Effect of Ecdysterone on Dry Eye Syndrome In Vitro and In Vivo

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Dry eye syndrome (DES) is caused by a lack of tear of severe evaporation of tear, also induced by various imaging devices such as mobile phone and computers. Ecdysterone (20-Hydroxyecdysone) is a naturally ecdysteroid hormone that have insect metamorphosis, antioxidant activity and anti-inflammation. Recently, in addition, ecdysterone has received the focus of attention for the role in disease prevention and health promotion. However, the exact mechanism for treatment of DES by ecdysterone is still unexploited. In the present study, we investigated whether ecdysterone has the protective effect against DES in hyperosmolar stress-stimulated human conjunctival cells and the extra-orbital lacrimal gland excised rats. In vitro, hyperosmotic media have significantly increased oxidative stress. However, antioxidant enzyme, superoxide dismutase (SOD), and antioxidant proteins, heme oxygenase-1 (HO-1), catalase (CAT), and Nuclear factor-erythroid factor 2-related factor 2 (NRF2) was considerably recovered by treatment of ecdysterone at 50 and 100 μM. Additionally, inflammatory genes, interleukin-1 beta (IL-1β), tumor necrosis factor-alpha (TNF-α), IL-33 and Matrix metallopeptidase 9 (MMP9), and apoptotic protein, Bcl-2-associated X protein (BAX) and activated caspase 3 were significantly inhibited by treatment of ecdysterone. In vivo, eye drops of formulated ecdysterone (FES) (0.01 %, and 0.05%) to the extra-orbital lacrimal gland excised rats were remarkably restored on tear volume. Moreover, damaged corneal condition by lacrimal gland excision was significantly attenuated by FES at 0.01 % and 0.05%. In conjunctival tissue, reduced goblet cells by DES was highly increased by eye drops of FES through inhibition on mRNA expression of inflammatory genes, IL-1β, TNF-α, interferon-gamma (IFN-γ) and IL-6. Taken together, these results demonstrated that ecdysterone prevents dry eye syndrome in human conjunctival cells and the extra-orbital lacrimal gland excised rats. Our study also proposes the insight to the mechanisms of ecdysterone underlying the regulation of oxidation, inflammation and apoptosis.