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IDENTIFYING NEW FGFR SIGNALING COMPONENTS IN *C. ELEGANS*

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Fibroblast growth factor receptors (FGFRs) are cell-surface receptor tyrosine kinases (RTKs) that regulate essential cellular processes. Mutations that disrupt FGFR signaling can cause developmental abnormalities and cancer. EGL-15 is the sole FGFR in the nematode *C. elegans*, and the genetic analysis of EGL-15 signal transduction has been useful for identifying new RTK signaling components. EGL-15 couples to downstream signaling pathways, such as the RAS/MAPK cascade, via the direct binding of the SEM-5/Grb2 adaptor protein. This signal transduction cascade regulates fluid levels within the worm's body, such that hyperactivation of this pathway causes excessive accumulation of clear fluid inside the worm's body. This dramatic effect is referred to as the Clr (clear) phenotype. The isolation of **S**uppressor **O**f **C**lr (Soc) mutants led to the identification of many of the core components of EGL-15 signaling. Surprisingly, eliminating the direct SEM-5 binding sites on EGL-15 does not confer a Soc phenotype, indicating that an alternate pathway can couple EGL-15 to SEM-5/Grb2. To identify components of this alternate pathway, we repeated the screen for Soc mutants in this *egl-15* mutant background. Of 25 mutations analyzed, 11 define two new *soc* genes, *soc-3* and *soc-4*, whose molecular identities were recently determined by a whole-genome sequencing approach. *soc-3* encodes a protein with structural features consistent with it acting within an alternate pathway that can couple EGL-15 to SEM-5/Grb2. Future experiments will test this hypothesis and probe the roles of these new FGFR signaling components.