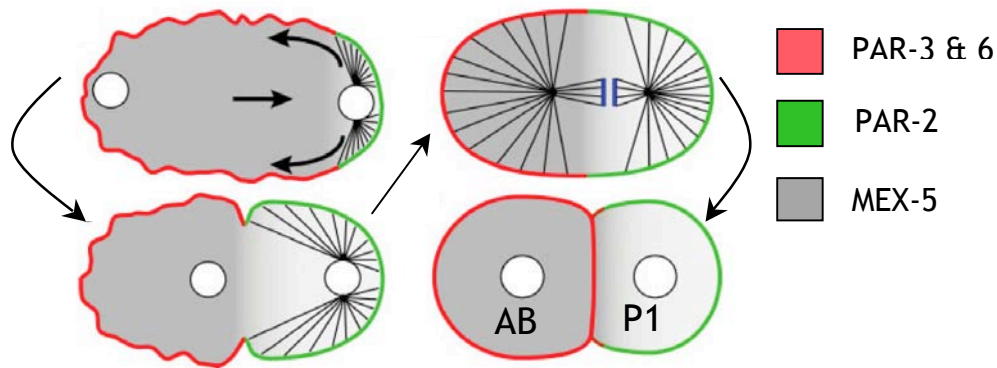


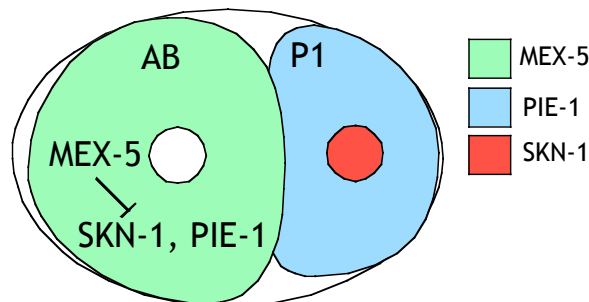
Step 2: Anterior and posterior differences arise in the cortex of the zygote, involving the PAR proteins



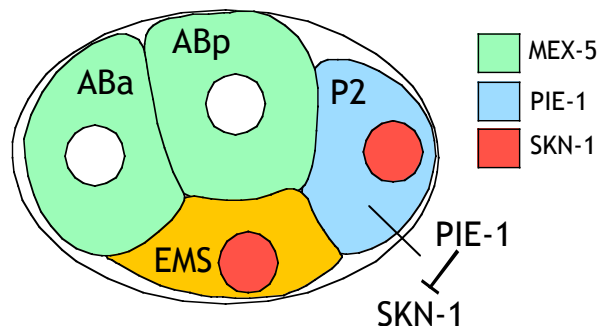
Adapted from Nance (2005). *Bioessays* 27:126-135.

Different PAR (for partitioning defective) proteins (PAR-3 and PAR-6 in the anterior, and PAR-2 in the posterior) localize to the anterior and posterior, reinforcing differences in these two ends of the zygote. PAR proteins promote accumulation of mRNAs and proteins in each end of the zygote, such as MEX-5 in the anterior.

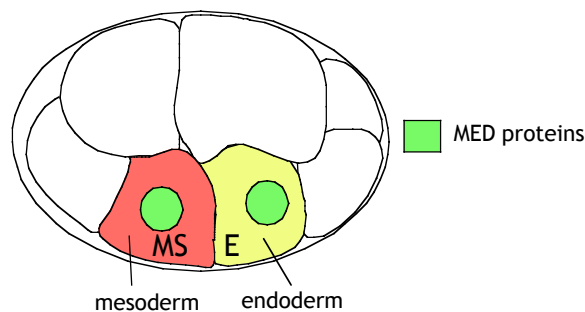
Step 3: SKN-1 and PIE-1 proteins accumulate in P1; MEX-5 and other proteins accumulate in AB. The MEX proteins keep SKN-1 and PIE-1 from being translated in the anterior cell of the two-cell embryo (AB).



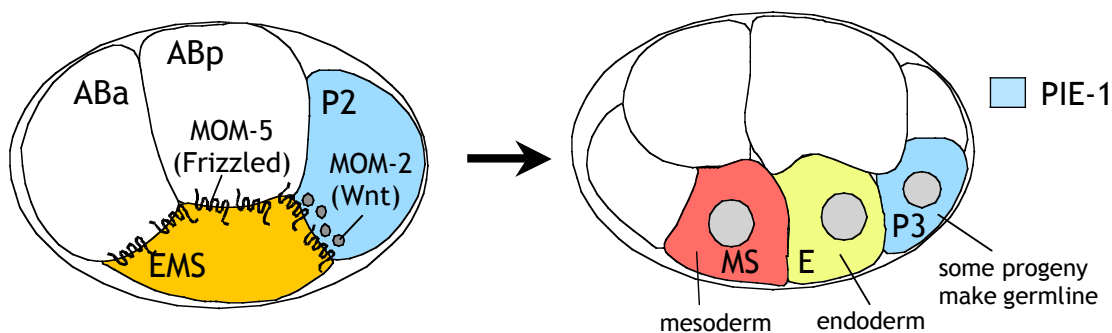
Step 4: PIE-1 represses the activity of SKN-1 in P2, resulting in a single cell in which SKN-1 acts (the EMS cell).



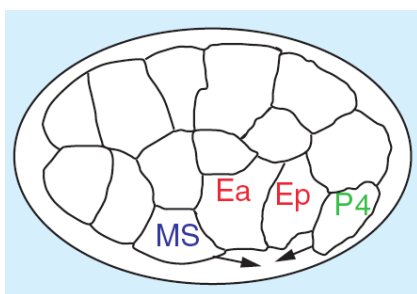
Step 5: SKN-1 activates transcription of genes (called MED genes), that allow differentiation of mesoderm and endoderm in the daughters of EMS (MS and E, respectively).



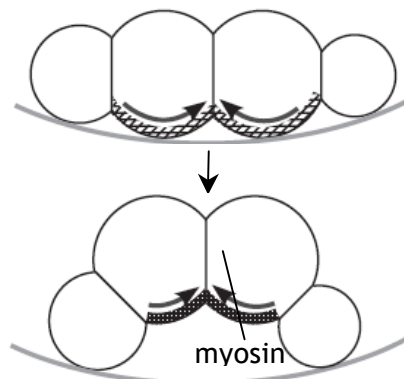
Step 6: A Wnt signal from P2 results in the posterior daughter of EMS becoming endoderm. Without this signal, E becomes a second mesodermal precursor instead. The Wnt signal, called MOM-2, is produced by P2. EMS has a Frizzled (MOM-5) that allows it to detect this signal. Without the activity of these two genes, no endoderm forms, and instead, extra mesoderm forms. Thus they are named "mom", for more mesoderm.



Step 7: Cells are internalized during gastrulation via multiple ingression movements. The endodermal precursor cells, Ea and Ep, ingress to internalize the endoderm. One proposed mechanism for ingression involves contraction of the outer, or apical, surfaces of Ea and Ep, causing them to undergo apical constriction. This constriction presumably involves actin and myosin.



Putzke & Rothman (2003). *Curr. Biol.* 13, R223-R225.



Adapted from Lee & Goldstein (2003). *Development* 130:307-320.