polytene chromosomes. Based on its overall phenotype, tentative location, and a weak interaction between it and the nw^2 allele in regard to wing shape, it was named $nw^{PZ[ry+]}$ *FlyBase* (1997) lists it as nw^{PZry+}.

Despite its proposed classification as a *nw* allele, uncertainty remained about the exact chromosomal location of the *PZ* element that had induced the so-called nw^{PZry+} mutation. This was due to a complex chromosomal aberration in the region of its insertion that made the insertion site difficult to interpret. In addition, the weak narrow-like wing phenotype of presumptive nw^{PZry+} / nw^2 flies often overlapped wild-type, raising doubts about the presence of the nw^2 allele in our test stocks. These stocks, which produce few homozygotes at either 18° or 25°C, had been obtained from the Bloomington, Mid-America, and Umeå Drosophila Stock Centers. In our hands, none of them yield homozygotes with a wing phenotype like that originally described for nw^2; rather, their wings appear wild-type. (Unfortunately, our laboratory had lost the nw^2 strain used by Doane and Clark [1984], which did produce homozygotes with the wing phenotype characteristic of this mutant.)

The only other gene on chromosome *2R* with a mutant phenotype similar to our *PZ*-induced mutation is *tapered* (*ta*; 2-56.6) which, prior to this report, had a single mutant allele called ta^1 (Lindsley and Zimm, 1992; *FlyBase*, 1997). Although this locus lies about 23 cM centromere-proximal to *nw*, the description of the ta^1 phenotype matched that of "nw^{PZry+}" almost perfectly. We therefore obtained several ta^1 stocks from the Mid-America Stock Center and tested the chromosome carrying ta^1 from each one over our "nw^{PZry+}"-bearing chromosome for potential allelic interaction. Contrary to expectation, the heterozygotes expressed the typical ta^1 mutant phenotype, suggesting the two mutations are alleles.

We now have conclusive evidence that "nw^{PZry+}" is actually a recessive mutant allele of the *tapered* gene (Doane, Bien-Willner and Scheel, in preparation) and, therefore, have renamed it ta^2. Supporting evidence includes: (1) a third mutant allele, ta^3, which was induced by excision of the *PZ* element from ta^2, behaves as a recessive lethal that is able to uncover the *tapered* mutant phenotype when tested in *trans* over either ta^1 or ta^2, (2) data from two different 3-point crossover analyses place the above three *ta* mutant alleles at the same locus on the genetic map, and the site of this locus is consistent with the published site of the *ta* gene, (3) three different deficiencies in the second chromosome, namely *Df(2R)B5*, *Df(2R)X3*, and *Df(2R)eve* (Bloomington Stock Center), uncover the mutant phenotypes of the above *ta* alleles, while four others, *Df(2R)cn76k3*, *Df(2R)cn88f34a*, *Df(2R)spleN3* (Michael Ashburner's laboratory, University of Cambridge) and *Df(2R)M41A4* (Mid-America Stock Center) do not. Thus, in addition to identifying two new *ta* mutations, we have defined the cytogenetic location of the *ta* gene, which lies within region 46C3-C4; 46C9-C11 of the polytene chromosome map for *2R*. Furthermore, we have collected preliminary data indicative of genetic interactions between the dominant nw^D allele of the *narrow* gene and the ta^1 and ta^2 alleles of *tapered*, suggesting that these two linked genes may be part of the same developmental pathway(s).

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Report of Mary Roberts and F. Rob Jackson. Department of Neuroscience, Tufts University School of Medicine, Boston, MA 02111.

A new *miniature-dusky* allele.

We report the isolation of a new spontaneous allele of the *miniature-dusky* (*m-dy*) gene complex. The new mutation was induced in a single male which carried an X chromosome marked with an existing *dy* allele (dy^{And}). It was identified among the progeny of a genetic cross designed to mobilize a *dy* transgene; (*y w* dy^{And}/*y w* dy^{And}; +/+; +/+ ♀ × +/Y; *P*{*dy12*}/+; *Sb P*{Δ2-3}/+ ♂); however, the mutant allele did not arise in a dysgenic individual. Genetic analysis indicated that the new mutation: (1) was recessive, (2) mapped to the X chromosome, and (3) failed to complement both *m* and *dy* alleles. We refer to this new *m-dy* allele as m^{MR}. The m^{MR} mutation causes reduced wing size similar to other *m-dy* alleles, but the reduction in wing surface area is more extreme than that observed in any other *m-dy* mutant with the exception of m^D, a dominant allele of the *m-dy* complex. Indeed, the phenotype associated with m^{MR} is similar to that previously described for *m dy* double mutants, consistent with the presence of the dy^{And} allele on the parental

chromosome. Southern blot analysis using DNA probes spanning the *m-dy* complex indicates that the m^{MR} mutation is a 19-25kb chromosomal deletion in the *m-dy* interval. Our unpublished molecular analysis of this region indicates the existence of separable *m* and *dy* transcription units, and we postulate that m^{MR} removes part or all of both transcription units.

Mutation Notes - Other Species

Report of E. Solé. Dept. Genètica, Facultat de Biologia, Universitat de Barcelona, Spain.
Spontaneous *yellow* mutation in the *ch cu* strain of *Drosophila subobscura.*

Two *yellow* male flies spontaneously arose in a homokaryotypic stock of *D. subobscura* kept in the laboratory for a long time. This stock bears the recessive mutations ch (*cherry*, bright red eyes) and *cu* (*curled*, wings curled concave upwards), both located on chromosome O. Another *yellow* male fly arose after some generations in a cross between a wild male and five *ch cu* females. Only the right half of this mutant individual was *yellow*; his half left was wild type. It was fertile and no mutant flies appeared either in the F1 or F2 of a cross with *ch cu* females.

The *yellow* mutation is recessive, located in the A (sexual) chromosome and has been previously described in *D. subobscura* (Krimbas, 1993; Mestres, 1996).

References: Krimbas, C.B., 1993, Drosophila subobscura: *Biology, Genetics, and Inversion Polymorphism.* Verlag Dr. Kovac, Hamburg; Mestres, F., 1996, Dros. Inf. Serv. 77: 148.

Report of Albert Kamping and Wilke van Delden. Department of Genetics, University of Groningen, P.O. Box 14, 9750 AA Haren, The Netherlands.
A rare *α-Gpdh* allele in *Drosophila simulans.*

In contrast to many other allozyme loci, *α-Gpdh* is remarkably invariable in *Drosophila.* The *α-Gpdh* locus is monomorphic for electrophoretic variation in almost all *Drosophila* species. Some species show alleles at very low frequencies and in only two out of almost 200 species that have been analyzed, the *α-Gpdh* locus is classified as polymorphic (*D. melanogaster* and *D. subarctica*). The low level of variation is ascribed to the important functions in energy metabolism of the enzyme. New mutants at this locus are assumed to be deleterious, and only under conditions without biochemical or physiological constraints new mutants may be maintained. Allele substitutions have taken place in the evolution of *α-Gpdh* in *Drosophila*, because different species carry different alleles. Alleles with identical electrophoretic mobility are restricted to certain species or species groups. The distribution and uniformity in alleles within and between species cannot be explained without the action of natural selection, where metabolic function of the enzyme and ecological niches of the species are assumed to be main factors in the evolutionary process of *α-Gpdh.*

Table 1. Changes of the rare $\alpha Gpdh^{S}$ allele frequency in laboratory *D. simulans* populations, started with different initial frequencies at 20°C and 29.5°C and raised under uncrowded conditions.

Temperature	Initial frequency	Generations 1	5	10	15
20°C	.25	.24	.25	.16	.14
	.50	.53	.52	.37	.31
	.75	.74	.69	.59	.59
29.5°C	.25	.28	.29	.21	.19
	.50	.51	.49	.41	.36
	.75	.72	.69	.65	.68

D. melanogaster is one of the exceptions concerning the level of variation at the *α-Gpdh* locus. Almost every wild population of *D. melanogaster* is polymorphic for two common alleles, Slow (*S*) and Fast (*F*). The sibling species *D. simulans* is monomorphic and carries an allele with identical electrophoretic mobility as the *D. melanogaster F*-allele. In consecutive years we observed an additional *α-Gpdh* variant in a wild population of *D. simulans* in The Netherlands. Electrophoretic mobility of this allele is comparable with the *S* allele of *D. melanogaster*, and its frequency reaches the level of polymorphism. Four out of 21 captured *D. simulans* females produced progeny (no hybrids) carrying the *S* allele in a frequency not significantly different from .25. We derived homozygous *S* and *F* strains, and laboratory populations with different