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A Computational Model of the Network that Controls Fate Determination and Cell Fusion in the Vulval Precursor Cells of *Caenorhabditis elegans*

The vulva of *Caenorhabditis elegans* has been used as a model for the study of cell differentiation and organogenesis for more than three decades. In this system the signaling cascades of WNT, Ras/MAPK and NOTCH interact with each other forming a molecular network, but the precise topology and dynamics of this network is incomplete. By making an exhaustive search of the experimental literature, we built a multivalued, discrete, synchronous model of the molecular network that controls cell fusion and fate determination in the vulval precursor cells of *Caenorhabditis elegans*. The proposed model is the first to include the Hox genes *lin-39, mab-5* and *ceh-13*, the WNT signaling pathway with the polarity defining genes, most of the components of the Ras/MAPK and NOTCH signaling pathways, the fusogen *eff-1*, and the genes that regulate its transcription. After simulating the dynamic behavior of the model we found steady patterns of gene expression and protein activity that correspond to those reported in the literature for each cell type; vulval precursor, first fate, second fate, second fate with reversed polarity, third fate and fusion fate. Moreover, we were able to simulate the fusion of cells, the determination of the first fate, second fate, the transition from the second fate to the first fate and the determination of the third fate under the right environmental conditions. Finally, the model was used to simulate all possible single gene loss- and gain-of-function mutants, as well as some double and triple mutants. We were able to associate most of these simulated mutants to multivulva (Muv), vulvaless (Vul), egg-laying defective (Egl), or defective polarity (Biv) phenotypes.