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果蠅族群中染色體逆位多態性和隱性不良因子累積之相關性

Association between Chromosomal Inversion Polymorphisms and Accumulation of Recessive Deleterious Mutations in *Drosophila melanogaster*

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本論文係董詩凡君（r99b44005）在國立臺灣大學生態學與演化生物學研究所完成之碩士學位論文，於民國 100 年 7 月 5 日承下列考試委員審查通過及口試及格，特此證明

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摘要

在自然族群中，染色體逆位多態性被認為和物種的適應有高度關係。目前認為族群維持染色體逆位多態性的機制，是透過其攜帶具選汰優勢的一組基因，因逆位異型合子抑制遺傳重組，而被同時保留在族群裡造成共適應的現象。然而，對於染色體逆位異型合子抑制重組造成的不良因子累積並增加突變負荷很少受到討論。不良因子的累積會隨著逆位同結合型在族群中被消除，進而增加逆位異型合子在族群裡的頻率。黃果蠅的熱帶非洲族群，同時具有高度的染色體逆位多態性和高頻的隱性不良因子累積，此現象提供了一個相當適合的研究材料。藉由定位隱性不良因子在染色體上累積的位置是否會真如預期在重組被高度抑制的位置，例如染色體逆位的端點，來探討染色體逆位和不良因子之間的關係，我們利用重組定位和基因缺失組定位出隱性不良因子累積的位置，結果在分別來自八個不同單雌系的八條三號染色體上定位出十四個不同的隱性致死因子，並且所有的致死因子和染色體逆位端點相距非常近，只有不到三個厘摩根。同時，隱性致死因子分佈在染色體上的位置和族群內的逆位多態性相當一致。本研究顯示隱性不良因子的累積受到染色體逆位多態性所抑制的重組率影響，在染色體上呈現不隨機的分佈。同時，也暗示染色體逆位和隱性不良因子之間強烈的相關性。

關鍵字：遺傳負荷、染色體逆位端點、穆勒氏棘輪、隱性致死因子、遺傳重組抑制。

Abstract

Chromosomal inversion polymorphisms have been demonstrated to play adaptive roles in natural populations by capturing local co-adapted alleles within recombination-suppressed regions of inversion heterozygotes. On the other hand, a less studied but important role of inversion polymorphisms is that recombination suppression by heterozygous chromosomal rearrangement may accumulate recessive deleterious mutations and thus cause a great amount of mutation load. Deleterious mutations will be eliminated when homozygotes and in turn increase heterozygosity of various inversions. The Afrotropical population of *Drosophila melanogaster* with high chromosomal inversion heterozygosity and high ratio of recessive lethals provides an ideal material to test any association between them by examining the accumulation pattern of recessive lethals. Recessive lethals are predicted to locate near the inversion breakpoints where the recombination is greatly suppressed by inversion heterokaryotypes. By using recombination and deficiency mappings, 14 recessive lethal alleles from eight lethal-bearing third chromosomes (each from eight distinct isofemale lines, respectively) were identified. All of recessive lethals were mapped into the regions close to (less than 3 centi-Morgan) the inversion breakpoints which were polymorphic in the African population. This result clearly shows that recessive lethal alleles are accumulated by recombination suppression and distributed

non-randomly along inverted chromosomes. The data also provide the strong association between chromosomal inversions and the accumulation of recessive deleterious mutations.

Key words: genetic load, inversion breakpoint, Muller’s ratchet, recessive lethal, recombination-suppression

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Introduction

Chromosomal inversions have been recognized as an aberration in which the order of a chromosomal segment is reversed. Sturtevant (1921) showed and later Caron (1946) proved the mechanism that inversions suppress recombination by eliminating abnormal crossover products of meiosis (dicentric and acentric chromosomes) (Sturtevant 1921; Caron 1946). Many hypotheses were proposed to explain the evolutionary significance of chromosomal inversion polymorphisms in nature, and six main explanations for the spread of inversions in population (reviewed in Hoffmann and Rieseberg 2008). The most accepted explanation is the reduction of recombination by inverted heterozygotes, causing a set of locally adapted genes captured by inverted regions. Combinations of these alleles at loci within inversions are assumed to be co-adapted by having epistatic effects on fitness, and favored by natural selection toward heterozygotes (Dobzhansky 1937, 1970); or even without epistatic effects, only by migration-selection balance toward locally favored alleles in populations (Kirkpatrick and Barton 2006). Previous studies, especially in *Drosophila pseudoobscura*, showed that genetic frequencies of distinct chromosomal rearrangements are involved in seasonal cycles and altitudes changes (Dobzhansky 1947). Schaeffer *et al*. (2003) proved the genomic evidence for maintaining positive epistatic relationships among loci within inversions of *D. pseudoobscura*. In other

Drosophila species, many traits, including viability, longevity, resistance to thermal extremes, mating success and fecundity, have been demonstrated to be linked to inversion polymorphisms (*e.g.*, Barnes 1983; reviewed in Hoffmann *et al.* 2004). In *D. melanogaster*, molecular markers linked to inversion *In(3R)Payne* are highly associated with body size and heat resistance (Weeks *et al.* 2002; Anderson *et al*. 2003). Recent studies of other species also have revealed that selective advantages of specific locally adapted alleles are highly associated with various chromosomal rearrangements among different local environments, *e.g.*, butterfly *Heliconius numata* (Joron *et al*. 2011), malaria mosquito *Anopheles* (Ayala *et al*. 2011; Rottschaefer *et al*. 2011), and yellow monkey flower *Mimulus guttatus* (Lowry and Wills 2010). Those studies strongly support that chromosomal inversion polymorphisms are maintained by adaptively selective advantages in local populations. In addition, the recombination suppression effect by inversion heterozygotes might cause genetic barrier within and/or between populations. Hence, chromosomal inversion polymorphisms have been suggested as a driving force on population divergence and even speciation process (reviewed in Krimbas and Powell 1992; King 1993; Noor *et al.* 2001; reviewed in Rieseberg 2001; Navarro and Barton 2003; Hoffmann *et al.* 2004). Stevison *et al*. (2011) tested the gene flow along the large inverted region of two closely related *Drosophila* species hybrids, and found that the nucleotide divergence differs in low recombination rate

regions, suggesting that inversion polymorphisms might drive the population divergence.

Another mechanism, though less studied, was first described in *Drosophila pseudoobscura* populations is that the genetic load might contribute to inversion polymorphisms (Epling *et al*. 1961; Dobzhansky *et al*. 1963; Mayhew *et al*. 1966). The recombination-suppressed region tends to have a higher probability to accumulate deleterious mutations, which could hide in inversion heterozygotes from elimination by natural selection. Some population surveys in *Drosophila subobscura* showed that a higher level of genetic load occurs in the population having a higher frequency of chromosomal rearrangements (Andjelković *et al*. 1998; Zivanović *et al*. 2000), while other studies revealed the opposite relationship that the frequency of chromosomal rearrangements in populations had a negative correlation with levels of genetic load (Watanabe 1969; Watanabe and Yamazaki 1976). On the other hand, some studies showed the non-random distribution of low frequency of recessive deleterious mutations on inverted chromosomes in different *Drosophila* species (Crumpacker and Salceda 1969; Mestres *et al*. 1990; Yang *et al*. 2002). Although without direct evidence, these studies suggested that the genetic load might contribute to the complex mechanism affecting the dynamics of genetic polymorphism in natural populations. In this study, I want to understand the distribution pattern on the chromosome of genetic

load and how genetic load contributes to the polymorphism of inversion populations.

High chromosomal inversion polymorphisms (Dobzhansky 1937) with accumulation of recessive deleterious mutations (Sturtevant 1937; Dubinin 1946) were reported in *Drosophila*, including Afrotropical (including southern and eastern Africa) populations of *D. melanogaster* (Aulard *et al.* 2002). A recent study (S. Fang unpublished data; also see Appendix A) on the inversion polymorphism of Afrotropical populations found that: (1) high inversion polymorphisms especially on right arms of 3rd chromosomes; (2) high inversion heterozygosity; (3) high ratio of recessive lethal-bearing chromosomes. These observations provide us a good material to study the genetic load mechanism and to test whether there is any association between accumulation deleterious mutations and inversion chromosomal inversion polymorphisms. If there is no association between chromosomal structural polymorphisms and genetic loads, mutations might distribute randomly on chromosomes; otherwise mutations might distribute in a given pattern. From the feature of chromosomal inversions which reducing crossover events, it was predicted that recessive deleterious mutations on the third chromosome were to accumulate non-randomly on highly recombination-suppressed regions, and in turns, to increase inversion heterozygosity. With the advent of fine mapping tools, such as deficiency mapping in *Drosophila*, it is possible to answer this long-lasting unsolved question. In

this study, I used recombination and deficiency mappings to precisely map recessive lethal alleles to examine the relationship between the recombination-suppression by complex chromosomal inversions and the accumulation of recessive deleterious mutations in the Afrotropical population of *D. melanogaster*.

Materials and Methods

Fly strains

Drosophila melanogaster isofemale lines were collected and established from Harare and Sengwa, Zimbabwe (referred hereafter as ZH and ZS, respectively) in 1990 (Wu *et al.*,1995) and from Malawi (MW) by J.W.O. Ballard (Iowa University) in 2002. Third chromosomes from each isofemale line are extracted (Appendix A, see Figure A3) and balanced with the balancer chromosome, *TM3*, to keep recessive lethals and recessive sterile alleles in stocks. The multimarker line *rucuca* (stock number: 576) was obtained from Bloomington Drosophila Stock Center. This line contains eight visible markers on the third chromosome: *roughoid* (*ru*), *hairy* (*h*), *thread* (*th*), *scarlet* (*st*), *curled* (*cu*), *stripe* (*sr*), *ebony* (*e*), and *claret* (*ca*). The cytological and physical positions of these markers were described by Lindsey and Zimm (1992) and summarized in Figure 1. Another similar multimarker line *ruPrca* (stock number 1711) also obtained from Bloomington Drosophila Stock Center, contains a marker *Prickly* (*Pr*) addition to above eight. All the deficiency strains were obtained from Drosophila Genomics Resource Center, Bloomington (http://dgrc.cgb.indiana.edu/) or Drosophila Genetic Resource Center, Kyoto (http://kyotofly.kit.jp/cgi-bin/stocks/index.cgi) (Appendix B, Table B1). Those stocks contain different genomic deletions with accurate coordinates and span the entire third chromosome (details in FlyBase http://flybase.org/). Flies were reared on standard

cornmeal medium at 22℃ under a 12h-12h light-dark cycle.

Recessive lethal alleles mapping

To map recessive lethal alleles, eight third lethal-bearing chromosomes (four Straight and four *In(3R)K*) were chosen: ZS2-3, ZS30-2, ZH12-6, ZH32-1, ZS53-3, ZH18-6, ZH21-1, and MW6-3 (four former are straight, later four are *In(3R)K*). Each lethal-bearing chromosome was examined allelism by complementation test (Appendix A, Figure A4). Distinct recessive lethals from eight third lethal-bearing chromosomes were then mapped by recombination and deficiency mappings. For recombination mapping, the multimarker line *rucuca* was used to construct the recombinants to narrow down the regions of recessive lethal alleles (Figure 2). In the cross scheme of recombination mapping, various genotypic recombinants (single male, for lack of recombination) at generation G_2 were collected and then backcrossed with the original lethal-bearing line (Figure 2). By scoring the G_3 offspring, we could exclude the lethal-free chromosomal regions (Appendix B, Table B2).

For further deficiency mapping (Figure 3), all deficiency lines which cover the candidate region were chosen. By scoring F_1 offspring numbers to examine whether or not recessive lethal alleles fail to complement deletions, the location of recessive lethal alleles can be mapped to the precise genomic regions of deletions which might only cover several genes (Appendix B, Table B3). Thus, recessive lethals can be

mapped to the gene level by deficiency mapping.

Marker	*ru*	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*
Cytology	61F5-F5	66D10-D10	72D1-D1	73A3-A3	86D7-D7	90E4-F1	93C1-D1	99C2-C2
GD (cM)	0.0	26.5	43.2	44.0	50.0	62.0	70.7	100.7

Figure 1 The distribution of eight markers on the third chromosome of *rucuca* strain.

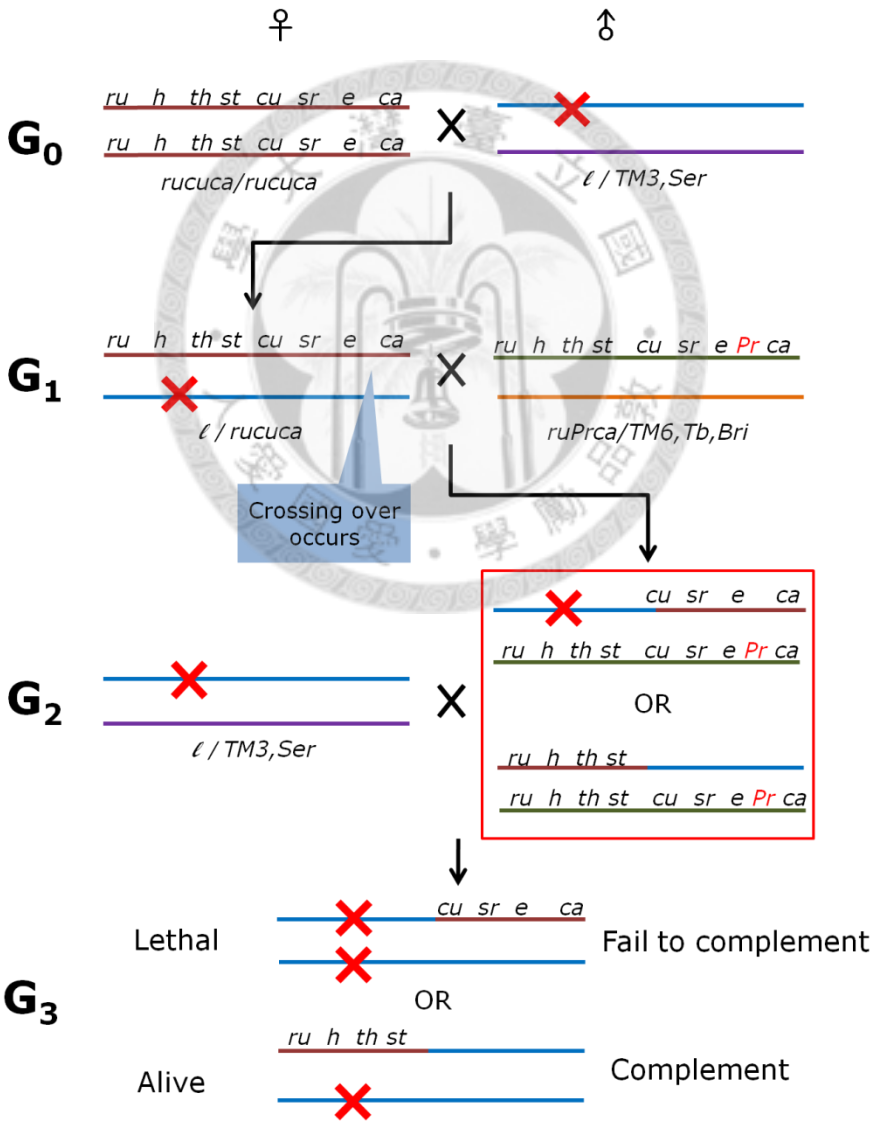

Figure 2 The cross scheme of recombination mapping. G_0, the *rucuca* strain cross with lethal-bearing chromosome lines. G_1, *rucuca*/lethal-bearing heterozygotes cross with *ruPrca*. Distinct recombinants male can be obtained from the G_1

rucuca/lethal-bearing female, then back cross with original lethal-bearing chromosome. By examining whether or not recombinants fail to complement lethal alleles (G_3), the rough regions of recessive lethals could be mapped. Red cross: recessive lethal allele.

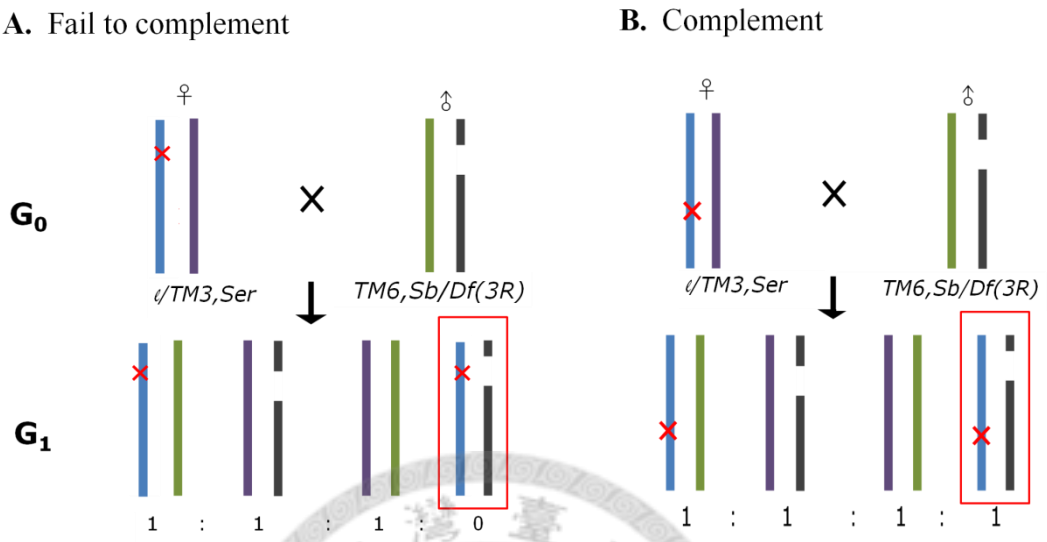

Figure 3 The cross scheme of deficiency mapping. These deletions with precise coordinates (empty space on the black bar) were utilized to narrow down the location of recessive lethal alleles to the gene level. Red cross: recessive lethal allele.

Estimation of genetic distance of recessive lethals and breakpoints of inversions

The genetic positions of recessive lethals and breakpoints of inversions were marked by genetic distance (cM) and were estimated by interpolation method as

$$\frac{\mathrm{R1}-\mathrm{R2}}{\mathrm{M1}-\mathrm{M2}}=\frac{\mathrm{RX}-\mathrm{R2}}{\mathrm{X}-\mathrm{M2}} \quad (1)$$

where M1 and M2 stand for genomic positions of the two nearest genetic markers, and R1 and R2 represent genetic positions of M1 and M2, respectively. X denotes the unknown genetic position of recessive lethals or inversion breakpoints. RX is the genomic position obtaining from experimental data. All the genomic position

information can be obtained from FlyBase.

Statistical test

With the help of Cheng-Ruei Lee, a R programming (Ihaka and Gentleman 1996) was used to test whether there is statistical significant in the distribution of recessive lethal alleles. The average true value of each distance between recessive lethals and nearest inversion breakpoints can be obtained by experimental results. I cut the 3rd chromosome (102 cM) into 1020 bins, each bins is 0.1 cM long. I then randomly sampled 14 locations on 3rd chromosome without replacement (1000 times), and also calculated the mean distance of each sample to nearest inversion breakpoints. I obtained the normal distribution from null hypothesis (that is, random distribution), with which the true average distance from experimental data can be compared.

Results

To map the recessive lethals on the 3rd chromosome of *D. melanogaster*, recombination mapping with *rucuca* multimarker line was applied to constrcut recombinants between the *rucuca* chromosome and the lethal-bearing chromosome. By scoring G_3 offspring produced by G_2 recombinants crossed with the parental lethal-bearing line (Appendix B, Table B3), the rough regions of recessive lethal alleles can be defined (Table 1). Because such mapping analyses can determine whether or not there is any recessive lethal but cannot determine how many recessive lethals located at the specific region, these regions with lethal alleles may harbor more than one lethals.

Table 1 Recombination mappirng result using recombinants between the *rucuca* multimarker chromosome and the lethal-bearing chromosome

G2 recombinant types								G3 offspring lethality of each lethal-bearing chromosome							
								ZS30-2	**ZS2-3**	**ZH32-1**	**ZH12-6**	**ZH18-6**	**ZH21-1**	**ZS53-3**	**MW6-3**
ru	+	+	+	+	+	+	+	L	L	L	L	L	L	L	L
ru	*h*	+	+	+	+	+	+	L	L	L	L	L	L	L	L
ru	*h*	*th*	*st*	+	+	+	+	-	L	L	L	L	L	L	L
ru	*h*	*th*	*st*	*cu*	+	+	+	A	L	L	L	-	-	-	-
ru	*h*	*th*	*st*	*cu*	*sr*	+	+	A	L	L	L	-	-	-	-
ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	A	A	A	A	-	-	-	-
+	+	+	+	+	+	+	*ca*	L	L	L	L	-	L	-	-
+	+	+	+	+	+	*e*	*ca*	L	L	L	A	-	-	-	-
+	+	+	+	+	*sr*	*e*	*ca*	L	L	L	A	-	-	-	-
+	+	+	+	*cu*	*sr*	*e*	*ca*	L	A	A	A	A	A	A	A
+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	L	A	A	A	A	A	A	A
+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	L	A	A	A	A	A	A	A
Rough regions of recessive lethals								*ru-sr*	*cu-ca*	*cu-ca*	*sr-ca*	*st-ca*	*st-ca*	*st-ca*	*st-ca*

"L" denotes the lethal-bearing recombinant. "A" denotes the lethal-free recombinant. "-"denotes that the recombinant type was not obtained due to recombination suppression.

For fine mapping, specific deficiency lines were chosen (Figures 5-18). Those deficiency lines contain a series of genomic deletions which cover previous predicted regions of recessive lethals (Table 1). By scoring on F_1 offspring, whether or not genomic deletions are complement to recessive lethal alleles can be determined (Appendix B, Table B3). The result showed that 10/14 recesssive lethals are located on the right arm of the 3rd chromosome, and the number of recessive lethal alleles on each eight chromosome ranging from one to four (Figure 4). Those recessive lethal alleles caused the flies died at different developmental stages, from the embryonic to pupal stages (Table 2). The precise genomic regions of recessive lethal alleles can be confirmed by intersection of overlapping smaller fragment of deletions which are both lack of complementation. In total, 14 recessive lethal alleles were mapped to the gene level on the 3rd chromosome (Figures 5-18 and Table 2).

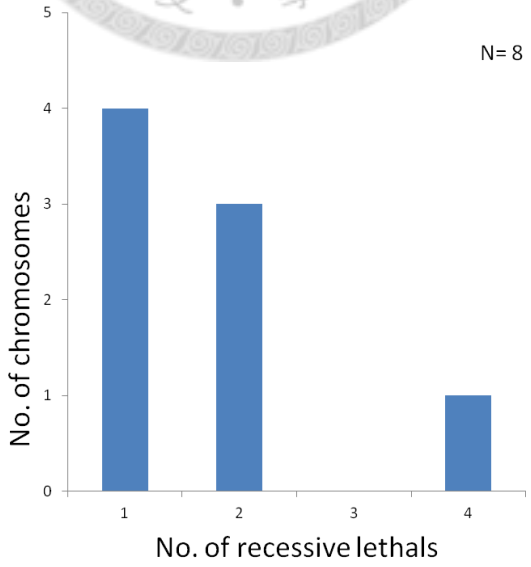

Figure 4 The distribution of recessive lethal number on eight 3rd chromosomes. X axis represents the number of recessive lethals of each distinct 3rd chromosome. Y axis represents the number of 3rd chromosomes bearing a specific number of lethals.

Table 2 Summary of 14 recessive lethal alleles

Lethal allele	Chromosome rearrangements in isofemale lines	Cytological position	Genomic ranges	Genetic position (cM)	Covering genes	Lethal stage
$\ell^{ZH18\text{-}I}$	*In(3R)K, In(3R)C*	85D19-23	3R:5,338,742..5,380,704	48.5	11	No visible larval lethality
$\ell^{ZH18\text{-}II}$		96A23-25	3R:20,639,446..20,663,321	85.2	10	2^{nd}-3^{rd} instar larval lethality
$\ell^{ZH21\text{-}I}$	*In(3R)K, In(3R)P*	76C5-6	3L:19,784,186..19,823,829	47.0	14	Eclosion failure
$\ell^{ZH21\text{-}II}$		82F6-7	3R:1,057,000..1,090,181	47.2	9	No visible larval lethality
$\ell^{ZS53\text{-}I}$	*In(3R)K, In(3R)93;96*	88E2-3	3R:10,983,368..11,075,682	56.5	12	1^{st}-2^{nd} instar larval lethality
$\ell^{MW6\text{-}I}$	*In(3R)K*	73E5-F2	3L:17,025,406..17,142,023	45.6	9	No visible larval lethality
$\ell^{MW6\text{-}II}$		82B1-2	3R:254,982..279,012	47.1	5	1^{st}-2^{nd} instar larval lethality
$\ell^{ZS30\text{-}I}$	*In(3R)K*, ST	61E1-2	3L:959651..1035182	0.0	6	Eclosion failure
$\ell^{ZS30\text{-}II}$		78E1	3L:21597878..21631024	47.0	8	No visible larval lethality
$\ell^{ZS30\text{-}III}$		84B4-E11	3R:2916249..3919805	47.6	~110	No visible larval lethality
$\ell^{ZS30\text{-}IV}$		87B11-C2	3R:8269738..8303300	51.6	9	No visible larval lethality
ℓ^{ZS2}	*In(3R)K*, ST	95E1	3R:19930781..19967091	84.5	10	2^{nd}-3^{rd} instar larval lethality
ℓ^{ZH12}	*In(3R)K*, ST	92B3-C1	3R:15662595..15716378	67.6	8	No visible larval lethality
ℓ^{ZH32}	*In(3R)K*, ST	95C13-14	3R:19747854..19768726	81.7	11	2^{nd}-3^{rd} instar larval lethality

Most of recessive lethal alleles can be mapped in the small genomic region containing less than dozens of genes, except the lethal allele $l^{ZS30\text{-}III}$ mapped in the large region covering more than one hundred genes (Figure 8 and Table 2). Interestingly, lethal allele $l^{ZS30\text{-}III}$ showed lethal phenotype when mapped with a deficiency line covering a large genomic region, but showed normal wild-type phenotypes when mapped with several lines covering a part of the large genomic region (Figure 8), suggesting that it was a synthetic lethal allele.

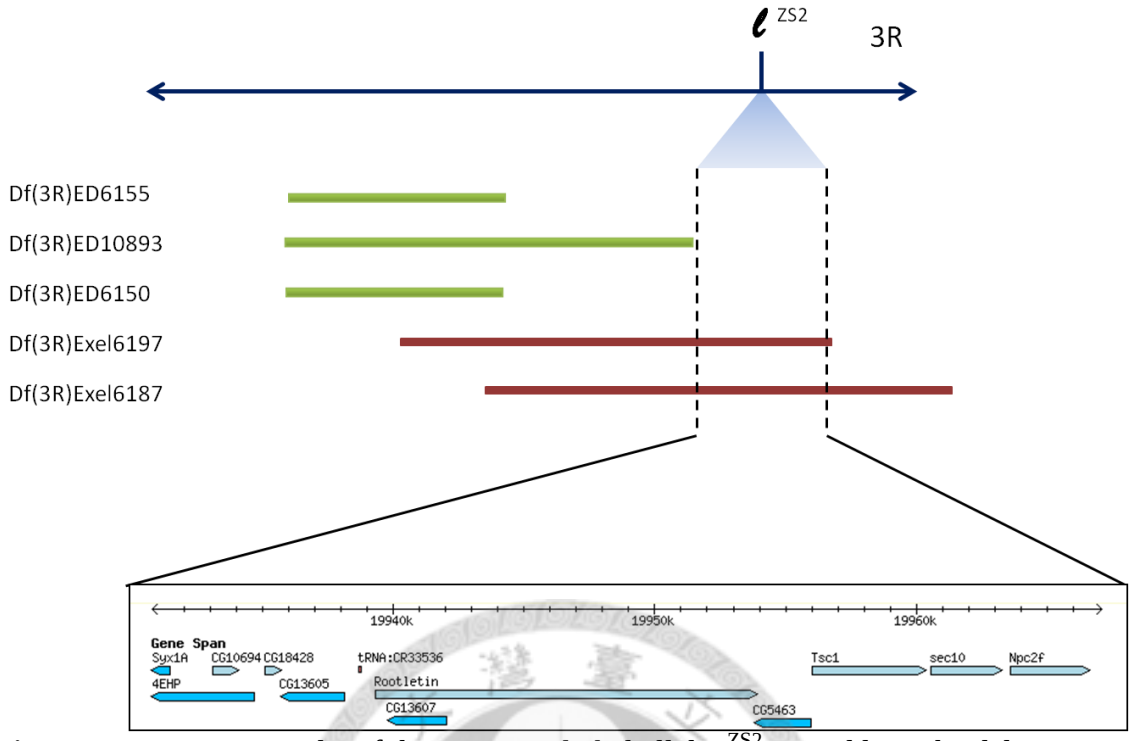

Figure 5 Mapping results of the recessive lethal allele ℓ^{ZS2}. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

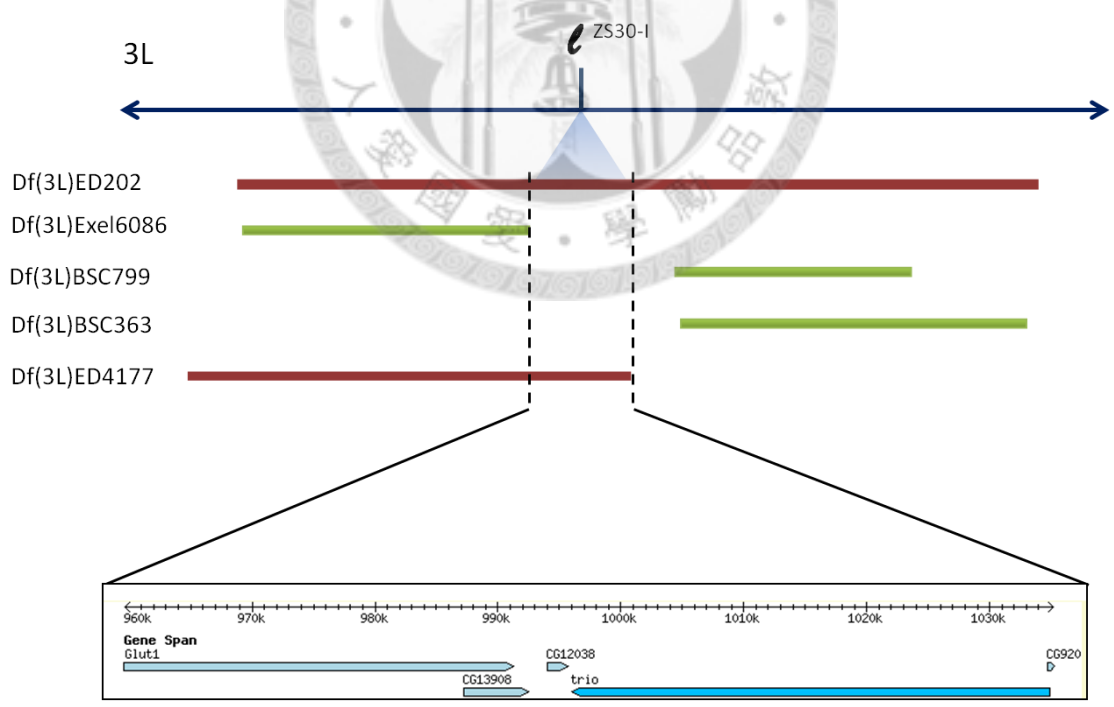

Figure 6 Mapping results of the recessive lethal allele $\ell^{ZS30\text{-}I}$. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

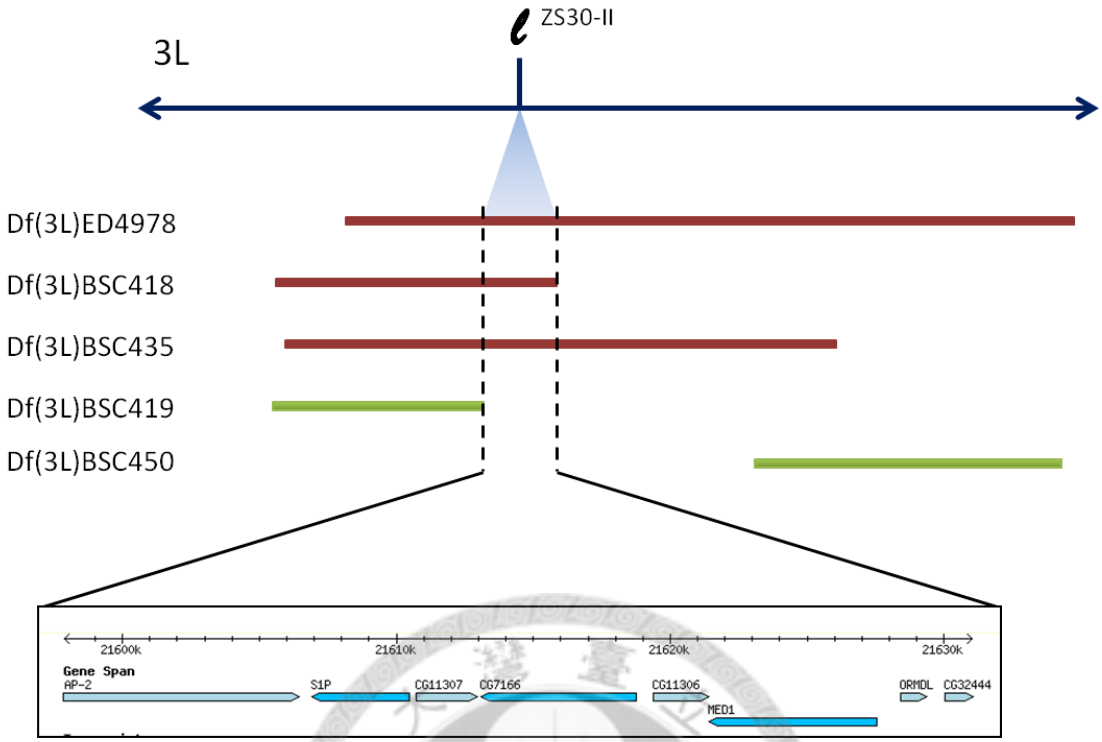

Figure 7 Mapping results of the recessive lethal allele $\ell^{ZS30\text{-}II}$. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

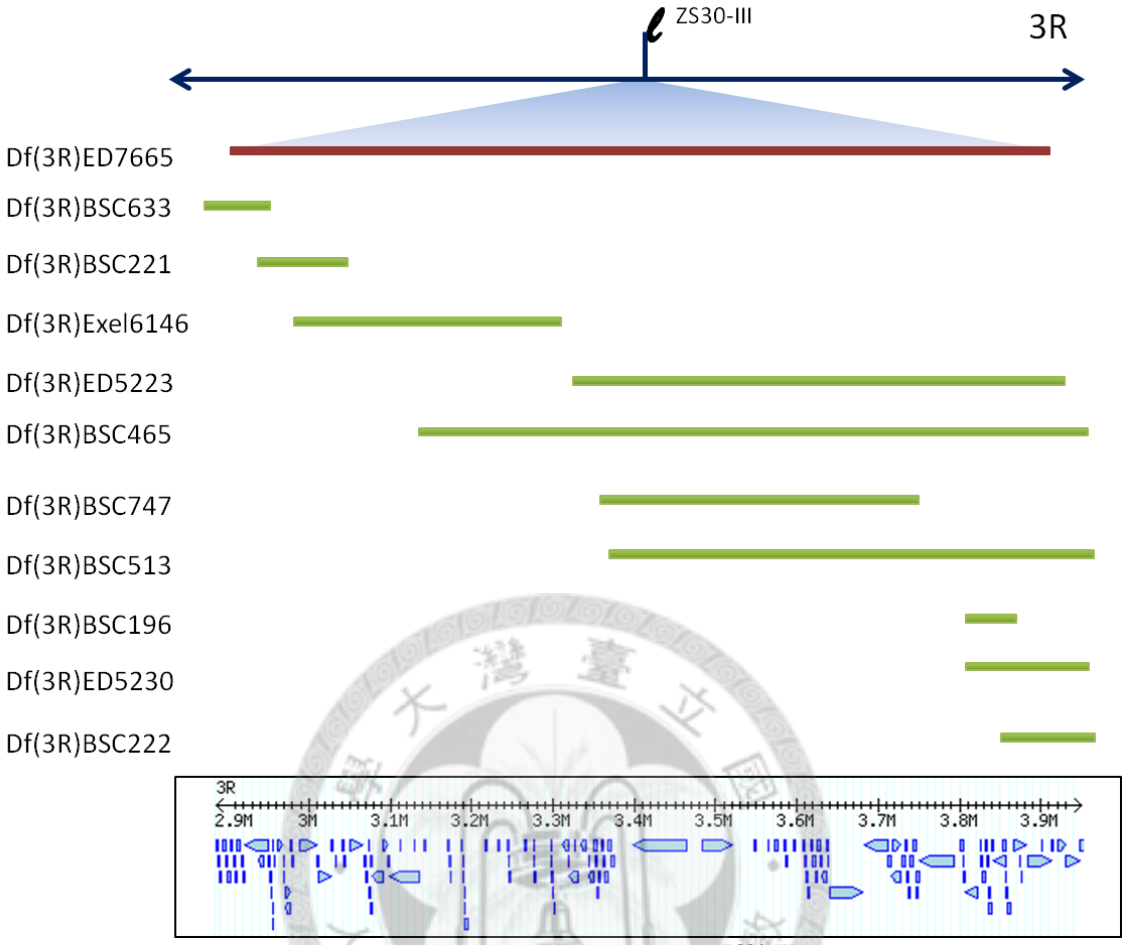

Figure 8 Mapping results of the recessive lethal allele $\ell^{ZS30\text{-}III}$. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

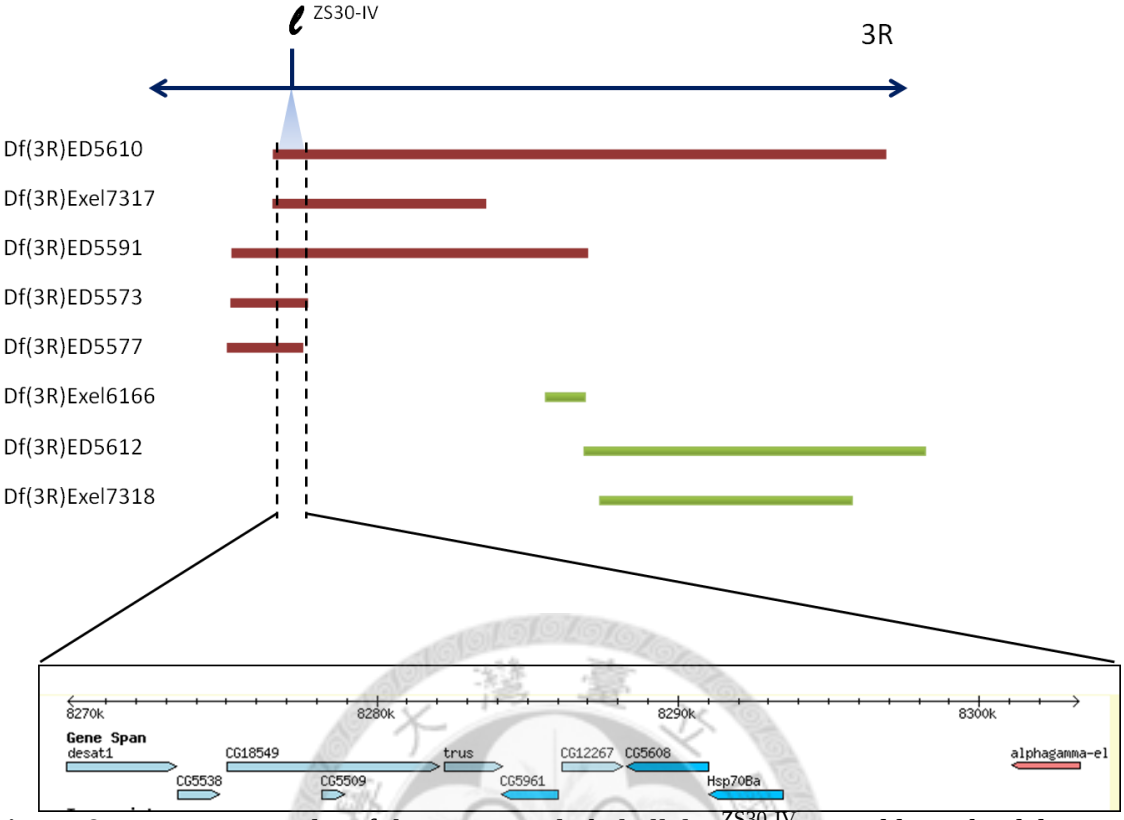

Figure 9 Mapping results of the recessive lethal allele $\ell^{ZS30\text{-}IV}$. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

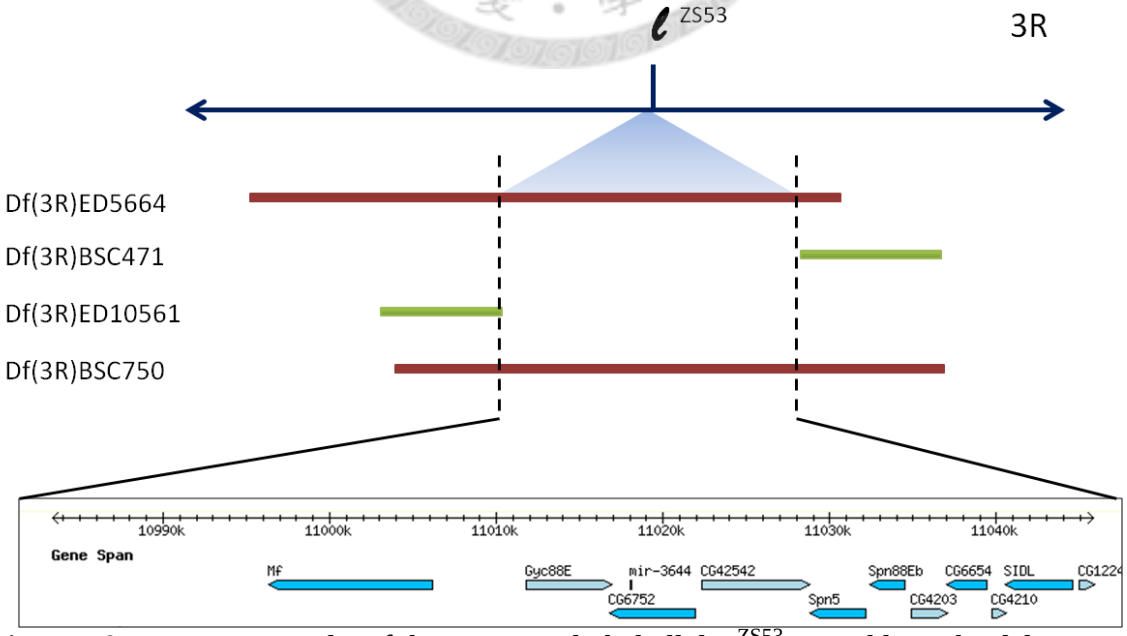

Figure 10 Mapping results of the recessive lethal allele ℓ^{ZS53}. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was

complementary to the recessive lethal.

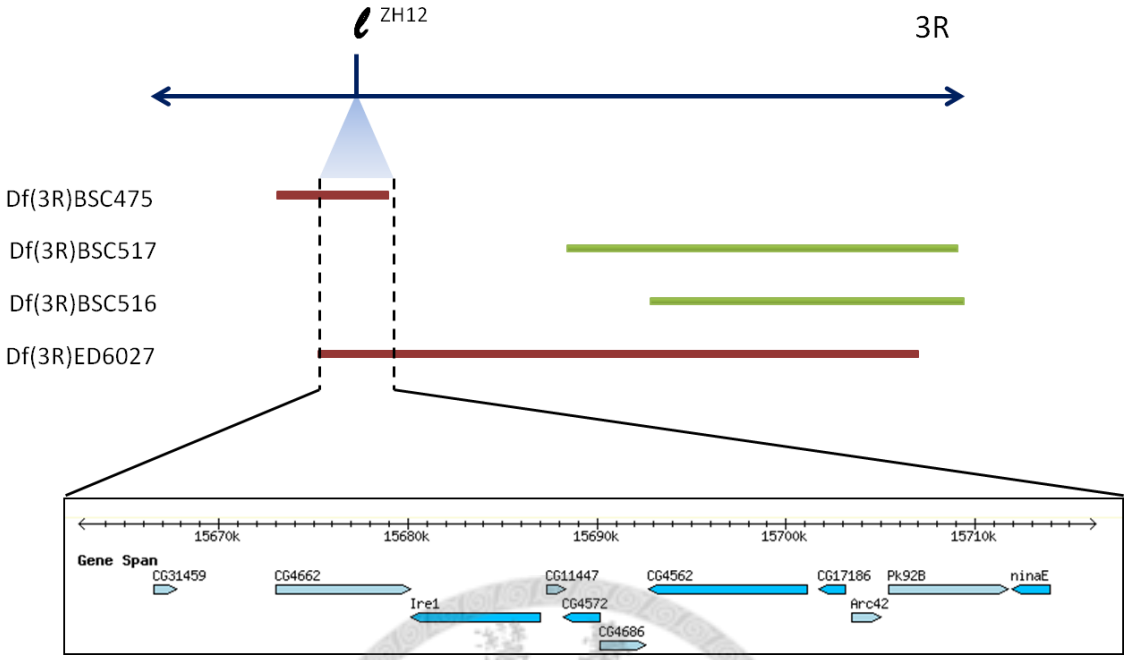

Figure 11 Mapping results of the recessive lethal allele ℓ^{ZH12}. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

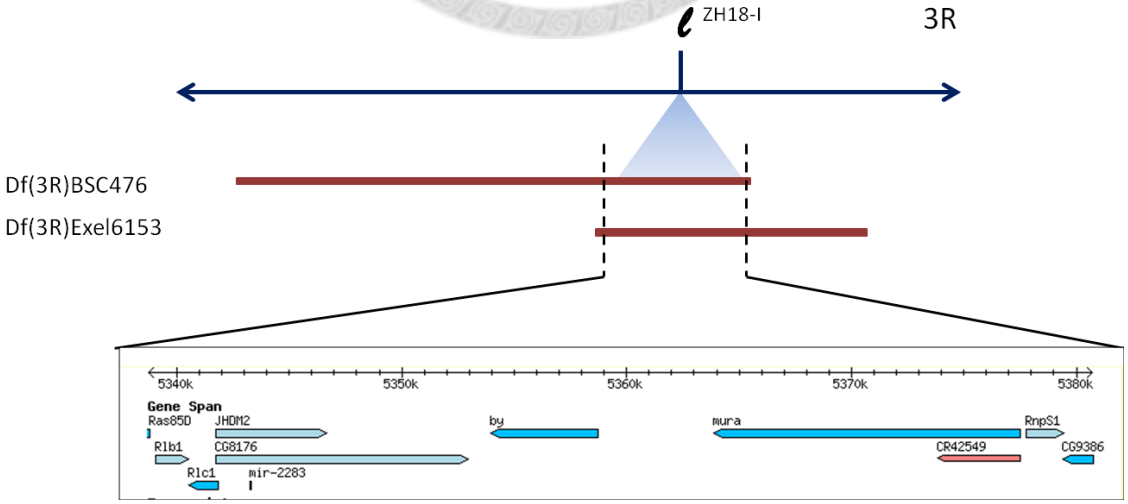

Figure 12 Mapping results of the recessive lethal allele ℓ^{ZH18-I}. Red line, the deletion region failed to complement the recessive lethal.

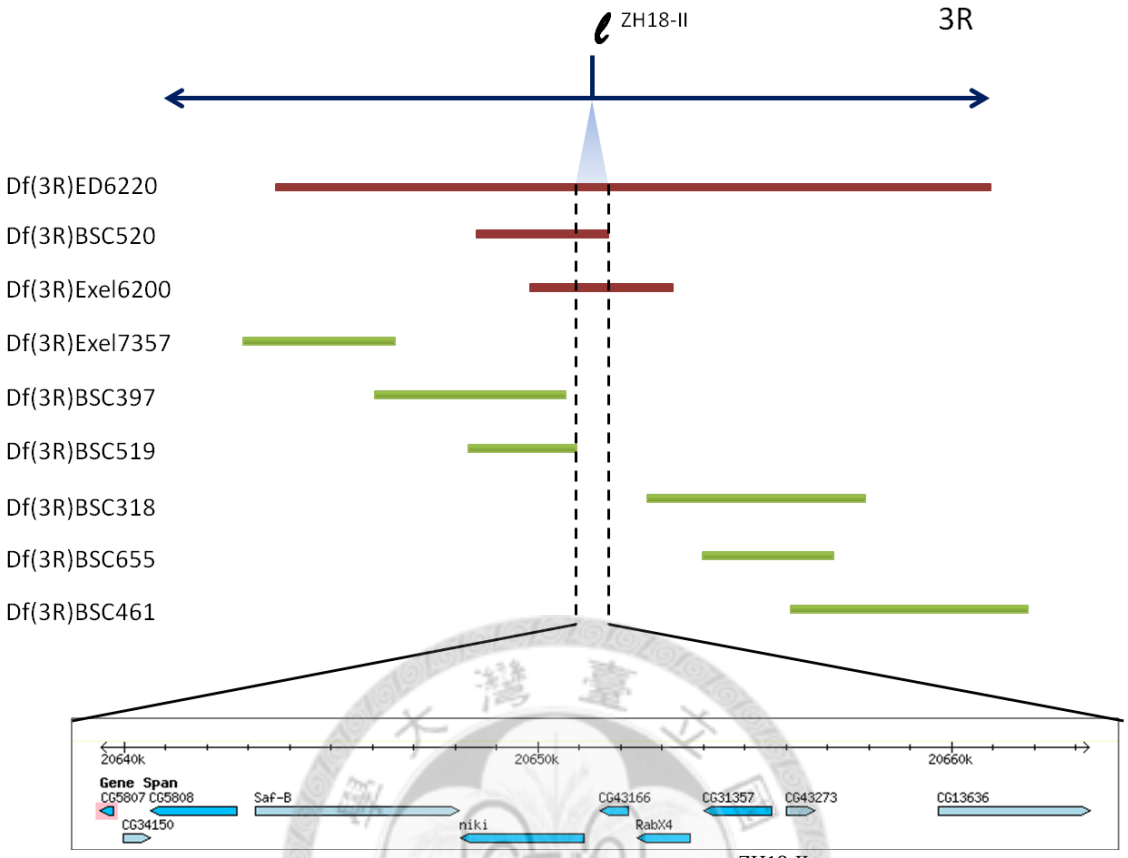

Figure 13 Mapping results of the recessive lethal allele $\ell^{ZH18\text{-}II}$. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

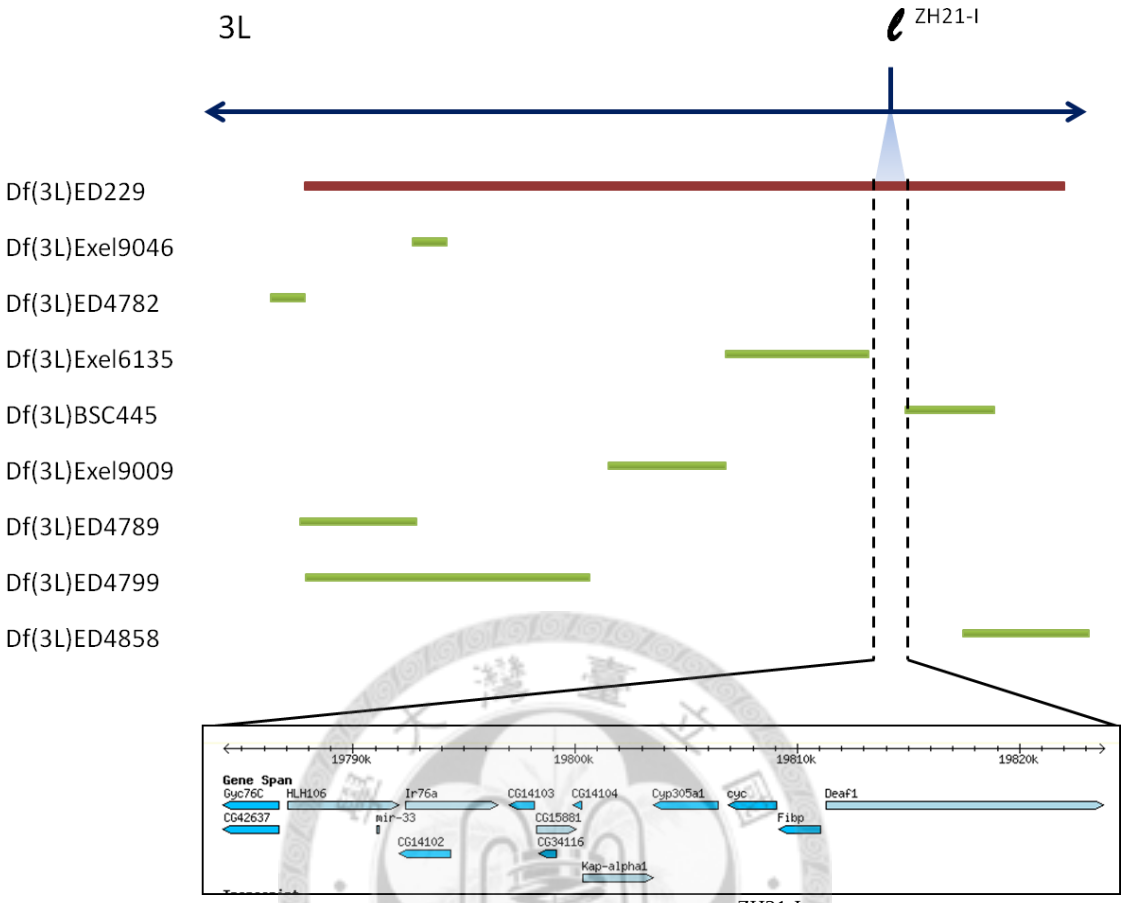

Figure 14 Mapping results of the recessive lethal allele ℓ^{ZH21-I}. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

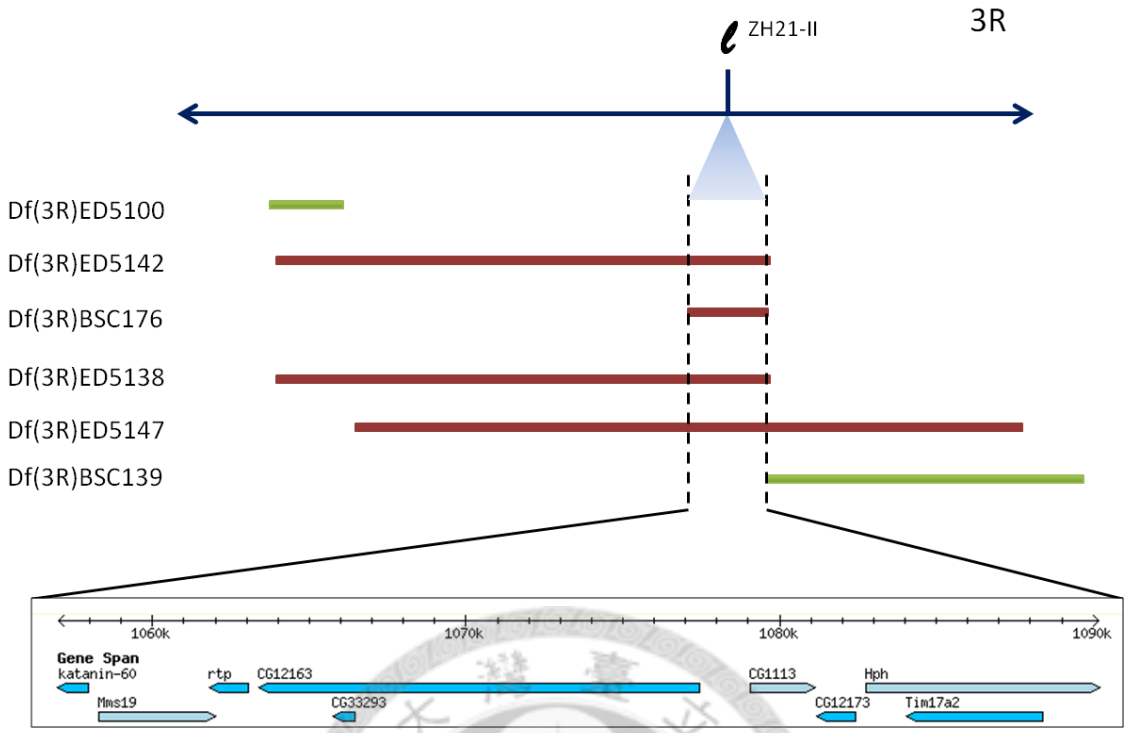

Figure 15 Mapping results of the recessive lethal allele $\ell^{ZH21\text{-}II}$. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

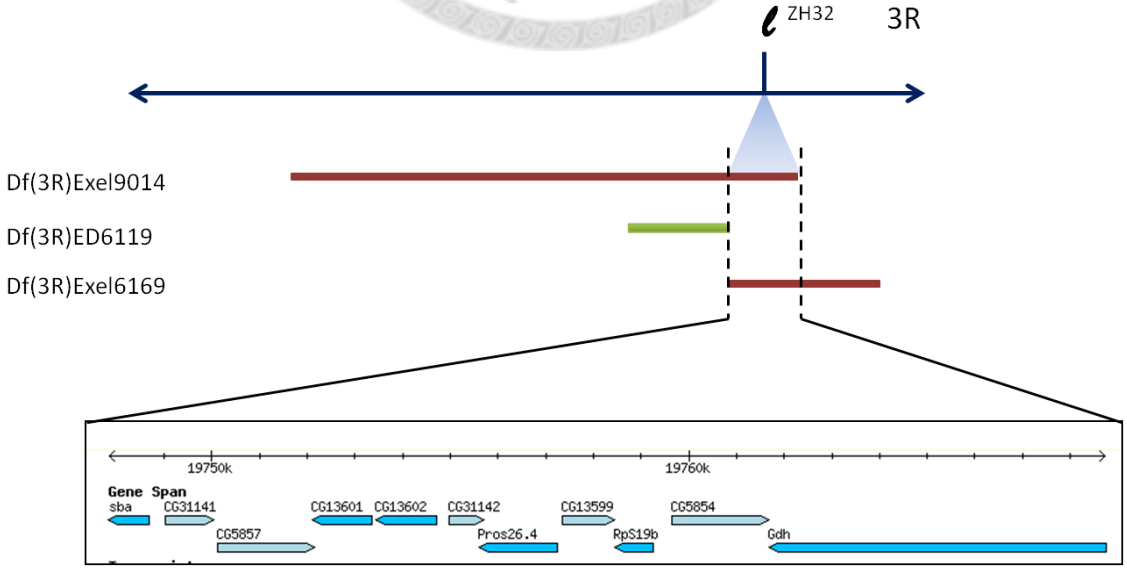

Figure 16 Mapping results of the recessive lethal allele ℓ^{ZH32}. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

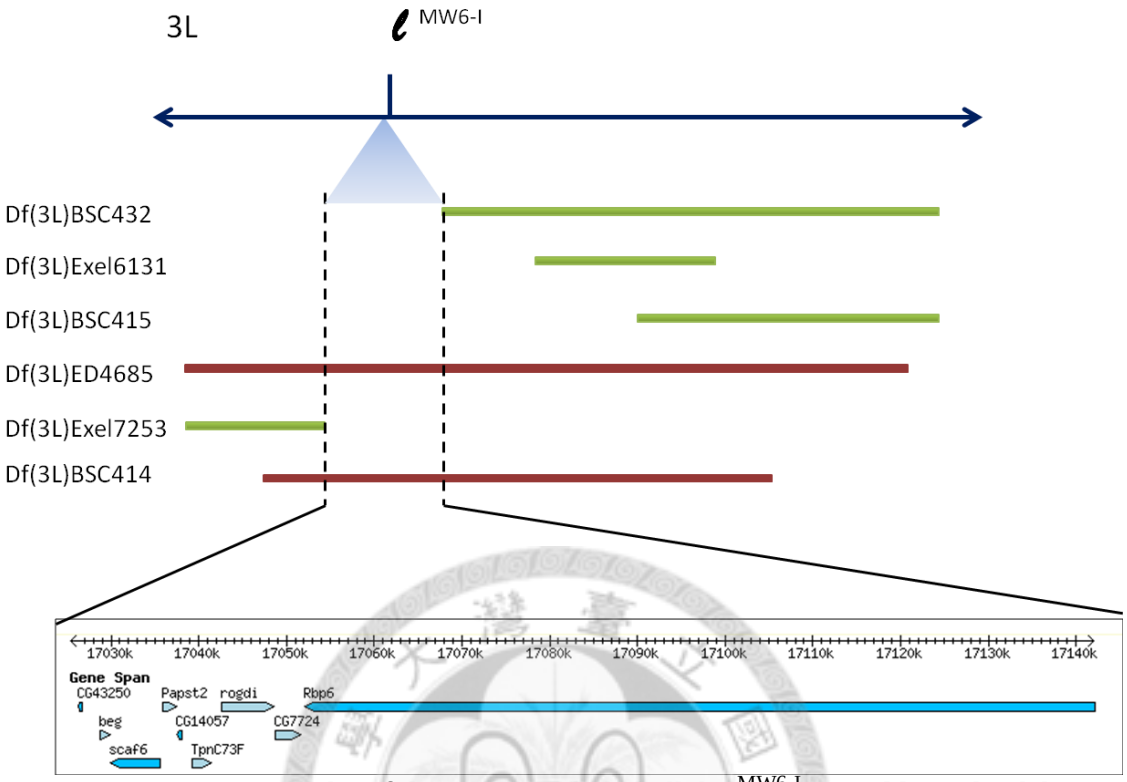

Figure 17 Mapping results of the recessive lethal allele $\ell^{MW6\text{-}I}$. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

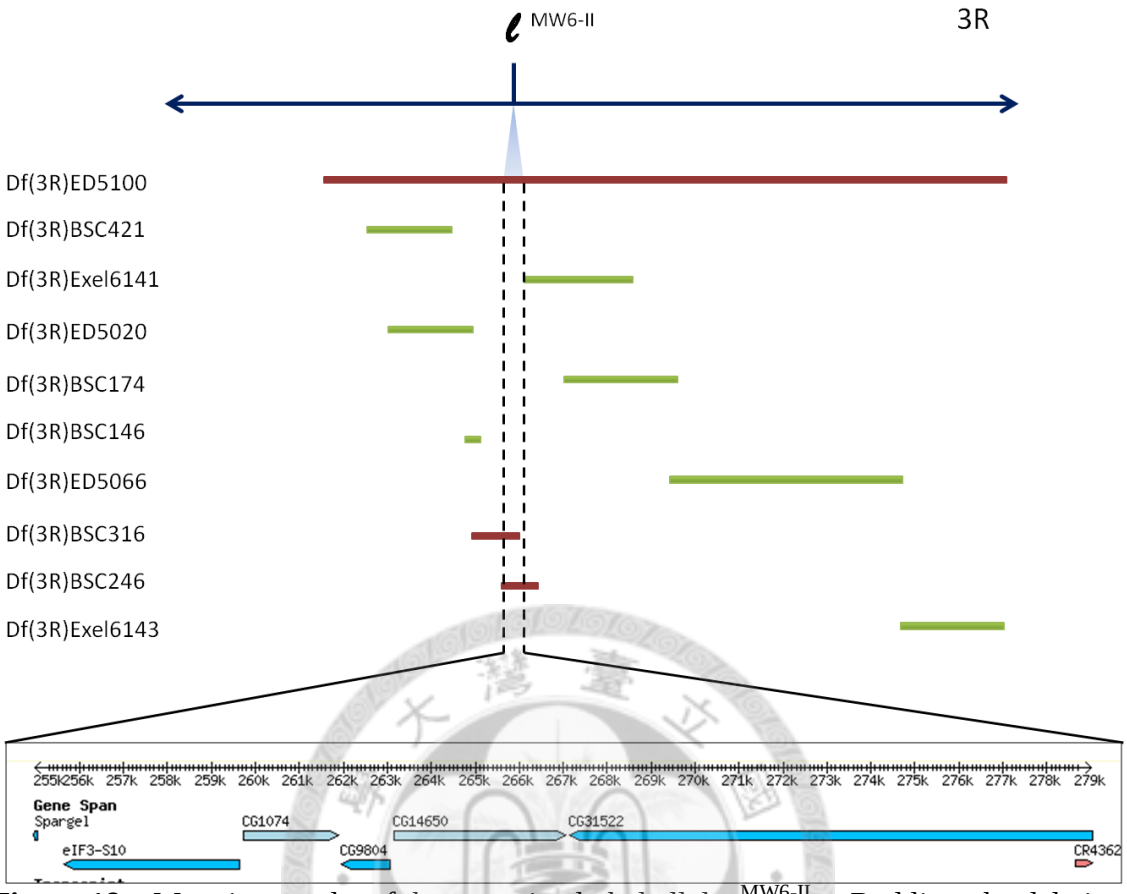

Figure 18 Mapping results of the recessive lethal allele $\ell^{MW6\text{-}II}$. Red line, the deletion region failed to complement the recessive lethal. Green line, the deletion region was complementary to the recessive lethal.

To understand how those recessive lethals were close to inversion breakpoints, we measured their genetic distances to estimate their recombination rate (Figure 19). By the interpolation method, genetic positions of recessive lethal alleles and inversion breakpoints were estimated (Figure 19), and then the distance between recessive lethals and the nearest breakpoints was calculated (Table 3). The mapping result showed that most of the recessive lethal alleles were close to the nearest breakpoints of the chromosomal inversions which were polymorphic in the Afrotropical *D. melanogaster* population. The extent of recombination reduction can be estimated by the distance between recessive lethal alleles and breakpoints of inversions which are polymorphic at three different levels (Table 4 and Figure 20). At the first level, the isofemale line level, the closest genetic distance is between ℓ^{ZS2} and the breakpoint of *In(3R)93;96*, 0.05 cM. When the level of inversion polymorphism enlarged the scale to the strong Z line level and even to the Afrotropical population level, all the recessive lethal alleles are accumulated nearby the breakpoints of inversions, and their genetic distances are less than 3.0 cM. The very short distances between recessive lethal alleles and breakpoints suggest that highly recombination-suppressed region in inversion heterozygotes might lead to the accumulation of recessive lethal alleles. Figure 20 shows the distribution pattern of recessive lethal alleles and chromosomal inversion polymorphism at three different levels. Most recessive lethal alleles (10/14) are located on the right arm of the 3rd chromosomes, consistent with the higher inversion polymorphism on the right arm than on the left arm (Figure 20).

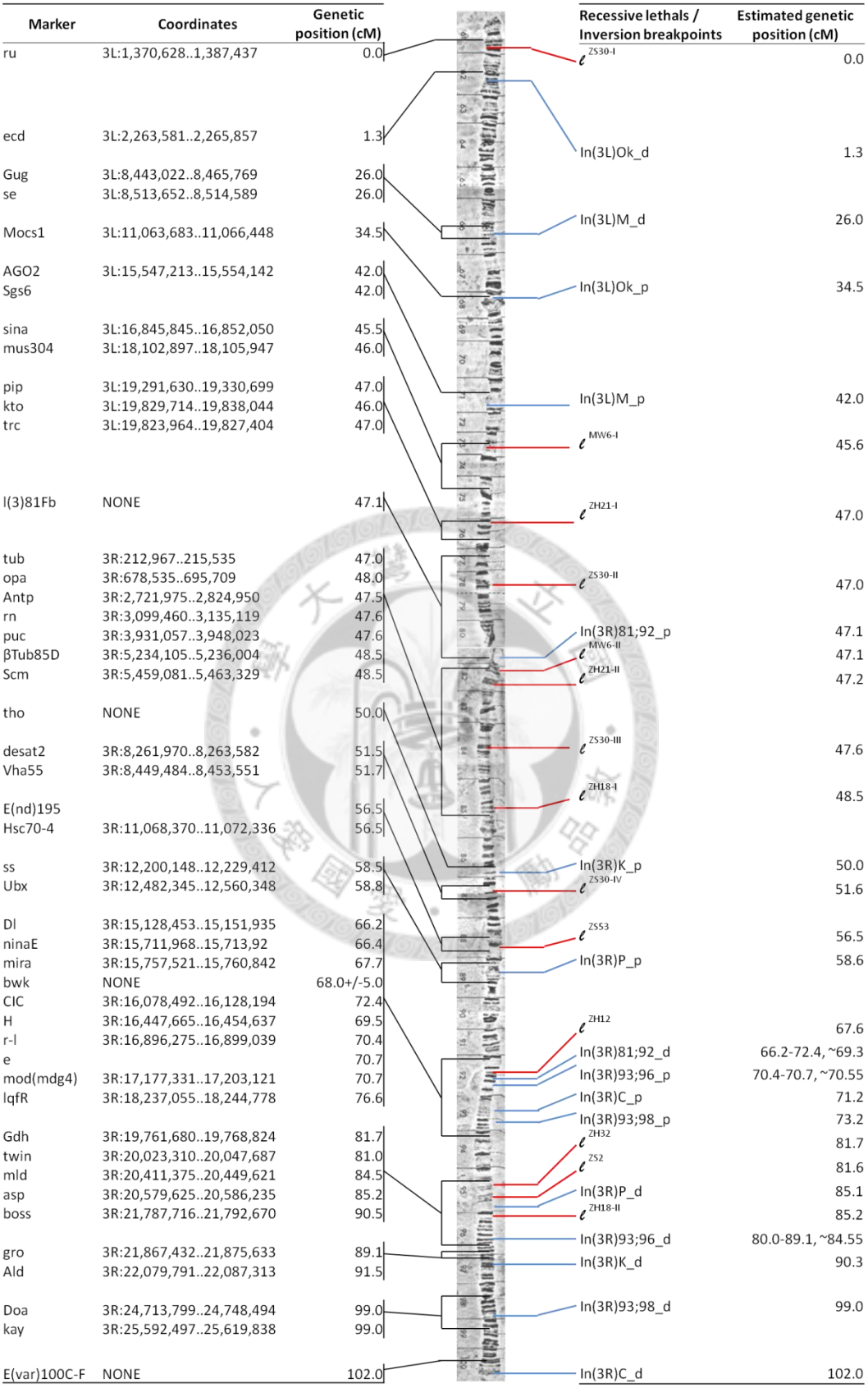

Figure 19 The estimation of genetic positions of recessive lethals and inversion breakpoints by interpolation of nearest genetic markers. p, proximal breakpoint of the inversion. d, distal breakpoint of the inversion.

Table 3 Genetic distances (cM) between each recessive lethals and each nearest inversion breakpoint

Lethal allele	Genetic position	Distance from breakpoints of inversions in Zimbabwe races							
		In(3R)K	***In(3R)P***	***In(3R)C***	***In(3R)93;96***	***In(3R)93;98***	***In(3L)Ok***	***In(3R)81;92***	***In(3L)M***
		50 cM;90.3cM	**58.6 cM;85.1 cM**	**71.2 cM;102.0 cM**	**~70.55cM;~84.55 cM**	**~73.2 cM;~99.0 cM**	**~1.3 cM;~34.5 cM**	**~47.1 cM;~69.3 cM**	**26.0 cM;42.0 cM**
$\ell^{ZH18\text{-}I}$	48.5	1.5	10.1	22.7	22.05	24.7	14.0	1.4	6.5
$\ell^{ZH18\text{-}II}$	85.2	5.1	0.1	14.0	0.65	12.0	50.7	15.9	43.2
$\ell^{ZH21\text{-}I}$	47.0	3.0	11.6	24.2	23.55	26.2	12.5	0.1	5.0
$\ell^{ZH21\text{-}II}$	49.0	1.0	9.6	22.2	21.55	24.2	14.5	1.9	7.0
ℓ^{ZS53}	56.5	6.5	2.1	14.7	14.05	16.7	12.0	9.4	14.5
$\ell^{MW6\text{-}I}$	45.6	4.4	13.0	25.6	24.95	27.6	11.1	1.5	3.6
$\ell^{MW6\text{-}II}$	47.1	2.9	11.5	24.1	23.45	26.1	12.6	0.0	5.1
$\ell^{ZS30\text{-}I}$	0.0	50.0	58.6	71.2	70.55	73.2	1.3	47.1	26.0
$\ell^{ZS30\text{-}II}$	50.6	0.6	8.0	20.6	19.95	22.6	16.1	3.5	8.6
$\ell^{ZS30\text{-}III}$	47.6	2.4	11.0	23.6	22.95	25.6	13.1	0.5	5.6
$\ell^{ZS30\text{-}IV}$	51.6	1.6	7.0	19.6	18.95	21.6	17.1	4.5	9.6
ℓ^{ZS2}	84.5	5.8	0.6	13.3	0.05	14.5	50.0	15.2	42.5
ℓ^{ZH12}	67.6	17.6	9.0	3.6	2.95	5.6	33.1	1.7	25.6
ℓ^{ZH32}	81.7	8.6	3.4	10.5	2.85	8.5	47.2	12.4	39.7

In(3L)Ok = In(3L)62D;68A. In(3L)M = In(3L)66D;71D. In(3R)K = In(3R)86E17;97A1-2. In(3R)P = In(3R)89B16;96A19. In(3R)C = In(3R)92D1-9;100F2-3.

Table 4 Genetic distances (cM) of recessive lethal alleles from the nearest breakpoint of chromosomal inversions at different levels

Lethal alleles	Cytological position	Lethal-bearing chromosomal rearrangements	Genetic distance from the breakpoints of nearest inversions					
			Isofemale line level		Strong Z line level		Afrotropical population level	
			(cM)	Nearest inversion type	(cM)	Nearest inversion type	(cM)	Nearest inversion type
$\ell^{MW6\text{-}I}$	73E5-F2	*In(3R)K*	4.40	*In(3R)K*	1.50	*In(3R)81;92*	1.50	*In(3R)81;92*
$\ell^{MW6\text{-}II}$	82B1-2	*In(3R)K*	2.90	*In(3R)K*	0.00	*In(3R)81;92*	0.00	*In(3R)81;92*
ℓ^{ZH12}	92B3-C1	ST	2.95	*In(3R)93;96*	1.70	*In(3R)81;92*	1.70	*In(3R)81;92*
$\ell^{ZH18\text{-}I}$	85D16-D18	*In(3R)K*	1.50	*In(3R)K*	1.40	*In(3R)81;92*	1.40	*In(3R)81;92*
$\ell^{ZH18\text{-}II}$	96A23-25	*In(3R)K*	0.10	*In(3R)P*	0.10	*In(3R)P*	0.10	*In(3R)P*
$\ell^{ZH21\text{-}I}$	76A1-D3	*In(3R)K*	3.00	*In(3R)K*	0.10	*In(3R)81;92*	0.10	*In(3R)81;92*
$\ell^{ZH21\text{-}II}$	82F6-7	*In(3R)K*	2.80	*In(3R)K*	0.10	*In(3R)81;92*	0.10	*In(3R)81;92*
ℓ^{ZH32}	95C13-14	ST	2.85	*In(3R)93;96*	2.85	*In(3R)93;96*	2.85	*In(3R)93;96*
ℓ^{ZS2}	95E1	ST	0.05	*In(3R)93;96*	0.05	*In(3R)93;96*	0.05	*In(3R)93;96*
$\ell^{ZS30\text{-}I}$	61E1-2	ST	50.00	*In(3R)K*	1.30	*In(3L)Ok*	1.30	*In(3L)Ok*
$\ell^{ZS30\text{-}II}$	78E1	ST	3.00	*In(3R)K*	3.00	*In(3R)K*	3.00	*In(3R)K*
$\ell^{ZS30\text{-}III}$	84B4-E11	ST	2.40	*In(3R)K*	0.50	*In(3R)81;92*	0.50	*In(3R)81;92*
$\ell^{ZS30\text{-}IV}$	87B11-C2	ST	1.60	*In(3R)K*	1.60	*In(3R)K*	1.60	*In(3R)K*
ℓ^{ZS53}	88E2-3	*In(3R)K*	2.10	*In(3R)P*	2.10	*In(3R)P*	2.10	*In(3R)P*

In(3L)Ok = In(3L)62D;68A. In(3L)M = In(3L)66D;71D. In(3R)K = In(3R)86E17;97A1-2. In(3R)P = In(3R)89B16;96A19. In(3R)C = In(3R)92D1-9;100F2-3.

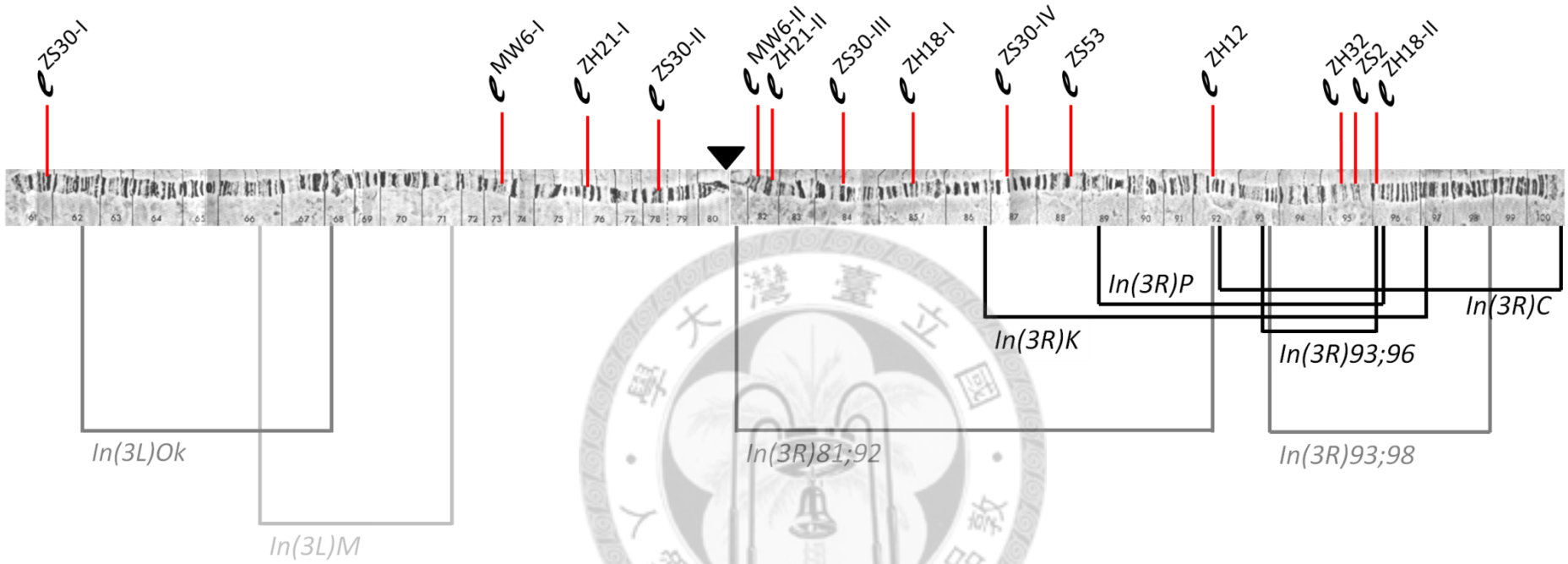

Figure 20 The distribution of recessive lethal alleles and inversions on the 3rd chromosome at three different levels. Black lines, inversions found in isofemale lines. Gray lines, inversions found in Strong-Z lines. Light gray lines, inversions found in the Afrotropical population. Reverse triangle, the centromere.

A R programming was run to test whether these recessive lethal alleles distribute randomly on the chromosome or not. The null hypothesis here is that all recessive lethals distribute randomly on the 3rd chromosome. Two data groups were compared. One is the true mean genetic distance between each 14 lethals and each nearest inversion breakpoint (from Table 4). The other is the expected mean genetic distance between 14 random-sampling locations on the 3rd chromosome to each nearest inversion breakpoint. With random sampling 1000 times, the null distribution was generated as a probability distribution. The statistical *P* value can be obtained by comparing the true mean distance to the null distribution. All *P* values from three different population levels are less than 0.05 (isofemale line level, $P = 0.012$; strong Z line level, $P = 0.001$; Afrotropical population level, $P = 0.001$), meaning that the null hypothesis was rejected (Figure 21). The statistical analyses strongly supported that highly non-random distribution of recessive lethals on the 3rd chromosome was the consequence of high recombination suppression by polymorphic inversions.

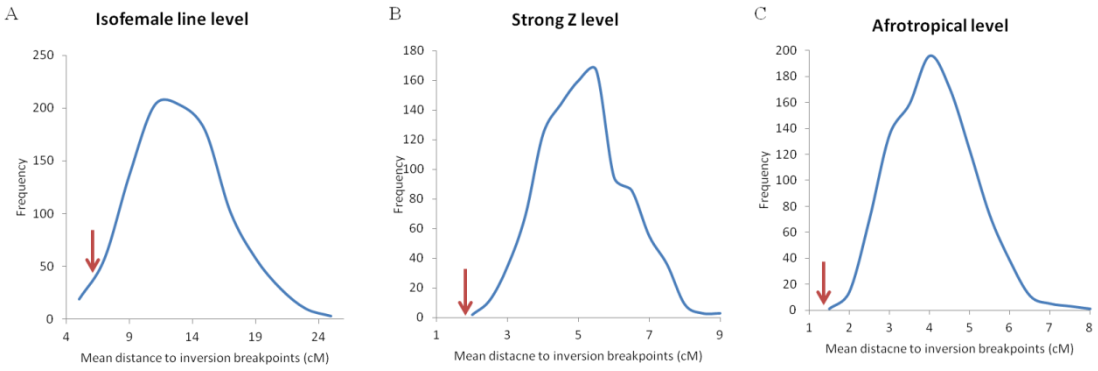

Figure 21 Comparison between null distribution of genetic distance and true mean distances. Red arrows are true mean values. (A) The isofemale line level, the true mean is 5.56 cM. (B) The strong Z level, the true mean is 1.02 cM. (C) The Afrotropical level, the true mean value is 1.02 cM.

Discussion

My data clearly shows that highly recombination-suppressed regions are likely to accumulate deleterious mutations (recessive lethal alleles in this case). All the 14 recessive lethal alleles mapped are very close to the nearest breakpoints of inversions found in Afrotropical populations. Here, I describe how inversions affect recombination rates along chromosomes, then possible pattern of deleterious mutations accumulating on the chromosomes with polymorphic inversions, and the association between recessive lethal alleles and chromosomal inversion polymorphisms in natural populations.

High recombination suppression due to inversion heterozygotes

Recombination rates in inversion heterozygotes are highly variable along the inverted chromosomal region. Outside the inversion, they decrease far from chromosomal inversion, and reach the lowest rate nearby breakpoints; inside the inversion, they go up gradually from the inversion breakpoints, and reach the highest peak in the middle region of the inversion (Navarro *et al.* 1997). My result is the first study to show the fine-scale mapping of recessive lethal alleles to be located nearby breakpoints of chromosomal inversion (Table 4), providing the evidence that deleterious mutation accumulation is in the low recombination region of inverted heterozygotes, as previous natural population surveys suggested (Epling *et al.* 1961; Dobzhansky *et al.*

1963; Zivanović *et al.* 2000). In addition, the reduced recombinational effects of the chromosomal inversions might extend to several megabases beyond the breakpoints of inversions. The inhibition ranges of recombination by chromosomal inversions have been described in some *Drosophila* species. In *D. melanogaster*, recombination suppression might extend to about over 1 Mb from the breakpoints of inversion *In(2L)t*, (Depaulis *et al.* 1999); similarly, recent genomic study showed large regions of linkage disequilibrium on both sides of inversion breakpoints of *In(3R)Mo* (0.5 Mb and 1.5 Mb), and the LD at long distances is overall greater for 3R then any other chromosome arms (Langley *et al.* 2012). From my experimental data, the recombination-suppressed region restricts to3 cM, roughly equal to 0.5Mb long, showing the consistence with the genomic study by Langley *et al.* (2012). In *D. subobscura*, the extents of recombination suppression in the regions outside the distal breakpoints are similar in different inversions (3.5 and 3.9 Mb for inversions *O6* and *O7*, respectively) (Pegueroles *et al*. 2010). In the F_1 hybrids of *D. pseudoobscura* and *D. persimilis*, the recombination suppression varies between 2.1 - 2.8 Mb outside the XR inversion (Kulathinal *et al*. 2009). Meanwhile, in my experiments, the recombination suppression is larger than 20 cM in *In(3R)K*/ST heterozygotes (Figure B1, Table B4). Though markers are limited, recombination is suppressed between two markers, *cu* and *sr*, which on both sides of *In(3R)K* proximal breakpoint (Table B4), also two markers, *e* and *ca* for distal breakpoints. Because those

recombination-suppressed regions contain a large amount of genes, it may have a greater effect on genetic loads once deleterious mutations occur. It was suggested that the size of inversion affected the recombination rate; large inversions (genetic distances > 20 cM) are expected to achieve an observable rate of double-crossover inside the inversion (Navarro *et al.* 1997). In the largest inversion in this study, *In(3R)K* which covers 40.3 cM, I observed no double-crossover inside the inversion and only observed one outside the inversion among 468 offspring (Table B4, grey). The recombination rate and amount of recombinant types in this inversion heterozygote is much lower and less than in the homozygote (Appendix B, Table B5), indicating that that recombination rate along the chromosome is dramatically inhibited by chromosomal inversions. The difference between the large region of recombination suppression revealed by my double crossing-over data and the small region genomic data suggests that gene conversion might play a role to increase the gene exchange inside the inversion (Chovnick 1973; Navarro *et al*. 1997).

Accumulation pattern of recessive lethals

The processes of deleterious mutation accumulation along the genome have been suggested in some theoretical works. Muller (1964) proposed a hypothesis that in an asexual population, due to no recombination, distinct deleterious mutations at various loci create a spectrum of genotypes carrying increment mutations from 0 to many.

Individuals in this population can carry more mutations than their ancestors did (Muller 1964). Moreover, because of genetic drift in a finite population, the lowest among class of deleterious mutations may be lost by chance from the population. At the same time, with no recombination, all remaining individuals acquire more mutations than last generation. Thus, individuals in this population undergo the irreversible process and cannot escape from bearing some among of deleterious mutations. This situation can also be applied to the non-recombination diploid sexual population, such as the Y chromosome in *Drosophila* species, which lacks of recombination in males (Charlesworth 1978). Similarly, chromosomal inversion heterozygotes could generate highly recombination-suppressed regions of the genome (breakpoints of inverted regions in this case), and initiate the Muller's ratchet as well. According to the distribution of the recessive lethal numbers on the eight 3rd chromosomes in my study (Figure 4), even with small sample size, the accumulation of deleterious mutations reveals that the population is undergoing Muller's ratchet process. The rate of Muller's ratchet turns faster when small population size because of stronger genetic drift effect. The simulation study of Charlesworth and Charlesworth (1997) also revealed the same situation in the non-recombination diploid sexual population. Combining with the empirical data of recessive lethal alleles, there are two possibilities for the accumulation of recessive lethal. One is that those recessive lethals are accumulated in laboratory stocks with the extreme small population; another is that the effective

population size in nature is small. For the first possibility, it is likely that very few, if not none, recessive lethals were accumulated in the isofemale line based on the following reasons. The simulation studies by Charlesworth *et al*. (1993) and Charlesworth and Charlesworth (1997) revealed that it took a very long time (more than 2000 generations) for deleterious mutations fixation with non-recombination and/or inbreeding populations (Charlesworth *et al.* 1993; Charlesworth and Charlesworth 1997). Taking the established time of our isofemale lines into consideration, 20 years (*i.e.*, 200-400 generations) is not long enough for all 14 recessive lethals occurring after collected from the wild. In addition, complex inversion *In(3LR)TM3*, which has been used as a balancer since first described in 1958 (Mitchell 1958), bears two to five recessive lethal alleles which was identified by my deficiency mapping (Table B3) as a by-product of my experiments (Table B6, Figure B). Compared to the small number of recessive lethal alleles accumulated in the extreme small laboratory population for over 50 years, most of the 14 recessive lethal alleles are more likely to be from the wild. Moreover, if recessive lethals occur after established in the vial with a small population size, they might be quickly eliminated by genetic drift. From the distribution data of these 14 recessive lethals (Figure 20), some recessive lethal alleles are located far from *In(3R)K*, such as $\ell^{ZS30\text{-}I}$, which still exist in the stock without the shelter by inversion breakpoints. This data strengthen that most of the recessive lethal alleles accumulated

in the wild. The last point, it is worth mentioning that mutations occur in the extreme small laboratory population and deleterious mutations accumulate around highly recombination-suppressed regions are not mutually exclusive. For second possibility, Chang and colleagues (Chang and Lin 1995; Chang *et al.* 1996; Yang *et al.*2002) hypothesized that in *D. albomicans*, deleterious mutation accumulation resulted from the bottleneck effect due to the population shrinking between seasons. The *D. melanogaster* populations used in this study are from Zimbabwe and the nearby regions and have strong sexual isolation with other populations (Wu *et al*. 1995; Hollocher *et al.* 1997). This strong behavior isolation may reduce the effective population size. Meanwhile, the high chromosomal inversion polymorphism in natural populations could build complex heterokaryotype combinations, which might cause a great variation of recombination rates along the chromosome and thus result in different mutation accumulation hotspots. In that case, distinct recessive lethal alleles will be sheltered in different chromosomal regions with great recombination suppression.

Chromosomal inversion polymorphisms vs. genetic load

Chromosomal inversion polymorphisms in natural populations may be maintained mainly by positive selection toward local co-adapted genes (Lowry and Wills 2010; Ayala *et al*. 2011; Joron *et al.* 2011; Rottschaefer *et al.* 2011) and also possibly by genetic load which eliminates the homozygous deleterious mutations, at least in the case

of some *Drosophila* species (Andjelković *et al.* 1998; Zivanović *et al.* 2000). In my study with the Afrotropical population of *D. melanogaster*, lethal mutations are accumulated in highly recombination-suppressed regions by inversion heterokaryotypes, providing a great amount of genetic loads in populations. With distinct chromosome rearrangements, deleterious mutations are produced independently by genetic drift at first, and then accumulate in different recombination-suppressed regions. Once the Muller's ratchet starts to work, because of the various mutation hotspots along the chromosome caused by various inversion heterozygotes, genetic loads are hardly fully eliminated from populations. However, the irreversible process of mutation accumulation could increase the effect of inbreeding depression and decrease the fitness in assortative mating populations; some deleterious mutations finally get loss in populations. In this situation, deleterious mutations are neither completely eliminated nor fixed in the population, but are sheltered by inverted heterozygotes in the population at low to moderate frequency. Meanwhile, the amount of genetic loads might keep a dynamic equilibrium in populations. By knowing the recombination-suppressed effects on deleterious mutation accumulation, my results provide the strong evidence that the accumulation of recessive lethal alleles is associated with high inversion polymorphism in the Afrotropical *D. melanogaster* population.

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Appendix A

Chromosomal inversion polymorphisms in Afrotropical populations

Chromosome rearrangements of Afrotropical *D. melanogaster* populations were examined by dissecting larval offspring of single males from each isofemale line crossed with females from the wild-type strain, Canton-S, with the standard chromosome arrangement (Figure A1 and A2). The right arm of third chromosome carried more inversion types compared to other chromosomal arms (Table A1). In total, six inversion types on the 3rd right arm of chromosome were found in our sampled populations: *In(3R)K* (= 86E17;97A1-2), *In(3R)P* (= 89B16;96A19), *In(3R)C* (= 92D1-9;100F2-3), *In(3R)81;92*, *In(3R)93;96*, and *In(3R)93;98*. The first three inversions have previously been found in Afrotropical populations of *D. melanogaster* (Aulard *et al.* 2002); the latter three are newly discovered in our laboratory. In addition, high heterozygosity of inversion was observed in some lines which show strong Z behavior (DI>3, data not shown) (Table A2). Further isogenization for the third chromosome (Figure A3) revealed that a high ratio (0.833, N = 12 lines) of lethal-bearing chromosomes as well as a few sterile-bearing chromosomes existed in these isofemale lines (Table A3). The complementation test was first conducted to examine the allelism of those recessive lethal alleles (Figure A4). If recessive lethal alleles fail to complement (in other words, they are the same locus), the wild type offspring would not be present. To examine whether the recessive lethal alleles carried

on different 3rd chromosomes are at the same locus, the complementation test was conducted. The result showed those recessive lethal alleles are distinct (Table A4).

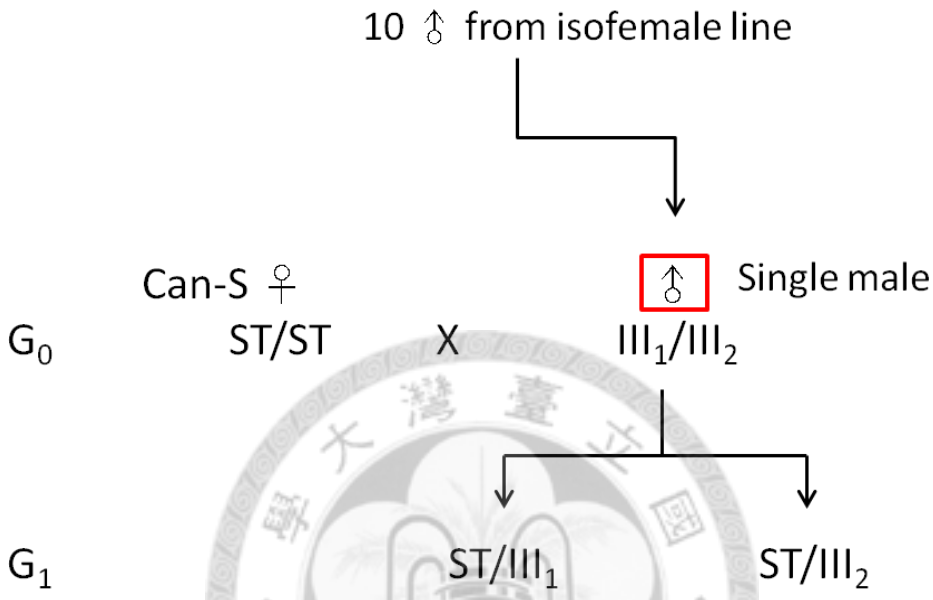

Figure A1 The cross scheme for chromosomal rearrangement examination. III_1 and III_2 denote different chromosomal rearrangements of the 3rd chromosomes in a isofemale line. ST, straight chromosome from the standard line Canton-S (Can-S).

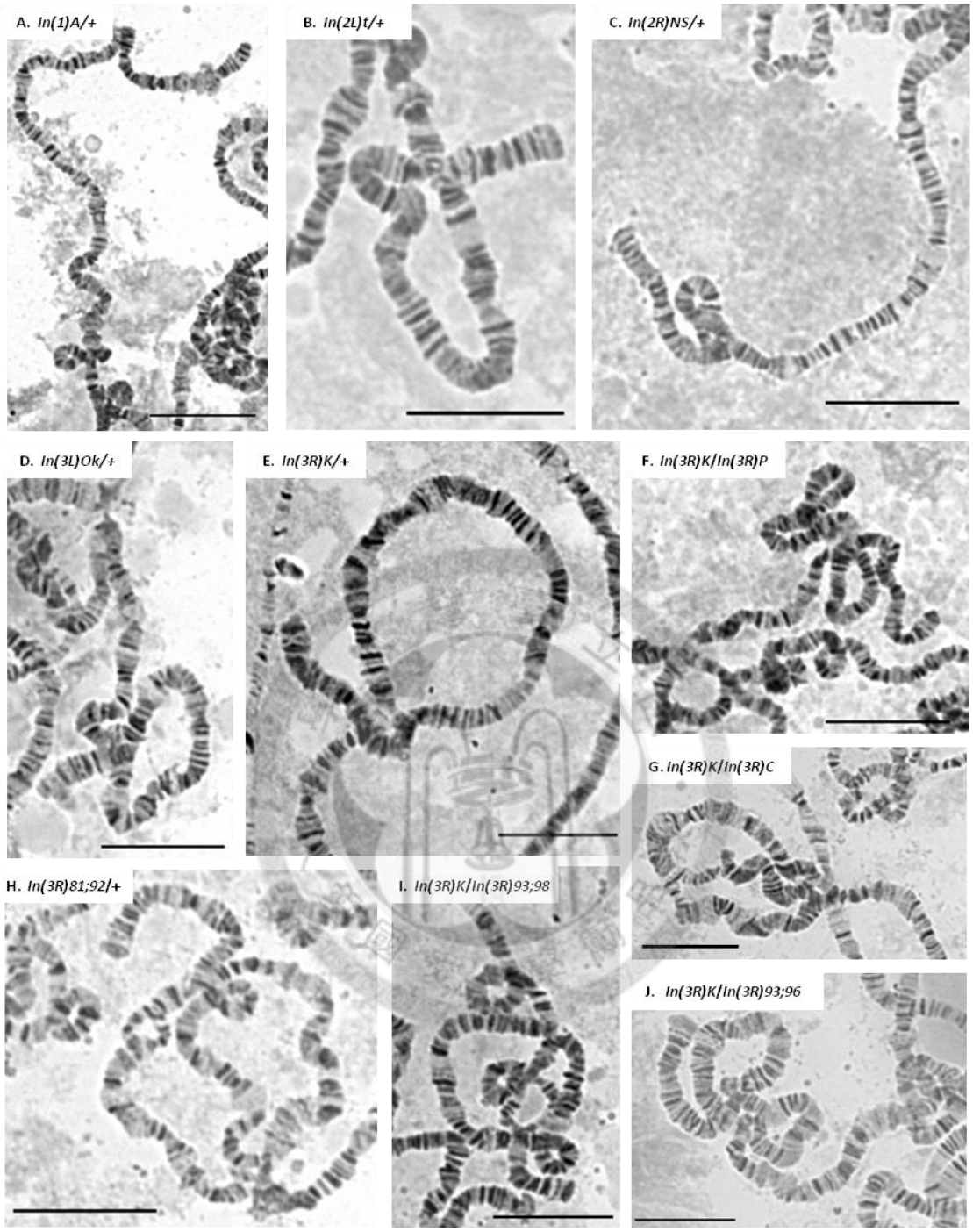

Figure A2 Chromosomal inversion polymorphism from isofemale line. **(**A) *In(1)A=In(1)12A;18D.* (B) *In(2L)t=In(2L)22D3-6;34A8-9.* (C) *In(2R)NS=In(2R)52A2-14;56F9-13*. (D)*In(3L)Ok=In(3L)62D;68A.* (E)*In(3R)K=In(3R)86E17;97A1-2*. (F)*In(3R)K/In(3R)P, In(3R)P=In(3R)89B16;96A19.* (G)*In(3R)K/In(3R)C, In(3R)C=In(3R) 92D1-9;100F2-3*. (H)*In(3R)81;92*. (I)*In(3R)K/In(3R)93;98*. (J)*In(3R)K/In(3R)93;96*. 40x for 20μm.

Table A1 High chromosomal inversion polymorphisms in Afrotropical lines

African lines	Chromosomal rearrangements														
	X		2L		2R		3L		3R						
	ST	*A*	ST	*t*	ST	*NS*	ST	*Ok*	ST	*K*	*P*	*93;96*	*93;98*	*C*	*81;92*
LA2	1	0	0.6	0.4	1	0	0.2	0.8	1	0	0	0	0	0	0
LA34	1	0	1	0	0.6	0.4	0.3	0.7	1	0	0	0	0	0	0
ZS2	0	1	0	1	0.9	0.1	1	0	0.3	0.7	0	0	0	0	0
ZS6	1	0	0.7	0.3	1	0	1	0	0	0.5	0.5	0	0	0	0
ZS8	0	1	1	0	1	0	0.7	0.3	0.2	0.8	0	0	0	0	0
ZS11	1	0	0	1	0.1	0.9	1	0	0.5	0.5	0	0	0	0	0
ZS30	1	0	0	1	1	0	1	0	0.3	0.7	0	0	0	0	0
ZS53	1	0	0.5	0.5	0.6	0.4	1	0	0	0.6	0	0.4	0	0	0
ZS56	1	0	0.5	0.5	0.5	0.5	1	0	0	0.8	0	0.2	0	0	0
ZH12	0.4	0.6	0.4	0.6	0.6	0.4	1	0	0.5	0.5	0	0	0	0	0
ZH16	1	0	0.6	0.4	0.6	0.4	1	0	0.5	0.5	0	0	0	0	0
ZH18	0.8	0.2	0.2	0.8	0.3	0.7	1	0	0	0.4	0	0	0	0.6	0
ZH21	1	0	0	1	0	1	1	0	0	0.5	0.5	0	0	0	0
ZH23	1	0	0.2	0.8	1	0	1	0	0.3	0.7	0	0	0	0	0
ZH32	1	0	0	1	0.1	0.9	1	0	0.5	0.5	0	0	0	0	0
ZH34	1	0	0.2	0.8	1	0	1	0	0	0.4	0	0	0.6	0	0
MW6	1	0	0	1	0.43	0.57	1	0	0	1	0	0	0	0	0
MW11	1	0	0.33	0.67	0.33	0.67	1	0	0.67	0.33	0	0	0	0	0
MW28	0.6	0.4	0	1	0	1	1	0	0	1	0	0	0	0	0
MW63	0.67	0.33	0	1	0.56	0.44	0.89	0.11	0.67	0	0	0	0	0	0.33

Sample size = 10 for each isofemale line except MW6 (7), MW11 (6), and MW63 (9).

Table A2 High observed heterozygosity in Afrotropical isofemale lines

African lines	Heterozygosity														
	X			2L			2R			3L			3R		
	Genotype	O	E	Genotype	O	E	Genotype	O	E	Genotype	O	E	Genotype	O	E
MW6	-	-	-	-	-	-	*NS*/ST	0.59	0.49	-	-	-	-	0	-
ZS2	-	-	-		-	-	*NS*/ST*	0.46	0.18	-	-	-	*K*/ST	0.50	0.42
ZS6	-	-	-	*t*/ST	0.23	0.42	-	-	-	-	-	-	*K/P*	0.55	0.50
ZS8	-	-	-	-	-	-	-	-	-	*Ok*/ST*	0.67	0.42	*K*/ST*	0.67	0.32
ZS11	-	-	-	-	-	-	*NS*/ST*	0.55	0.18	-	-	-	*K*/ST*	0.85	0.50
ZS30	-	-	-	-	-	-	-	-	-	-	-	-	*K*/ST*	0.73	0.42
ZS53	-	-	-	*t*/ST*	1.00	0.50	*NS*/ST*	0.95	0.48	-	-	-	*K/93;96**	0.85	0.48
ZS56	-	-	-	*t*/ST*	0.96	0.50	*NS*/ST*	0.91	0.50	-	-	-	*K/93;96**	0.63	0.32
ZH12	*A*/ST	0.40	0.48	*t*/ST	0.54	0.48	*NS*/ST	0.45	0.48	-	-	-	*K*/ST	0.55	0.50
ZH18	*A*/ST	0.41	0.32	*t*/ST	0.65	0.32	*NS*/ST	0.40	0.42	-	-	-	*K/C*	0.50	0.48
ZH21	-	-	-	-	-	-	-	-	-	-	-	-	*K/P*	0.42	0.50
ZH32	-	-	-	-	-	-	*NS*/ST	0.04	0.18	-	-	-	*K*/ST*	0.83	0.50
ZH34	-	-	-	*t*/ST	0.52	0.32	-	-	-	-	-	-	*K/93;98**	0.95	0.48

O: observed heterozygosity; E: expected heterozygosity; "-": chromosomal rearrangements on specific chromosomes of an isofemale line are monomorphic and are thus inappropriate for heterozygosity comparison.
Sample size N>20 for estimating observation heterozygosity.
*Chi-square test significant at single isofemale line level.

	♀		♂	
G_0	I_Z; II_Z; TM3/Sb	x	I_Z;II_Z; III_1/III_2	
G_1	I_Z; II_Z; TM3/Sb	x	I_Z;II_Z; TM3/III_1 (Single) or I_Z;II_Z; TM3/III_2 (Single)	
G_2	I_Z; II_Z; TM3/III_1	x	I_Z; II_Z; III_1/Sb	
G_3	I_Z; II_Z; TM3/III_1	x	I_Z; II_Z; TM3/III_1	
G_4	I_Z; II_Z; TM3/III_1	and	I_Z; II_Z; III_1/III_1	
	2	:	1	No recessive lethals
	2	:	0	With recessive lethals

Figure A3 The cross scheme for isogenizing 3rd chromosomes. III_1 and III_2 denote different homologous 3rd chromosomes, which may bear recessive lethal or not.

Table A3 Existence of recessive lethals and sterile alleles on the third chromosome isogenized from Zimbabwe isofemale lines

African lines	Chromosomal rearrangement	Existence of recessive lethal	Female sterility
ZS2	ST	v	
	In(3R)K		
ZS6	*In(3R)K*		
	In(3R)P	v	
ZS8	ST		
	In(3L)Ok	v	
ZS11	ST		
	In(3R)K		v
ZS30	ST	v	
	In(3R)K		v
ZS53	*In(3R)K*	v	
	In(3R)93;96		v
ZS56	*In(3R)93;96*		
	In(3R)K	NA	NA
ZH12	ST	v	
	In(3R)K		v
ZH18	*In(3R)C*		
	In(3R)K	v	
ZH21	*In(3R)K*	v	
	In(3R)P	NA	NA
ZH32	ST	v	
	In(3R)K		v
ZH34	*In(3R)93;98*	v	
	In(3R)K	NA	NA

v : existence of recessive lethals or female sterility. NA: not assayed

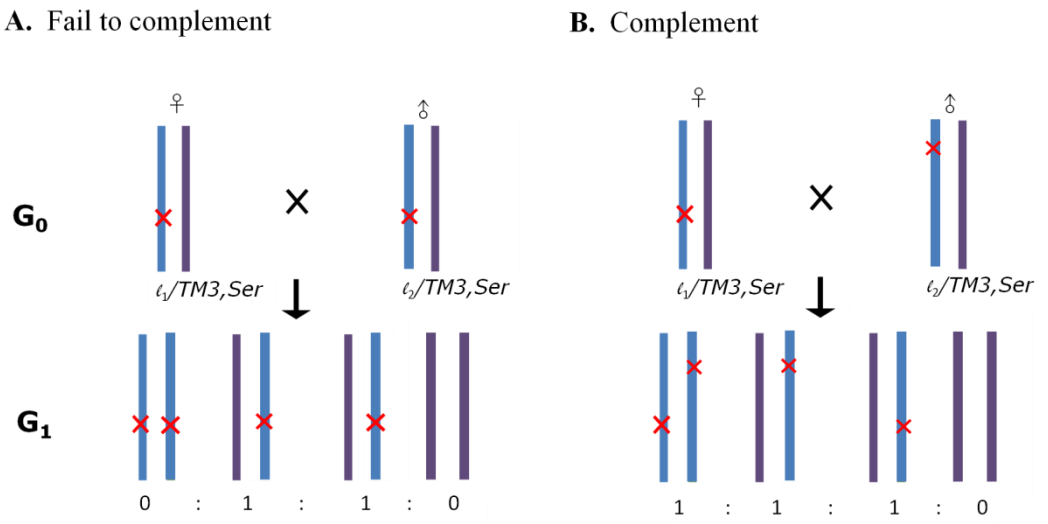

Figure A4 Complementation test for the allelism between two recessive lethal alleles. If recessive lethal alleles fail to complement, the wild type pffspring would not be present. The *TM3,Ser*/*TM3,Ser* homozygote is lethal.

Table A4 Complementation test for the allelism of each recessive lethal allele

Isogenized chromosomes		ZH18-6	ZH21-1	ZH32-1	ZS2-3	ZS30-2	ZS53-3
	Chromosome rearrangements	*In(3R)K*	*In(3R)K*	ST	ST	ST	*In(3R)K*
ZH18-6	*In(3R)K*		+	+	+	+	+
ZH21-1	*In(3R)K*	+		+	+	+	+
ZH32-1	ST	+	+		+	+	+
ZS2-3	ST	+	+	+		+	+
ZS30-2	ST	+	+	+	+		+
ZS53-3	*In(3R)K*	+	+	+	+	+	

"+" denotes wild type offspring of each cross between two isogenic lines.

Appendix B

Supplementary data

Table B1 list the genotype of all deficiency lines which used in deficiency mapping. Table B2 represents the scoring of G_2 and G_3 from recombination mapping. Table B3 shows F_1 scoring of deficiency mapping. From scoring recombinants (G_2 of recombination mapping, Figure2), the recombination-suppressed region in the inverted/straight heterozygous can be roughly estimated (Table B4), though the marker is limited (Figure B1). For example, *In(3R)K* is covered from marker *cu* to *ca*, but the there is no double crossing over occurs within *In(3R)K* (see discussion). Table B5 shows the comparison of recombination rates between inversion heterozygotes and straight homozygotes. In addition, Table B6 and Figure B2 show the state of recessive lethal alleles bearing by balancer, *TM3*.

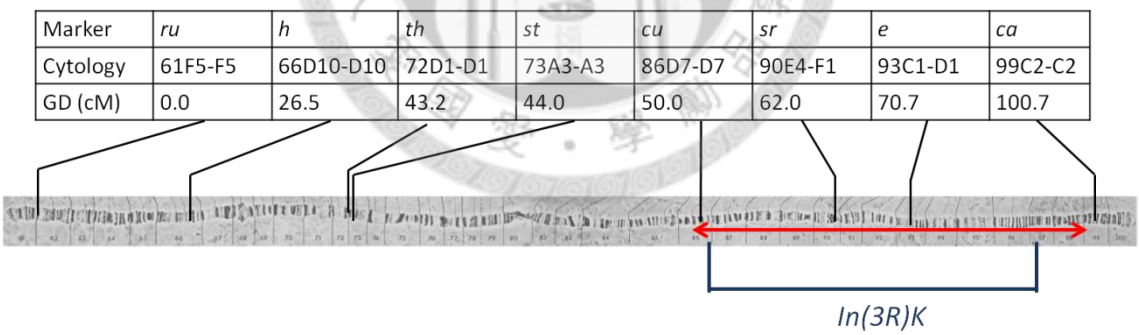

Figure B1 Genomic position of both *rucuca* markers and the inversion, take In(3R)K as example. The recombination-suppressed region in the inverted/straight heterozygous 3rd chromosome extends to larger than 20 cM. Red double arrows denote recombination-suppressed region by inversion heterozygotes.

Table B1 Deficiency lines of 3rd arm chromosome used in mapping

Stock number	Cytological position	Deletion breakpoints	Genotype
8048	61C1;61E2	3L:319846;1035182	*w*^{1118}; *Df(3L)ED4177, P{3'.RS5+3.3'}ED4177/TM6C, cu*^{1} *Sb*^{1}
8051	61C9;61F7	3L:738739;1336381	*w*^{1118}; *Df(3L)ED202, P{3'.RS5+3.3'}ED202/TM6C, cu*^{1} *Sb*^{1}
8052	61C9;62A4	3L:738739;1546931	*w*^{1118}; *Df(3L)ED4238, P{3'.RS5+3.3'}ED4238/TM6C, cu*^{1} *Sb*^{1}
8053	61C9;62A6	3L:738739;1568108	*w*^{1118}; *Df(3L)ED207, P{3'.RS5+3.3'}ED207/TM2*
8054	62A3;62A6	3L:1546104;1586663	*w*^{1118}; *Df(3L)ED4256, P{3'.RS5+3.3'}ED4256/TM6C, cu*^{1} *Sb*^{1}
8056	62B4;62B12	3L:1795442;1963552	*w*^{1118}; *Df(3L)ED4284, P{3'.RS5+3.3'}ED4284/TM6C, cu*^{1} *Sb*^{1}
8096	62B4;62E5	3L:1795442;2551761	*w*^{1118}; *Df(3L)ED4287, P{3'.RS5+3.3'}ED4287/TM6C, cu*^{1} *Sb*^{1}
8057	63A6;63B7	3L:3070827;3149091	*w*^{1118}; *Df(3L)ED4288, P{3'.RS5+3.3'}ED4288/TM6C, cu*^{1} *Sb*^{1}
8058	63C1;63C1	3L:3226338;3250564	*w*^{1118}; *Df(3L)ED4293, P{3'.RS5+3.3'}ED4293/TM6C, cu*^{1} *Sb*^{1}
8059	63C1;63F5	3L:3249148;3893148	*w*^{1118}; *Df(3L)ED208, P{3'.RS5+3.3'}ED208/TM6C, cu*^{1} *Sb*^{1}
8060	63F6;64B9	3L:3905091;4542236	*w*^{1118}; *Df(3L)ED4341, P{3'.RS5+3.3'}ED4341/TM6C, cu*^{1} *Sb*^{1}
8062	64A12;64B12	3L:4277987;4625372	*w*^{1118}; *Df(3L)ED4342, P{3'.RS5+3.3'}ED4342/TM6C, cu*^{1} *Sb*^{1}
8061	64B9;64C13	3L:4544234;5348442	*w*^{1118}; *Df(3L)ED210, P{3'.RS5+3.3'}ED210/TM6C, cu*^{1} *Sb*^{1}
25119	64C7;64E1	3L:5129360;5627605	*w*^{1118}; *Df(3L)BSC557/TM6C, Sb*^{1}
30589	64D6;64E7	3L:5601375;5770185	*w*^{1118}; *Df(3L)BSC884/TM6C, Sb*^{1} *cu*^{1}
24914	64E7;65B3	3L:5763773;6483285	*w*^{1118}; *Df(3L)BSC410/TM6C, Sb*^{1} *cu*^{1}
8063	65A9;65B4	3L:6211235;6545859	*w*^{1118}; *Df(3L)ED211, P{3'.RS5+3.3'}ED211/TM6C, cu*^{1} *Sb*^{1}
150518*	65A9;65D5	3L:6211753;6957841	*w*^{1118}; *Df(3L)ED212 / TM6B, P{Ubi-GFP.S65T}PAD2, Tb*^{1}
9701	65D5;65E6	3L:6957557;7150109	*w*^{1118}; *Df(3L)BSC224/TM6C, Sb*^{1} *cu*^{1}
8065	66A22;66C5	3L:7972207;8292674	*w*^{1118}; *Df(3L)ED4408, P{3'.RS5+3.3'}ED4408/TM6C, cu*^{1} *Sb*^{1}
9194	66D12;66E6	3L:8759071;8972087	*w*^{1118}; *Df(3L)ED4415, P{3'.RS5+3.3'}ED4415/TM6C, cu*^{1} *Sb*^{1}
9070	66D12;66E6	3L:8759532;8972087	*w*^{1118}; *Df(3L)ED4413, P{3'.RS5+3.3'}ED4413/TM6C, cu*^{1} *Sb*^{1}

Table B1 (Continued)

Stock number	Cytological position	Deletion breakpoints	Genotype
8066	66D12;67B3	3L:8738426;9377175	*w*^{1118}; *Df(3L)ED4421, P{3'.RS5+3.3'}ED4421/TM6C, cu*^{1} *Sb*^{1}
9221	66E1;67B1	3L:8820579;9342724	*w*^{1118}; *Df(3L)ED4416, P{3'.RS5+3.3'}ED4416/TM6C, cu*^{1} *Sb*^{1}
8970	67B1;67B5	3L:9342609;9416591	*w*^{1118}; *Df(3L)BSC113/TM6B, Tb*^{1}
24415	67B7;67C5	3L:9439870;9690291	*w*^{1118}; *Df(3L)BSC391/TM6C, Sb*^{1} *cu*^{1}
24416	67C4;67D1	3L:9671803;9892355	*w*^{1118}; *Df(3L)BSC392/TM6C, Sb*^{1} *cu*^{1}
26525	67C7;67D10	3L:9756714;10174058	*w*^{1118}; *Df(3L)BSC673, P+PBac{XP3.WH3}BSC673/TM6C, Sb*^{1} *cu*^{1}
9355	67E2;68A7	3L:10357051;11118909	*w*^{1118}; *Df(3L)ED4457, P{3'.RS5+3.3'}ED4457/TM6C, cu*^{1} *Sb*^{1}
8068	68A6;68E1	3L:11090089;11826330	*w*^{1118}; *Df(3L)ED4470, P{3'.RS5+3.3'}ED4470/TM6C, cu*^{1} *Sb*^{1}
8069	68C13;69B4	3L:11580140;12401701	*w*^{1118}; *Df(3L)ED4475, P{3'.RS5+3.3'}ED4475/TM6C, cu*^{1} *Sb*^{1}
8070	69A5;69D3	3L:12270320;12686314	*w*^{1118}; *Df(3L)ED4483, P{3'.RS5+3.3'}ED4483/TM6C, cu*^{1} *Sb*^{1}
8071	69B5;69C4	3L:12410653;12497398	*w*^{1118}; *Df(3L)ED215, P{3'.RS5+3.3'}ED215/TM6C, cu*^{1} *Sb*^{1}
8072	69C4;69F6	3L:12507519;13025585	*w*^{1118}; *Df(3L)ED4486, P{3'.RS5+3.3'}ED4486/TM6C, cu*^{1} *Sb*^{1}
8097	70A3;70C10	3L:13220865;13986651	*w*^{1118}; *Df(3L)ED4502, P{3'.RS5+3.3'}ED4502/TM6C, cu*^{1} *Sb*^{1}
9214	70C11;70D3	3L:13995861;14198424	*w*^{1118}; *Df(3L)ED4536, P{3'.RS5+3.3'}ED4536/TM6C, cu*^{1} *Sb*^{1}
9072	70C15;70D2	3L:14030141;14070123	*w*^{1118}; *Df(3L)ED4528, P{3'.RS5+3.3'}ED4528/TM6C, cu*^{1} *Sb*^{1}
9074	70C15;70D3	3L:14030141;14186794	*w*^{1118}; *Df(3L)ED4534, P{3'.RS5+3.3'}ED4534/TM6C, cu*^{1} *Sb*^{1}
9071	70C6;70C15	3L:13932272;14030132	*w*^{1118}; *Df(3L)ED4515, P{3'.RS5+3.3'}ED4515/TM6C, cu*^{1} *Sb*^{1}
9073	70C6;70D2	3L:13932272;14070123	*w*^{1118}; *Df(3L)ED4529, P{3'.RS5+3.3'}ED4529/TM6C, cu*^{1} *Sb*^{1}
8073	70C6;70F4	3L:13928325;14751140	*w*^{1118}; *Df(3L)ED4543, P{3'.RS5+3.3'}ED4543/TM6C, cu*^{1} *Sb*^{1}
8074	70F4;71E1	3L:14751170;15582196	*w*^{1118}; *Df(3L)ED217, P{3'.RS5+3.3'}ED217/TM6C, cu*^{1} *Sb*^{1}
8075	71B1;71E1	3L:15007168;15582196	*w*^{1118}; *Df(3L)ED218, P{3'.RS5+3.3'}ED218/TM6C, cu*^{1} *Sb*^{1}
27888	71D3;72A1	3L:15504128;15819023	*w*^{1118}; *Df(3L)BSC845/TM6C, Sb*^{1} *cu*^{1}

Table B1 (Continued)

Stock number	Cytological position	Deletion breakpoints	Genotype
27346	71F1;72D10	3L:15693003;16233373--16233380	*w*^{1118}; *Df(3L)BSC774/TM6C, Sb*^{1} *cu*^{1}
8077	72D4;72F1	3L:16080584;16404777	*w*^{1118}; *Df(3L)ED220, P{3'.RS5+3.3'}ED220/TM6C, cu*^{1} *Sb*^{1}
8078	72D4;73C4	3L:16080584;16773223	*w*^{1118}; *Df(3L)ED4606, P{3'.RS5+3.3'}ED4606/TM6C, cu*^{1} *Sb*^{1}
8079	73A1;73D5	3L:16444925;16883977	*w*^{1118}; *Df(3L)ED223, P{3'.RS5+3.3'}ED223/TM6C, cu*^{1} *Sb*^{1}
8099	73D5;74E2	3L:16884176;17605270	*w*^{1118}; *Df(3L)ED4685, P{3'.RS5+3.3'}ED4685/TM6C, cu*^{1} *Sb*^{1}
8100	74D1;75B11	3L:17480563;18132399	*w*^{1118}; *Df(3L)ED4710, P{3'.RS5+3.3'}ED4710/TM2*
8080	75B1;75C6	3L:17962303;18391619	*w*^{1118}; *Df(3L)ED224, P{3'.RS5+3.3'}ED224/TM6C, cu*^{1} *Sb*^{1}
8081	75C1;75D4	3L:18179245;18614437	*w*^{1118}; *Df(3L)ED225, P{3'.RS5+3.3'}ED225/TM6C, cu*^{1} *Sb*^{1}
8082	75F2;76A1	3L:18988994;19163802	*w*^{1118}; *Df(3L)ED4782, P{3'.RS5+3.3'}ED4782/TM6C, cu*^{1} *Sb*^{1}
8087	76A1;76E1	3L:19163806;19995811	*w*^{1118}; *Df(3L)ED229, P{3'.RS5+3.3'}ED229/TM6C, cu*^{1} *Sb*^{1}
8088	76D3;77C1	3L:19888473;20394920	*w*^{1118}; *Df(3L)ED4858, P{3'.RS5+3.3'}ED4858/TM2*
27369	77C3;78A1	3L:20445923;20942833	*w*^{1118}; *Df(3L)BSC797/TM6C, Sb*^{1} *cu*^{1}
24953	77F2;78C2	3L:20850015--20850088;21196030	*w*^{1118}; *Df(3L)BSC449/TM6C, Sb*^{1} *cu*^{1}
24923	78C2;78D8	3L:21218032;21597878	*w*^{1118}; *Df(3L)BSC419/TM6C, Sb*^{1} *cu*^{1}
8101	78D5;79A2	3L:21526907;21873785	*w*^{1118}; *Df(3L)ED4978, P{3'.RS5+3.3'}ED4978/TM6C, cu*^{1} *Sb*^{1}
7616	78F4;79A4	3L:21836682;21948191	*w*^{1118}; *Df(3L)Exel6137, P{XP-U}Exel6137/TM6B, Tb*^{1}
9700	79A3;79B3	3L:21909520--21909525;22078536	*w*^{1118}; *Df(3L)BSC223/TM6C, Sb*^{1} *cu*^{1}
23149	79B2;79D1	3L:22069193--22069195;22200952	*w*^{1118}; *Df(3L)BSC249/TM6C, Sb*^{1} *cu*^{1}
8089	79C2;80A4	3L:22127751;22827471	*w*^{1118}; *Df(3L)ED230, P{3'.RS5+3.3'}ED230/TM6C, cu*^{1} *Sb*^{1}
8102	80A4;80C2	3L:22828597;22991401	*w*^{1118}; *Df(3L)ED5017, P{3'.RS5+3.3'}ED5017/TM6C, cu*^{1} *Sb*^{1}
9226	81F6;82E7	3R:22995;912807	*w*^{1118}; *Df(3R)ED5100, P{3'.RS5+3.3'}ED5100/TM6C, cu*^{1} *Sb*^{1}
8967	82E7;83A1	3R:912842;1193526	*w*^{1118}; *Df(3R)ED5147, P{3'.RS5+3.3'}ED5147/TM6C, cu*^{1} *Sb*^{1}

Table B1 (Continued)

Stock number	Cytological position	Deletion breakpoints	Genotype
9159	83A7;83B4	3R:1344078;1426000	*w*^{1118}; *Df(3R)ED10257, P{3'.RS5+3.3'}ED10257/TM6C, cu*^{1} *Sb*^{1}
8103	83B4;83B6	3R:1426351;1449817	*w*^{1118}; *Df(3R)ED5177, P{3'.RS5+3.3'}ED5177/TM6C, cu*^{1} *Sb*^{1}
9339	83B7;83D2	3R:1474504;1833866	*w*^{1118}; *Df(3R)ED5197, P{3'.RS5+3.3'}ED5197/TM6C, cu*^{1} *Sb*^{1}
24968	83B7;83E1	3R:1474083;2037668	*w*^{1118}; *Df(3R)BSC464/TM6C, Sb*^{1} *cu*^{1}
26533	83E2;83E5	3R:2111067;2206257	*w*^{1118}; *Df(3R)BSC681, P+PBac{XP3.RB5}BSC681/TM6C, Sb*^{1} *cu*^{1}
26843	83E2;83E5	3R:2129696;2206257	*w*^{1118}; *Df(3R)BSC745, P+PBac{XP3.WH3}BSC745/TM6C, Sb*^{1} *cu*^{1}
26844	83E2;83E6	3R:2138798--2139080;2231048	*w*^{1118}; *Df(3R)BSC746/TM6C, Sb*^{1} *cu*^{1}
26842	83E4;83E6	3R:2168401;2231048	*w*^{1118}; *Df(3R)BSC744/TM6C, Sb*^{1} *cu*^{1}
9620	83E5;83F4	3R:2206257;2444575	*w*^{1118}; *Df(3R)BSC193/TM6B, Tb*$^{+}$
26836	83E5;84A1	3R:2206257;2510873	*w*^{1118}; *Df(3R)BSC738/TM6C, Sb*^{1} *cu*^{1}
24971	83F1;84B2	3R:2365827;2824771	*w*^{1118}; *Df(3R)BSC467/TM6C, Sb*^{1} *cu*^{1}
24926	84A5;84B2	3R:2532402;2824771	*w*^{1118}; *Df(3R)BSC422/TM6C, Sb*^{1} *cu*^{1}
25724	84B2;84C3	3R:2906110;2949098	*w*^{1118}; *Df(3R)BSC633/TM6C, cu*^{1} *Sb*^{1}
8685	84B4;84E11	3R:2916249;3919805	*w*^{1118}; *Df(3R)ED7665, P{3'.RS5+3.3'}ED7665/TM6C, cu*^{1} *Sb*^{1}
8682	84E6;85A5	3R:3803496;4478856	*w*^{1118}; *Df(3R)ED5230, P{3'.RS5+3.3'}ED5230/TM6C, cu*^{1} *Sb*^{1}
9338	84F6;85C3	3R:4076143;4882413	*w*^{1118}; *Df(3R)ED5296, P{3'.RS5+3.3'}ED5296/TM6C, cu*^{1} *Sb*^{1}
9203	85C3;85D1	3R:4859916;5055517	*w*^{1118}; *Df(3R)ED5331, P{3'.RS5+3.3'}ED5331/TM6C, cu*^{1} *Sb*^{1}
9204	85D1;85D11	3R:5052798;5178097	*w*^{1118}; *Df(3R)ED5339, P{3'.RS5+3.3'}ED5339/TM6C, cu*^{1} *Sb*^{1}
25011	85D6;85D15	3R:5084968;5220302	*w*^{1118}; *Df(3R)BSC507/TM6C, Sb*^{1} *cu*^{1}
24980	85D16;85D24	3R:5243395;5380704	*w*^{1118}; *Df(3R)BSC476/TM6C, Sb*^{1} *cu*^{1}
25696	85F5;85F14	3R:5826632--5826961;5970476	*w*^{1118}; *Df(3R)BSC621/TM6C, cu*^{1} *Sb*^{1}
8919	85D24;85E5	3R:5376427;5530672	*w*^{1118}; *Df(3R)ED5429, P{3'.RS5+3.3'}ED5429/TM2*

Table B1 (Continued)

Stock number	Cytological position	Deletion breakpoints	Genotype
9082	85F11;86B1	3R:5935134;6176446	*w*^{1118}; *Df(3R)ED5474, P{3'.RS5+3.3'}ED5474/TM6C, cu*^{1} *Sb*^{1}
9215	85F16;86C7	3R:5996223;6712482	*w*^{1118}; *Df(3R)ED5495, P{3'.RS5+3.3'}ED5495/TM6C, cu*^{1} *Sb*^{1}
150152*	86C7; 86E11	3R:6710720;7394975.	*w*^{1118}; *Df(3R)ED5514 / TM6C, Sb*^{1}
8920	86E11;87B11	3R:7394904;8269738	*w*^{1118}; *Df(3R)ED5559, P{3'.RS5+3.3'}ED5559/TM2*
9087	87B11;87D7	3R:8269738;8821397	*w*^{1118}; *Df(3R)ED5610, P{3'.RS5+3.3'}ED5610/TM6C, cu*^{1} *Sb*^{1}
7972	87C7;87D5--6	3R:8549577;8772996--8801525	*w*^{1118}; *Df(3R)Exel7318/TM6B, Tb*^{1}
7973	87D8;87D10	3R:8838435;8877507	*w*^{1118}; *Df(3R)Exel8157/TM6B, Tb*^{1}
7646	87D10;87E3	3R:8877179;9085101	*w*^{1118}; *Df(3R)Exel6167, P{XP-U}Exel6167/TM6B, Tb*^{1}
8921	87E3;88A4	3R:9085471;9809634	*w*^{1118}; *Df(3R)ED5623, P{3'.RS5+3.3'}ED5623/TM2*
9090	88A4;88C9	3R:9843625;10451431	*w*^{1118}; *Df(3R)ED5644, P{3'.RS5+3.3'}ED5644/TM6C, cu*^{1} *Sb*^{1}
29025	88C6;88D2	3R:10384103;10576978	*w*^{1118}; *Df(3R)BSC841/mwh*^{1} *kni*$^{ri-1}$ *snk*^{4} *red*^{1} *e*^{1} *Tl*^{3} *ca*^{1}*/TM6C, Sb*^{1} *cu*^{1}
7742	88D1;88D7	3R:10549487;10743982	*w*^{1118}; *Df(3R)Exel6275, P{XP-U}Exel6275/TM6B, Tb*^{1}
24137	88D1;88E3	3R:10523031;11054571	*w*^{1118}; *Df(3R)ED5664, P{3'.RS5+3.3'}ED5664/TM6C, cu*^{1} *Sb*^{1}
26848	88E2;88E5	3R:10960881;11075682	*w*^{1118}; *Df(3R)BSC750/TM6C, Sb*^{1} *cu*^{1}
26839	88E8;88F1	3R:11090909--11090913;11166385	*w*^{1118}; *Df(3R)BSC741/TM6C, Sb*^{1} *cu*^{1}
9152	88E12;89A5	3R:11117380;11619518	*w*^{1118}; *Df(3R)ED5705, P{3'.RS5+3.3'}ED5705/TM2*
7982	89A8;89B1	3R:11727155--11727156;11867284	*w*^{1118}; *Df(3R)Exel7327/TM6B, Tb*^{1}
7983	89A12;89B6	3R:11835140;11983178	*w*^{1118}; *Df(3R)Exel7328/TM6B, Tb*^{1}
9481	89B7;89B18	3R:12038635;12306942	*w*^{1118}; *Df(3R)ED10639, P{3'.RS5+3.3'}ED10639/TM6C, cu*^{1} *Sb*^{1}
9482	89B17;89D5	3R:12279479;12450993	*w*^{1118}; *Df(3R)ED10642, P{3'.RS5+3.3'}ED10642/TM6C*
8104	89E11;90C1	3R:12882199;13507523	*w*^{1118}; *Df(3R)ED5780, P{3'.RS5+3.3'}ED5780/TM2*
27362	90B6;90E2	3R:13432489;13875093--13875130	*w*^{1118}; *Df(3R)BSC790, P+PBac{XP3.WH3}BSC790/TM6C, Sb*^{1} *cu*^{1}

Table B1 (Continued)

Stock number	Cytological position	Deletion breakpoints	Genotype
7657	90F4;91A5	3R:13992149;14223078	*w*^{1118}; *Df(3R)Exel6178, P{XP-U}Exel6178/TM6B, Tb*^{1}
6962	91A5;91F1	3R:14224953;14922493	*w*^{1118}; *Df(3R)BSC177/TM6B, Tb*^{1}
8922	91F12;92B3	3R:15052016;15660809	*w*^{1118}; *Df(3R)ED5942, P{3'.RS5+3.3'}ED5942/TM6C, cu*^{1} *Sb*^{1}
9479	92B3;92E2	3R:15662595;16135241	*w*^{1118}; *Df(3R)ED6027, P{3'.RS5+3.3'}ED6027/TM6C, cu*^{1} *Sb*^{1}
25021	92C1;92F13	3R:15856989;16609520	*w*^{1118}; *Df(3R)BSC517/TM2*
24992	92F2;92F13	3R:16392669;16616293	*w*^{1118}; *Df(3R)BSC488/TM2*
9501	92F2;93A1	3R:16420602;16632118	*w*^{1118}; *Df(3R)BSC141/TM6B, Tb*$^{+}$
27580	93A2;93B8	3R:16677299;16886325	*w*^{1118}; *Df(3R)BSC819, P+PBac{XP3.RB5}BSC819/TM6C, Sb*^{1} *cu*^{1}
7739	93A4;93B13	3R:16783425;16938056	*w*^{1118}; *Df(3R)Exel6272, P{XP-U}Exel6272/TM6B, Tb*^{1}
9487	93B9;93D4	3R:16890893;17122221	*w*^{1118}; *Df(3R)ED10845, P{3'.RS5+3.3'}ED10845/TM6C, cu*^{1} *Sb*^{1}
9480	93D4;93D8	3R:17122205;17191074	*w*^{1118}; *Df(3R)ED6052, P{3'.RS5+3.3'}ED6052/TM6C, cu*^{1} *Sb*^{1}
24140	93D4;93F6	3R:17122217;17545322	*w*^{1118}; *Df(3R)ED6058, P{3'.RS5+3.3'}ED6058/TM6C, cu*^{1} *Sb*^{1}
8962	93E10;94A1	3R:17459227;17868550	*w*^{1118}; *Df(3R)ED6076, P{3'.RS5+3.3'}ED6076/TM6C, cu*^{1} *Sb*^{1}
8924	94A2;94C4	3R:17959510;18552029	*w*^{1118}; *Df(3R)ED6093, P{3'.RS5+3.3'}ED6093/TM2*
8684	94B5;94E7	3R:18413403;19047691	*w*^{1118}; *Df(3R)ED6096, P{3'.RS5+3.3'}ED6096/TM6C, cu*^{1} *Sb*^{1}
8963	94D3;94E9	3R:18724275;19084137	*w*^{1118}; *Df(3R)ED6103, P{3'.RS5+3.3'}ED6103/TM6C, cu*^{1} *Sb*^{1}
7673	94F1;95A4	3R:19210900;19467128	*w*^{1118}; *Df(3R)Exel6194, P{XP-U}Exel6194/TM6B, Tb*^{1}
7674	95A4;95B1	3R:19467128;19549624	*w*^{1118}; *Df(3R)Exel6195, P{XP-U}Exel6195/TM6B, Tb*^{1}
7991	95B1;95B5	3R:19548559;19610564	*w*^{1118}; *Df(3R)Exel9013/TM6B, Tb*^{1}
7992	95B1;95D1	3R:19598843;19768726	*w*^{1118}; *Df(3R)Exel9014/TM6B, Tb*^{1}
7675	95C12;95D8	3R:19747854--19747855;19857149	*w*^{1118}; *Df(3R)Exel6196, P{XP-U}Exel6196/TM6B, Tb*^{1}
7676	95D8;95E1	3R:19857149;19967091	*w*^{1118}; *Df(3R)Exel6197, P{XP-U}Exel6197/TM6B, Tb*^{1}
9347	95D10;96A7	3R:19877370;20369665	*w*^{1118}; *Df(3R)ED6187, P{3'.RS5+3.3'}ED6187/TM2*
9211	96A7;96C3	3R:20369520;21009495	*w*^{1118}; *Df(3R)ED6220, P{3'.RS5+3.3'}ED6220/TM6C, cu*^{1} *Sb*^{1}

Table B1 (Continued)

Stock number	Cytological position	Deletion breakpoints	Genotype
24965	96B15;96D1	3R:20829131;21118634--21118719	*w*^{1118}; *Df(3R)BSC461/TM6C, Sb*^{1} *cu*^{1}
7680	96C2;96C4	3R:20963561;21022720	*w*^{1118}; *Df(3R)Exel6201, P{XP-U}Exel6201/TM6B, Tb*^{1}
7994	96C4;96C5	3R:21022721;21035008--21035010	*w*^{1118}; *Df(3R)Exel9056/TM6B, Tb*^{1}
7681	96D1;96D1	3R:21118719;21341395	*w*^{1118}; *Df(3R)Exel6202, P{XP-U}Exel6202/TM6B, Tb*^{1}
7682	96E2;96E6	3R:21341620;21463598	*w*^{1118}; *Df(3R)Exel6203, P{XP-U}Exel6203/TM6B, Tb*^{1}
7683	96F9;97A6	3R:21832109;22087161	*w*^{1118}; *Df(3R)Exel6204, P{XP-U}Exel6204/TM6B, Tb*^{1}
8105	96F10;97D2	3R:21862598;22624704	*w*^{1118}; *Df(3R)ED6232, P{3'.RS5+3.3'}ED6232/TM6C, cu*^{1} *Sb*^{1}
9210	97D2;97F1	3R:22624758;23107623	*w*^{1118}; *Df(3R)ED6255, P{3'.RS5+3.3'}ED6255/TM6C, cu*^{1} *Sb*^{1}
8960	97E2;98A7	3R:22937981;23405492	*w*^{1118}; *Df(3R)ED6265, P{3'.RS5+3.3'}ED6265/TM6C, cu*^{1} *Sb*^{1}
25001	97E6;98B5	3R:22991339;23731307	*w*^{1118}; *Df(3R)BSC497/TM6C, Sb*^{1} *cu*^{1}
25390	98B6;98E5	3R:23763552;24627253	*w*^{1118}; *Df(3R)BSC567/TM6C, Sb*^{1}
7688	98E1;98F5	3R:24500683;24816740	*w*^{1118}; *Df(3R)Exel6210, P{XP-U}Exel6210/TM6B, Tb*^{1}
27378	98F1;98F10	3R:24696033;24938249	*w*^{1118}; *Df(3R)BSC806, P+PBac{XP3.RB5}BSC806/TM6C, Sb*^{1} *cu*^{1}
25005	98F10;99B9	3R:24938249;25550407	*w*^{1118}; *Df(3R)BSC501/TM6C, Sb*^{1} *cu*^{1}
8961	98F12;99B2	3R:24964617;25337875	*w*^{1118}; *Df(3R)ED6310, P{3'.RS5+3.3'}ED6310/TM6C, cu*^{1} *Sb*^{1}
8925	99A5;99C1	3R:25081045;25608389	*w*^{1118}; *Df(3R)ED6316, P{3'.RS5+3.3'}ED6316/TM6C, cu*^{1} *Sb*^{1}

All stocks were obtained from Drosophila Genomics Resource Center, Bloomington, except "*" were from Drosophila Genetic Resource Center, Kyoto.

Table B2-a G2 and G3 scoring of recombination mapping--ZS2-3

Recombinants								G2 Number	G3 scoring											
									Genotype	*ℓ*/rec.			*TM3,Ser* /rec.			*TM3,Ser* /*ruPrca*			*ℓ*/*ruPrca*		
									Replicate	1	2	3	1	2	3	1	2	3	1	2	3
ru	+	+	+	+	+	+	+	55		0	0		67	76		69	63		64	51	
ru	*h*	+	+	+	+	+	+	31		0	0		82	91		82	66		80	64	
ru	*h*	*th*	+	+	+	+	+	0													
ru	*h*	*th*	*st*	+	+	+	+	6		0	0		85	64		84	68		107	64	
ru	*h*	*th*	*st*	*cu*	+	+	+	12		0	0		48	67		36	87		47	81	
ru	*h*	*th*	*st*	*cu*	*sr*	+	+	7													
ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	57		60	15		46	18		42	16		49	17	
+	+	+	+	+	+	+	*ca*	83		0	0		53	72		69	57		66	69	
+	+	+	+	+	+	*e*	*ca*	22		0	0		73	63		67	51		89	57	
+	+	+	+	+	*sr*	*e*	*ca*	25		0	0		104	83		88	101		100	116	
+	+	+	+	*cu*	*sr*	*e*	*ca*	2													
+	+	+	*st*	*cu*	*sr*	*e*	*ca*	0													
+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	27		63	49		50	39		48	45		77	54	
+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	26		20	45		19	38		18	37		22	49	
Others								475													
Total								828													

Table B2-b G2 and G3 scoring of recombination mapping--ZS30-2

Recombinants								G2 Number	Genotype	G5 scoring rec(ℓ)/rec(ℓ)			G5 scoring *TM3,Ser* /rec.		
									Replicate	1	2	3	1	2	3
ru	+	+	+	+	+	+	+	42		0	0	0	46	65	37
ru	*h*	+	+	+	+	+	+	20		0	0	0	37	54	54
ru	*h*	*th*	+	+	+	+	+	0							
ru	*h*	*th*	*st*	+	+	+	+	0							
ru	*h*	*th*	*st*	*cu*	+	+	+	3		2	3	1	33	25	50
ru	*h*	*th*	*st*	*cu*	*sr*	+	+	1		2			62		
ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	5		10	15		47	46	
+	+	+	+	+	+	+	*ca*	51		0	0	0	20	9	47
+	+	+	+	+	+	*e*	*ca*	9		0	0		54	9	
+	+	+	+	+	*sr*	*e*	*ca*	19		0	0	0	40	22	14
+	+	+	+	*cu*	*sr*	*e*	*ca*	2		0			27		
+	+	+	*st*	*cu*	*sr*	*e*	*ca*	0							
+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	4		0	0	0	40	61	27
+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	7		0	0		36	6	
Others								182							
Total								345							

The recessive lethal alleles are examined by G_3 balancing with the *TM3* balancer and then G_4 crossing (Z30;Z30;rec./TM3 ♀ × Z30;Z30;rec./TM3 ♂)

Table B2-c G2 and G3 scoring of recombination mapping--ZS53-3

Recombinants								G2 Number	G3 scoring												
									Genotype	*ℓ*/rec.			*TM3,Ser* /rec.			*TM3,Ser* /*ruPrca*			*ℓ*/*ruPrca*		
									Replicate	1	2	3	1	2	3	1	2	3	1	2	3
ru	+	+	+	+	+	+	+	65		1	10	7	80	51	53	81	55	51	82	50	59
ru	*h*	+	+	+	+	+	+	64		7	7	3	48	39	21	38	71	27	41	63	24
ru	*h*	*th*	+	+	+	+	+	1													
ru	*h*	*th*	*st*	+	+	+	+	9		1	4	2	69	70	46	95	52	40	103	68	47
ru	*h*	*th*	*st*	*cu*	+	+	+	0													
ru	*h*	*th*	*st*	*cu*	*sr*	+	+	0													
ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	0													
+	+	+	+	+	+	+	*ca*	0													
+	+	+	+	+	+	*e*	*ca*	0													
+	+	+	+	+	*sr*	*e*	*ca*	0													
+	+	+	+	*cu*	*sr*	*e*	*ca*	10		27	38	55	22	48	41	24	46	24	23	58	38
+	+	+	*st*	*cu*	*sr*	*e*	*ca*	1		60			49			55			56		
+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	45		34	32	42	35	36	40	40	57	37	33	41	40
+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	49		59	60	58	33	50	31	39	46	32	49	62	56
Others								463													
Total								707													

Table B2-d G2 and G3 scoring of recombination mapping--ZH12-6

Recombinants								G2 Number	G3 scoring												
									Genotype	*ℓ*/rec.			*TM3,Ser* /rec.			*TM3,Ser* /*ruPrca*			*ℓ*/*ruPrca*		
									Replicate	1	2	3	1	2	3	1	2	3	1	2	3
ru	+	+	+	+	+	+	+	42		5	18	1	94	180	55	111	156	54	100	201	45
ru	*h*	+	+	+	+	+	+	45		0	1	0	2	74	1	2	79	0	2	84	2
ru	*h*	*th*	+	+	+	+	+	0													
ru	*h*	*th*	*st*	+	+	+	+	4		1	8		64	65		61	74		65	82	
ru	*h*	*th*	*st*	*cu*	+	+	+	19		1	2	10	27	29	21	27	47	27	33	49	39
ru	*h*	*th*	*st*	*cu*	*sr*	+	+	8		0	3	20	21	17	16	16	24	22	13	29	28
ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	61		59	139	27	46	84	19	42	76	18	35	127	22
+	+	+	+	+	+	+	*ca*	78		0	6	3	113	156	61	87	161	48	98	190	62
+	+	+	+	+	+	*e*	*ca*	19		82	64	20	132	144	67	117	123	52	141	142	48
+	+	+	+	+	*sr*	*e*	*ca*	22		72	143	53	84	130	47	86	109	33	102	122	54
+	+	+	+	*cu*	*sr*	*e*	*ca*	6		92	144	54	84	132	31	68	106	46	93	124	47
+	+	+	*st*	*cu*	*sr*	*e*	*ca*	0													
+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	32		16	30	58	14	28	33	21	28	49	21	26	42
+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	39		79	154	17	56	114	15	31	95	19	71	115	16
Others								573													
Total								948													

Table B2-e G2 and G3 scoring of recombination mapping--ZH18-6

Recombinants								G2 Number	G3 scoring												
									Genotype	*ℓ*/rec.			*TM3,Ser* /rec.			*TM3,Ser* /*ruPrca*			*ℓ*/*ruPrca*		
									Replicate	1	2	3	1	2	3	1	2	3	1	2	3
ru	+	+	+	+	+	+	+	186		9	0	2	66	36	49	62	38	47	69	40	54
ru	*h*	+	+	+	+	+	+	128		2	1	26	59	68	28	67	76	46	73	78	60
ru	*h*	*th*	+	+	+	+	+	0													
ru	*h*	*th*	*st*	+	+	+	+	9		45	11	30	43	41	30	72	60	70	84	61	94
ru	*h*	*th*	*st*	*cu*	+	+	+	0													
ru	*h*	*th*	*st*	*cu*	*sr*	+	+	0													
ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	0													
+	+	+	+	+	+	+	*ca*	0													
+	+	+	+	+	+	*e*	*ca*	0													
+	+	+	+	+	*sr*	*e*	*ca*	0													
+	+	+	+	*cu*	*sr*	*e*	*ca*	19		22			16			24			16		
+	+	+	*st*	*cu*	*sr*	*e*	*ca*	2		14			15			15			16		
+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	117		53	21	19	42	18	9	45	16	10	42	18	12
+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	153		8	13	27	4	14	19	1	12	16	4	20	28
Others								887													
Total								1501													

Table B2-f G2 and G3 scoring of recombination mapping--ZH21-1

Recombinants								G2 Number	G3 scoring												
									Genotype	*ℓ*/rec.			*TM3,Ser* /rec.			*TM3,Ser* /*ruPrca*			*ℓ*/*ruPrca*		
									Replicate	1	2	3	1	2	3	1	2	3	1	2	3
ru	+	+	+	+	+	+	+	136		0	0	0	12	46	44	12	43	42	18	34	44
ru	*h*	+	+	+	+	+	+	114		0	8	7	45	59	53	49	55	48	51	59	52
ru	*h*	*th*	+	+	+	+	+	1		8			42			47			56		
ru	*h*	*th*	*st*	+	+	+	+	16		3	1	3	27	32	51	32	45	42	37	50	61
ru	*h*	*th*	*st*	*cu*	+	+	+	0													
ru	*h*	*th*	*st*	*cu*	*sr*	+	+	0													
ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	0													
+	+	+	+	+	+	+	*ca*	1		0			20			23			23		
+	+	+	+	+	+	*e*	*ca*	0													
+	+	+	+	+	*sr*	*e*	*ca*	0													
+	+	+	+	*cu*	*sr*	*e*	*ca*	14		33	28	1	32	24	5	35	29	3	43	45	5
+	+	+	*st*	*cu*	*sr*	*e*	*ca*	2													
+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	89		17	8	8	23	14	10	17	15	8	22	17	9
+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	100		12	26	13	11	26	14	15	27	19	19	36	12
Others								673													
Total								1146													

Table B2-g G2 and G3 scoring of recombination mapping--ZH32-1

Recombinants								G2 Number	G3 scoring												
									Genotype	*ℓ*/rec.			*TM3,Ser* /rec.			*TM3,Ser* /*ruPrca*			*ℓ*/*ruPrca*		
									Replicate	1	2	3	1	2	3	1	2	3	1	2	3
ru	+	+	+	+	+	+	+	90		0	0	0	39	55	26	50	43	38	58	47	33
ru	*h*	+	+	+	+	+	+	46		0	0	0	44	53	104	37	55	87	38	53	87
ru	*h*	*th*	+	+	+	+	+	0													
ru	*h*	*th*	*st*	+	+	+	+	9		0	4	1	44	46	91	32	71	72	47	64	91
ru	*h*	*th*	*st*	*cu*	+	+	+	20		0	0	0	23	26	29	35	33	37	20	23	39
ru	*h*	*th*	*st*	*cu*	*sr*	+	+	5		0	0	0	40	41	74	55	56	100	55	55	88
ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	48				80			72			72			95
+	+	+	+	+	+	+	*ca*	122		2	0	13	40	52	85	66	42	85	58	51	96
+	+	+	+	+	+	*e*	*ca*	25		3	9	8	56	52	99	59	56	95	48	57	111
+	+	+	+	+	*sr*	*e*	*ca*	39		0	6	10	44	61	101	44	44	84	31	36	113
+	+	+	+	*cu*	*sr*	*e*	*ca*	7			45	20		54	13		40	16		44	20
+	+	+	*st*	*cu*	*sr*	*e*	*ca*	0													
+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	39		40	47	53	45	42	56	34	39	61	34	52	67
+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	44		17	13	60	14	9	81	15	4	58	19	9	66
Others								658													
Total								1152													

Table B2-h G2 and G3 scoring of recombination mapping--MW6-3

Recombinants								G2 Number	G3 scoring												
									Genotype	*ℓ*/rec.			*TM3,Ser* /rec.			*TM3,Ser* /*ruPrca*			*ℓ*/*ruPrca*		
									Replicate	1	2	3	1	2	3	1	2	3	1	2	3
ru	+	+	+	+	+	+	+	68		2	0	0	34	19	33	36	16	28	35	20	38
ru	*h*	+	+	+	+	+	+	78		2	2	0	42	44	37	33	32	42	42	35	38
ru	*h*	*th*	+	+	+	+	+	0													
ru	*h*	*th*	*st*	+	+	+	+	6		15	2	6	45	43	28	38	41	23	43	45	28
ru	*h*	*th*	*st*	*cu*	+	+	+	0													
ru	*h*	*th*	*st*	*cu*	*sr*	+	+	0													
ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	0													
+	+	+	+	+	+	+	*ca*	0													
+	+	+	+	+	+	*e*	*ca*	0													
+	+	+	+	+	*sr*	*e*	*ca*	0													
+	+	+	+	*cu*	*sr*	*e*	*ca*	12		35	25		39	26		44	26		50	35	
+	+	+	*st*	*cu*	*sr*	*e*	*ca*	0													
+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	49		34	23		30	26		28	32		42	38	
+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	63		37	32	6	29	33	7	34	37	11	31	31	5
Others								433													
Total								709													

Table B3-a F1 scroing sheet of deficiency mapping--ZH12-6

Deficiency lines		Genotype (Phenotype)				Total
	1	Df/ℓ (WT)	TM6C, cu^1 Sb^1/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu^1 Sb^1 (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb^1/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb^1 (Ser, Tb, e)	
Df(3R)ED6310	1	**65**	79	65	68	277
Df(3R)ED6232	1	**64**	66	57	61	248
Df(3R)ED6052	1	**125**	89	85	100	399
Df(3R)ED6027	1	**0**	78	75	81	234
Df(3R)ED5942	1	**66**	51	64	57	238
Df(3R)ED6096	1	**46**	46	48	59	199
Df(3R)BSC177	3	**57**	76	66	70	269
Df(3R)ED10845	1	**48**	55	56	44	203
Df(3R)ED6316	1	**79**	65	72	79	295
Df(3R)ED6220	1	**50**	32	37	33	152
Df(3R)ED6076	1	**70**	84	83	77	314
Df(3R)BSC517	2	**34**	73	0	41	148
Df(3R)BSC475	1	**0**	74	66	67	207
Df(3R)BSC636	1	**14**	52	46	69	181
Df(3R)BSC516	2	**71**	113	8	73	265

Red colour denotes that the deletion and the recessive lethal allele fail to complement.

Table B3-b F1 scroing sheet of deficiency mapping--ZH32-1

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3L)ED223	1	**65**	91	75	73	304
Df(3L)ED4685	1	**29**	31	29	20	109
Df(3L)ED224	1	**41**	39	35	39	154
Df(3L)ED225	1	**47**	62	62	51	222
Df(3L)ED4782	1	**147**	112	78	93	430
Df(3L)ED4978	1	**134**	121	117	106	478
Df(3L)ED230	1	**63**	50	41	58	212
Df(3L)ED5017	1	**110**	103	110	98	421
Df(3R)ED5147	1	**134**	103	115	98	450
Df(3R)ED10257	1	**80**	81	51	62	274
Df(3R)ED5177	1	**113**	145	52	118	428
Df(3R)ED5197	1	**114**	88	82	61	345
Df(3R)ED7665	1	**118**	114	85	116	433
Df(3R)ED5296	1	**27**	26	20	16	89
Df(3R)ED5331	1	**131**	113	93	87	424
Df(3R)ED5339	1	**188**	126	92	138	544
Df(3R)ED5474	1	**91**	86	65	65	307
Df(3R)ED5518	1	**20**	31	22	19	92
Df(3R)ED5610	1	**112**	122	77	97	408
Df(3R)ED5644	1	**146**	141	123	105	515
Df(3R)ED10639	1	**145**	100	39	74	358
Df(3R)BSC177	3	**70**	79	79	76	304
Df(3R)ED5942	1	**66**	76	62	78	282
Df(3R)ED6027	1	**131**	116	82	88	417
Df(3R)ED6052	1	**133**	134	82	106	455
Df(3R)ED6076	1	**100**	82	62	80	324
Df(3R)ED6096	1	**106**	97	91	93	387
Df(3R)ED6220	1	**100**	129	110	79	418
Df(3R)ED6232	1	**107**	115	114	98	434
Df(3R)ED6310	1	**128**	117	103	124	472
Df(3L)ED4710	2	**64**	69	75	56	264
Df(3L)ED229	1	**70**	71	56	73	270
Df(3L)ED4858	1	**73**	67	51	63	254
Df(3R)ED5100	1	**85**	114	50	100	349
Df(3R)ED5623	2	**96**	102	89	80	367
Df(3R)ED5705	2	**86**	93	72	91	342

Table B3-b (Continued)

Deficiency lines		Genotype (Phenotype)				Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)ED10642	1	**118**	125	93	116	452
Df(3R)ED5780	2	**60**	54	44	35	193
Df(3R)ED10845	1	**41**	70	48	59	218
Df(3R)ED6316	1	**133**	126	119	143	521
Df(3R)ED5429	2	**138**	52	138	42	370
Df(3R)ED5559	2	**7**	11	9	8	35
Df(3R)ED6093	2	**104**	78	54	66	302
Df(3R)ED6255	1	**97**	101	77	103	378
Df(3L)BSC797	1	**87**	80	70	70	307
Df(3L)BSC449	1	**64**	60	64	65	253
Df(3L)BSC419	1	**44**	26	26	36	132
Df(3R)ED5230	1	**174**	106	87	114	481
Df(3R)ED5495	1	**51**	50	22	40	163
Df(3R)ED5664	1	**88**	90	71	99	348
Df(3R)BSC750	1	**58**	79	59	61	257
Df(3R)BSC741	1	**33**	29	31	28	121
Df(3R)BSC790	1	**38**	49	33	61	181
Df(3R)BSC819	1	**83**	105	75	90	353
Df(3R)ED6058	1	**70**	87	67	78	302
Df(3R)BSC461	1	**52**	131	80	79	342
Df(3R)ED6265	1	**85**	96	61	81	323
Df(3R)BSC497	1	**42**	53	36	56	187
Df(3R)BSC567	1	**48**	49	52	55	204
Df(3R)BSC806	1	**74**	64	67	89	294
Df(3R)BSC501	1	**97**	107	75	103	382
Df(3L)Exel6136	3	**81**	90	84	55	310
Df(3R)Exel7318	3	**88**	123	87	71	369
Df(3R)Exel8157	3	**51**	95	69	45	260
Df(3R)Exel6167	3	**23**	90	79	57	249
Df(3R)Exel7327	3	**74**	118	3	62	257
Df(3R)Exel7328	3	**57**	77	8	59	201
Df(3R)Exel6178	3	**52**	64	42	34	192
Df(3R)BSC517	2	**54**	67	31	36	188
Df(3R)ED6187	1	**39**	77	28	38	182
Df(3R)Exel6272	3	**68**	54	47	44	213
Df(3R)Exel6194	3	**40**	33	52	44	169

Table B3-b (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)Exel6195	3	**75**	82	67	53	277
Df(3R)Exel6195	3	**45**	57	49	47	198
Df(3R)Exel9014	3	**0**	44	40	30	114
Df(3R)Exel6196	3	**0**	49	60	50	159
Df(3R)Exel6197	3	**39**	60	33	39	171
Df(3R)Exel6201	3	**95**	84	82	60	321
Df(3R)Exel9056	3	**90**	103	105	68	366
Df(3R)Exel6202	3	**67**	58	71	69	265
Df(3R)Exel6203	3	**78**	87	80	57	302
Df(3R)Exel6204	3	**67**	73	50	66	256
Df(3R)Exel621C	3	**22**	42	23	44	131
Df(3R)BSC56	2	**52**	56	50	54	212
Df(3R)BSC141	3	**50**	44	15	40	149
Df(3R)ED6103	1	**65**	75	58	83	281
Df(3R)BSC488	2	**111**	152	1	89	353
Df(3R)BSC633	1	**92**	102	77	99	370
Df(3R)BSC681	1	**150**	146	120	112	528
Df(3R)BSC745	1	**145**	124	124	140	533
Df(3R)BSC193	3	**150**	120	104	123	497
Df(3R)BSC738	1	**159**	136	96	119	510
Df(3R)BSC467	1	**94**	92	76	95	357
Df(3R)BSC422	1	**78**	72	57	66	273
Df(3R)BSC621	1	**57**	71	72	52	252
Df(3R)BSC744	1	**138**	136	107	128	509
Df(3R)ED6119	1	**49**	50	22	34	155
Df(3R)ED6144	1	**45**	84	70	81	280

Red colour denotes that the deletion and the recessive lethal allele fail to complement. Green colour denotes the deletion fails to complement the recessive lethal bearing by balancer, TM3.

Table B3-c F1 scoring sheet of deficiency mapping--ZS2-3

Deficiency lines		Genotype (Phenotype)				Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)ED5177	1	**195**	107	49	94	445
Df(3R)ED5197	1	**94**	61	61	56	272
Df(3R)ED7665	1	**69**	74	52	56	251
Df(3R)ED5296	1	**40**	39	26	40	145
Df(3R)ED5331	1	**56**	74	58	65	253
Df(3R)ED5339	1	**144**	96	30	99	369
Df(3R)ED5518	1	**37**	38	39	29	143
Df(3R)ED5610	1	**290**	238	88	168	784
Df(3R)ED5644	1	**150**	130	65	107	452
Df(3R)ED5942	1	**42**	27	26	44	139
Df(3R)ED6027	1	**101**	95	83	97	376
Df(3R)ED10845	1	**23**	12	18	24	77
Df(3R)ED6052	1	**101**	79	48	64	292
Df(3R)ED6076	1	**32**	29	25	33	119
Df(3R)ED6096	1	**69**	72	59	49	249
Df(3R)ED6220	1	**76**	61	46	62	245
Df(3R)ED6232	1	**87**	103	89	81	360
Df(3R)ED6310	1	**56**	37	37	56	186
Df(3R)ED6316	1	**117**	100	74	114	405
Df(3L)ED5017	1	**114**	98	84	81	377
Df(3R)ED5100	1	**41**	71	28	50	190
Df(3R)ED5147	1	**35**	57	43	51	186
Df(3R)ED10257	1	**103**	74	47	79	303
Df(3R)ED10639	1	**24**	22	2	16	64
Df(3R)BSC177	3	**41**	55	46	55	197
Df(3L)ED223	1	**25**	17	12	18	72
Df(3L)ED4685	1	**55**	49	62	47	213
Df(3L)ED224	1	**119**	121	44	96	380
Df(3L)ED225	1	**40**	20	47	61	168
Df(3L)ED4782	1	**73**	79	65	78	295
Df(3L)ED229	1	**36**	29	19	26	110
Df(3L)ED4978	1	**65**	72	67	58	262
Df(3L)ED230	1	**28**	39	35	32	134
Df(3R)ED5474	1	**78**	76	64	134	352
Df(3R)ED10642	1	**107**	65	50	92	314
Df(3L)ED4710	2	**49**	62	44	43	198

Table B3-c (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3L)ED4858	2	**71**	75	45	43	234
Df(3R)ED5623	2	**72**	74	52	53	251
Df(3R)ED5705	2	**127**	178	80	89	474
Df(3R)ED5780	2	**80**	77	48	66	271
Df(3R)ED5429	2	**244**	82	129	75	530
Df(3R)ED5559	2	**3**	7	3	5	18
Df(3R)ED6093	2	**110**	90	42	62	304
Df(3R)ED6255	1	**96**	76	79	76	327
Df(3L)BSC797	1	**94**	77	88	77	336
Df(3L)BSC449	1	**96**	115	79	85	375
Df(3L)BSC419	1	**50**	61	81	62	254
Df(3R)ED5230	1	**152**	96	31	83	362
Df(3R)ED5495	1	**34**	47	32	55	168
Df(3R)ED5664	1	**111**	87	37	78	313
Df(3R)BSC750	1	**68**	54	48	52	222
Df(3R)BSC741	1	**48**	58	38	57	201
Df(3R)BSC790	1	**28**	57	33	49	167
Df(3R)BSC819	1	**81**	100	63	84	328
Df(3R)ED6058	1	**121**	103	89	123	436
Df(3R)BSC461	1	**72**	90	84	80	326
Df(3R)ED6265	1	**65**	76	60	58	259
Df(3R)BSC497	1	**42**	29	27	29	127
Df(3R)BSC567	1	**38**	40	26	47	151
Df(3R)BSC806	1	**91**	89	58	64	302
Df(3R)BSC501	1	**130**	102	30	79	341
Df(3L)Exel6136	3	**81**	66	65	39	251
Df(3R)Exel7318	3	**81**	109	62	60	312
Df(3R)Exel8157	3	**78**	95	65	58	296
Df(3R)Exel6167	3	**72**	87	69	44	272
Df(3R)Exel7327	3	**53**	62	0	54	169
Df(3R)Exel7328	3	**84**	86	9	88	267
Df(3R)Exel6178	3	**67**	96	79	61	303
Df(3R)BSC517	3	**74**	65	37	33	209
Df(3R)ED6187	3	**1**	44	34	17	96
Df(3R)Exel6272	3	**57**	61	50	34	202
Df(3R)Exel6194	3	**55**	50	54	40	199

Table B3-c (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)Exel6195	3	**72**	77	49	65	263
Df(3R)Exel9013	3	**51**	54	43	52	200
Df(3R)Exel9014	3	**19**	39	20	21	99
Df(3R)Exel6196	3	**42**	55	56	53	206
Df(3R)Exel6197	3	**0**	56	65	66	187
Df(3R)Exel6201	3	**52**	57	53	33	195
Df(3R)Exel9056	3	**38**	48	33	34	153
Df(3R)Exel6202	3	**64**	45	37	53	199
Df(3R)Exel6203	3	**112**	104	112	72	400
Df(3R)Exel6204	3	**75**	59	49	42	225
Df(3R)Exel6210	3	**42**	56	19	31	148
Df(3R)BSC56	2	**61**	90	61	53	265
Df(3R)BSC141	3	**37**	38	7	26	108
Df(3R)ED6103	1	**80**	67	47	65	259
Df(3R)BSC488	2	104	111	3	66	284
Df(3R)BSC633	1	96	97	63	64	320
Df(3R)BSC681	1	102	95	95	102	394
Df(3R)BSC745	1	148	145	119	112	524
Df(3R)BSC193	3	123	75	75	73	346
Df(3R)BSC738	1	116	82	61	93	352
Df(3R)BSC467	1	77	72	43	48	240
Df(3R)BSC422	1	130	90	32	57	309
Df(3R)BSC621	1	86	60	53	57	256
Df(3R)BSC744	1	138	126	67	101	432
Df(3R)ED10893	1	**83**	65	59	57	264
Df(3R)ED6150	1	**133**	131	119	100	483
Df(3R)ED6155	1	**111**	115	94	101	421

Red colour denotes that the deletion and the recessive lethal allele fail to complement. Green colour denotes the deletion fails to complement the recessive lethal bearing by balancer, TM3.

Table B3-d F1 scoring sheet of deficiency mapping--ZS30-2

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3L)ED223	1	**57**	67	58	54	236
Df(3L)ED4685	1	**103**	103	72	98	376
Df(3L)ED4710	1	**79**	73	6	65	223
Df(3L)ED224	1	**134**	116	83	101	434
Df(3L)ED225	1	**173**	174	163	183	693
Df(3L)ED4782	1	**156**	132	100	123	511
Df(3L)ED229	1	**21**	21	16	21	79
Df(3L)ED4858	1	**45**	21	33	23	122
Df(3L)ED4978	1	**0**	56	37	60	153
Df(3L)ED230	1	**83**	66	78	75	302
Df(3L)ED5017	1	**48**	63	45	49	205
Df(3R)ED5100	1	**62**	68	42	63	235
Df(3R)ED5147	1	**66**	51	66	61	244
Df(3R)ED10257	1	**138**	69	34	74	315
Df(3R)ED5177	1	**69**	75	11	69	224
Df(3R)ED5197	1	**101**	91	97	77	366
Df(3R)ED7665	1	**2**	58	72	64	196
Df(3R)ED5296	1	**42**	34	0	36	112
Df(3R)ED5331	1	**95**	109	75	93	372
Df(3R)ED5339	1	**46**	56	39	54	195
Df(3R)ED5429	1	**61**	33	50	28	172
Df(3R)ED5474	1	**128**	63	91	82	364
Df(3R)ED5518	1	**44**	32	35	34	145
Df(3R)ED5610	1	**3**	71	54	65	193
Df(3R)ED5623	2	**107**	105	91	89	392
Df(3R)ED5644	1	**73**	78	63	66	280
Df(3R)ED5705	1	**88**	63	66	68	285
Df(3R)ED10639	1	**49**	21	4	29	103
Df(3R)ED10642	1	**154**	119	85	108	466
Df(3R)ED5780	1	**79**	54	61	53	247
Df(3R)BSC177	3	**66**	90	80	83	319
Df(3R)ED5942	1	**81**	77	63	76	297
Df(3R)ED6027	1	**126**	128	78	94	426
Df(3R)ED10845	1	**63**	57	61	50	231
Df(3R)ED6052	1	**151**	111	44	97	403
Df(3R)ED6076	1	**109**	57	64	55	285

Table B3-d (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1] Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1] Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)ED6093	1	**70**	62	46	39	217
Df(3R)ED6096	1	**59**	51	60	73	243
Df(3R)ED6220	1	**26**	42	31	36	135
Df(3R)ED6232	1	**82**	67	63	73	285
Df(3R)ED6255	1	**84**	81	72	77	314
Df(3R)ED6310	1	**105**	86	56	78	325
Df(3R)ED6316	1	**109**	104	86	93	392
Df(3L)ED202	1	**0**	73	44	44	161
Df(3L)ED4238	1	**0**	42	43	46	131
Df(3L)ED207	2	**0**	103	56	39	198
Df(3L)BSC419	1	**39**	43	40	29	151
Df(3R)ED5230	1	**169**	112	34	101	416
Df(3R)Exel7318	3	**33**	60	26	27	146
Df(3R)Exel8157	3	**66**	64	45	50	225
Df(3R)Exel6167	3	**61**	60	54	63	238
Df(3L)BSC450	1	**62**	77	56	69	264
Df(3L)BSC418	1	**0**	42	47	38	127
Df(3R)Exel6146	3	**32**	37	34	40	143
Df(3R)BSC747	1	**63**	68	52	48	231
Df(3R)Exel7317	3	**0**	26	34	15	75
Df(3L)Exel6086	3	**49**	65	58	44	216
Df(3L)BSC363	1	**56**	63	54	44	217
Df(3L)BSC799	1	**50**	38	50	49	187
Df(3L)BSC435	1	**0**	42	31	27	100
Df(3R)BSC221	3	**86**	98	89	85	358
Df(3R)ED5223	1	**50**	61	56	56	223
Df(3R)Exel6166	3	**41**	66	36	29	172
Df(3R)BSC513	1	**5**	93	66	72	236
Df(3R)BSC633	2	**110**	117	90	101	418
Df(3R)BSC196	3	**62**	69	30	49	210
Df(3R)BSC222	3	**71**	31	44	31	177
Df(3L)ED4177	1	**2**	19	13	17	51
Df(3R)ED5612	1	**60**	46	49	35	190
Df(3R)ED5577	1	**0**	29	30	28	87
Df(3R)ED5573	2	**0**	46	27	34	107
Df(3L)ED4177	1	**0**	45	48	37	130

Table B3-d (Continued)

	Genotype (Phenotype)					
Deficiency lines	1	Df/ℓ (WT)	TM6C, cu^1 Sb^1/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu^1 Sb^1 (Ser, Sb, e)	Total
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb^1/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb^1 (Ser, Tb, e)	

Red colour denotes that the deletion and the recessive lethal allele fail to complement. Green colour denotes the deletion fails to complement the recessive lethal bearing by balancer, TM3.

Table B3-e F1 scoring sheet of deficiency mapping--ZS53-3

Deficiency lines		Genotype (Phenotype)				Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3L)ED223	1	**101**	110	73	74	358
Df(3L)ED4685	1	**62**	47	25	39	173
Df(3L)ED224	1	**119**	69	9	78	275
Df(3L)ED225	1	**139**	121	54	95	409
Df(3L)ED4782	1	**181**	101	32	78	392
Df(3L)ED229	1	**32**	31	19	17	99
Df(3L)ED4858	2	**19**	10	4	14	47
Df(3L)Exel6136	3	**68**	55	28	44	195
Df(3L)BSC449	1	**45**	82	52	62	241
Df(3L)ED4978	1	**116**	90	49	92	347
Df(3L)BSC419	1	**52**	61	68	64	245
Df(3L)ED4710	2	**36**	32	35	16	119
Df(3L)BSC223	1	**74**	66	56	53	249
Df(3L)ED230	1	**72**	64	79	87	302
Df(3L)ED5017	1	**64**	41	58	59	222
Df(3R)ED5147	1	**45**	45	55	36	181
Df(3R)ED10257	1	**118**	64	41	47	270
Df(3R)ED5177	1	**90**	91	19	88	288
Df(3R)ED5197	1	**108**	81	67	71	327
Df(3R)ED7665	1	**31**	26	10	20	87
Df(3R)ED5230	1	**113**	100	19	59	291
Df(3R)ED5296	1	**41**	40	43	31	155
Df(3R)ED5331	1	**138**	134	116	113	501
Df(3R)ED5339	1	**183**	107	42	67	399
Df(3R)ED5429	2	**139**	35	129	36	339
Df(3R)ED5474	1	**108**	112	103	106	429
Df(3R)ED5514	1	**50**	63	44	69	226
Df(3R)ED5610	1	**179**	124	76	112	491
Df(3R)Exel7318	3	**138**	102	55	7	302
Df(3R)Exel8157	3	**42**	70	50	50	212
Df(3R)Exel6167	3	**39**	46	41	45	171
Df(3R)ED5623	2	**133**	117	120	96	466
Df(3R)Exel6275	3	**120**	87	60	63	330
Df(3R)ED5664	1	**25**	109	111	105	350
Df(3R)BSC750	1	**2**	135	109	91	337
Df(3R)BSC741	1	**54**	68	59	66	247

Table B3-e (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)ED5705	2	**44**	45	46	59	194
Df(3R)Exel7328	1	**79**	90	0	97	266
Df(3R)ED10639	1	**150**	108	22	99	379
Df(3R)ED10642	1	**132**	101	61	103	397
Df(3R)BSC177	3	**94**	92	86	78	350
Df(3R)ED5942	1	**48**	56	42	46	192
Df(3R)ED6027	1	**152**	99	78	120	449
Df(3R)BSC141	3	**24**	22	4	10	60
Df(3R)BSC819	1	**144**	121	57	84	406
Df(3R)ED6052	1	**135**	111	56	94	396
Df(3R)ED6058	1	**101**	118	84	125	428
Df(3R)ED6093	2	**60**	67	58	61	246
Df(3R)ED6103	1	**77**	79	65	76	297
Df(3R)BSC56	2	**87**	70	56	51	264
Df(3R)Exel6194	3	**121**	91	77	82	371
Df(3R)Exel6195	3	**121**	99	57	91	368
Df(3R)Exel6196	3	**71**	82	72	85	310
Df(3R)ED6187	2	**23**	28	36	33	120
Df(3R)BSC461	1	**75**	64	59	66	264
Df(3R)Exel6201	3	**33**	41	96	46	216
Df(3R)Exel9056	3	**112**	63	57	73	305
Df(3R)Exel6202	3	**41**	33	36	43	153
Df(3R)Exel6204	3	**43**	30	22	50	145
Df(3R)ED5780	2	**80**	120	78	80	358
Df(3R)Exel6272	3	**82**	68	47	57	254
Df(3R)ED6076	1	**102**	99	92	73	366
Df(3R)Exel9013	3	**83**	87	82	89	341
Df(3R)Exel9014	3	**9**	14	19	10	52
Df(3R)ED6220	1	**68**	143	79	82	372
Df(3R)Exel6203	3	**17**	19	17	15	68
Df(3R)ED6265	1	**88**	81	78	70	317
Df(3R)Exel6210	3	**113**	112	26	111	362
Df(3R)ED6310	1	**54**	52	40	63	209
Df(3R)ED6316	1	**78**	77	61	78	294
Df(3R)ED5100	1	**40**	35	39	41	155
Df(3R)BSC744	1	**60**	67	53	68	248

Table B3-e (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)BSC193	3	**80**	50	53	51	234
Df(3R)BSC422	1	**57**	67	40	27	191
Df(3R)BSC507	1	**96**	89	46	95	326
Df(3R)BSC476	1	**55**	63	38	55	211
Df(3R)BSC621	1	**115**	76	59	89	339
Df(3R)BSC806	1	**63**	96	23	75	257
Df(3R)BSC501	1	**130**	66	28	66	290
Df(3L)BSC797	1	**63**	148	74	105	390
Df(3R)BSC745	1	**159**	97	102	97	455
Df(3R)BSC746	1	**24**	13	19	13	69
Df(3R)BSC633	2	**68**	41	25	31	165
Df(3R)BSC738	1	**52**	51	33	41	177
Df(3R)ED6096	1	**52**	52	28	44	176
Df(3L)BSC249	1	**89**	74	72	86	321
Df(3R)BSC464	1	**63**	73	55	45	236
Df(3R)BSC681	1	**98**	105	100	86	389
Df(3R)BSC467	1	**69**	66	19	50	204
Df(3R)ED5644	1	**75**	69	55	46	245
Df(3R)Exel7327	3	**66**	72	0	61	199
Df(3R)ED10845	1	**43**	54	57	49	203
Df(3R)Exel6197	3	**91**	86	92	78	347
Df(3R)ED6232	1	**42**	52	50	53	197
Df(3R)BSC497	1	**27**	43	14	21	105
Df(3R)ED5495	1	**100**	88	75	98	361
Df(3R)ED6255	1	**93**	114	77	95	379
Df(3R)Exel6178	3	**77**	64	64	62	267
Df(3R)BSC567	1	**37**	54	26	49	166
Df(3L)Exel6137	3	**66**	79	60	36	241
Df(3R)BSC488	2	**65**	84	16	46	211
Df(3R)BSC790	1	**15**	29	12	16	72
Df(3R)ED10561	1	**41**	82	73	67	263
Df(3R)BSC471	1	**83**	84	68	68	303

Red colour denotes that the deletion and the recessive lethal allele fail to complement. Green colour denotes the deletion fails to complement the recessive lethal bearing by balancer, TM3.

Table B3-f F1 scoring sheet of deficiency mapping--ZH21-1

Deficiency lines		Genotype (Phenotype)				Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3L)ED223	1	**49**	51	50	38	188
Df(3L)ED4685	1	**145**	99	85	109	438
Df(3L)ED224	1	**169**	96	47	100	412
Df(3L)ED225	1	**132**	92	56	82	362
Df(3L)ED4782	1	**177**	155	108	95	535
Df(3L)ED229	1	**12**	87	34	57	190
Df(3L)ED4858	2	**45**	57	25	46	173
Df(3L)Exel6136	3	**63**	51	54	45	213
Df(3L)BSC449	1	**142**	145	102	75	464
Df(3L)ED4978	1	**102**	100	86	89	377
Df(3L)BSC419	1	**57**	50	46	47	200
Df(3L)ED4710	2	**39**	32	15	24	110
Df(3L)BSC223	1	**25**	30	9	22	86
Df(3L)ED230	1	**60**	69	46	54	229
Df(3L)ED5017	1	**51**	50	42	55	198
Df(3R)ED5147	1	**0**	19	19	12	50
Df(3R)ED10257	1	**19**	12	4	9	44
Df(3R)ED5177	1	**75**	37	10	41	163
Df(3R)ED5197	1	**25**	15	4	22	66
Df(3R)ED7665	1	**58**	62	48	58	226
Df(3R)ED5230	1	**62**	46	17	40	165
Df(3R)ED5296	1	**14**	23	21	16	74
Df(3R)ED5331	1	**40**	24	11	25	100
Df(3R)ED5339	1	**46**	51	15	19	131
Df(3R)ED5429	2	**106**	44	34	32	216
Df(3R)ED5474	1	**54**	41	26	18	139
Df(3R)ED5514	1	**10**	16	8	14	48
Df(3R)ED5610	1	**67**	76	39	46	228
Df(3R)Exel7318	3	**76**	45	22	36	179
Df(3R)Exel8157	3	**62**	72	64	42	240
Df(3R)Exel6167	3	**51**	39	42	39	171
Df(3R)ED5623	2	**105**	96	57	74	332
Df(3R)Exel6275	3	**65**	57	36	35	193
Df(3R)ED5664	1	**190**	98	18	115	421
Df(3R)BSC750	1	**111**	81	71	88	351
Df(3R)BSC741	1	**68**	72	47	89	276

Table B3-f (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)ED5705	2	**42**	65	30	50	187
Df(3R)Exel7328	1	**75**	75	1	74	225
Df(3R)ED10639	1	**238**	121	14	98	471
Df(3R)ED10642	1	**71**	46	18	45	180
Df(3R)BSC177	3	**51**	49	50	54	204
Df(3R)ED5942	1	**68**	57	67	48	240
Df(3R)ED6027	1	**94**	76	51	64	285
Df(3R)BSC141	3	**43**	42	3	36	124
Df(3R)BSC819	1	**87**	54	37	74	252
Df(3R)ED6052	1	**83**	72	14	59	228
Df(3R)ED6058	1	**103**	80	34	50	267
Df(3R)ED6093	2	**124**	93	40	80	337
Df(3R)ED6103	1	**208**	132	63	108	511
Df(3R)BSC56	2	**144**	140	64	78	426
Df(3R)Exel6194	3	**220**	90	19	76	405
Df(3R)Exel6195	3	**164**	90	7	70	331
Df(3R)Exel6196	3	**101**	67	83	68	319
Df(3R)ED6187	2	**83**	60	39	36	218
Df(3R)BSC461	1	**78**	127	93	68	366
Df(3R)Exel6201	3	**112**	93	27	81	313
Df(3R)Exel9056	3	**136**	100	46	64	346
Df(3R)Exel6202	3	**141**	82	48	77	348
Df(3R)Exel6204	3	**50**	79	45	50	224
Df(3R)ED5780	2	**161**	96	59	90	406
Df(3R)Exel6272	3	**89**	66	44	49	248
Df(3R)ED6076	1	**94**	77	20	63	254
Df(3R)Exel9013	3	**95**	117	78	84	374
Df(3R)Exel9014	3	**39**	78	48	48	213
Df(3R)ED6220	1	**71**	52	35	38	196
Df(3R)Exel6203	3	**71**	55	30	34	190
Df(3R)ED6265	1	**94**	94	46	96	330
Df(3R)Exel6210	3	**32**	33	5	22	92
Df(3R)ED6310	1	**173**	89	30	89	381
Df(3R)ED6316	1	**138**	92	68	100	398
Df(3R)ED5100	1	**86**	67	19	47	219
Df(3R)BSC744	1	**79**	60	46	63	248

Table B3-f (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)BSC193	3	**113**	93	14	58	278
Df(3R)BSC422	1	**74**	52	22	44	192
Df(3R)BSC507	1	**83**	49	15	40	187
Df(3R)BSC476	1	**104**	67	27	60	258
Df(3R)BSC621	1	**84**	96	60	75	315
Df(3R)BSC806	1	**33**	29	9	15	86
Df(3R)BSC501	1	**95**	63	15	48	221
Df(3L)BSC797	1	**91**	101	79	86	357
Df(3R)BSC745	1	**146**	115	45	73	379
Df(3R)BSC746	1	**100**	84	44	63	291
Df(3R)BSC633	2	**181**	137	29	79	426
Df(3R)BSC738	1	**95**	71	5	49	220
Df(3R)ED6096	1	**148**	119	71	91	429
Df(3L)BSC249	1	**39**	33	22	34	128
Df(3R)BSC464	1	**43**	50	2	39	134
Df(3R)BSC681	1	**91**	122	79	93	385
Df(3R)BSC467	1	**32**	35	2	20	89
Df(3R)ED5644	1	**61**	65	45	64	235
Df(3R)Exel7327	3	**68**	70	1	46	185
Df(3R)ED10845	1	**67**	80	66	63	276
Df(3R)Exel6197	3	**88**	94	79	59	320
Df(3R)ED6232	1	**93**	83	47	72	295
Df(3R)BSC497	1	**69**	83	38	57	247
Df(3R)ED5495	1	**68**	93	64	70	295
Df(3R)ED6255	1	**84**	99	74	88	345
Df(3R)Exel6178	3	**46**	49	36	35	166
Df(3R)BSC567	1	**34**	28	19	37	118
Df(3L)Exel6137	3	**82**	93	66	53	294
Df(3R)BSC488	2	**96**	96	4	54	250
Df(3R)BSC790	1	**61**	68	29	58	216
Df(3R)BSC139	3	**74**	76	38	50	238
Df(3R)BSC176	3	**1**	81	85	78	245
Df(3R)ED5138	1	**6**	95	47	55	203
Df(3R)ED5142	1	**7**	109	58	91	265
Df(3L)ED4799	1	**56**	67	59	56	238
Df(3L)ED4789	1	**49**	46	44	39	178

Table B3-f (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3L)Exel6135	3	**77**	72	34	51	234
Df(3L)Exel9009	3	**58**	54	34	30	176
Df(3L)BSC445	1	**63**	46	7	41	157
Df(3L)Exel9046	3	**84**	56	61	57	258

Red colour denotes that the deletion and the recessive lethal allele fail to complement. Green colour denotes the deletion fails to complement the recessive lethal bearing by balancer, TM3.

Table B3-g F1 scoring sheet of deficiency mapping--ZH18-6

Deficiency lines		Genotype (Phenotype)				Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3L)ED223	1	**83**	62	66	57	268
Df(3L)ED4685	1	**114**	95	96	86	391
Df(3L)ED224	1	**109**	77	28	49	263
Df(3L)ED225	1	**108**	84	82	100	374
Df(3L)ED4782	1	**118**	121	91	98	428
Df(3L)ED229	1	**55**	61	44	60	220
Df(3L)ED4858	2	**69**	78	58	59	264
Df(3L)Exel6136	3	**90**	58	89	66	303
Df(3L)BSC449	1	**103**	81	62	61	307
Df(3L)ED4978	1	**79**	77	95	76	327
Df(3L)BSC419	1	**77**	76	63	68	284
Df(3L)ED4710	2	**55**	61	44	43	203
Df(3L)BSC223	1	**45**	55	33	51	184
Df(3L)ED230	1	**68**	72	63	57	260
Df(3L)ED5017	1	**24**	34	38	30	126
Df(3R)ED5147	1	**73**	95	63	81	312
Df(3R)ED10257	1	**101**	56	31	47	235
Df(3R)ED5177	1	**56**	34	18	33	141
Df(3R)ED5197	1	**6**	5	7	3	21
Df(3R)ED7665	1	**42**	37	25	23	127
Df(3R)ED5230	1	**208**	104	25	102	439
Df(3R)ED5296	1	**47**	71	0	44	162
Df(3R)ED5331	1	**93**	86	48	71	298
Df(3R)ED5339	1	**40**	36	19	20	115
Df(3R)ED5429	2	**43**	40	36	34	153
Df(3R)ED5474	1	**79**	68	51	62	260
Df(3R)ED5514	1	**73**	83	62	73	291
Df(3R)ED5610	1	**102**	88	87	79	356
Df(3R)Exel7318	3	**72**	89	19	49	229
Df(3R)Exel8157	3	**80**	72	70	75	297
Df(3R)Exel6167	3	**56**	58	49	53	216
Df(3R)ED5623	2	**73**	55	68	57	253
Df(3R)Exel6275	3	**118**	96	70	70	354
Df(3R)ED5664	1	**163**	86	46	126	421
Df(3R)BSC750	1	**74**	102	44	87	307
Df(3R)BSC741	1	**38**	53	36	42	169

Table B3-g (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)ED5705	2	**22**	33	30	32	117
Df(3R)Exel7328	1	**106**	93	3	74	276
Df(3R)ED10639	1	**75**	48	4	34	161
Df(3R)ED10642	1	**114**	75	59	69	317
Df(3R)BSC177	3	**142**	94	72	92	400
Df(3R)ED5942	1	**60**	52	56	57	225
Df(3R)ED6027	1	**116**	111	88	102	417
Df(3R)BSC141	3	**35**	38	9	31	113
Df(3R)BSC819	1	**64**	53	46	48	211
Df(3R)ED6052	1	**110**	69	48	94	321
Df(3R)ED6058	1	**67**	73	62	57	259
Df(3R)ED6093	2	**52**	46	40	23	161
Df(3R)ED6103	1	**78**	71	62	71	282
Df(3R)BSC56	2	**79**	46	31	59	215
Df(3R)Exel6194	3	**144**	68	83	91	386
Df(3R)Exel6195	3	**80**	70	44	55	249
Df(3R)Exel6196	3	**50**	62	54	53	219
Df(3R)ED6187	2	**55**	60	0	34	149
Df(3R)BSC461	1	**53**	57	55	64	229
Df(3R)Exel6201	3	**19**	20	15	8	62
Df(3R)Exel9056	3	**97**	78	47	53	275
Df(3R)Exel6202	3	**78**	58	52	60	248
Df(3R)Exel6204	3	**40**	34	26	36	136
Df(3R)ED5780	2	**64**	71	56	58	249
Df(3R)Exel6272	3	**68**	56	51	38	213
Df(3R)ED6076	1	**82**	49	72	52	255
Df(3R)Exel9013	3	**85**	96	63	95	339
Df(3R)Exel9014	3	**23**	58	35	39	155
Df(3R)ED6220	1	**5**	52	41	40	138
Df(3R)Exel6203	3	**153**	103	90	91	437
Df(3R)ED6265	1	**92**	78	71	108	349
Df(3R)Exel6210	3	**40**	79	13	47	179
Df(3R)ED6310	1	**150**	94	46	85	375
Df(3R)ED6316	1	**72**	78	54	62	266
Df(3R)ED5100	1	**36**	39	14	43	132
Df(3R)BSC744	1	**102**	112	96	83	393

Table B3-g (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)BSC193	3	**73**	68	39	41	221
Df(3R)BSC422	1	**92**	72	34	59	257
Df(3R)BSC507	1	**92**	61	52	58	263
Df(3R)BSC476	1	**1**	58	28	37	124
Df(3R)BSC621	1	**91**	115	72	89	367
Df(3R)BSC806	1	**41**	46	16	28	131
Df(3R)BSC501	1	**39**	40	18	23	120
Df(3L)BSC797	1	**123**	121	108	118	470
Df(3R)BSC745	1	**82**	79	70	78	309
Df(3R)BSC746	1	**56**	60	52	60	228
Df(3R)BSC633	2	**40**	31	21	20	112
Df(3R)BSC738	1	**144**	99	63	67	373
Df(3R)ED6096	1	**81**	75	70	69	295
Df(3L)BSC249	1	**62**	77	49	50	238
Df(3R)BSC464	1	74	78	59	57	268
Df(3R)BSC681	1	64	70	61	41	236
Df(3R)BSC467	1	58	62	7	54	181
Df(3R)ED5644	1	71	66	46	53	236
Df(3R)Exel7327	3	45	45	1	53	144
Df(3R)ED10845	1	56	62	72	64	254
Df(3R)Exel6197	3	87	83	66	37	273
Df(3R)ED6232	1	55	45	33	40	173
Df(3R)BSC497	1	109	116	82	104	411
Df(3R)ED5495	1	72	84	51	54	261
Df(3R)ED6255	1	75	71	61	48	255
Df(3R)Exel6178	3	61	66	53	48	228
Df(3R)BSC567	1	29	35	12	40	116
Df(3L)Exel6137	3	61	72	61	36	230
Df(3R)BSC488	2	82	89	20	44	235
Df(3R)BSC790	1	31	22	23	30	106
Df(3R)BSC519	1	**50**	45	50	41	186
Df(3R)Exel6200	3	**0**	80	89	77	246
Df(3R)BSC520	1	**0**	57	62	64	183
Df(3R)Exel7357	3	**96**	108	81	107	392
Df(3R)BSC655	1	**105**	92	88	93	378
Df(3R)BSC318	1	**67**	54	56	54	231

Table B3-g (Continued)

Deficiency lines		Genotype (Phenotype)				Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)Exel6153	3	**0**	36	35	31	102
Df(3R)BSC397	1	**82**	67	54	78	281

Red colour denotes that the deletion and the recessive lethal allele fail to complement. Green colour denotes the deletion fails to complement the recessive lethal bearing by balancer, TM3.

Table B3-h F1 scroing sheet of deficiency mapping--MW6-3

Deficiency lines		Genotype (Phenotype)				Total
	1	Df/ℓ (WT)	TM6C, cu^1 Sb^1/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu^1 Sb^1 (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb^1/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb^1 (Ser, Tb, e)	
Df(3L)ED223	1	**65**	73	65	64	267
Df(3L)ED4685	1	**1**	51	66	65	183
Df(3L)ED224	1	**84**	46	17	52	199
Df(3L)ED225	1	**103**	69	57	55	284
Df(3L)ED4782	1	**100**	70	63	92	325
Df(3L)ED229	1	**45**	47	56	51	199
Df(3L)ED4858	2	**34**	12	9	11	66
Df(3L)Exel6136	3	**12**	13	9	10	44
Df(3L)BSC449	1	**77**	89	56	89	311
Df(3L)ED4978	1	**77**	105	96	83	361
Df(3L)BSC419	1	**40**	33	35	29	137
Df(3L)ED4710	2	**24**	21	20	29	94
Df(3L)BSC223	1	**66**	61	57	57	241
Df(3L)ED230	1	**106**	98	100	86	390
Df(3L)ED5017	1	**75**	76	56	67	274
Df(3R)ED5147	1	**23**	28	28	22	101
Df(3R)ED10257	1	**137**	97	49	87	370
Df(3R)ED5177	1	**75**	74	56	80	285
Df(3R)ED5197	1	**80**	55	88	83	306
Df(3R)ED7665	1	**83**	79	82	83	327
Df(3R)ED5230	1	**68**	79	49	64	260
Df(3R)ED5296	1	**28**	32	22	25	107
Df(3R)ED5331	1	**65**	70	58	64	257
Df(3R)ED5339	1	**48**	37	30	42	157
Df(3R)ED5429	2	**58**	39	49	46	192
Df(3R)ED5474	1	**13**	21	24	10	68
Df(3R)ED5514	1	**53**	46	30	37	166
Df(3R)ED5610	1	**55**	57	58	70	240
Df(3R)Exel7318	3	**96**	85	58	53	292
Df(3R)Exel8157	3	**80**	112	106	89	387
Df(3R)Exel6167	3	**41**	48	41	35	165
Df(3R)ED5623	2	**84**	59	75	57	275
Df(3R)Exel6275	3	**81**	77	79	82	319
Df(3R)ED5664	1	**145**	110	95	134	484
Df(3R)BSC750	1	**120**	98	82	125	425
Df(3R)BSC741	1	**56**	63	52	53	224

Table B3-h (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)ED5705	2	**44**	56	36	43	179
Df(3R)Exel7328	1	**82**	77	11	50	220
Df(3R)ED10639	1	**135**	115	78	84	412
Df(3R)ED10642	1	**31**	35	38	37	141
Df(3R)BSC177	3	**88**	79	70	75	312
Df(3R)ED5942	1	**107**	115	36	107	365
Df(3R)ED6027	1	**72**	82	83	78	315
Df(3R)BSC141	3	**22**	16	8	18	64
Df(3R)BSC819	1	**31**	31	35	32	129
Df(3R)ED6052	1	**71**	72	63	52	258
Df(3R)ED6058	1	**41**	42	42	41	166
Df(3R)ED6093	2	**61**	48	48	43	200
Df(3R)ED6103	1	**94**	103	64	75	336
Df(3R)BSC56	2	**62**	86	56	52	256
Df(3R)Exel6194	3	**232**	135	72	112	551
Df(3R)Exel6195	3	**106**	81	53	66	306
Df(3R)Exel6196	3	**103**	98	66	61	328
Df(3R)ED6187	2	**36**	47	38	44	165
Df(3R)BSC461	1	**62**	122	57	73	314
Df(3R)Exel6201	3	**54**	48	44	46	192
Df(3R)Exel9056	3	**133**	132	114	85	464
Df(3R)Exel6202	3	**76**	46	56	70	248
Df(3R)Exel6204	3	**41**	35	39	46	161
Df(3R)ED5780	2	**93**	71	84	59	307
Df(3R)Exel6272	3	**44**	46	37	35	162
Df(3R)ED6076	1	**43**	45	44	28	160
Df(3R)Exel9013	3	**34**	32	24	37	127
Df(3R)Exel9014	3	**27**	20	20	10	77
Df(3R)ED6220	1	**82**	81	74	74	311
Df(3R)Exel6203	3	**84**	83	54	61	282
Df(3R)ED6265	1	**106**	109	95	90	400
Df(3R)Exel6210	3	**91**	85	34	77	287
Df(3R)ED6310	1	**57**	73	54	75	259
Df(3R)ED6316	1	**72**	69	63	77	281
Df(3R)ED5100	1	**0**	57	34	48	139
Df(3R)BSC744	1	**65**	61	69	72	267

Table B3-h (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)BSC193	3	**60**	59	56	54	229
Df(3R)BSC422	1	**99**	109	76	67	351
Df(3R)BSC507	1	**52**	50	46	42	190
Df(3R)BSC476	1	**91**	105	89	88	373
Df(3R)BSC621	1	**88**	81	54	83	306
Df(3R)BSC806	1	**76**	57	58	54	245
Df(3R)BSC501	1	**56**	70	47	55	228
Df(3L)BSC797	1	**50**	41	115	44	250
Df(3R)BSC745	1	**67**	83	83	70	303
Df(3R)BSC746	1	**25**	22	23	16	86
Df(3R)BSC633	2	**77**	83	62	58	280
Df(3R)BSC738	1	**96**	49	48	57	250
Df(3R)ED6096	1	**77**	89	69	82	317
Df(3L)BSC249	1	**75**	101	66	77	319
Df(3R)BSC464	1	**42**	34	36	34	146
Df(3R)BSC681	1	**102**	145	99	94	440
Df(3R)BSC467	1	**63**	54	22	45	184
Df(3R)ED5644	1	**111**	122	41	82	356
Df(3R)Exel7327	3	**50**	50	6	48	154
Df(3R)ED10845	1	**43**	49	31	29	152
Df(3R)Exel6197	3	**72**	84	68	32	256
Df(3R)ED6232	1	**67**	56	72	72	267
Df(3R)BSC497	1	**82**	99	89	106	376
Df(3R)ED5495	1	**74**	86	68	72	300
Df(3R)ED6255	1	**101**	92	104	107	404
Df(3R)Exel6178	3	**55**	61	61	60	237
Df(3R)BSC567	1	**24**	24	15	27	90
Df(3L)Exel6137	3	**100**	110	95	45	350
Df(3R)BSC488	2	**71**	83	42	69	265
Df(3R)BSC790	1	**64**	59	38	61	222
Df(3L)Exel7253	3	**60**	80	78	63	281
Df(3L)BSC414	1	**0**	105	93	109	307
Df(3L)Exel6131	1	**72**	77	57	60	266
Df(3L)BSC415	1	**73**	76	77	87	313
Df(3R)BSC421	1	**41**	52	38	44	175
Df(3R)BSC316	3	**0**	68	63	53	184

Table B3-h (Continued)

Deficiency lines	Genotype (Phenotype)					Total
	1	Df/ℓ (WT)	TM6C, cu[1], Sb[1]/ℓ (Sb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6C, cu[1], Sb[1] (Ser, Sb, e)	
	2	Df/ℓ (WT)	TM2/ℓ (Ubx)	TM3, Ser/Df (Ser)	TM3, Ser/TM2 (Ser, Ubx, e)	
	3	Df/ℓ (WT)	TM6B,Tb[1]/ℓ (Tb)	TM3, Ser/Df (Ser)	TM3, Ser/TM6B, Tb[1] (Ser, Tb, e)	
Df(3R)BSC174	3	**53**	56	45	48	202
Df(3R)Exel6141	3	**52**	59	49	37	197
Df(3R)Exel6143	3	**63**	61	52	72	248
Df(3L)BSC432	1	**143**	120	121	107	491
Df(3R)ED5020	1	**57**	80	65	47	249
Df(3R)ED5066	1	**63**	86	78	71	298
Df(3R)BSC146	3	**65**	102	73	73	313
Df(3R)BSC246	3	**0**	54	46	42	142

Red colour denotes that the deletion and the recessive lethal allele fail to complement.

Table B4 G_2 scoring of recombinant types

Crossover events	**G_2 Recombinants**								**Number**
Single crossover	*ru*	+	+	+	+	+	+	+	63
	ru	*h*	+	+	+	+	+	+	47
	ru	*h*	*th*	+	+	+	+	+	0
	ru	*h*	*th*	*st*	+	+	+	+	9
	ru	*h*	*th*	*st*	*cu*	+	+	+	0
	ru	*h*	*th*	*st*	*cu*	*sr*	+	+	0
	ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	0
	+	+	+	+	+	+	+	*ca*	0
	+	+	+	+	+	+	*e*	*ca*	0
	+	+	+	+	+	*sr*	*e*	*ca*	0
	+	+	+	+	*cu*	*sr*	*e*	*ca*	12
	+	+	+	*st*	*cu*	*sr*	*e*	*ca*	0
	+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	55
	+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	49
Double crossover	*ru*	+	+	+	*cu*	*sr*	*e*	*ca*	7
	+	*h*	*th*	*st*	+	+	+	+	6
	+	*h*	+	+	+	+	+	+	5
	+	+	*th*	*st*	+	+	+	+	2
	ru	*h*	+	+	*cu*	*sr*	*e*	*ca*	6
	ru	*h*	+	*st*	*cu*	*sr*	*e*	*ca*	1
	ru	*h*	*th*	*st*	+	+	+	*ca*	1
Other types									205
Total offspring									468

Grey light in table represents only one double crossover occurs around *In(3R)K*.

Table B5 The comparison of recombination rate between inversion heterozygotes and straight homozygotes.

Recombinants								G2 Number	
								ST/*rucuca*	*In(3R)K*/*rucuca*
ru	+	+	+	+	+	+	+	42	63
ru	*h*	+	+	+	+	+	+	20	47
ru	*h*	*th*	+	+	+	+	+	0	0
ru	*h*	*th*	*st*	+	+	+	+	0	9
ru	*h*	*th*	*st*	*cu*	+	+	+	3	0
ru	*h*	*th*	*st*	*cu*	*sr*	+	+	1	0
ru	*h*	*th*	*st*	*cu*	*sr*	*e*	+	5	0
+	+	+	+	+	+	+	*ca*	51	0
+	+	+	+	+	+	*e*	*ca*	9	0
+	+	+	+	+	*sr*	*e*	*ca*	19	0
+	+	+	+	*cu*	*sr*	*e*	*ca*	2	12
+	+	+	*st*	*cu*	*sr*	*e*	*ca*	0	0
+	+	*th*	*st*	*cu*	*sr*	*e*	*ca*	4	55
+	*h*	*th*	*st*	*cu*	*sr*	*e*	*ca*	7	49
ru	+	+	+	+	+	+	*ca*	21	0
ru	+	+	+	+	+	*e*	*ca*	7	0
ru	+	+	+	+	*sr*	*e*	*ca*	4	0
ru	+	+	+	*cu*	*sr*	*e*	*ca*	1	7
ru	*h*	+	+	+	+	+	*ca*	12	0
ru	*h*	+	+	+	+	*e*	*ca*	1	0
ru	*h*	+	+	+	*sr*	*e*	*ca*	3	0
ru	*h*	+	+	*cu*	*sr*	*e*	*ca*	0	6
ru	*h*	+	*st*	*cu*	*sr*	*e*	*ca*	0	1
ru	*h*	*th*	*st*	+	+	+	*ca*	0	1
ru	*h*	*th*	*st*	*cu*	+	*e*	*ca*	2	0
+	*h*	*th*	*st*	*cu*	*sr*	*e*	+	2	0
+	*h*	*th*	*st*	*cu*	*sr*	+	+	1	0
+	*h*	*th*	*st*	+	+	+	+	1	6
+	*h*	+	+	+	+	+	+	3	5
+	+	*th*	*st*	*cu*	*sr*	*e*	+	2	0
+	+	*th*	*st*	*cu*	*sr*	+	+	2	0
+	+	*th*	*st*	*cu*	+	+	+	1	0
+	+	*th*	*st*	+	+	+	+	0	2
+	+	+	+	+	*sr*	*e*	+	1	0
+	+	+	+	+	+	*e*	+	4	0
Total								345	468

Table B6 Recessive lethal alleles bearing by the *TM3* balancer.

African lines	Recessive lethal mutations on the *TM3* balancer (cytological positions)						
	89A12;89B1	92F13;93A1	85A5;85C3	84A1;84A5	95E1;96A7	83D2;83E1	74E2;75B1
ZS30-2	●	●	●	NA			●
ZS2-3	●	●		NA			
ZH32-1	●	●		NA			
ZH12-6	●	●		NA			
ZS53-3	●	●					
ZH18-6	●	●	●	●	●		
ZH21-1	●	●		●		●	
MW6-3	●	●		●			

●: Recessive lethal mutation on balancer. NA: not applicable

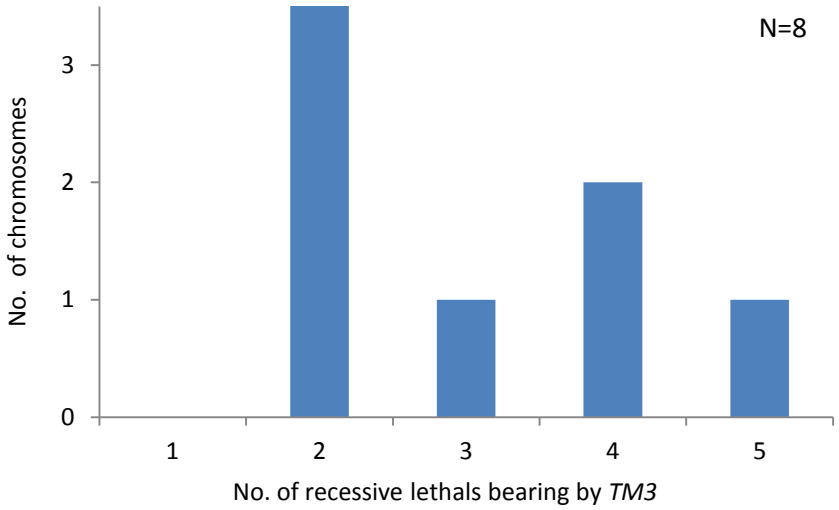

Figure B2 The accumulation pattern of recessive lethal lethals bearing by balancer, *TM3*.