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Naringenin prevents diabetic retinopathy via inhibition of apoptosis, oxidative stress and inflammation through heme oxygenase-1 upregulation

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Despite the high prevalence of diabetic retinopathy, early interventions remain limited due to a lack of therapies targeting oxidative stress and inflammation simultaneously. This study investigates naringenin, a flavonoid with potential antioxidant and anti-inflammatory properties, as a novel therapeutic candidate targeting HO-1 upregulation to mitigate retinal damage. In this regard, a rat model of streptozocin-induced diabetic retinopathy was established and naringenin was administrated (40 mg/Kg/day and 80 mg/Kg/day). Two other untreated groups of diabetic and non-diabetic rats were used to compare with naringenin-treated groups. The findings revealed that naringenin decreased the overexpression of the pro-inflammatory factors IL-1β, IL-6, and TNF-α in the retinal tissue. Moreover, naringenin inhibited the overexpression of TLR4, NF-κB, and CASP-3, caused heme oxygenase-1 (HO-1) overexpression, upregulated BCL-2, reduced the levels of malondialdehyde, and elevated the levels of superoxide dismutase, catalase, and glutathione peroxidase (P-value$\leq$0.05). Intraperitoneal injection of the HO-1 inhibitor zinc protoporphyrin (ZnPP) blocked the protective effect of naringenin. These findings suggest that naringenin exerts therapeutic effects in diabetic retinopathy possibly by inducing HO-1 expression.

Keywords: Flavonoid, Retinal damage, TLR4, NF-κB, Streptozotocin, Signaling pathway