Role of Adenosine Receptor A2A in Traumatic Optic Neuropathies

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PUBLIC ABSTRACT

Soldiers at war suffering from head injury may become blind because of the pressure of the skull damages the fragile nerve that connects the eyes and the brain. The pressure causes oxygen radicals to form in the retina to induce inflammation that may destroy the neurons and eventually the vision. As soon as head injury occurs, it is necessary to stop inflammation in order to protect vision.

During the search for effective treatments to protect the retinal neurons and vision, it is realized that in many diseases the oxygen radicals that cause inflammation also triggers a self-defense system that uses self-released adenosine to block inflammation. However, it is not clear if this system occurs in the injured eyes. Moreover, this system may lack sustained effectiveness due to the limited release of adenosine; this problem is compounded with time, since the tendency to accumulate a much larger amount of destructive oxygen radicals is increased. Scientists are now experimenting with drugs designed to prolong the activity of this system in other inflammatory diseases. Recently identified drugs that prolong a similar self-defense system in the diseased retina models may tell whether these drugs can be applied in the injured retina. It is hypothesized that drugs that enhance the adenosine self-defense system will protect retinal neurons. This hypothesis will be tested in cells and animal models. This study will explain how the drugs work and help to find more drugs that preserve vision in our soldiers.