Caenorhabditis elegans Vulva formation

→ why C. elegans? → nematode
- simple system
- few cells with already known identities (cell fate map)
- small gene number for Ras & Notch signalling

→ hermaphrodite → creates vulva to lay egg after self-fertilization.

→ Notch-Dependant Lateral Inhibition
SEQUENTIAL SIGNALLING
↳ tells one cell that it is the organizer

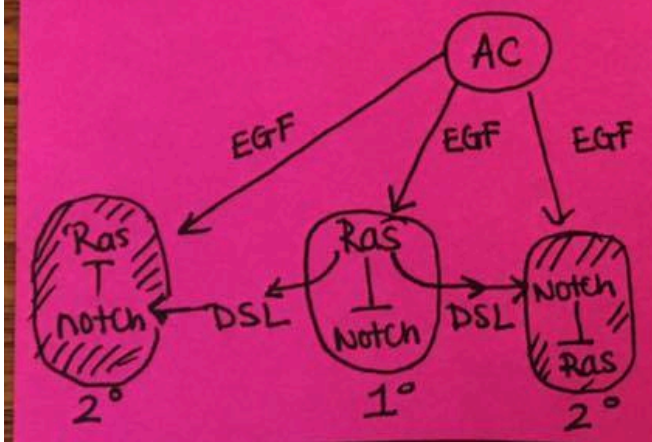
1. ANCHOR CELL is organizer in gonad.
↓
2. releases EGF
↳ signals to neighboring cells & forms vulval pre-cursor cells (VPC)
→ 1° cells → certain divisions + certain position
→ 2° cells → diff. position + helps egg pushed out
→ 3° cells

→ determined by Notch lateral inhibition → makes lots of delta and induces notch signalling to stop neighbors from being anchor cells too.

important to have ONE anchor cell otherwise multiple vulva.

secretes EGF (epithelial growth factor) → activates Ras pathway in neighbor and stimulate their differentiation → Ras promotes expression of ligands to activate Notch signalling in adjacent cells

tells other neighboring cells what to do.



But remember how 1° cell have high EGF and Ras pathway?
→ Ras through kinase pathway
→ transcription factor that TURN OFF NOTCH
→ and make DSL ligand
→ on surface
→ turn on notch in neighbor.

How formed?
- cells in developing Gonad express DSL ligand AND notch receptor

Step 1:
notch + DSL ↔ notch + DSL ↔ notch + DSL

Step 2:
Notch ← AC → notch

1° and 2° cells divide multiple times to form vulva

cells induced by EGF signalling from Anchor cell and can form either 1° (primary) or 2° (secondary) cell fates.
→ cell closest to Anchor cell is induced by AC to form primary cell
→ because it recieves most EGF and highest amount of Ras onswitch
→ releases signals and says you be 2° and I am 1°

3° cells furthest from AC and didn't recieve any EGF signal → become ventral epidermis

What happens when you ablate the Anchor Cell?
- everything becomes 3° cell → no vulva but only always ventral epidermis

important?
- Ras/notch disrupted in cancer
- birth defects
- regulate stem cell

NOTCH Activated → Ras off → 2° fate

Anchor cell: high levels of DSL → activate notch signalling in neighbor → notch ligand ⊣ Anchor cell fate

1. After NICD is released and translocate to nucleus
2. NICD displaces CSL transcription factor, switching it to activator
3. Target: Hes genes: transcriptional repressors inhibit DSL

(Hes activated → ⊣ DSL)

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