Taurine deficiency can cause visual loss by photoreceptor and ganglion cell degeneration: Role in glaucoma and diabetic retinopathy

Taurine has become a well-known amino acid as a constituent of energy drinks. In the 70s, its deficiency was shown to cause photoreceptor loss in cats on a casein diet. Retinal alterations were also reported in non-human primate babies receiving taurine-free milk. In humans, taurine deficiency was found to induce functional retinal change during parenteral nutrition. Taurine addition is now added to artificial baby milk and parenteral nutrition. However, no taurine deficiency was so far associated to any retinal diseases with photoreceptor degeneration. Working on the retinal toxicity of the anti-epileptic drug, vigabatrin, we found that retinal ganglion cells (RGCs) also degenerate under taurine deficiency. Surprisingly, the retinal toxicity of vigabatrin is associated to a loss of RGCs and their optic fibers sending visual information to the brain. The relation to taurine deficiency was indicated by the decrease in plasma taurine for both vigabatrin-treated animals and patients. Neuroprotection of both photoreceptors and RGCs was obtained by taurine supplementation in vigabatrin-treated animals. These observations suggested that taurine is a major molecule for RGC survival. We have therefore investigated this hypothesis in models of glaucoma, the second cause of blindness worldwide. Purified RGCs survived better in the presence of taurine. One risk factor for triggering glaucoma is an increase in intraocular pressure. We therefore used two rodent glaucoma models with such an increase in intraocular pressure and showed that RGCs were preserved in animals receiving taurine in their water despite no effect on the increase in intraocular pressure. These studies indicate that taurine deficiency could contribute to diseases with RGC degeneration like glaucoma and diabetic retinopathy. This conclusion is further supported by the decrease in blood taurine in diabetic patients. Taurine supplementation may therefore become a potential treatment for diabetes and glaucoma.

Biography

Serge Picaud has expertise in Retinal Physiology and Physiopathology. He has characterized retinal cell function at the cellular and molecular levels while developing different cell, tissue and animal models of retinal diseases such as purified photoreceptors and purified RGCs or even human postmortem retinal explants. His team has contributed to the understanding of different mechanisms leading to photoreceptor and RGC death. The present study on taurine deficiency has emerged following fundamental studies on GABA retinal function because the antiepileptic drug, Vigabatrin, is a blocker of the GABA-transaminase. Recently, his interest on Translational Research has moved to the development of devices for restoring vision in blind patients.

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