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Ginkgo biloba extract protects human melanocytes from H_2 O_2 -induced oxidative stress by activating Nrf2.

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Abstract

Vitiligo is a common skin depigmenting disorder characterized by the loss of functional melanocytes. Its pathogenesis is complicated and oxidative stress plays a critical role in the development of vitiligo. Thus, antioxidant therapy is a promising therapeutic strategy to prevent or even reverse the progression of depigmentation. Ginkgo biloba extract EGb761 has been confirmed to have protective effects on neurons against oxidative stress. Notably, several clinical trials have shown that patients with stable vitiligo achieved repigmentation after taking EGb761. However, the exact mechanism underlying the protective effects of EGb761 on melanocytes against oxidative stress has not been fully elucidated. In the present study, we found that EGb761 effectively protected melanocytes against oxidative stress-induced apoptosis and alleviated the excessive accumulation of reactive oxygen species (ROS) and lipid peroxidation by enhancing the activity of antioxidative enzymes. Furthermore, the antioxidative effect of EGb761 was achieved by activating Nrf2 and its downstream antioxidative genes. In addition, interfering Nrf2 with siRNA abolished the protective effects of EGb761 on melanocytes against oxidative damage. In conclusion, our study proves that EGb761 could protect melanocytes from H₂ O₂ -induced oxidative stress by activating Nrf2. Therefore, EGb761 is supposed to be a potential therapeutic agent for vitiligo.

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KEYWORDS: EGb761; Melanocyte; Nrf2; Oxidative stress; Vitiligo

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