Autonomous Specification in Tunicate development

Autonomous & Conditional Specification in *C. elegans* Embryonic Development

**Figure 8.36** Bilateral Symmetry in the Egg of the Tunicate *Styela partita*

**Figure 8.37** Cytoplasmic Rearrangement in the Fertilized Egg of *Styela partita*
Fig. 8.37 Cytoplasmic Rearrangement in the Fertilized Egg of Styela partita

(C) Clear cytoplasm

(D) Yellow cytoplasm

Sperm pronucleus

Yolk material

Yellow crescent

Figure 8.38(1) Cytoplasmic Segregation in the Egg of Styela partita.

Figure 3.8 Autonomous Specification in the Early Tunicate Embryo
Figure 3.9 Acetylcholinesterase in Progeny of Muscle Lineage Blastomeres

Normal larva - AChE stain in muscles
From B4.1 pair
From remaining 6 blastomeres

Figure 3.10 Microsurgery on Tunicate Eggs

Animal pole
Vegetal pole
Muscle-forming region
Tissue including muscle cells
Needle pushes muscle-forming cytoplasm into animal cells

Figure 8.39 Autonomous Specification by a Morphogenetic Factor

macho-1 mRNA localization
Caenorhabditis elegans embryonic development

Autonomous specification:

- P granule segregation

Endomesoderm specification
- skn-1 gene function

Conditional specification:

- Endomesoderm specification
  - Wnt signaling
Caenorhabditis elegans complete (embryonic & post-embryonic) cell lineage

Figure 8.44(1) Segregation of the P-granules into the Germ Line Lineage of the C. elegans Embryo

Figure 8.44(2) Segregation of the P-granules into the Germ Line Lineage of the C. elegans Embryo
Segregation of the P-granules into the Germ Lineage

C. elegans 4 cell embryo

Autonomous specification of cell lineages

Normal cell lineage
Deficiencies of Intestine and Pharynx in Skn-1 Mutants of C. elegans

**Figure 8.45**

**A** Wild-type

Pharynx muscle antigen

Posterior pharynx is derived from MS

**B** skn-1 mutant

No pharynx

**C** Gut-specific granules

Gut derived from E

**D** No gut

Transformation of EMS blastomere into P2-like cell

Fate transformation in progeny of skn-1(-/-) mothers (maternal effect)

Transformation of P2 blastomere into EMS-like cell

pie-1(-/-) mothers (maternal effect)

EMS -> “P2-like”

pie = pharynx in excess
Normal segregation of Skn-1 & Pie-1 proteins in EMS & P2

Skn-1 is a bZIP TF

Pie-1 is also a TF

Segregation of Pie-1 protein into P cell germ line precursors

Blue: DAPI (nuclei)  Red: Pie-1 protein

Pie-1 is a component of P granules
Segregation of Pie-1 movie

Transformation of blastomeres in mex-1 mutants

Fate transformation in progeny of mex-1(-/-) mothers (maternal effect)

Misregulation of Skn-1 protein in mex-1 mutant embryos

Mex-1 is a TF
Caenorhabditis elegans embryonic development

Endomesoderm specification also depends on cell-cell signaling

P2 signals EMS to promote endoderm formation in one of its progeny (E)

Figure 8.46 Experiments Show that Cell-Cell Interactions Are Required for the EMS Cell to Form Intestinal Lineage Determinants

Separation

(Figure 8.46) Fate of EMS progeny

Separation + recombination

P2 signals EMS to make gut!

Time of separation (minutes before EMS cleavage)

Figure 8.47 Cell-Cell Signaling in the 4-Cell Embryo of C. elegans

P2 also signals ABp to specify its fate (not covered)

P2 signal to EMS causes daughter cell next to P2 to be endoderm (E cell)
C. elegans Wnt signaling pathway in blastomere specification

Maternal effect mutants affecting P2 -> EMS signal:

Mom - “more of MS” (E transformed to MS)

Pop - “posterior pharynx defect” - opposite phenotype to Mom (MS transformed to E)

pop-1 mutant embryo phenotype

mutant embryo  wild type embryo

posterior pharynx missing  excess gut
C. elegans Wnt signaling pathway in blastomere specification

P2 to EMS signal uses a Wnt signaling pathway

- **Mom-2**: Wnt-like (secreted signal from P2)
- **Mom-5**: Frizzled (receptor)
- **Gsk-3**: GSK3 (kinase)
- **Wrm-1**: β-catenin (transcription factor)
- **Pop-1**: TCF (transcription factor)

No apparent Dishevelled (Dsh) homolog

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**Canonical vs. Worm Embryo comparison of the Wnt Pathway**

**A** Canonical Wnt pathway

- Wnt
- Frizzled
- Dishevelled
- GSK3
- β-catenin

→ TCF/LEF target genes

**B** P2 to EMS signal in C. elegans

- EMS
- **Mom-2**
- **Mom-5**
- **Gsk-3**
- **Wrm-1**
- **Pop-1** (HIGH)
- **Pop-1** (LOW)

→ posterior pharynx
- body muscle
- intestine

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**Fig. 6.24(1) The Wnt Signal Transduction Pathway**

How β-catenin typically works as a transcription factor in the nucleus:

- β-catenin binds to TCF to help it turn on transcription in downstream target genes
Wnt Signaling in C. elegans vulval development

Wnt signaling in vulval development is canonical:
BAR-1 binds and activates POP-1 (TCF homolog)

\[
\begin{align*}
\text{BAR-1} & \rightarrow \text{TCF} \\
\downarrow & \\
\text{POP-1} & \rightarrow \text{Wnt target gene} (e.g., \text{lin-39}) \\
\end{align*}
\]

BAR-1/POP-1 complex turns on downstream target genes

Non-canonical Wnt Signaling in C. elegans embryogenesis

The β-catenin homolog Wrm-1 works differently in embryonic Wnt signaling pathway:

Wrm-1 activates "Nemo-like" kinase Lit-1
Lit-1 phosphorylates Pop-1, which blocks its nuclear localization

Loss of nuclear localization

Variations on the Wnt Pathway
Variations on the Wnt Pathway

<table>
<thead>
<tr>
<th>VULVA FORMATION IN C. ELEGANS</th>
<th>CANCER IN MAMMALS</th>
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<td>Targets (lin-39)</td>
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