

Figure S1: Model for β -catenin regulation in the seam cells of a wild type worm.

Two pools of APR-1/APC are present in the anterior daughter, a microtubule-associated WRM-1-regulating pool (wrmAPR-1, shown in blue) and a KIN-19/CKIα associated SYS-1-regulating pool (sysAPR-1, shown in red). Polarized localization of both sysAPR-1 and wrmAPR-1 is controlled by PRY-1/Axin. Frizzled and Dishevelled presumably localize to the posterior daughter, but how they translate the polarity and activity signal to APR-1 is unknown (denoted by question marks). S, SYS-1/β-catenin (red); W, WRM-1/β-catenin (blue); Ax, SYS-1-regulating PRY-1/Axin (red) Ax, WRM-1-regulating PRY-1/Axin (blue); Ap, SYS-1-regulating APR-1/APC (red); Ap, WRM-1-regulating APR-1/APC (blue); K, KIN-19/CKIα (red); MT, microtubules; Fz, Frizzled; D, DSH-2, MIG-5/Dishevelled.

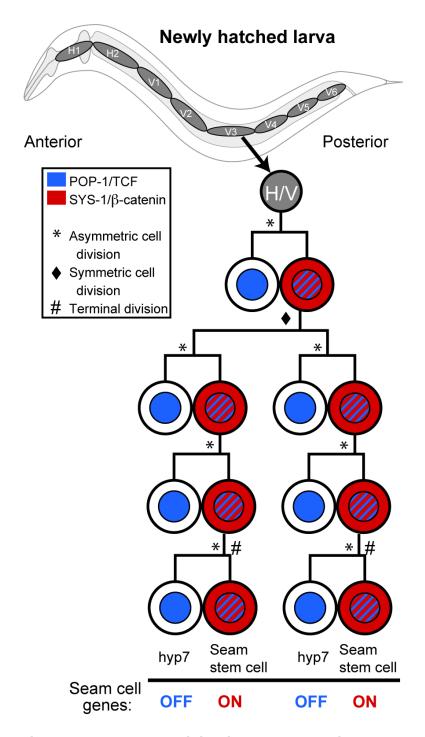


Figure S2: The epidermal seam cells divide in a stem cell-like pattern dependent on the WβA signaling pathway. A cartoon lineage of a representative seam cell. Nuclear POP-1/TCF is high in the anterior nuclei, resulting in target gene repression. Nuclear POP-1 is low in posterior in response to WRM-1/ β -catenin-mediated nuclear export, SYS-1/ β -catenin levels are high in response to stabilization, and Wnt targets are activated that result in adoption of the seam cell fate. Note that the "#" symbol marks the terminal/final seam cell division, upon which we focus our studies.

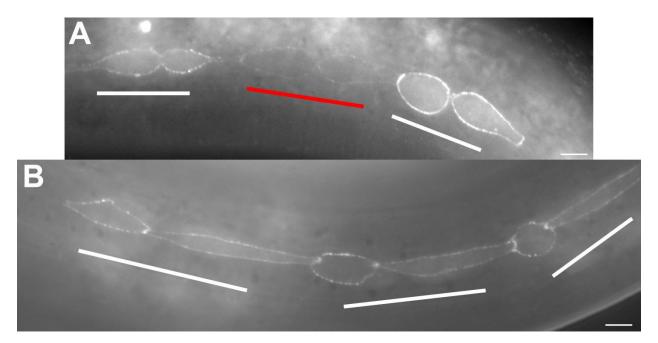


Figure S3: PRY-1::GFP localizes symmetrically to seam cell daughter cortices and does not alter cell fate specification. A, $osEx229(p_{pry-1}::PRY-1::GFP)$ in wild-type seam cells. Lines indicate daughter pairs. The red line marks a daughter pair with lower osEx229 expression that still displays symmetric PRY-1::GFP localization. B, osEx229 expression after division and fate specification. White lines mark daughter pairs. The anterior daughters have correctly adopted the hyp7 fate and have become smaller and more rounded in anticipation of fusion with hyp7, while the posterior daughters have adopted the seam fate and begun elongation. Scale bars = 5 μ m.

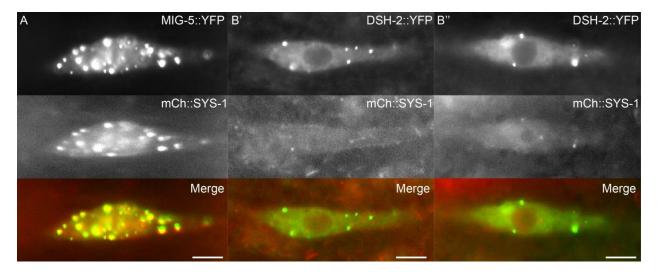


Figure S4: Dvl puncta are present prior to division. All images are representative of seam cell mothers in interphase that are expression high/punctate levels of Dvl overexpression. A, top panel: $osEx233(p_{SCM}::MIG-5::YFP)$, middle panel: $uiwls4(p_{sys-1}::mCherry::SYS-1)$, bottom panel: merge. B' & B'', top panels: $osEx225(p_{SCM}::DSH-2::YFP)$, middle panels: $uiwls4(p_{sys-1}::mCherry::SYS-1)$, bottom panels: merge. Scale bars = 5 μ m.

Table S1: DSH-1 does not regulate seam cell fate. Seam cell counts were obtained in early adulthood using the *wls51*(*scm*::GFP) reporter. EV = empty vector.

Genotype	RNAi treatment	Average seam cell number ± standard error	N- value
wild-type	EV	15.98 ± 0.07	62
wild-type	dsh-1 RNAi	15.96 ± 0.05	62
dsh-1(ok1445)	EV	16 ± 0.05	61
dsh-2(or302) mig-5(tm2639)	EV	17.66 ± 0.28	54
	dsh-1 RNAi	17.72 ± 0.27	59