

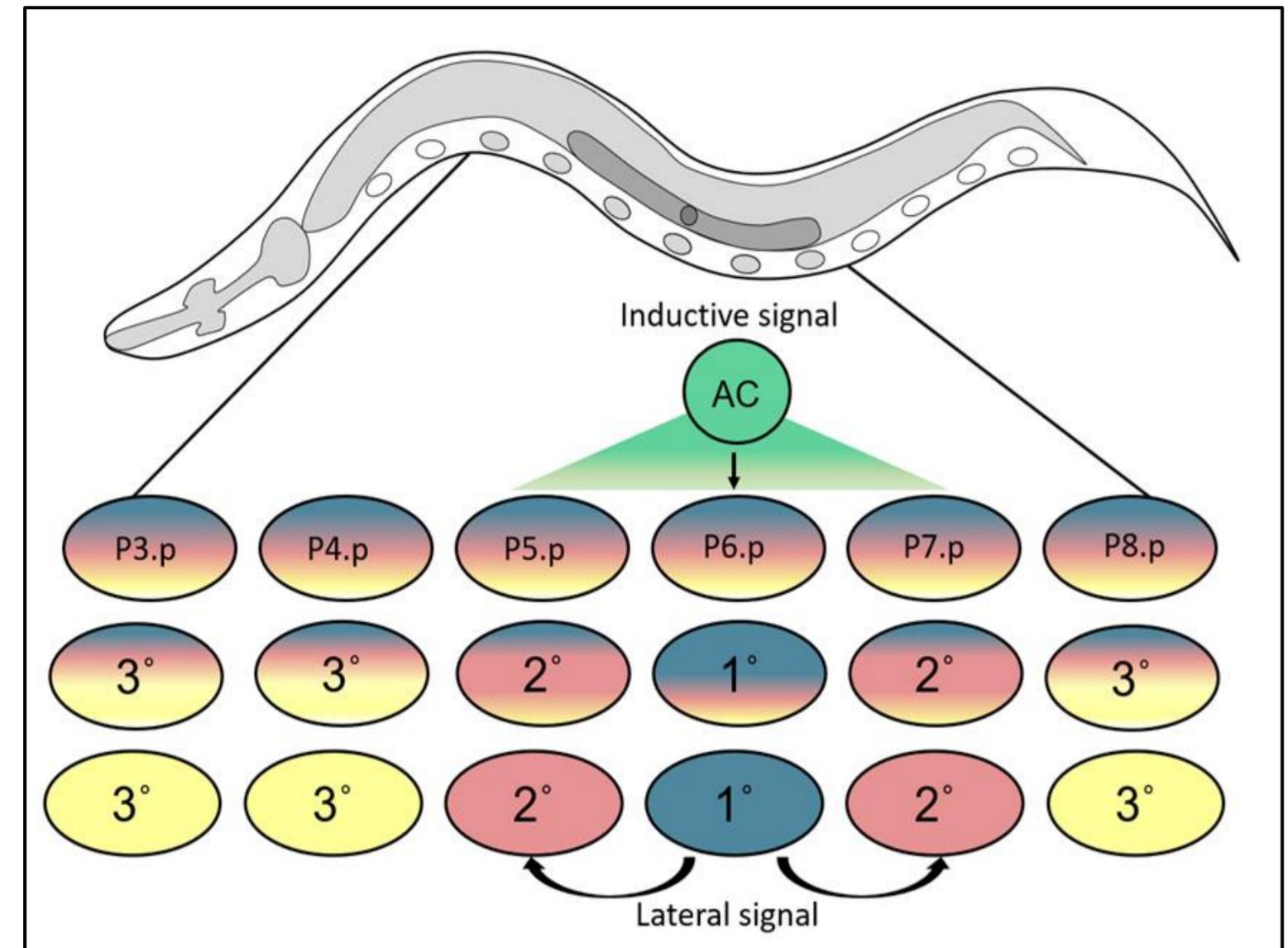


The MIG-15 MAP4 kinase promotes tertiary fate of *C. elegans* Vulva Precursor Cells Patterning

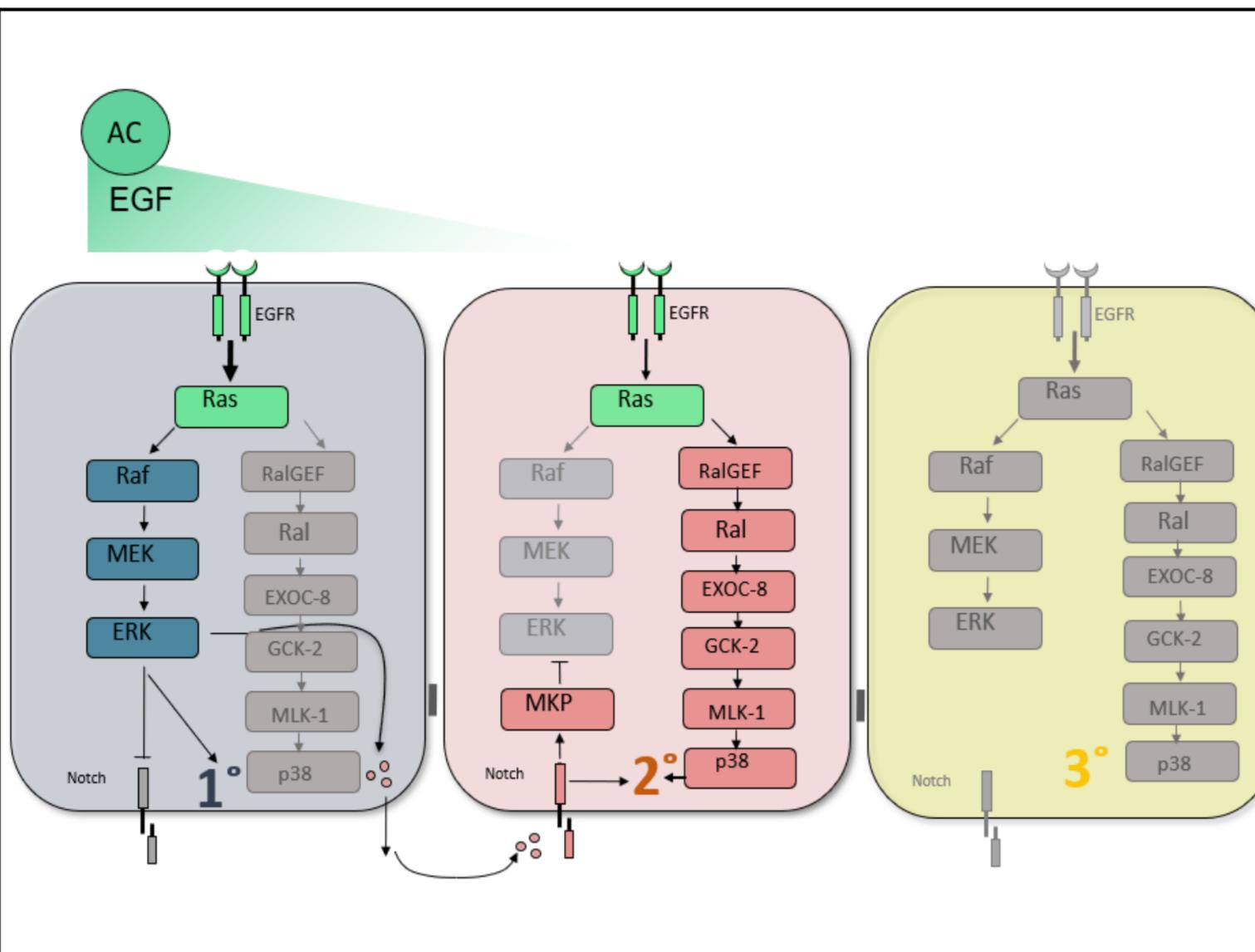


INSTITUTE OF BIOSCIENCES
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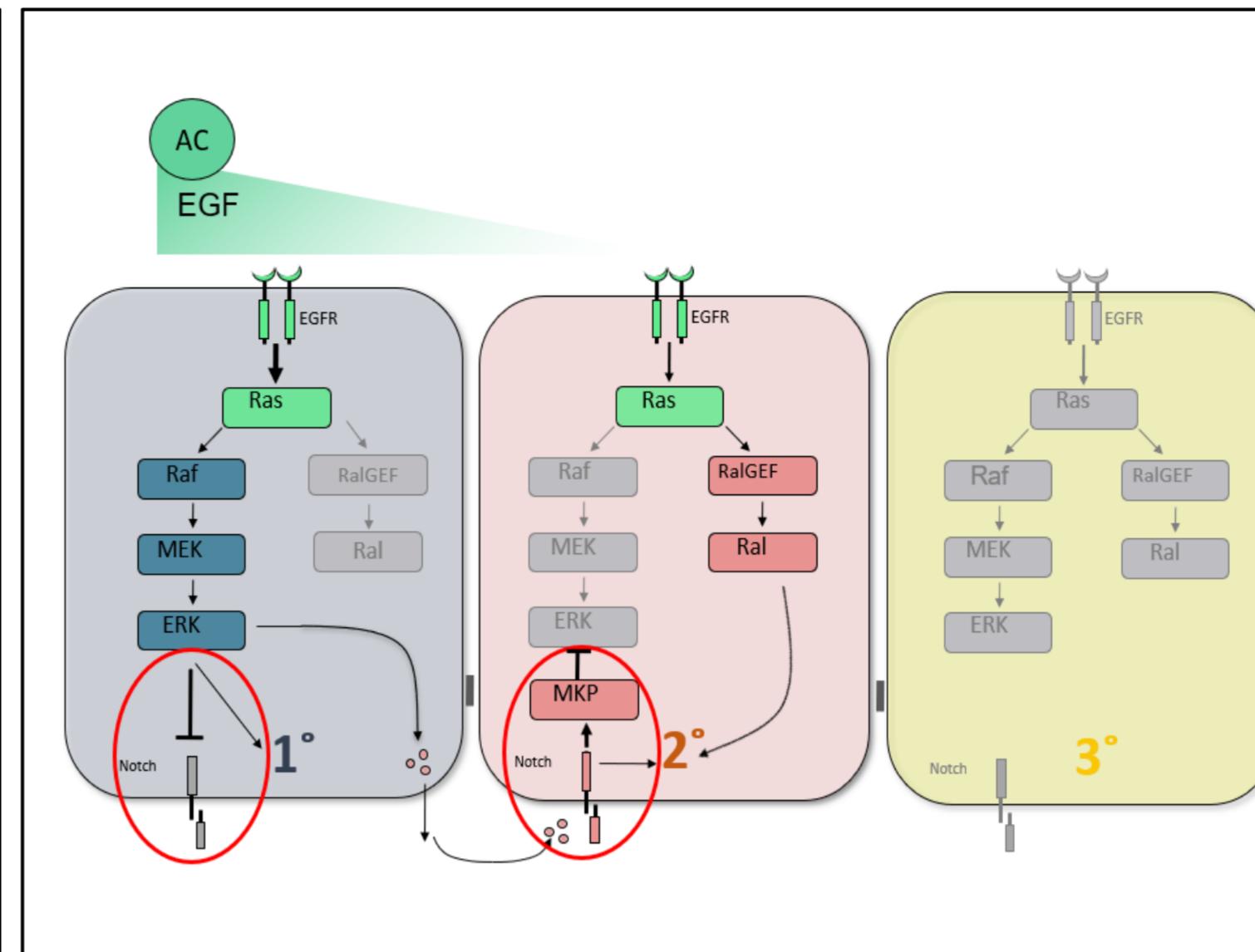
Equipotent to Committed



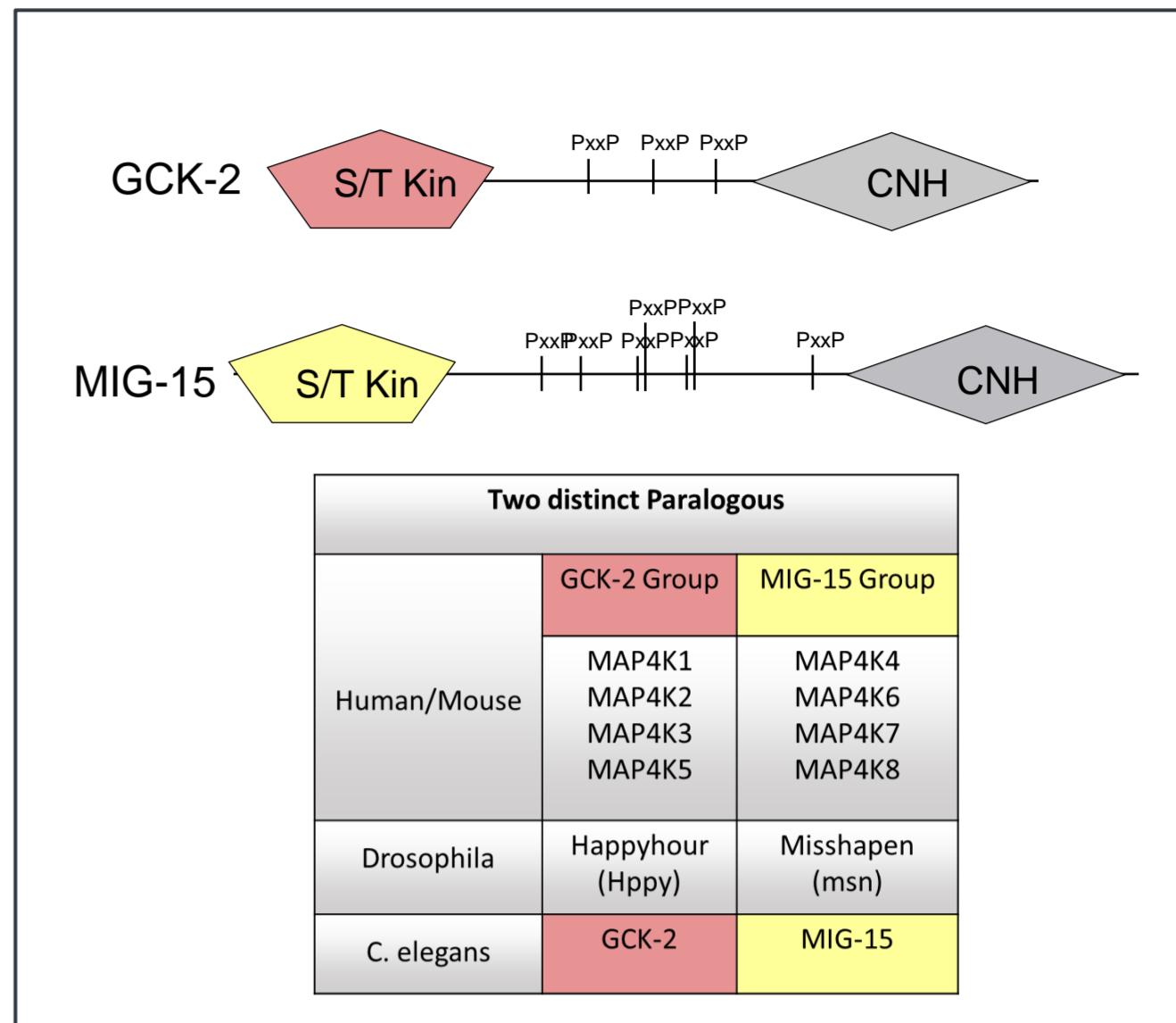
Signaling Network



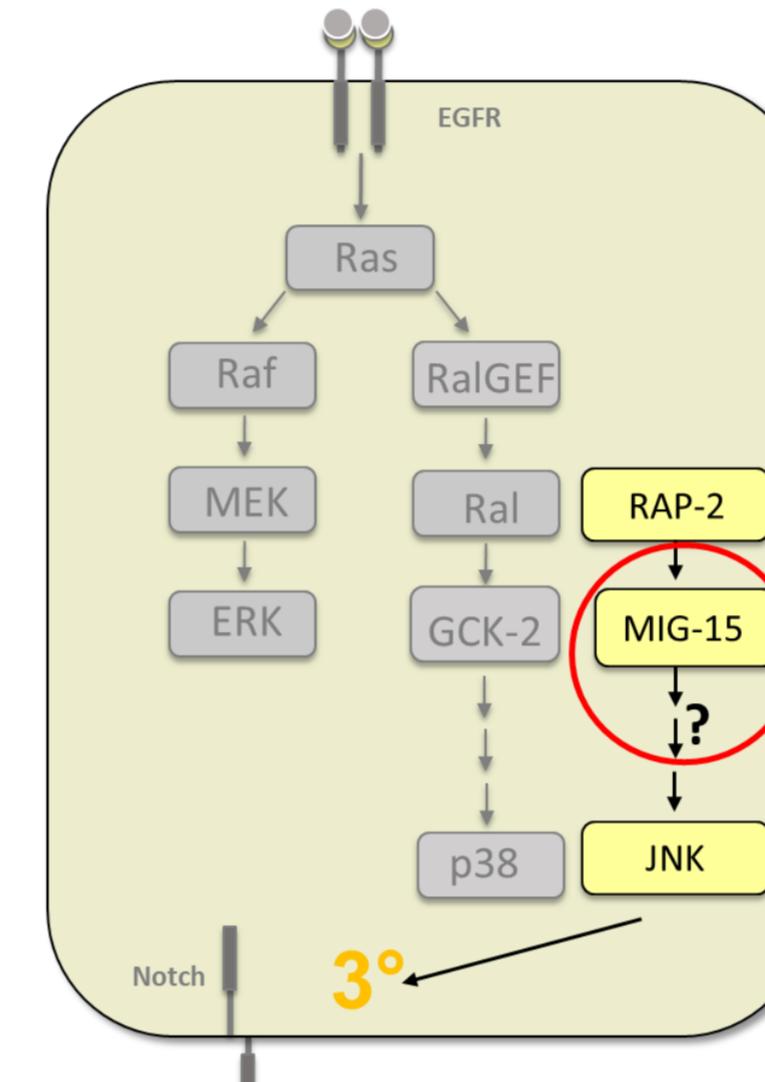
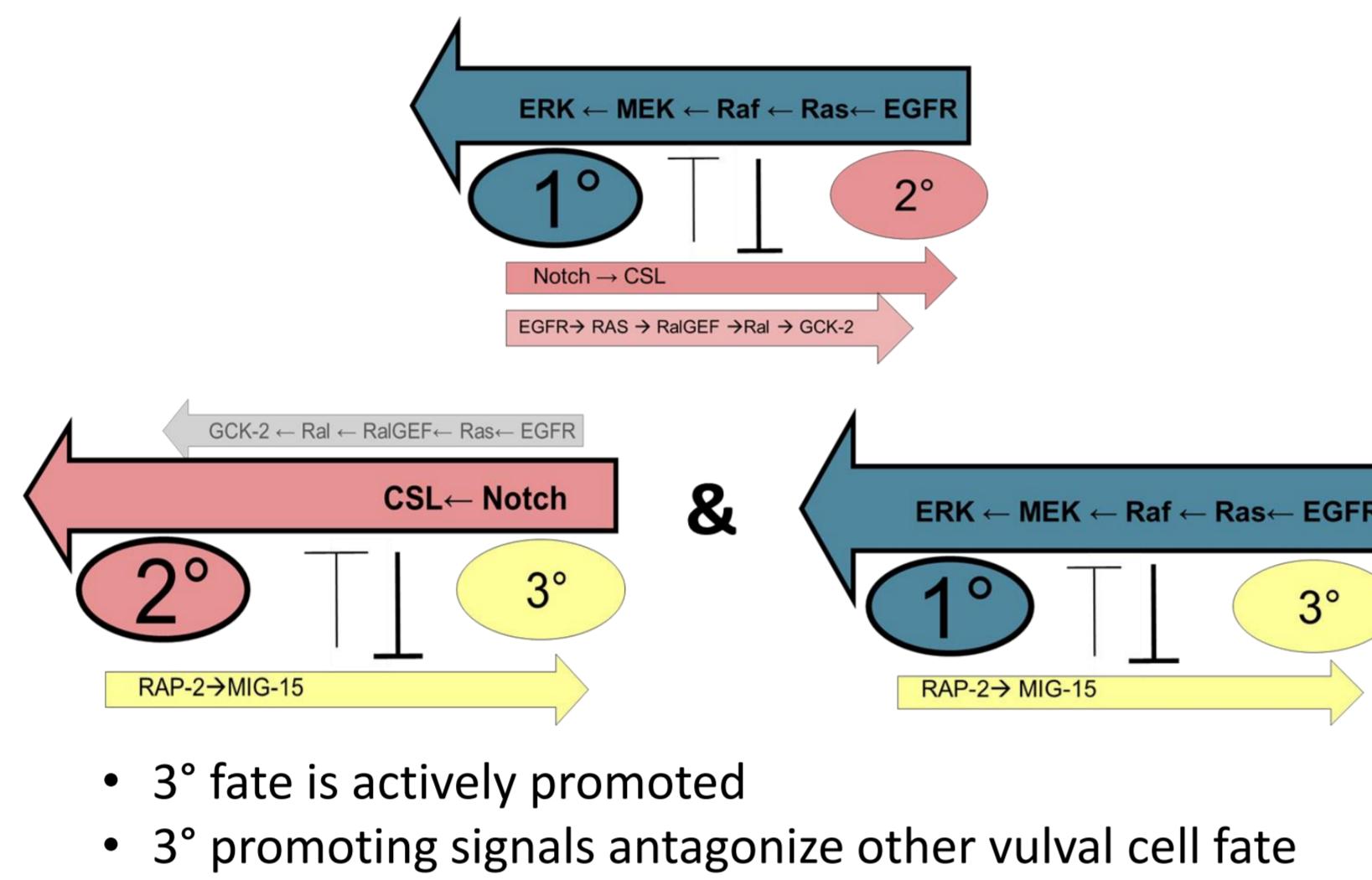
Mutual Antagonism



CNH MAP4 Kinase



Hypothesis

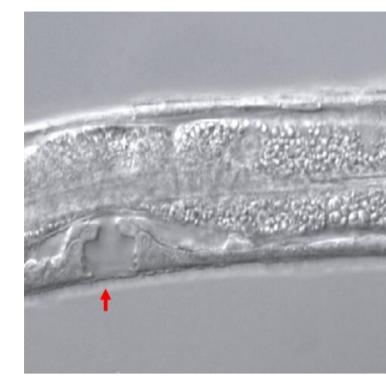


Aims

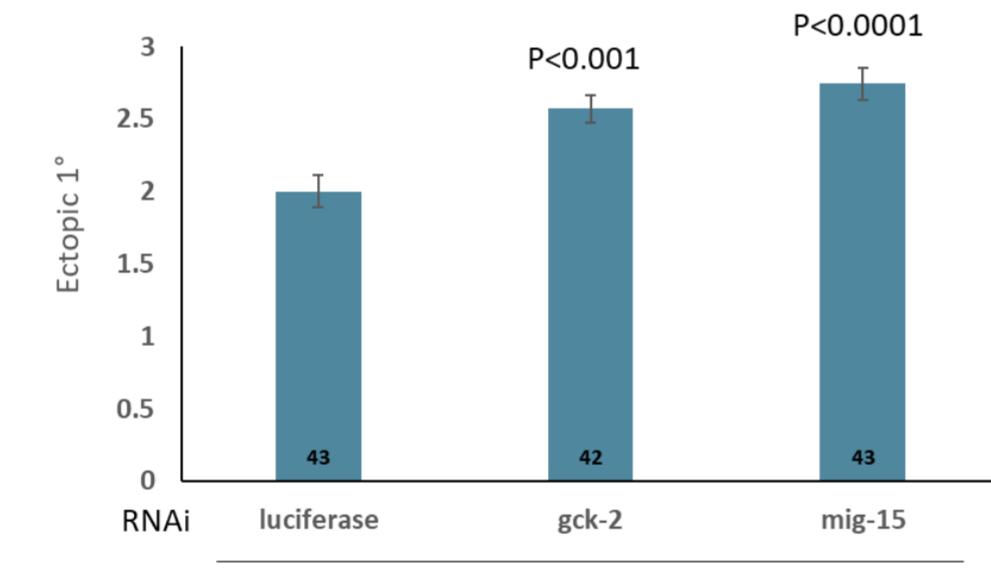
1. Elucidate the 3°-promoting role of MIG-15
2. Test whether MIG-15 functions cell autonomously in VPCs
3. Determine whether RAP-2 → MIG-15 → JNK functions as a 3°-promoting signaling cascade.

MIG-15 and RAP-2 are 3° Promoting Proteins

A Wild Type

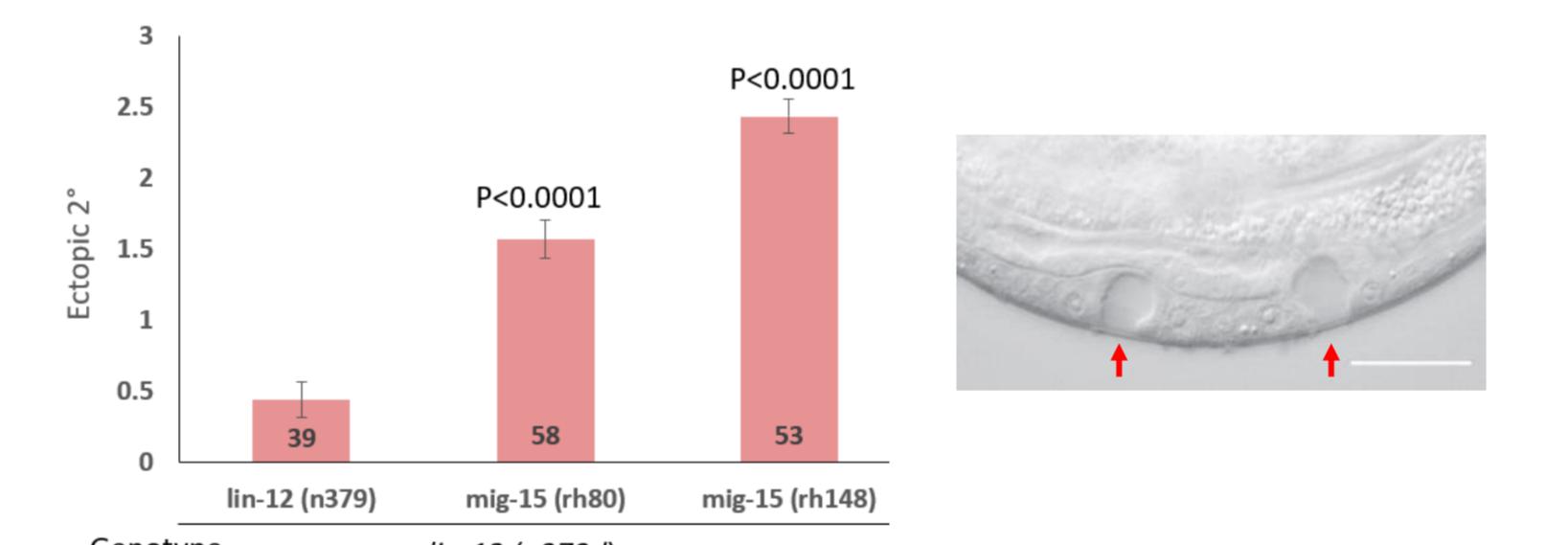


B Increase in ectopic 1° with MIG-15 RNAi depletion



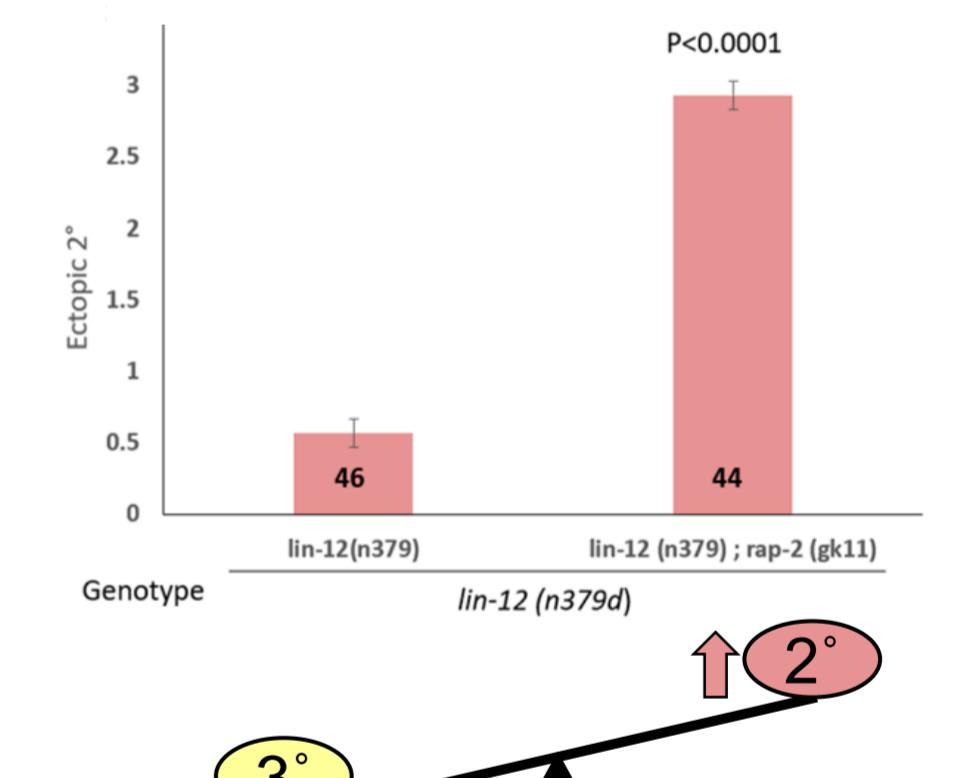
C

Increase in ectopic 2° with MIG-15 mutant and RNAi depletion

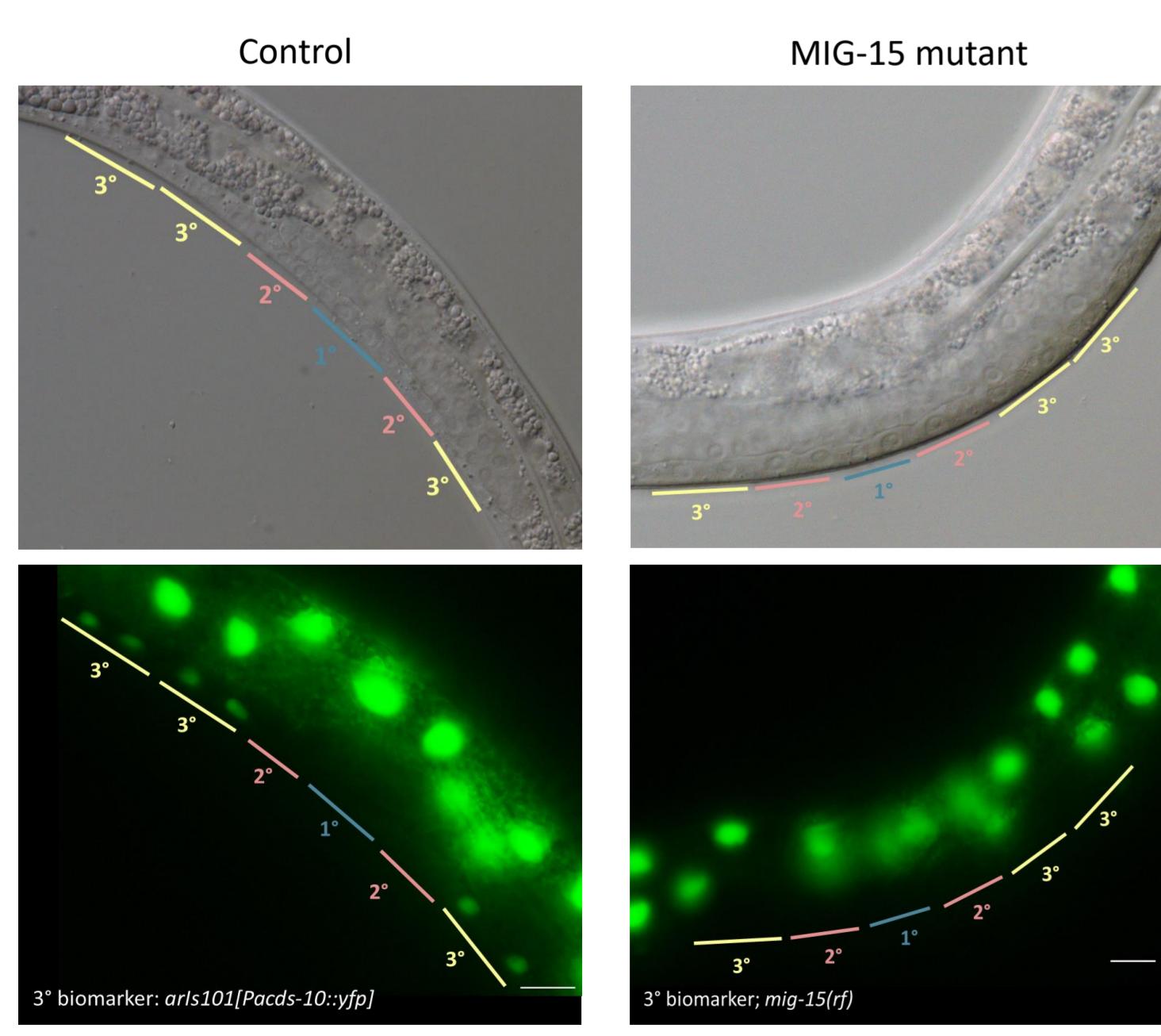


D

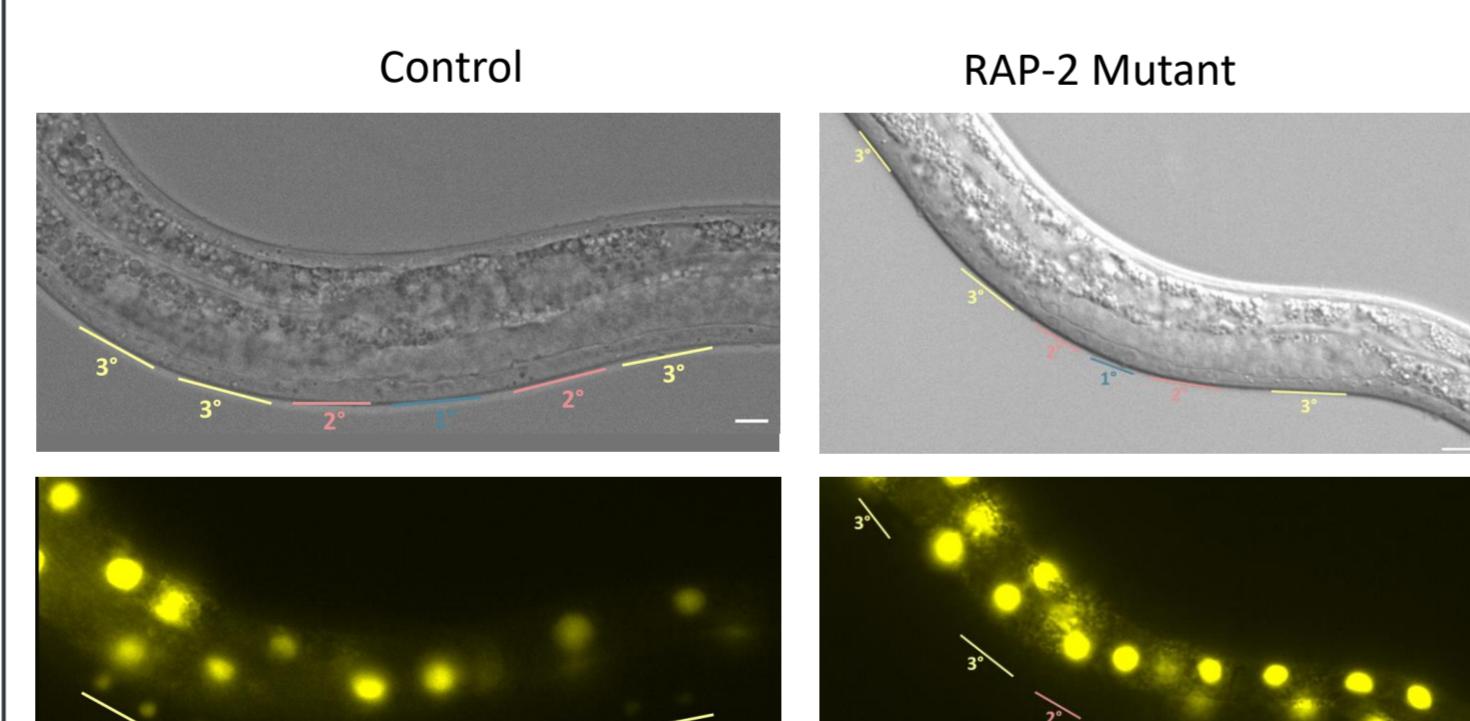
Increase in ectopic 2° with RAP-2 mutant



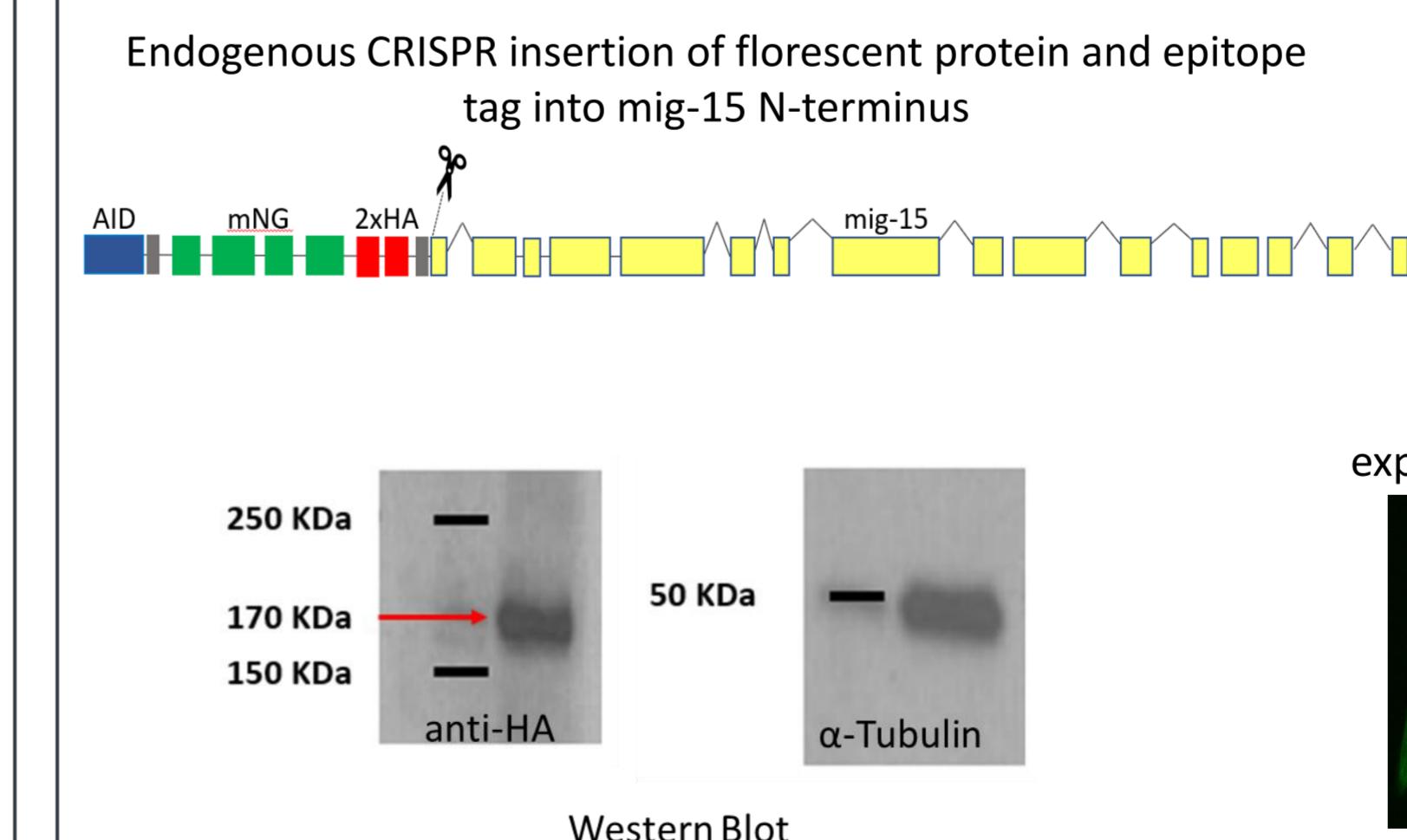
3° Biomarker diminished in MIG-15 mutant



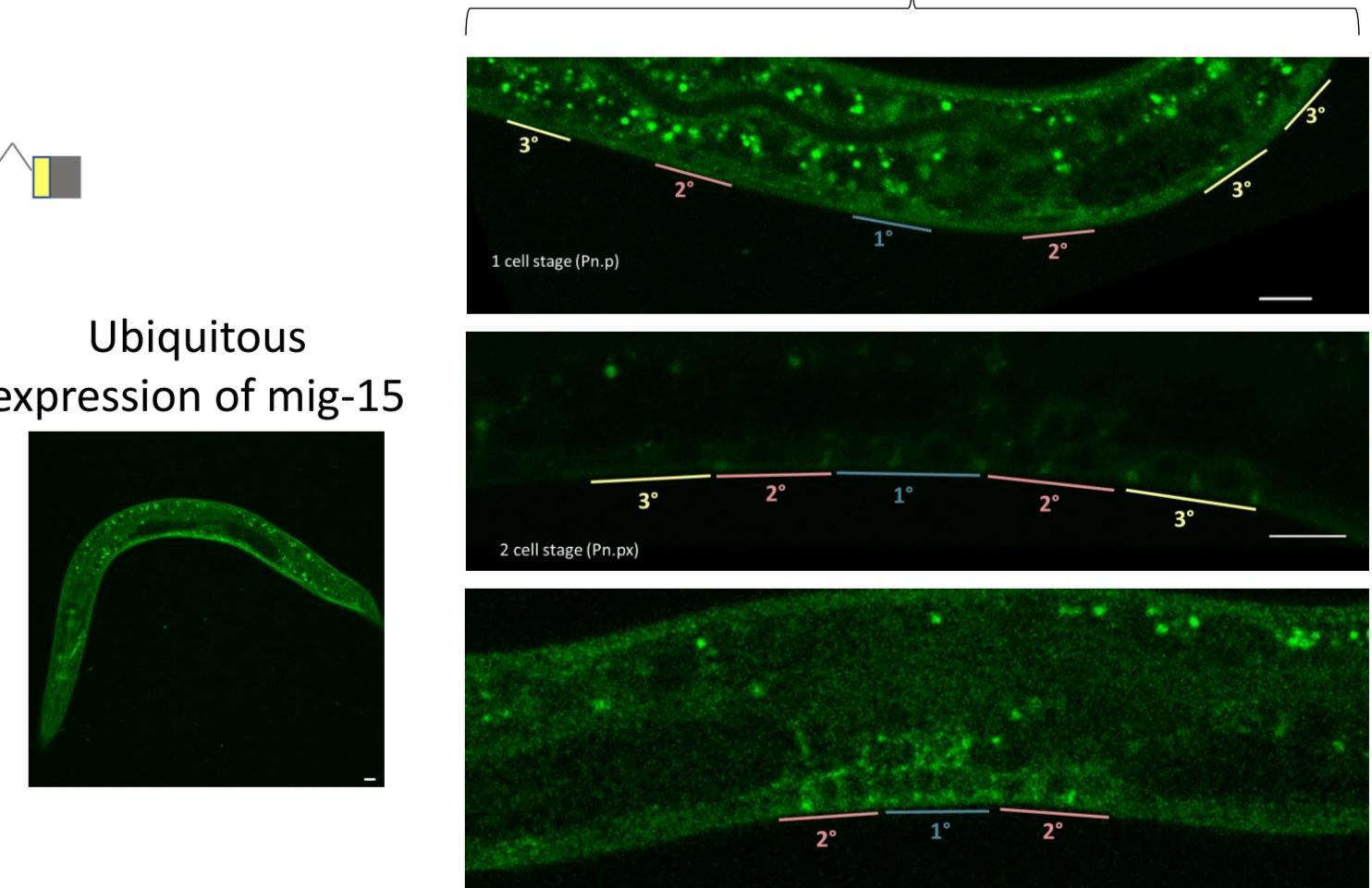
RAP-2 phenocopies MIG-15 Loss



MIG-15 is Expressed Ubiquitously and Localizes to Cytosol and Junctions in VPCs

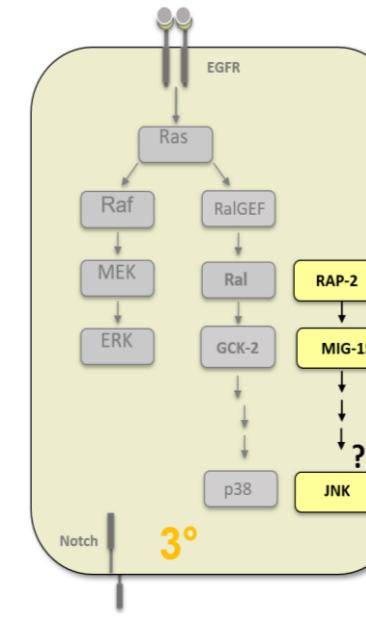


Expression of mig-15 in VPCs



Summary & Future Plans

- MIG-15 acts as anti-1° and 2° to promote 3° fate
- RAP-2 represses 2° signal
- Loss of MIG-15 and RAP-2 reduce expression of 3° fate reporter



- Activation of MIG-15 through mid-deletion
- VPC-specific rescue of MIG-15 mutant
- VPC-specific depletion of tagged MIG-15
- Genetic testing of RAP-2-MIG-15 pathway relationship