Retina is a critical component of the central nerve system that is responsible for the conversion of light stimulus into electrical spikes. Retinitis pigmentosa (RP) comprises a heterogeneous group of inherited retinal dystrophies leading to blindness. We examined retinal neuroglobin (Ngb) expression in a pharmacologically induced RP animal model, the N-Methyl-N-nitrosourea (MNU) administered mice. The retinal Ngb expression in MNU administered mice attenuated following a time dependent manner, suggesting Ngb was involved in the photoreceptor degeneration. Conversely, the intravenous delivery of Hemin, a Ngb up-regulator, enhanced the Ngb expressions in the retinas of MNU administered mice. Optokinetic behavioral tests and Electroretinogram (ERG) examination suggested that the Hemin treatment could improve the visual function of MNU administered mice. The retinal morphology of the Hemin treated group was much more intact than the MNU group as evidenced by retinal sections and optical coherence tomography (OCT) examinations. Moreover, immunostaining experiments showed the cone photoreceptors in the MNU administered mice were also rescued by Hemin treatment. Furthermore, mechanism studies suggested the Hemin treatment not only alleviated the oxidative stress, but also rectified the apoptotic changes in the retinas of MNU administered mice. In conclusion, the intraperitoneally delivery of Hemin can enhance the Ngb expressions in the MNU administered retinas, thereby ameliorating the photoreceptor degeneration and associated visual impairments. These findings would shed light on the opportunity to develop Ngb into a therapeutic molecular against RP [1)].

Tauroursodeoxycholic acid TUDCA plays an important role in protecting against cell death in certain retinal disorders, such as retinitis pigmentosa.

Retinal stimulation can restore limited visual perception to patients with retinitis pigmentosa, however loss of retinal ganglion cells precludes this approach.

Intravitreal delivery of αB -crystallin (αBC) could alleviate N-methyl- N-nitrosourea (MNU) induced photoreceptor degeneration and visual impairment. Further refinement of the αBC induced protection would afford a novel therapeutic strategy for retinitis pigmentosa [2)].

1)
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2)
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