This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Ophthalmic disease				
Glaucoma; optic neuropathy	Gap junction protein δ 236 kDa (GJD2); CX36; connexin-36); gap junction protein γ 145 kDa (GJC1; CX45)	<i>In vitro</i> and mouse studies suggest inhibiting gap junction components CX36 or CX45 could help treat neurodegenerative ocular disorders, including ischemic retinopathy and glaucoma. In mouse retina eyecup explant cultures, gap junction blockers decreased NMDA-induced cell death compared with vehicle. In mouse retinas, the gap junction blockers decreased ischemia-induced cell death compared with vehicle. In mouse retinas, Cx36 knockout protected against NMDA-induced cell death and Cx45 knockout protected against ischemic cell death. Next steps include testing whether targeting gap junctions can protect retinal ganglion cells from injury in glaucoma.	Patent application filed; available for licensing	Akopian, A. <i>et al. J. Neurosci.</i> ; published online Aug. 6, 2014; doi:10.1523/JNEUROSCI.1912-14.2014 Contact: Stewart A. Bloomfield, State University of New York College of Optometry, New York, N.Y. e-mail: sbloomfield@sunyopt.edu
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