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# Inhibition of Touch Cell Fate by *egl-44* and *egl-46*

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In wild-type *C.elegans* six cells develop as receptors for gentle touch. In *egl-44* and *egl-46* mutants (which were first identified because of defects in the HSN neurons; Desai *et al.*, 1988, Nature 336: 638), however, the FLP neurons also possess touch receptor-like features and express touch receptor genes (Mitani *et al.*, 1993, Development 119: 773). Because these genes appear to repress the expression of touch cell fate, we have cloned them and characterized their action on touch cell-specific genes. *egl-44* encodes a TEF(transcription enhancer factor)-like protein, and like TEF proteins, EGL-44 contains a TEA/ATTS DNA-binding domain and a putative transcriptional regulation domain. A *gfp::egl-44* rescuing fusion is expressed in nuclei of neurons (including the FLP and HSN cells), hypodermis and interstine, but not in touch cells. *egl-46* encodes a zinc-finger protein that with two *Drosophila* proteins and two proteins from humans and mice defines a novel zinc-finger subfamily. A *gfp::egl-46* rescuing fusion is expressed predominantly and transiently (except for in a few cells) in nuclei of neurons, including FLP, HSN, and the touch cells. The late and transient expression of *gfp::egl-46* in the Q lineages and the previously identified lineage defects in *egl-46* mutants, suggest that the gene may control the production and differentiation of terminal cells in this lineage. In contrast to its expression in many other cells, *egl-46* is continually expressed in the FLP neurons; this expression is dependent on *egl-44*.

To test whether coexpression of *egl-44* and *egl-46* prevented the expression of touch cell characteristics, we ectopically expressed *egl-44* and *egl-46* in the touch cells. This expression resulted in touch insensitivity and the loss of *mec-7*, *mec-4*, and *mec-18* expression. In addition, as with the FLP cells, coexpression of *egl-44* and *gfp::egl-46* led to the continued expression of the fluorescent protein. These defects were not seen when *egl-44* was expressed in the touch cells of *egl-46* mutant animals. These results suggest that expression of both genes are needed to repress touch cell fate in the FLP cells.

EGL-44 and EGL-46 are likely to repress the expression of touch cell function genes by binding to their promoters and preventing the binding of the MEC-3::UNC-86 complex. EGL-44 and EGL-46 bind to each other in *in vitro* S-Tag pull down experiments. In addition, the binding of EGL-44 to specific sites in the *mec-4* and *mec-7* promoters is enhanced by the presence of EGL-46. These binding sites are near those of UNC-86 and MEC-3, and the binding of these latter proteins is prevented by the presence of EGL-44 and EGL-46. We are currently examining the roles of different domains of EGL-44 and EGL-46 in this binding to understand further the action of these genes in the combinatorial control of touch cell fate.