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Non-canonical Ras output: RalGEF⟶Ral and segregating signals

Abstract:
We investigate novel components and organizational principles of signal transduction networks. EGF induces certain C. elegans epithelial cells to form the 3˚-3˚-2˚-1˚-2˚-3˚ pattern of cell fates. Interpretation of a dose-sensitive EGF-EGFR gradient is mediated by LET-60/Ras switching effectors, from the canonical Raf-MEK-ERK MAP kinase cascade to promote 1˚ fate to the non-canonical RalGEF-Ral to promote 2˚ fate. We observe that Ras activates its two effectors in spatially segregated subcellular compartments: Raf is recruited basolaterally while RalGEF is recruited apically. We have also discovered that Ral signaling through Exo84 of the exocyst complex leads to activation of GCK-2, a CNH domain-containing MAP4 kinase, which in turn signals through a PMK-1/p38 MAP kinase cascade to promote 2˚ fate. RalGEF, an inessential component, orchestrates opposing signals; its deletion increases patterning errors by 15x, suggesting a role in mitigating signaling noise. Thus, we have found novel components and wiring mechanisms in signaling networks.

Keck Seminar

Friday, February 16, 4pm

BioScience Research Collaborative

Room 280 (2nd Floor)

Gulf Coast Consortia
QUANTITATIVE BIOMEDICAL SCIENCES

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