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 α B -crystallin (α BC) belongs to the family of small heat shock proteins that are necessary for maintaining oxygen homeostasis. This study was designed to explore the possible effects of α BC on N-methyl- N-nitrosourea (MNU) induced retinal degeneration and the underlying mechanisms.

METHODS: The α BC was injected into the vitreous bodies of MNU administered mice. The retinal morphology and visual function of experimental animals were analyzed by electroretinography (ERG), Spectral domain optical coherence tomography (SD-OCT), fundus photographs, optokinetic testing and immunohistochemistry assay.

Optokinetic behavioural tests and ERG examination suggested that the visual impairments of the MNU administered mice were ameliorated effectively by α BC treatment. OCT analysis showed that the major retinal architecture of the MNU administered mice was efficiently rescued by α BC treatment. Fundus examination suggested that the lesion size of the MNU administered mice was decreased by α BC treatment. MNU induced photoreceptor loss was also mitigated by α BC treatment as shown by hematoxylin and eosin staining. In particular, the immunostaining study suggested that M-cone photoreceptors, rather than the S-cone photoreceptors, were preferentially rescued, indicating that the photoreceptor populations have different sensitivities to α BC. The mechanism study suggested that the anti-apoptotic, anti-oxidative and neurotrophic function of α BC collectively contributed to these therapeutic effects.

Intravitreal delivery of α BC could alleviate MNU induced photoreceptor degeneration and visual impairment. Further refinement of the α BC induced protection would afford a novel therapeutic strategy for retinitis pigmentosa ¹⁾.

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