

TABLE S1

**Analysis of heteroallelic combinations between *psq<sup>rum</sup>* and other *psq* alleles or deficiencies uncovering *psq***

<i>psq</i> alleles or deficiencies	homozygous phenotype	maternal-effect phenotype of heteroallelic combination with <i>psq<sup>rum</sup></i>	nature of mutation	reference
<i>psq[rum]</i>	All embryos show terminal defects (lack of structures posterior to A7; n=100). In addition embryos frequently show deletions of one or more abdominal segments.		EMS-induced; G to A nucleotide substitution affecting the splicing donor site of intron 6, which is common to all known <i>psq</i> transcripts. This leads to partial retention of the intron in the mRNA and is predicted to cause the addition of 16 amino acids followed by a stop codon.	this work
<i>psq[8109]</i>	Decreased viability of adults, female sterile, posterior group, dorsalized eggshell and embryo, early oogenesis defects.	96% of embryos show little cuticle, 4% of embryos develop contiguous cuticle and lack terminal structures (n=100)	P{PZ} insertion into the largest intron of <i>psq-1</i> . Aberrant fusion protein created.	HOROWITZ and BERG 1995
<i>psq[0115]</i>	Decreased viability of adults, female sterile, posterior group, dorsalized eggshell and embryo, early oogenesis defects.	94% of embryos show little or no cuticle, 6% of embryos develop contiguous cuticle and lack terminal structures (n=100)	P{PZ} insertion into the largest intron of <i>psq-1</i> . Aberrant fusion protein created.	HOROWITZ and BERG 1995
<i>psq[fs1]</i>	Female sterile, posterior group, grandchildless	Embryos do not hatch. 80% of embryos lack segment A8 and/or spiracles (n=50)	P{lacW} insertion into the first intron of <i>psq-1</i> .	SIEGEL <i>et al.</i> 1993
<i>psq[rev2]</i>	n.d. *	Fertile	Isolated as EMS-induced revertant of <i>psq</i> overexpression in the eye. Deletion of 11 bp, resulting in a premature stop codon in the BTB domain. The P{GSV1}lola[GS88A8] insertion is still present on the chromosome.	FERRES-MARCO <i>et al.</i> 2006

<i>psq[rev4]</i>	n.d. *	Many collapsed eggs. 6.25% of embryos develop contiguous cuticle. These embryos lack abdominal segments (n=80).	Isolated as EMS-induced revertant of <i>psq</i> overexpression in the eye. Nucleotide substitution causing a Q530>stop mutation. The P{GSV1}lola[GS88A8] insertion is still present on the chromosome.	FERRES-MARCO <i>et al.</i> 2006
<i>psq[rev7]</i>	n.d. *	Fertile	Isolated as EMS-induced revertant of <i>psq</i> overexpression in the eye. Amino acid replacement M1I in the start Methionine of Psq1. The P{GSV1}lola[GS88A8] insertion is still present on the chromosome.	FERRES-MARCO <i>et al.</i> 2006
<i>psq[rev9]</i>	n.d. *	Weakly fertile. Adults show narrow blistered wings with extra veins. Embryos do not show terminal or other morphological defects (n=50).	Isolated as EMS-induced revertant of <i>psq</i> overexpression in the eye. G to A nucleotide substitution affecting the splicing donor site of intron 2 (72 bp), leading to retention of the intron in the mRNA. This causes the in-frame addition of 24 amino acids within the BTB/POZ domain, presumably disrupting the conserved BTB pocket domain, which is important for dimerization. The P{GSV1}lola[GS88A8] insertion is still present on the chromosome.	FERRES-MARCO <i>et al.</i> 2006
<i>psq[rev12]</i>	n.d. *	90% of embryos show little or no cuticle, 10% of embryos develop contiguous cuticle and lack terminal structures (n=80)	Isolated as EMS-induced revertant of <i>psq</i> overexpression in the eye. Amino acid replacement G867D in the third PSQ repeat. The P{GSV1}lola[GS88A8] insertion is still present on the chromosome.	FERRES-MARCO <i>et al.</i> 2006

<i>psq[rev14]</i>	n.d. *	98% of embryos show little or no cuticle, 2% of embryos develop contiguous cuticle and show posterior and terminal defects (n=100)	Isolated as EMS-induced revertant of <i>psq</i> overexpression in the eye. Nucleotide substitution causing a Q582>stop mutation. The P{GSV1}lola[GS88A8] insertion is still present on the chromosome.	FERRES-MARCO <i>et al.</i> 2006
<i>psq[KG02404]</i>	Viable, female-sterile	Embryos do not hatch, 60% of embryos lack segment A8 and/or spiracles (n=50)	P{SUPor-P} insertion in <i>psq-1</i> 5'-UTR	BELLEN <i>et al.</i> 2004
<i>psq[KG00811]</i>	Lethal	Fertile	P{SUPor-P} insertion in the largest intron of <i>psq-1</i> , 0.7 kb downstream of the transcription start site of <i>psq-3</i> transcripts	BELLEN <i>et al.</i> 2004
<i>psq[EP2011]</i>	Viable, female-sterile. Homozygous females lay no eggs.	Embryos do not develop cuticle (n=100).	P{EP} insertion in the largest intron of <i>psq-1</i> , 71 bp upstream of the transcription start site of <i>psq-3</i> transcripts	BELLEN <i>et al.</i> 2004
<i>Df(2R)47A</i>		Most embryos undeveloped, few embryos with little cuticle		FlyBase
<i>Df(2R)E3363</i>		Most embryos undeveloped, few embryos develop and show terminal defects		FlyBase

Phenotypes of embryos from females carrying heteroallelic combinations of *psq<sup>um</sup>* are indicated. \*: We were unable to determine homozygous phenotypes of the *psq* revertant alleles, all of which are lethal in homozygous state and in all trans-heterozygous combinations tested. Lethality of these chromosomes is presumably due to the presence of additional mutations or due to the P{GSV1}lola[GS88A8] P-element insertion.

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