k02206k02206 |
| P{lacW}l(2)k03201 | Df(2)Rl(2)BSC296, Df(2)Rl(2)BSC354 | | P{lacW}WSec6ia6203 | (2)k03201k03201 | |
| P{lacW}l(2)k04003 | Df(2)Rl(2)ED235 | | P{lacW}WSec6ia6203 | (2)k04003k04003 | |
| P{lacW}l(2)k04308 | Df(2)Rl(2)BSC281, Df(2)Rl(2)BSC303 | | P{lacW}Wgenek04308 | (2)k04308k04308 | |
| P{lacW}l(2)k05448 | Df(2)Rl(2)BSC290, Df(2)Rl(2)BSC30, Df(2)Rl(2)BSC302, Df(2)Rl(2)BSC68, Df(2)Rl(2)BSC68, Df(2)Rl(2)BSC68, Df(2)Rl(2)BSC68, Df(2)Rl(2)BSC68 | | P{lacW}Wgenek05448 | (2)k05448k05448 | |
| P{lacW}l(2)k06204A06204 | Df(2)Rl(2)BSC158, l(2)kD020406204 | | P{lacW}Sec24ABk06204 | CR45467k06204 | (2)k06204A06204 |
| P{lacW}l(2)k06205A06205 | Df(2)Rl(2)BSC158, l(2)kD0204A06205 | | P{lacW}Sec24ABk06205 | CR45467k06205 | (2)k06205A06205 |
| P{lacW}l(2)k06502A06502 | Df(2)Rl(2)BSC158, l(2)kD0204A06502 | | P{lacW}l(2)G11030k06502 | CR45467k06502 | (2)k06502A06502 |
| P{lacW}l(2)k07215A07215 | Df(2)Rl(2)BSC158, l(2)kD0204A07215 | | P{lacW}l(2)G924d07215 | CR45467k07215 | (2)k07215A07215 |
| P{lacW}l(2)k07408A07408 | Df(2)Rl(2)BSC359 | | P{lacW}AsnRs407408 | (2)k07408A07408 | |
| P{lacW}l(2)k08601A08601 | Df(2)Rl(2)ED2308 | | P{lacW}WMeF2k08601 | (2)k08601A08601 | |
| P{lacW}l(2)k09328A09328 | Df(2)Rl(2)BSC245 | | P{lacW}l(2)CG17574k09328 | (2)k09328A09328 | |
| P{lacW}l(2)k10105A10105 | Df(2)Rl(2)Exel7077 | | P{lacW}WGenek10105 | (2)k10105A10105 | |
| P{lacW}l(2)k10371A10371 | Df(2)Rl(2)BSC360 | | P{lacW}Wic4k10371 | (2)k10371A10371 | |
| P{lacW}l(2)k12402A12402 | Df(2)Rl(2)BSC280 | | P{lacW}Wgenek12402 | (2)k12402A12402 | |
| P{lacW}l(2)k13014A13014 | Df(2)Rl(2)BSC289 | | P{lacW}Wgenek13014 | (2)k13014A13014 | |
| P{lacW}l(2)k13142A13142 | Df(2)Rl(2)BSC280 | | P{lacW}Wgenek13142 | (2)k13142A13142 | |
| P{lacW}l(2)k13604A13604 | Df(2)Rl(2)BSC289 | | P{lacW}Wgenek13604 | (2)k13604A13604 | |
| P{lacW}l(2)k16204A16204 | Df(2)Rl(2)Exel7164 | | P{lacW}Wgenek16204 | (2)k16204A16204 | |
| P{lacW}l(2)k16805A16805 | Df(2)Rl(2)Exel7164 | | P{lacW}Wgenek16805 | (2)k16805A16805 | |
| P{lacW}l(2)k17002A17002 | Df(2)Rl(2)Exel7164 | | P{lacW}Wgenek17002 | (2)k17002A17002 | |

Insertion and lethality may not be separable, but separate insertion and lethal locus named.

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less ambiguous genotypes, and to revise several potentially misleading FlyBase entries. We expect this information to be valuable to Drosophila geneticists using these insertions in experiments examining the effects of disrupting specific gene regions.

Phenotype in complementation tests: I, lethal; pl, partially lethal; v-l, homozygous viable but lethal in combination with deletions.

When an insertion lies in the region of gene overlap, FlyBase uses the symbol of one gene in the P insertion symbol and lists associated alleles for the other genes.

Original allele symbol.
Table 4 Insertions with refined mapping

| Original Symbol | Noncomplementing Deletions | Phenotype | Complementing Deletions | Refined Mapping |
|-----------------|-----------------------------|-----------|------------------------|----------------|
| P[PZ]l(2)43Bb04614a | Df(2R)BSC263 | l/fs | Df[2R]BSC106, Df[2L]ED19, Df[2L]ED50001 | 43A4–B2, 2R:7326951–7395885 |
| P[PZ]l(2)02836a02836a | Df(2R)Exel6063, Df[2L]Exel7080 | l/fs | Df[2L]BSC264, Df[2R]BSC106, Df[2L]ED19, Df[2L]ED50001 | 21B1–3, 2L:72671–159063 |
| P[hsneo]l(2)neo4 | Df(2R)BSC263, Df[2R]ED1715 | l/fs | Df[2R]BSC264, Df[2L]ED19, Df[2L]ED50001 | 40A5–D3, 2L:21828252–22019106 |
| P[hsneo]l(2)neo5 | Df(2L)Exel6049 | l | Df[2L]BSC213 | 55D1–E2, 2R:18503870–18621522 |
| P[lacW](2)21E4 | Df(2R)Exel7066 | l/fs | BSC213 | 21E4–F1, 2L:1074079–1158137 |
| P[lacW](2)120685 | Df[2L]Exel6004 | l | Df[2R]BSC213 | 32B1–C1, 2L:10809118–11001451 |
| P[BS2.7B]l(2)32B1C4 | Df[2R]Exel6054, Df[2R]Exel7092 | l | Df[2L]BSC213 | 43E9–12, 2R:7665795–7708707 |
| P[lacW](2)52B8A1604b | Df[2L]BSC302, Df[2L]Exel7080 | l | Df[2R]BSC213 | 39A1–2, 2L:21070044–21102742 |
| P[rny]l(2)rny | Df(2L)BSC107 | l/fs | Df[2L]BSC213 | 21C2–E2, 2L:431096–574741 |
| P[rny]l(2)111 | Df(2L)BSC107 | l | Df[2L]BSC213 | 21C2–E2, 2L:431096–574741 |
| P[PZl](2)125026 | Df(2R)Exel6063 | l | Df[2R]BSC213 | 52F6–53C4, 2R:16187888–16368515 |
| P[PZl](2)003832a003832a | Df(2R)Exel1466 | l | Df[2R]BSC213 | 39E7–40A5, 2L:21676796–21828548 |
| P[PZl](2)04535b04535b | Df[2L]ED1466 | l | Df(2L)BSC213 | 42A4–C7, 2R:6236062–6746030 |
| P[PZl](2)43Bb04614a | Df(2R)BSC263 | l | Df(2L)BSC213 | 42F2–43A4, 2R:7148684–7326951 |
| P[lacW](2)125026b125026b | Df(2R)Kr10 | l | Df(2R)BSC213 | 60F2–S, 2R:25061964–25288936 |

a Phenotype in complementation tests: l, lethal; l/fs, lethal with female sterile escapers; ms, male sterile.
b Figure 2 in Han et al. (2003) suggests insertion lies to the right of Df[2L]BSC103.

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