Proceedings of the Joint Congress SIBV-SIGA
Milano, Italy – 8/11 September, 2015
ISBN **978-88-904570-5-0**

Poster Communication Abstract – C.15

ENGINEERING OF A LIGHT-GATED POTASSIUM CHANNEL

ALBERIO L.*, COSENTINO C.*, GAZZARRINI S.*, AQUILA M.*, ROMANO E., CERMENATI S.*, ZUCCOLINI P.*, PETERSEN J.**, BELTRAME M.*, VAN ETTEN J.L.***, CHRISTIE J.M.**, THIEL G.****, MORONI A.*

*) Department of Biosciences, University of Milan, Via Celoria 26, 20133 Milano (Italy)
**) Institute of Molecular Cell and Systems Biology, University of Glasgow (UK)
***) Department of Plant Pathology and Nebraska Center for Virology, University of Nebraska-Lincoln, NE (USA)
****) Membrane Biophysics, Technical University of Darmstadt, Darmstadt (Germany)

Viral K^+ channel Kcv, LOV domain, optogenetic

The present palette of opsin-based optogenetic tools lacks a light-gated potassium (K^+) channel desirable for silencing of excitable cells. Hence, we decided to engineer a genetically encoded light-activated K^+ channel by fusing the LOV (light oxygen voltage) domain of the plant blue-light receptor phototropin (Christie, 2007) to the viral K^+ channel Kcv (Plugge *et al,* 2000). The resulting chimeric channel BLINK1 (Blue Light INduced K^+ channel) is reversibly activated by blue light and maintains biophysical features of Kcv, including K^+ selectivity and high single channel conductance. Preliminary tests to verify in vivo applicability of this channel were performed on zebrafish embryos: ectopic expression of BLINK1 reversibly inhibits the escape response in light-exposed larvae. BLINK1 therefore provides a single-component optogenetic tool that can establish prolonged, physiological hyperpolarization of cells at low light intensities (Cosentino *et al*, 2015). We are currently optimizing cellular trafficking and surface expression of BLINK1 in several expression systems, including yeast, Sf9 and HEK 293 cells.