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Oral Sessions

An apelin receptor agonist prevents retinal ganglion cell death in the diabetes model mouse fed the high-fat diet

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Diabetic retinopathy results in visual dysfunction through retinal ganglion cell (RGC) death. We have previously shown that APJ, an apelin receptor, is expressed in the RGCs and intravitreal injection of apelin protects against NMDA-induced RGC death. In the present study, we investigated whether systemic administration of an APJ agonist prevents RGC death in the retina of the diabetes model mouse fed the high fat diet (HFD). We used Insulin2 mutant (Ins2+/-) Akita mouse, which is a mouse model of type 1 diabetes. The Akita mouse was fed the HFD from 5 weeks after birth. The APJ agonist ML233 (5 mg/kg) was administered intraperitoneally on every other day for 4 weeks from 5 weeks after birth. The electro-responses of the RGCs were measured by electroretinography. ML233 suppressed the reduction of both the electro-response and the number of Brn-3a positive RGCs in the retina of the Akita mice fed HFD for 4 weeks. These effects were blocked by an APJ antagonist ML221 (5 mg/kg, i.p.). The present study showed that the APJ agonist protected against RGC death via APJ in the diabetes model mouse fed the HFD, suggesting that systemic administration of APJ agonists may prevent RGC degeneration in diabetic retinopathy.