Biochemistry & Physiology: Open Access
ISSN: 2168-9652

Abstract

Oxidative stress is one of the significant factors in the pathogenesis of several retinal diseases, viz. age-related macular degeneration, diabetic retinopathy, etc. Available treatments are not fully effective in attenuating tissue damage and the associated vision loss. Hence development of newer therapeutic compounds is highly desirable. We have previously demonstrated the effectiveness of metabolic and nutritional antioxidants such as pyruvate and caffeine in preventing oxidative damage to the lens. However, so far, studies investigating the protective effect of caffeine on the neural retina exposed to direct oxidative stress are lacking. The primary goal of this study was therefore to examine the efficacy of caffeine in preventing biochemical damage to the neural retina exposed to oxyradicals, in terms of maintaining the concentration of glutathione (GSH), a major endogenous antioxidant reserve. In vitro short-term tissue culture studies were conducted using freshly isolated neural retinas exposed to H_2O_2 in a medium with/without caffeine
neural retina has been shown for the first time. The results
investigating its other possible mechanisms of action, and

compounds that could be e ective in preventing oxidative damage to the retina, and can become potentially useful for therapeutic purpose.

(RPE) and Bruch's membrane visible ophthalmoscopically in the macula and also o en in the peripheral retina. is is called the dry AMD, which can eventually progress to formation of excessive drusen, degeneration of the overlying RPE, degeneration of the photoreceptors which depend on the RPE for their maintenance, and consequent damage to the neural retina, choroidal neovascularization with leaky blood vessels (wet AMD) and scarring of the retina. e associated vision loss is permanent and is usually progressive. e Age-related Eye Disease Study (AREDS) concluded that an antioxidant formulation consisting of ascorbate, beta-carotene, vitamin E, copper, zinc, lutein and zeaxanthin may have some role in retarding progression of dry AMD (atrophic AMD) [23]. However, even early intervention with this treatment has limited bene t which is seen only in a small percentage of patients. is could be attributable to the possible tendency of some of these antioxidants to become pro-oxidant, especially in the presence of trace metals, and also due to the involvement of other factors such as genetic predisposition and smoking in the pathogenesis of this disease. Hence there is a need for the development of newer antioxidants which