qed^{1}, *qed*^{S140V} – probably ommatoreductum

Both lines were saved from the mutagenesis done in Florence and McGinnis (1998). They are EMS induced alleles originally on a y.YL chromosome. The first allele qed^{1} has been "cleaned-up" by transferring it first to a wildtype chromosome and then to a y cv v f chromosome. The second allele, qed^{S140V}, is still on the original mutagenized chromosome. My mutation maps to 5A6-9 based on its meiotic recombination map position of 12.5 mu and the following deficiency complementation.

Complements: Df(1)HC244, Df(1)bi-DL1, Df(1)RC40, Df(1)bi-DL2, Df(1)JC70, Df(1)C149, Df(1)N73, Df(1)5D, Df(1)JF5, Df(1)dx81, Df(1)B56^{D}J102^{P}, Df(1)J102^{D}B119^{P}

The only gap is between Df(1)J102^{D}B119^{P} (proximal breakpoint: 5A6-9) and Df(1)C149 (5A8-9) suggesting qed is in 5A8-9. I tried imprecisely hopping out and locally hopping EP(X)496 but could not get a hit in qed.

In many ways, the qed head phenotype is similar to both the Dfd hypomorphic and the Dfd ectopic expression phenotypes seen in adults. Maxillary palps are reduced (as in Dfd hypomorphs) and the ventral eye is reduced (as in Dfd^{D}). When qed is combined with Dfd^{D} the vibrissae are transformed to maxillary palps.

qed shows a recessive genetic interaction with Dfd^{3}/Dfd^{13} (see screen in Florence and McGinnis, 1998). NB: This interaction is NOT dominant as were the other loci reported in Florence and McGinnis. nej^{Q7} shows a dominant enhancement of the qed recessive phenotype.

txl: *taxi-like*

These alleles were obtained by mobilizing EP(X)1303 and EP(X)1033 (only one allele from the latter). By meiotic recombination, I have placed txl proximal to mys.

Complements: Df(1)RA2, Df(1)GE202, Df(1)HA11, mys, fsh, sn

Fails to complement: Df(1)C128, Df(1)Desi^{S3}, Df(1)fh1516

Some alleles are pupal lethal, some are viable. Wings are outstretched (like taxi), cuticle is dark (like ptg). Since Haynes et al. mapped the end of Df(1)C128 within ~15kb of the proximal end of mys, there is a very small region where txl could be.

Oce^{WC1}: *Ocellarless*

Fails to complement Oce^{1}, but is completely wildtype in the heterozygous state. Can be separated from and is proximal to the Dfd-interacting lethal on the WC1 chromosome: l(1)WC1. The l(1)WC1 chromosome still has the Oce^{WC1} allele on it.

l(1)7Ad

Assumed to be l(1)7Ad by failure to complement Df(1)Sxl^{RA} and because of the hemizygous phenotype which is similar to that described for l(1)7Ad^{1}. This chromosome has at least two mutations, one of which appears to be an Ax allele of N. Either Ax or another mutation distal to 7Ad strongly enhances the split thorax phenotype of l(1)7Ad. When this enhancing locus is removed, the phenotype is much less penetrant and not nearly as severe.

Dp(1;Y)dx+

From Abe Schalet. This is a transcription of his summary:

Dp(1;Y)dx's were made by irradiating the $In(1)sc^7$ XYL•YS chromosome and selecting for Y chromosomes that complemented dx.

Genetic/ Cytological coverage	Dp(1;Y)dx+Y #1	Dp(1;Y)dx+Y #2	Dp(1;Y)dx+Y #3	Dp(1;Y)dx+Y #4	Dp(1;Y)dx+Y #5	Dp(1;Y)dx+Y #6	Dp(1;Y)dx+Y #7	Dp(1;Y)dx+Y #8
Tip of X to distal break of $In(1)sc^7$	1A1;1B3-4	1A1;1B3-4	1A1;1B3-4	1A1;1B3-4	1A1;1B3-4	1A1;1B3-4	1A1;1B3-4	1A1;1B3-4
Proximal break of $In(1)sc^7$	6D8	6D8	6D8	6D8	6D8	6D8	6D8	6D8
dx	+	+	+	+	+	+	+	+
rux	+	+	+	+	+	+	+	-
cv	+	ND	+	+	+	+	+	-
omm/qed	+	+	+	+	+	+	±	±
cx	-	+	+	-	+	+	+	ND
Df(1)JC70	-	-	-	-	+	-	ND	ND
cho	-	-	-	ND	-	ND	ND	ND
su(f)	ND	-	ND	ND	ND	ND	ND	+
INFERRED CYTOLOGY	5A8-9;6D8	5A7-5C2;6D8	5A7-5C2;6D8	5A6-9;6D8	4C11;6D8	5A7-5C2;6D8	5A7-5C2;6D8	6D8

Df(1)dx^{81} (5C3-10;6C3-12 – uncovers dx).

I don't know how $Df(1)dx^{81}$ was constructed and I only have its cytology. I know Abe also made $Df(1)dx^{58}$ and $Df(1)dx^{67}$. Spyros Artavanis might have more information since Abe was working for him when he made these lines.