

Antioxidant activity and inhibitory effect of L. viridis extract on Fe2+-induced lipid peroxidation in brain homogenates

P Costa 1, S Gonçalves 1, A Romano 1

• ¹University of Algarve, Faculty of Sciences and Technology and Institute for Biotechnology and Bioengineering (IBB-CGB/UTAD), Campus de Gambelas, Ed. 8, 8005–139 Faro, Portugal

Congress Abstract

The brain is particularly susceptible to oxidative stress damaging effects due such events as the high consumption of oxygen, limited concentration of antioxidants and a relatively high degree of polyunsaturated fatty acids that are particularly good substrates for peroxidation reactions [1-3]. Oxidative stress could lead to damage biological target molecules, affecting the cellular function and integrity [4]. The ability of natural antioxidants, mainly phenolic compounds, to protect cells from oxidative stress has been previously demonstrated [5]. In this work, the methanol extract from Lavandula viridis L'Hér. (Lamiaceae), a xerophytic aromatic shrub endemic to the south-west Iberian Peninsula [6], was investigated for its effect on deoxyribose degradation, its reducing properties, Fe²⁺-chelating ability and total phenol content. The capacity of this extract to prevent Fe2+-induced lipid peroxidation in mouse brain (in vitro) was also evaluated. L. viridis extract showed Fe2+ chelating activity, reducing power and the ability to prevent Fe²⁺/H₂O₂-induced decomposition of deoxyribose in a dose-dependent manner. This extract also revealed a high phenol content (893.01±17.09µmol gallic acid equivalents/g extract) evaluated by Folin-Ciocalteu method. Moreover, in brain homogenates, the methanol extract of L. viridis caused a high decrease in the MDA production in both the basal and the Fe²⁺-induced lipid peroxidation. The effective protective properties of L. viridis could be attributed to its higher phenol content, Fe^{2*} chelating ability, reducing properties and HO· radical scavenging ability. The findings suggest that methanol extract from L. viridis could be a potential source of natural antioxidants.

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