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Title: Dpr and DIP cell surface protein interactions control selection & survival of amacrine neurons in color vision circuits

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Abstract: Sperry's chemoaffinity hypothesis proposes that assembly of neural circuits involves interactions among cell-surface proteins (CSPs) that label individual neurons or neuronal types. This includes heterophilic interacting partners expressed on synaptically connected neurons. Among these are Dprs and DIPs, which are members of an interacting network of immunoglobulin superfamily (IgSF) CSPs. Here we examine the functions of the DIP-gamma and Dpr11 interaction pair in the development of color vision circuits in the optic lobe. The Drosophila compound eye is composed of ~750 ommatidial units. Each ommatidium contains eight photoreceptors (PRs) that express different rhodopsins (Rh). Outer PRs (R1-R6) are used for motion detection, while the inner PRs (R7 and R8) transmit chromatic information. Visual information received in the retina is transmitted to the optic lobe (OL), which consists of neuropils: lamina, medulla and lobula complex. The medullary neuropil is arranged in ~750 columns with 10 layers. There are two major types of ommatidia – pale (p) and yellow (y), which are randomly distributed in the retina. p ommatidia detect shorter wavelengths and have R7 (pR7) that express the Rh3 (shorter-wave UV) rhodopsin; y ommatidia detect longer wavelengths and have R7 (yR7) that express Rh4 (longer-wave UV). Axons of R7 PRs grow through the lamina and into the medulla, where they find their synaptic targets in layer M6. One of the major targets of R7 PRs in the medulla is Dm8, a wide-field amacrine neuron that pools information from 12 to 16 R7s. In our study, we found that Dpr11 and DIP-gamma CSPs define 'yellow' and 'pale' color vision circuits. yR7 PRs specifically connect to DIP-gamma-expressing Dm8 neurons (yDm8), while pR7 PRs connect to DIP-gamma-negative Dm8 neurons (pDm8) in their respective y and p 'home columns' in the medulla. We examined Dm8 neurons in these circuits by electron microscopic reconstruction and expansion microscopy. Mutations in both DIP-gamma and its binding partner dpr11, alter the morphologies of yDm8 dendrites. Dpr11 and DIP-gamma also control survival of yDm8 neurons. During early development, these amacrine neurons are generated in excess and compete for presynaptic yR7 PRs. Their survival depends on interactions between Dpr11 and DIP-gamma that allow yDm8 neurons to find their appropriate presynaptic yR7 PRs. Manipulation of R7 subtype fate in the retina alters Dm8 fates in the medulla by changing the relative representation of yDm8 and pDm8 neurons. Thus, Dpr11-DIP-gamma interactions control yDm8 survival and R7-Dm8 connectivity.