Cataract is the leading cause of visual impairment worldwide, with an estimated 17 million people affected by bilateral cataract (Congdon et al, 2003). Cataract results from aggregation of the crystallin proteins in the fiber cells of the eye lens, leading to opacification. The aggregation is a result of oxidation; with age, disease, or genetic mutation, reactive oxygen species can desolubilize the crystallins (Truscott, 2005). The two most prevalent protective factors in the eye are the α-crystallins and glutathione. α-crystallin, one of the three forms of crystallins in the human lens, exhibits a chaperone-like activity; it is able to bind to the denatured β- and γ-crystallins to stabilize and resuspend them, but cannot truly renature the proteins, instead leaving them in a suspended soluble aggregate.

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Currently, the only approved treatment for cataract is surgical replacement of the cloudy lens with an artificial one. This is infeasible among people of lower socioeconomic status or in areas with poor healthcare. A non-surgical control measure would thus greatly reduce the prevalence of cataract. To find or create a drug suitable for this role, one must first elucidate the genetic functions leading to the onset of cataract. It is difficult to obtain lenses for experimentation, and evidence suggests that the etiology of human cataract is different from that in other, more easily obtainable animals (Truscott, 2005). Thus, using computational tools can provide significant insight and illuminate possible targets for further study.