mainly collected in human habitation. On the other hand, *D. daruma* was found only in the wild forest of Iriomotejima. This species is usually rare and collected in riversides of southern Japan. *D. lacteicornis* and *D. formosana* were collected only in wild forests of Ishigakigjima and Iriomotejima although they were not dominant in the islands.

The collection of *D. bocki* in Ishigakijima, Iriomotejima, Haterumajima and Yonagunijima is a new record of the distribution in Japan. *D. bocki* is a closely related species to *D. kikkawai* which is a domestic and world wide species. The first collection of Japanese *D. bocki* was in Iriomotejima in 1979 by the one of the authors (M.W.). Since morphological classification of the species was actually difficult at that time, it was identified as *D. bocki* by mating experiments, two-dimensional electrophoresis and allozyme electrophoresis (Ohnishi *et al.*, 1983). The present study shows that *D. bocki* dwells in the four Sakishima islands and prefers the wild environment rather than human habitation. *D. bocki* may not be a recent colonizer in Japan. A difficult identification might have missed the species as in the case in Taiwan (Baimai, 1979; Baimai *et al.*, 1980).

D. simulans was a colonizing species in mainlands of Japan (Honsyu, Kyusyu, Shikoku and Hokkaido), and had never been found in Okinawa and Sakishima islands (Watanabe and Kawanishi, 1978). Many *D. simulans* were once collected in Miyakojima in 1982. However, this species seems to be suffering from settlement there. Only 8 flies are *D. simulans* in a total of 986 *Drosophila* flies in spring of 1999. New colonization of *D. simulans* was found in Nago (Okinawa island) by sweeping. But the species was not collected in Naha. A further and precise survey is needed for the study of colonization of *D. simulans* in Okinawa and Sakishima islands.

Table 1. Number of *Drosophila* collected in human habitation of Okinawa (Naha and Nago) and Miyakojima in 1982 and 1983.

	Nana (H)	Nago (P)	Miyakojima (H)	
	82/10	82/10	82/10	83/8
bryani	0	6	18	79
dorsocentralis	1	3	31	10
takahashii	119	6	69	19
melanogaster	1	341	7	5
simulans	0	0	137	5
ficusphila	3	0	22	11
ananassae	2	168	2	21
bipectinata	3	0	508	199
kikkawai	23	32	6	8
triauraria	2	0	0	0
albomicans	7	6	7	0
Total	161	562	807	356

(H) and (P) show collection sites of human habitation and pineapple yard, respectively.

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Why is *mama* not *adipose*?

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I reported earlier (Doane, 1996) that *fs(2)lto5DF6*, a recessive mutation in the gene *female sterile (2) late oogenesis5*, does not complement adp^{fs}, a female sterility mutation previously believed to be an allele of *adipose* (*adp*), the first obesity gene of *Drosophila melanogaster* to be described. The former was isolated in a second chromosome saturation screen for EMS-induced female-steriles (Schupbach and Wieschaus, 1989, 1991; T. Schupbach *in* Lindsley and Zimm, 1992, page 237). The latter was described nearly 40 years ago as a spontaneous mutation derived from a natural population in Kaduna, Nigeria (Doane, 1960a, b).[1] It became apparent that *fs(2)lto5DF6* and adp^{fs} are recessive alleles of the same gene based on genetic location, failure to complement one another, and similarities in their female sterility phenotypes (Doane, 1996). I have named this gene *maternal metaphase arrest* (genetic symbol, *mama*). Its name reflects the maternal effect lethality of eggs laid by females homozygous for either of these female sterility mutations and the meiotic or mitotic arrest

associated with the first of them to be described. Late stages of oogenesis are affected by both mutations (see Lindsley and Zimm, 1992).

The *mama* gene has three known alleles to which I have assigned the following genetic symbols: $mama^{+}$ for its wild-type allele; $mama^{1}$ for the spontaneous mutation formerly called adp^{fs} (synonym, *fs(2)adp*); and $mama^{lto5}$ for the EMS-induced allele previously known as *fs(2)lto5DF6*. These symbols conform to the guidelines of *FlyBase* (M. Ashburner, personal communication) and will be used throughout the remainder of this report. The primary question addressed here is: Are *mama* and *adp* the same or different genes? The facts that $mama^{lto5}$ mutants do not express an obese phenotype and that the obese adp^{60} mutants are fertile suggest that they are discrete (Doane, 1996). If true, *adipose* has only two known alleles, adp^{+} and adp^{60}, and it can be assumed that adp^{60} was linked to $mama^{1}$ when the latter was first isolated from the Kaduna population. Evidence for this already exists (Doane, 1961).

The relationship between *mama* and *adp* is clarified in this report by a series of relatively large-scale, classical recombination experiments. The goals were to determine (1) whether or not *mama* and *adp* are separate genes, (2) what the genetic distance between them might be if they are different genes, and (3) what their gene order might be relative to closely linked markers. First, a pilot experiment was conducted to test the hypothesis that *adp* and *mama* are separate genes. Heterozygous females of the genotype $cn^{1}\ adp^{+}\ mama^{lto5}\ bw^{1}/cn^{+}\ adp^{60}\ mama^{+}\ bw^{+}$ were mated to males homozygous for $cn^{1}\ adp^{60}\ mama^{1}\ bw^{1}$. The progeny from this test cross numbered 1,235 flies of both sexes. These were scored for eye color, but only females displaying brown eyes (115) were scored for *mama* and *adp* mutant phenotypes to avoid the tedium of aging and testing all flies individually for these traits. The results were consistent with crossing-over having occurred between *adp* and *mama* at an estimated frequency of ~0.73%, but this was based on only two *adp-mama* recombinants and double crossover events could not be scored.

The second experiment was a 3-point recombination analysis that did score for double crossover progeny. Here $adp^{60}\ mama^{1}\ bw^{1}/adp^{+}\ mama^{+}\ bw^{+}$ females were crossed to homozygous $adp^{60}\ mama^{1}\ bw^{1}$ males, and the female test cross progeny were counted (N = 1955) and classified for eye color and fertility. To correct for flies that might express sterility unrelated to *mama*, only fertile females were tested for *adp* phenotypes. These numbered 1,031 and included: 10 single crossovers between *adp* and *mama*; 196 singles crossovers between *mama* and *bw*; and one double crossover between *cn* and *bw* (Interference, 53.8%). The results confirmed that *adp* and *mama* are separate genes and established the gene order as *adp - mama - bw*, with *adp* centromere-proximal to *mama*. Also, a better estimate of the map distance between *adp* and *mama* was calculated, namely ~1.07 cM.

Table 1. Recombination analyses performed to map *adp* relative to *stau* and *Pcl* and to determine the map distances between them.

Test Cross	Test Cross Progeny: Phenotypic Classes	Number	% Crossing-over
A: $c^{+}\ stau^{ry9}\ adp^{+}$ / cl $stau^{+}\ adp^{60}$ female	Parental : wild-type	1,781	—
x	Single Crossovers: c-stau	99	5.23
$c^{1}\ stau^{+}\ adp^{60}$ / $c^{1}\ stau^{+}\ adp^{60}$ male	Single Crossovers: stau-adp	12	0.63
	Total number scored:	1,892	
B: $c^{+}\ Pcl^{Pl}\ adp^{+}$ / cl $Pcl^{+}\ adp^{60}$ female	Parental: wild-type	1,418	—
x	Single Crossovers: c-Pcl	101	6.61
$c^{1}\ Pcl^{+}\ adp^{60}$ / $c^{1}\ Pcl^{+}\ adp^{60}$ male	Single Crossovers: Pcl-adp	10	0.65
	Total number scored:	1,529	

Two additional 3-point analyses were performed (Table 1, test crosses A and B). All gene loci involved were located within a span of 10 cM, thus eliminating double crossover classes and simplifying genetic calculations. The *adp* gene had been located on the genetic map at 2-83.4, *i.e.*, 7.9 map units distal to *curved* (*c*, 2-75.5) (see Appendix *in* Doane, 1969). Because there is a paucity of genetically well defined, visible markers in this region of the genome (*FlyBase*, 1999), stable *P* element-induced *staufen* (*stau*, 2-83.5) and *Polycomblike* (*Pcl*, 2-84) mutations were used as markers. The mutant alleles employed were $stau^{ry9}$ and Pcl^{Pl}. These are associated with the transposon *P{ry11}* (St. Johnston *et al.*, 1991; Lonie *et al.*, 1994) which carries a wild-type *rosy* gene (ry^{+}). Test crosses were set up so that all flies were homozygous for $ry^{506}\ e^{1}$.

This permitted heterozygous progeny carrying a single copy of *stau*^{ry9} or *Pcl*^{P1} to be detected by their wild-type eye color. Flies without these mutations had rosy-colored eyes.

Only one-half of the progeny from each test cross in Table 1 were counted since no double crossing-over was anticipated. Reciprocal classes were discarded. This greatly simplified the tasks of aging the flies one week and classifying their fat body phenotypes. In test cross A, only flies with wild-type wings were counted because curved-wing flies tended to become stuck in the food medium and die during the required 1-week aging period. For test cross B, special care was taken to prevent this from happening so both wild-type and curved-wing flies were scored. To half the number of progeny scored, only males were scored because the *adp* phenotype can be determined with better accuracy in males than females.

The molecular studies of Lonie *et al.* (1994) indicate that *stau* and *Pcl* are separated by about 8 kb of DNA and that *stau* is located proximal to *Pcl*, which is consistent with the genetic map positions given above. Taking this and the results of both test crosses in Table 1 into account, it now appears that the *adp* gene lies about 0.6 cM distal to the *stau-Pcl* region on the genetic map and that the combined gene order is *c - stau - Pcl - adp*. Despite the limitations of gene mapping based on recombination data, the percentages of crossing-over between *stau* and *adp* and between *Pcl* and *adp* listed in Table 1 (0.63 % and 0.65%, respectively) are remarkably similar. The crossing-over distances between *c* and *adp* derived from test crosses A and B were calculated to be 5.86 and 7.26 map units, respectively. Both of these distances are less than the 7.9 cM reported earlier (Doane, 1969), but only by ~0.6 cM in the latter case.

The recessive *mama*^{1} and *adp*^{60} phenotypes are both expressed in deficiency heterozygotes carrying *Df(2R)PC4, Df(2R)Pcl-W5, Df(2R)Pcl7B, Df(2R)Pcl11B* and *Df(2R)Pcl*^{XM82}, but not *Df(2R)PC29, Df(2R)P34* or *Df(2R)PC66* (Doane and Dumapias, 1987; Doane, 1994, 1996 and unpublished data). They also fail to complement a deletion that apparently exists at the left breakpoint in the inversion chromosome *In(2R)Pcl*^{11} (Doane, 1994). This deletion also fails to complement *stau* and *Pcl*, but it does complement *thr* (Doane, 1994 and unpublished data), which is in region 55A1 of the polytene chromosome map (*FlyBase*, 1999). This implies that the left breakpoint of the deletion is between 55A1 and 55A4, wherein *stau* is located (*FlyBase*, 1999). Clearly, the deletion in the *In(2R)Pcl*^{11} chromosome is much larger than originally supposed since it spans the region containing *stau* and *mama*, which are ~1.6 cM apart on the genetic map. The latter suggests that *mama* may be cytogenetically located in 55B and not 55A, wherein *adp* is still likely to be found. Furthermore, earlier speculation that *PpY-55A* and *adp* might be the same gene (Foehr and Doane, 1994) is negated by the fact that *PpY-55A* is in 55A1-3, which is proximal to *stau*, while *adp* is distal to *stau* and *Pcl*.

I have knowingly omitted reference to the report by Tearle (1996) that is archived in *FlyBase* in its original, unedited draft form. The EMS-induced lethal and female-sterile mutations isolated by Tearle should eventually prove very useful for analyzing the part of the genome spanned by *Df(2R)PC4*, which includes the *mama* and *adp* genes. However, attempts to interpret this report with regard to these two genes proved fruitless at this time.

Conclusions: The following conclusions may be drawn from the data presented here: (1) *mama* and *adp* are different genes with a genetic distance of ~1 cM between them; (2) *adp* is located ~0.6 cM distal to the *stau-Pcl* region on the genetic map; (3) the gene order for the loci included in this study is *c - stau - Pcl - adp - mama*; (4) *adp* is probably located cytogenetically toward the distal end of 55A, while *mama* is likely in regions 55B; and (5) *PpY-55* and *adp* are clearly different genes.

Acknowledgments: The source of the *mama*^{hto5} and *stau*^{ry9} mutations was the Bloomington, Indiana Stock Center. *Pcl*^{P1} was provided by the laboratory of R. Saint, University of Adelaide, Australia. Other mutations used in test crosses, including *adp*^{60} and *mama*^{1}, have been maintained in my laboratory for many years. Two different stocks containing *In(2R)Pcl*^{11} were tested with the same results; one came from the Bloomington Stock Center and the other was provided by Daniel Moore while at the Whitehead Institute for Biomedical Research, Cambridge, MA.

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[1]*Errata*: S. J. Counce gave me the adp^{fs} mutation (syn. *fs(2)adp*) in 1956. My original name for it was *female-sterile(2)adipose* but the gene locus was renamed *adipose* after my isolation of the fertile adp^{60} mutation (Doane, 1963). Counce has been credited with discovery of adp^{fs} (Lindsley and Grell, 1968; Lindsley and Zimm, 1992), but she recently pointed out that C. Auerbach had isolated it from the Kaduna wild population maintained at the University of Edinburgh (S. J. Counce, personal communication). To further complicate its history, my first description of *fs(2)adp* as a new mutant appeared in *Dros. Inf. Serv.* as the "Report of S. Counce" (see Counce, 1960). This error was corrected a year later when the same description appeared in a report under my own name (Doane, 1961). An editorial note accompanying it stated: "This report supersedes that in *Dros. Inf. Serv.*-34 inadvertently attributed to S. Counce." Unfortunately, *FlyBase* (1999) has perpetuated the prior error in authorship by including reference FBrf0094540 in its bibliography without comment or correction.

Drosophila hormone receptor 38: phenotypic analysis of mutations generated by P-element excision.

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DHR38 is a member of the steroid receptor superfamily in *Drosophila* sharing homology with vertebrate NGF1-B-type orphan receptors. As a monomer, DHR38 interacts with the USP component of the ecdysone receptor complex *in vitro* in yeast and in *Drosophila* cell line, suggesting that DHR38 might modulate ecdysone triggered signals.

Fine functional and structural analyses of this gene were carried out as described in Sutherland *et al.* (1995), Fisk and Thummel (1995), and Kozlova *et al.* (1998). The gene spans more than 40kb in length, has a complex genomic organization and produces multiple mRNA species developmentally regulated. Four mutations in this gene are known: one P(ry+, lacZ) insertion (named *l(2)02306*) and three EMS induced. All mutations result in local fragility of the adult cuticle: the cuticle in the leg joints is ruptured by mechanical stress, this leads to melanization of the damaged spots. Subsequently flies die as pharate adult or within a few hours after eclosion. The phenotypic abnormalities and effective lethal phase suggest an important role of DHR38 in late stage of epidermal metamorphosis.

It is known that the *Dhr38* gene expresses during most *Drosophila* developmental stages, suggesting that this gene may perform a critical function during another stage (more early). To test this hypothesis we induced the new mutations by imprecise removal of the P element.

To generate mutations in the *Dhr38* gene P(ry+, lacZ) transpozon related with *l(2)02306* mutation localized 34 bp upstream of cTK61 isoform (Figure 1, from Kozlova *et al.*, 1998) was mobilized according to standard genetic scheme, using Δ2,3 (ry+) on the third chromosome as a source of transposase. ry-excisions were checked for viability in combinations with Df(2)DS9, Df(2)KetelRX32 or *l(2)02306*. From more than 10,000 analyzed flies we obtained 106 ry-excisions and 18 of them were lethal during pharate adult/adult stages when hemizygous with deletions. The lethal phenotypes exhibited by new mutations in hemizygous condition are similar to the ones described earlier.

Determination of the lethal phases for 14 selected lines shows that most of them are adult lethals like the original P insertion (Table 1), but some are derivatives with more severe phenotypes- larval, prepupal or pupal lethals. v9 and v27 homozygotes show delayed pupariation up to four days. It is interesting to note that