Transmembrane protein VSD is required for axon crossing for the formation of central complex

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Neuronal connections between the hemispheres are considered important for organisms to organize information to adapt to variable environments. In central complex split flies, the locomotor activity and the visual learning and memory are defect. Axon midline crossing is an event that requires many components to regulate each other. We found a transmembrane protein VSD (visual system disorganization) is essential for the formation of the central complex. Nonsense mutation in the extracellular domain of VSD contributed to the mutant phenotype. Biochemistry results showed the protein can be cleaved by an unknown protease and release the N terminal from the membrane. It has genetic interaction with another transmembrane protein NRG. Overexpression the dominant negative form of NRG can partially rescue the mutant phenotype caused by knockdown of *vsd*. We proposed that NRG can inactivate VSD through an unknown pathway to regulate the axon midline crossing.