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The retinal degeneration disease Stargardt macular dystrophy (STGD1) causes the loss of central vision during the first two decades of life. In cases of STGD1 it has been observed that circulating levels of the peptide inflammatory cytokines, tumor necrosis factor alpha (TNF $\alpha$ ) and interleukin-6 (IL-6), are elevated. Attempts to treat STGD1 by reducing the levels of circulating TNF $\alpha$  and IL-6 have been unsuccessful. Many studies have suggested that treating STGD1 could be accomplished by modulating levels of TNF $\alpha$  in the retina. The most robust methods of modulating TNF $\alpha$  levels in the retina are by using anti-TNF $\alpha$  neutralizing monoclonal antibodies and soluble TNF $\alpha$  receptors (TNF $\alpha$ RII). The therapeutic effects of anti-TNF $\alpha$  monoclonal antibodies in STGD1 are variable and not well-understood. However, small clinical trials of anti-TNF $\alpha$  and TNF $\alpha$ RII have shown promise. Experimental evidence suggests that TNF $\alpha$ RII may have an important role in the treatment of STGD1. The goal of this study is to determine whether TNF $\alpha$ RII is a major contributor to the efficacy of anti-TNF $\alpha$  in the treatment of STGD1. TNF $\alpha$  and IL-6 are important cytokines in diseases of the

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retina and brain. Their serum levels are increased in patients with many retinal diseases. They can contribute to disease by having deleterious effects on the retina. The effect of TNF $\alpha$  and IL-6 on the retina is dependent upon their interactions with their receptors, TNF $\alpha$ R and IL-6R. We hypothesize that the blockage of the interaction between TNF $\alpha$ R and IL-6R will prevent the actions of TNF $\alpha$  and IL-6 in the retina. t know it was there. There