For instance, *m* is a symbol of the *miniature* locus in *D. melanogaster*, but in *D. virilis* this is a symbol for the another gene - *magenta*. We propose to unify the genetic symbols of these two species and to symbolize, in particular, the *miniature* locus in *Drosophila virilis* as *m*.

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Suppressing apoptosis fails to cure "extra-joint syndrome" or to stop sex-comb rotation.

Held, Lewis I., Jr. Department of Biological Sciences, Texas Tech University, Lubbock, Texas 79409.

One of the strangest fly phenotypes ever described is an "extra-joint syndrome," where the leg has an extra, inverted joint in all but one of its five tarsal segments (Held *et al.*, 1986). This defect is extreme for the *spiny legs 1* allele at the *prickle* locus (*pk* ^{sple1}) and milder for other mutations in the planar cell polarity (PCP) pathway that also cause disoriented bristles (*e.g.*, *frizzled*). Its resemblance to the segment-polarity syndrome of the embryo is intriguing, but its etiology remains a mystery.

A possible clue comes from a similar phenotype obtained with surgical experiments on abdominal segments of the milkweed bug. When Wright and Lawrence (1981) excised more than half the length of a tergite, the cut edges healed together, and the next instar had an extra, inverted segment boundary in the new tissue that grew between the edges.

If leg and body segmentation are homologous processes, as some authors have proposed (Minelli, 2003), then defects in the PCP pathway might evoke extra leg joints by a similar route. To wit, a mutation might cause more than half of the cells along each affected tarsal segment to die, and healing might elicit intercalation of reversed-polarity tissue containing an extra joint.

To test whether cell death is an essential part of the *pk* ^{sple1} syndrome's etiology, I used the baculovirus *p35* gene to suppress apoptosis, as has been done for the eye (Hay *et al.*, 1994). To express *p35* in the tarsal region and incidentally in the arista, I used *Distal-less Gal4* (*Dll-Gal4*) to drive a *UAS-p35* construct in flies that were also homozygous for *pk* ^{sple1}.

The desired genotype was created as follows. *Dll-Gal4*/*CyO* males were mated with *pk* ^{sple1} females, and non-*Curly* (*Dll-Gal4*/*pk* ^{sple1}) F_1 virgins were collected, wherein crossing over between *Dll* and *pk* should yield recombinant *Dll-Gal4 pk* ^{sple1} 2nd chromosomes (25% total). These virgins were mated to F_1 males from a cross between females carrying *UAS-p35* on the 3rd chromosome and *pk* ^{sple1} males. F_2 flies carrying both *Dll-Gal4* and *UAS-p35* were distinguished by short or missing claws, though rarely an extra (third) claw was present. Their aristae were swollen at the base, with extra posterior lateral branches near the tip, as is typical for loss-of-function *hid* alleles that suppress apoptosis (Cullen and McCall, 2004). Such flies comprised 28% of the total eclosed F_2 (52/187), and 21% of them (11/52) had disoriented bristles (as per *pk* ^{sple1}) — thus evincing no lethality for the

combined genotype (5.9% actual ≈ 6.3% expected based on 100% viability). Flies were cultured at 25˚C, and pupae were transferred to humid, empty petri dishes to prevent adults from getting mired in the food. The legs of 10 desired flies were mounted between cover slips in Faure's solution and examined at 200×.

All 20 second legs of these *Dll-Gal4 pk* ^{sple1} / *pk* ^{sple1}; *UAS-p35*/+ flies displayed at least one extra, inverted joint, as assessed by a ball-and-socket articulation (without regard to the presence of an intersegmental membrane). Six of them had the full complement of four extra, inverted joints. The overall average was 3.0 extra, inverted joints per leg. In contrast, 20 control second legs of *pk* ^{sple1} F_2 siblings (disoriented bristles but normal claws and aristae) had 4.0 extra joints, while 20 control second legs of *Dll-Gal4* (*pk* ^{sple1} / +?); *UAS-p35*/+ F_2 siblings (normal bristles but defective claws and aristae) had no extra joints.

These results argue that apoptosis is not needed for the extra-joint syndrome. The lower expressivity of extra, inverted joints in *Dll-Gal4 pk* ^{sple1} /*pk* ^{sple1}; *UAS-p35*/+ flies (3.0 *vs.* 4.0) may be due to synergism between polarity disruption and apoptosis suppression. In about half the legs (9/20) the T5 segment was deformed (swollen with vesicles, invaginations, or blebs), and the normal joint between T4 and T5 was missing, in contrast to the relatively normal controls.

I also studied the sex combs of 10 *Dll-Gal4* (*pk* ^{sple1} / +?); *UAS-p35*/+ F_2 males to see whether suppressing apoptosis impedes rotation of the sex comb. Among their 20 first legs, only one comb had an odd angle. It was split, and each half had rotated ~ 60˚ (*vs.* the normal ~ 90˚). There was no effect on tooth number (mean = 10.25, N = 20), though 13/20 combs had 1-3 teeth apart from the main row. These results imply that apoptosis is not needed to "make room" for rotation of the sex comb into proximal territory on the basitarsus during normal development (Held *et al.*, 2004), nor to refine the number of teeth (Held, 2002). How the sex comb rotates remains unclear. Apoptosis had seemed a plausible guess, since flies do use it to adjust patterns (Meier *et al.*, 2000), but the present data rule it out.

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A new paracentric inversion in the left arm of the third chromosome of *Drosophila ananassae.*

Singh, P., and B.N. Singh*. Genetics Laboratory, Department of Zoology, Banaras Hindu University, Varanasi-221005, India. *E- mail: bnsingh@bhu.ac.in.

Drosophila ananassae exhibits a high degree of chromosomal polymorphism. A total of 70 paracentric and 17 pericentric inversions and 13 translocations have been described in *D. ananassae*