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May Renalase Deficiency Catalyzing Catecholamines be One of the Reasons for Glaucoma?

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Editorial

Glaucoma (eye tension) is increasing of intraocular pressure which is caused by reasons such as anemia, long-lasting cortisone treatment, intraocular inflammation (uveitis), advanced age, diabetes, eye injuries, hypertension or hypotension, high myopia or hypermetropia and deficiency in discharging of eye fluid produced by eyes with balanced suction. Eye tension below 20-21 mmHg is deemed as normal. It is notified that glaucoma may occur even in the lower level of eye tensions. No matter the reason for the eye tension is, tension imbalance comes into question [1].

We want to hypothesize the possibility of a relationship between eye tension and renalase which might have a role in the regulation of eye pressure [2]. Recombinant human renalase application was reported to decrease arterial pressure in Sprague-Dawley rats [3]. In addition, plasma renalase levels are lower in hypertension patients (n=50) as against control group (n=50) in surgical repairment of aorta coarctation [3]. Possible renalase mechanisms causing a decrease of tension have not been clarified fully yet. However, it has been stated until the discovery of renalase that it regulates tension by catabolizing catecholamines (epinephrine, norepinephrine, and dopamine) [4]. The antihypertensive effect of renalase in circulation is attributed to FAD-dependent monoaminoxidase activity by some researchers. If the amount of renalase enzyme in the fluid produced by eyes is inadequate, it may cause eye tension as epinephrine, norepinephrine and dopamine cannot be removed by catabolization within an appropriate time unit. We think that revealing suggested mechanisms clinically and experimentally may help the elucidation of underlying mechanisms of eye tension.

Although renalase was detected to be secreted by kidney tissues, thereafter it was also reported to be secreted by various biological tissues including the heart tissue. It is not known yet for sure whether it is produced by eye tissue or not. Normal concentrations of renalase in circulation is reported as 3-5 µg/ml [5,6]. Amount of renalase in circulation may transport to eye fluid as it reaches to saturation level or it may be produced directly by the eye tissue and transferred to eye fluid or if

renalase is detected in eye fluid, the origin of it may be either blood or eye tissue. In the case that amount of renalase decreases in eye fluid, this decrease is predicted to relate to glaucoma. When we remember that there are various types of synaptic transmitters at retinae such as acetylcholine, dopamine, serotonin, GABA, glycine, substance P, somatostatin, TRH, GnRH, enkephaline, β-endorphine, CCK, VIP and neurotensin [7], it seems inevitable that there is a relationship between renalase amounts in eye fluid and glaucoma. We should remember that intracellular renalase contains N-terminal peptides whereas intercellular renalase does not contain N-terminal peptides. Researchers claim that absence of N-terminal peptide which is the structural prerequisite for FAD binding, the independent behavior of intercellular (circulatory) renalase from FAD and effect mechanism is not related to FAD [8]. No matter the effect mechanism of renalase is, there is a relationship between renalase amounts and blood pressure. Consequently, eye tension is probably related to eye tension which is measured over 21 mm Hg. In brief, all this data refers the necessity for revealing the relationship between eye tension and renalase. If the linkage between renalase and intraocular pressure is revealed, renalase can be a new laboratory parameter for detecting intraocular pressure and there may be a possibility to prevent vision loss by treating intraocular pressure without harming eye nerves.

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