SUBMITTED VERSION

Robert J. Casson, Glyn Chidlow, John Wood

Comment on 'A method to quantify regional axonal transport blockade at the optic nerve head after short term intraocular pressure elevation in mice by A. Korneva et al.' (Exp. Eye Res. doi: https://doi.org/10.1016/j.exer.2020.108035) Experimental Eye Research, 2020; 197:108073-1

© 2020 Elsevier Ltd. All rights reserved.

Published at: http://dx.doi.org/10.1016/j.exer.2020.108073

PERMISSIONS

https://www.elsevier.com/about/policies/sharing

Preprint

- Authors can share their preprint anywhere at any time.
- If accepted for publication, we encourage authors to link from the preprint to their formal publication via its Digital Object Identifier (DOI). Millions of researchers have access to the formal publications on ScienceDirect, and so links will help your users to find, access, cite, and use the best available version.
- Authors can update their preprints on arXiv or RePEc with their accepted manuscript.

Please note:

- Some society-owned titles and journals that operate double-blind peer review have different preprint policies. Please check the journals Guide for Authors for further information
- Preprints should not be added to or enhanced in any way in order to appear more like, or to substitute for, the final versions of articles.

1 July 2021

http://hdl.handle.net/2440/130879

Comment on 'A method to quantify regional axonal transport blockade at the optic nerve head after short term intraocular pressure elevation in mice by A. Korneva et al. (Exp. Eye Res. doi: https://doi.org/10.1016/j.exer.2020.108035)

Dear Editor,

We read with interest the study by Korneva et al. (Korneva et al. 2020, Exp Eye Res, 108035), reporting the use of amyloid precursor protein (APP) as a marker of axonal transport blockade in experimental glaucoma. We acknowledge the quantification of the immunofluorescence as a novel methodology and the important pioneering work of Professor Quigley on axonal transport disruption in glaucoma. However, the implication that the use of APP as a marker of axonal transport blockade is a novel finding is not consistent with the evidence. Thirty years ago, Koo et al. reported that APP is a cargo for the fast anterograde transport system, (Koo et al., 1990, Proc Natl Acad Sci USA 87, 1561-1565) and in 1994, Blumbergs, et al. reported that APP accumulation was a marker of fast axonal transport disruption in concussion. (Blumbergs et al., 1994, Lancet 344, 1055-1056).

We first described accumulation of APP on the proximal side of an optic nerve infarct in a rodent vascular occlusion model. (Chidlow et al., 2010, Invest Ophthalmol Vis Sci 51, 1483-1497.) We then investigated APP as an endogenous marker to visualize and quantify anterograde fast axonal transport disruption at the optic nerve head (ONH) in a rodent model of glaucoma. (Chidlow et al., 2011, Acta Neuropathol 121, 737-751) Its spatial distribution was compared to that of the neural tracer, cholera toxin β -subunit, and described in relation to myelin basic protein in the post-laminar region. In subsequent studies, we demonstrated accumulation of interleukin-6 (Chidlow et al., 2012, Neurobiol Dis 48, 568-581) and mitogenactivated protein kinases (Mammone et al., 2018, Mol Cell Neurosci 88, 270-291) at the ONH in the rodent model of glaucoma, in each case using APP as the gold standard comparative marker. We are reassured that the utility of APP as a marker of anterograde fast axonal transport disruption has been validated by Korneva et al., but their results should be taken in historical context.

Robert J Casson Glyn Chidlow John Wood

Ophthalmic Research Laboratory, University of Adelaide