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*forever young*: a gene facilitating the study of the third larval instar of *D. melanogaster*.

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In our study of the gene *sarah* (genetic symbol *sra*; synonym *nebula*, genetic symbol *nla*; publication in preparation), we discovered two alleles of the gene *forever young* (genetic symbol *fey*). When balanced over *TM6B*, *Tb e*, homozygous *fey* larvae are identifiable by their *Tb*<sup>+</sup> phenotype. *Tubby* (*Tb*) causes short, thick individuals; the phenotype is visible in larvae, pupae and adults. *fey*<sup>1</sup> homozygotes, *fey*<sup>2</sup>/*Df(3R)sbd* (see below), and *fey*<sup>1</sup>/*fey*<sup>2</sup> trans-heterozygotes develop to the third instar larvae. Some eventually pupate. Homozygous *fey*<sup>2</sup> animals die at an earlier stage.

The *fey* phenotype allows studies on behavior and tumorigenesis of third instar larvae. When placed on food containing 20-hydroxyecdysone (1 µg/ml), most *fey* homozygotes pupate though morphogenesis is arrested early. Mixed in the substrate, any chemical can be studied for its potential of inducing pupation.

The two *fey* alleles were obtained from a P-element reversion experiment where the P-elements in the *sra* alleles *sra*<sup>P1</sup> and *sra*<sup>P2</sup> were removed. Originally, *fey*<sup>1</sup> and *fey*<sup>2</sup> had been referred to as *sra*<sup>11</sup> and *sra*<sup>12</sup>, respectively, (Czank 1998) as the result of the fact that P-element removal was a "*sra* allele" that was homozygous lethal and had a *sra* phenotype in trans-heterozygous *sra/sra*<sup>11</sup> or *sra/sra*<sup>12</sup> animals. A genomic rescue with a construct containing the *sra* genomic DNA including the 5' regulatory sequences was performed. This rescue construct reverted the *sra* phenotype (studied in trans-heterozygotes) but not the lethality. Therefore, we believe that the original P-element removal was imprecise and thus causing a mutant *sra* allele, and, as an additional effect, caused a mutation in *fey*. This is possible as the P-elements in *sra*<sup>P1</sup> and *sra*<sup>P2</sup> have inserted in the large *sra* intron that might harbor regulatory sequences for *fey*. However, we cannot exclude that our rescue construct was lacking stage specific regulatory sequences for *sra*. If so, *fey* would be a lethal *sra* allele.

The two genes, *fey* and *sra*, lie in the same genomic region 89B12. When *fey*<sup>1</sup> or *fey*<sup>2</sup> is crossed to flies with *Df(3R)sbd*<sup>26</sup>, *Df(3R)sbd*<sup>45</sup> or *Df(3R)sbd*<sup>104</sup>, no adult *fey/Df(3R)sbd* trans-heterozygotes are found. These *sbd* deletions also uncover *sra*. Two stocks are available, UM3 and UM4, the flies have the genotype *cn; ry fey*<sup>1</sup> *sra*<sup>3</sup>/*TM6B*, *Tb e* and *w; fey*<sup>2</sup> *sra*<sup>4</sup>/*TM6B*, *Tb e*, respectively.

References: Czank, A., 1998, *sarah*, a novel gene affecting reproduction of *Drosophila melanogaster* females: Phenotype analysis and cloning. Ph.D. thesis, University of Zurich, Switzerland.