SMART Teams 2015-2016 Research and Design Phase

Cedarburg High School SMART Team

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Opsin: *To See or Not to See*

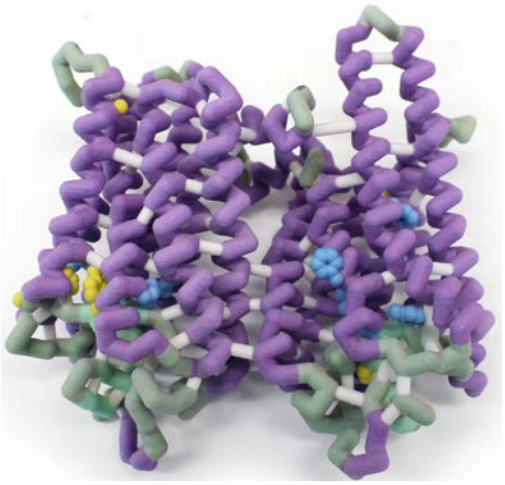
PDB: 3CAP

Primary Citation:
Jung Hee Park, Patrick Scheerer, Klaus Peter Hofmann, Hui-Woog Choe, and Oviver Peter Ernst. 2008. Crystal Structure of the Ligand-free G-protein-coupled Receptor Opsin. *Nature*, v454, 183-188.

Joseph Carroll, Alfredo Dubra, Jessica C. Gardner, Liliana Mizrahi-Meissonnier, Robert F. Cooper, Adam M. Dubis, Rick Nordegren, Mohamed Genead, Thomas B. Connor. Jr, Kimberly E. Stepien, et al. The Effect of Cone Opsin Mutations on Retinal Structure and the Integrity of the Photoreceptor Mosaic. *ARVO*, v53,8006-8015.

Format: Alpha carbon backbone

RP: Zcorp with plaster



Description:
Colorblindness, a genetic disorder affecting millions of people worldwide, is caused by mutations in a retinal protein, opsin. Opsin is a G-protein coupled receptor which binds to light sensitive retinal. When light enters the cell, the retinal isomerizes from the *cis* form to the *trans* form, and opsin is activated; nerves then signal the brain. The opsin complex, sensitive to specific wavelengths of light, is identified by the wavelength that activates it; long (L), middle (M), or short (S). Mutations in opsin may lead to visual problems, including the inability to distinguish between red and green. The Cedarburg SMART (Students Modeling A Research Topic) Team has used 3D printing technology to construct a model of opsin which contains two monomers consisting of several helices that are stabilized by interactions between Lys231-Glu247 and Tyr223-Arg135. Two openings in opsin can be found in the retinal-binding pocket; one allows the *cis* form of retinal to enter and bind opsin, while another between allows the *trans* form to exit opsin. Common forms of colorblindness are characterized by mutations in amino acid residues. Three specific mutations responsible for the common red-green form of colorblindness, identified by using the single letter amino acid codes LIAVA, LIAVS, and LVAVA, involve the amino acids at positions 153, 171, 174, 178 and 180. These residues are particularly important for differentiating between M cone opsin and L cone opsin. Because the effect of different opsin mutations on the cell is unknown, a major research focus is to determine whether these mutations can be overcome to restore spectral sensitivity in colorblind people.

Specific Model Information:

- The alpha carbon backbone is colored grey.
- The alpha helices are colored dark magenta.
- The beta sheets are colored aquamarine.
- The hydrogen bonds are colored old lace.
- The amino acids involved in retinal binding (Glu113, Glu181, Tyr191, Met207, Phe261, Trp 265, Lys296, and Phe293) are displayed as spacefill and colored Dodger blue.
- Sites of mutations commonly associated with colorblindness (Ala153, Pro171 Gly174, Tyr178, and Pro180) are displayed in spacefill and colored yellow.
- Structural supports are colored white.

http://cbm.msoe.edu/smartTeams/index.php

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