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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

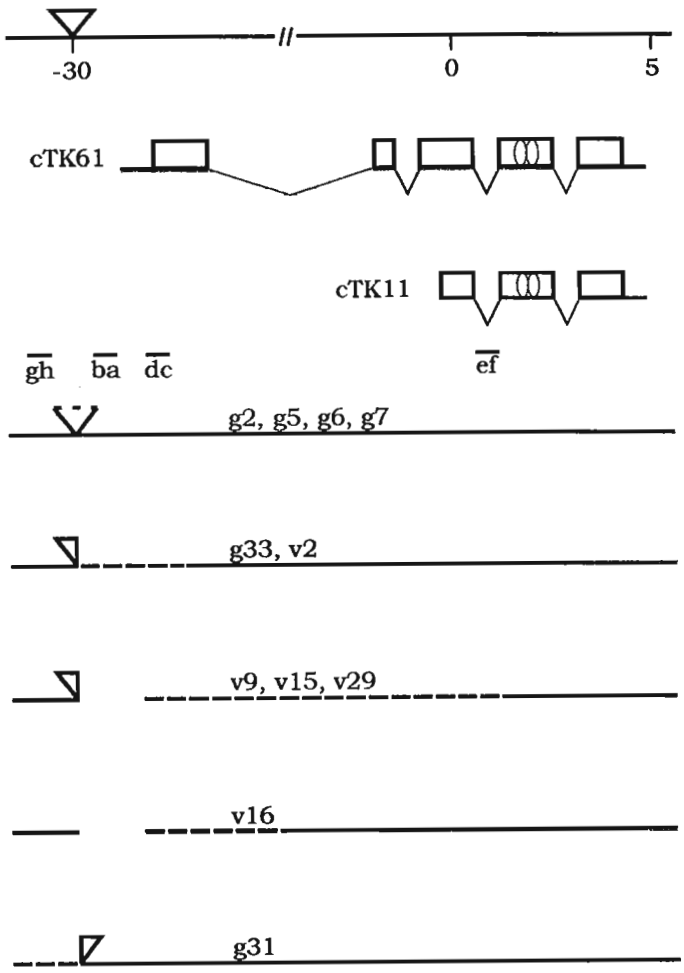

Figure 1. Molecular mapping of the new excision alleles. Molecular map of the *Dhr38* gene is adapted from Kozlova *et al.* (1998) and positions of exons and introns are indicated. Protein coding regions are represented by open rectangles, ovals show the position of Zn fingers. Untranslated sequences are shown by solid lines. Solid triangle represents insertion site of *l(2)02306* P-element insertion line. Fragments amplified by the primers used for molecular mapping of the rearrangements are shown below molecular map (gh, ba, dc, ef). Genomic sequences still present in new rearrangements are shown by solid lines, dashed areas represent an uncertainty of mapping. Part of the P-element construct retained in rearrangements (if any) is shown by an open triangle.

many new mutations such as g6, v15, v29, g33 behave as earlier lethals when homozygous rather than in combination with deficiency. For example, 64% of hemizygous Df(2)/g6 flies survive until the adult stage and die displaying melanization in the leg joints. However the majority of g6 homozygous flies (91%) die at prepupal and early pupal stages with only 1% surviving until adulthood. A few excision mutations such as v16 behave as hypomorphic, showing 86% survival to adulthood when homozygous and lethality throughout larval and pupal stages with only 8% of adults eclosing when hemizygous.

PCR analysis of selected lethal ry- chromosomes was performed on DNA templates prepared from heterozygous and homozygous flies using several sets of primers based on genomic and cDNA sequences (Figure 1) as well as a primer corresponding to the terminal repeats of the P element. In most of the excisions part of the P-element construct is still present (v9, v15, v29, g33, v2, g31). In others, both P element ends and adjacent genomic sequences are intact (g2, g5, g6, g7), suggesting that ry- phenotype is caused simultaneously by internal rearrangement in the P(ry+, lacZ), and possibly by inversion or duplication, frequently generated in P mutagenesis (Zhang and Spradling, 1993; Dorer and Henikoff, 1994).

We identified four excisions (v9, v15, v29, v16) removing the first exon of the *Dhr38* gene, containing 5 UTR sequences and part of the A/B domain present in cTK61 isoform (Figure 1). None of these eliminated the DNA-binding and ligand-binding sequences of *Dhr38*. Since the distance between the insertion site of P element and 5' end of the cTK61 is only 34 bp, all the excisions generated probably affect only this cDNA isoform and therefore are not complete loss-of-function alleles of *Dhr38*. Our lethal phase analysis and molecular mapping suggest that excision alleles retaining part of the P element construct (in contrast to the original P element insertion) show dominant phenotype manifested in earlier larval and pupal lethality, but

Table 1. Viability of mutants at different developmental stages.

Genotype	Larvae %	Pupae %	Pharate adults %	Eclosed adults %	Lph
P/CyO, y+	100	98	96	96	—
P/P	100	96	94	89	A
v2/v2	100	95	60	34	P, A
v2Df	100	74	70	64	A
v8/v8	100	97	78	62	A
v9/v9	100	62	0	0	L, P
v15/Df	100	64	62	59	L,A
v15/v15	100	97	0	0	P
v16/v16	100	86	86	86	A
v16/Df	100	44	44	8	L, P, A
v27/v27	100	60	0	0	L, P
v29/v29	100	26	0	0	L, P
v29/DF	100	60	53	53	L, A
g2/g2	100	90	86	86	A
g5/g5	100	95	92	92	A
g6/g6	100	92	1	1	P
g6/Df	100	66	66	64	L, A
g7/g7	100	94	94	94	A
g31/g31	100	95	33	0	P
g31/Df	100	50	50	46	L, A
g33/g33	100	96	34	0	P
g33/Df	100	68	68	64	L,A
g36/g36	100	92	0	0	P

The respective genotypes of animals homozygous or hemizygous in combination with the deficiency Df(2)KetelRX32 or Df(3L) DS9 are shown in the left column; P represents the insertion allele l(2)02306, and P/CyO, y+ is the control. Homozygous or hemizygous mutant first instar larvae, pupal cases, pharate adults (stage P15, Bainbridge and Bownes, 1981) and eclosed adults were scored. All eclosed adults with the exception of controls in P/CyO, y+ died within a few hours after eclosion displaying melanization in appendage joints. Lethal Phase (LPh) of corresponding genotypes is summarized in the right column: L-larval, P-pupal and pharate adults, A-eclosed adults.

also represent loss-of-function alleles at later pharate adult/adult stage when *Dhr38* function is required (Kozlova *et al.*, 1998). We believe that these ry- derivatives may be considered as gain-of-function alleles at larval and pupal stages due to the activity of the remaining P-element promoter sequences which probably cause ectopic *Dhr38* transcription. This suggestion is in agreement with earlier observations that *Dhr38* is transcribed at a very low level at larval stages and overexpression of *Dhr38* under hsp70 promoter at this stage causes ectopic lethality (Kozlova *et al.*, 1998; Kozlova, T.Yu., unpublished).

In conclusion, we described here several new excision alleles of *Dhr38*, some of which represent true hypomorphs (v16), and others (g6, v15, v29) posses both gain-of–function and loss-of-function characteristics.

Acknowledgments: The work was supported in part by grant of the Program "Frontier Program of Genetics" and RFBR grants 96-15-97749 and 99-04-49270.

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