A237 Evaluation of toxic and protective effects of an essential oil of *Salvia officinalis* L. on liver cells

C. Lima a, F. Carvalho b, E. Fernandes b, M. Bastos b, P. Santos-Gomes a, M. Fernandes-Ferreira a and C. Pereira a

a Departamento de Biologia, Centro de Ciências do Ambiente, Universidade do Minho, 4710-057 Braga, Portugal. b ICETA/CEQUP, Serviço de Toxicologia, Faculdade de Farmácia, Universidade do Porto, 4050-047 Porto, Portugal.

The widespread use of sage (*Salvia officinalis* L.) in herbal teas and as a food condiment requires that studies of their biological effects are conducted in order to prevent ill effects on human health. It is known that the essential oil (EO) of this plant is neurotoxic, but in higher concentrations than those used in the applications referred above.

In this study we have isolated and characterized the EO of *S. officinalis* and studied its toxic/protective effects in rat hepatocytes isolated by collagenase perfusion. The aims were to determine: 1. whether the use of the *S. officinalis* EO for human consumption has any adverse effects to the liver in the concentration range likely to be ingested; 2. verify the often attributed antioxidant effects (protective) on liver cells challenged with an oxidant agent (tertbutyl hydroperoxide tBHP) and compare it to the effects of the reference antioxidant quercetin.

The EO was obtained by hydrodistillation of fresh aerial parts of sage plants harvested in April 2002 in Arouca experimental farms in northern Portugal and then analyzed by GC and GC-MS. We obtained a total yield of 12.07 mg of EO per g of plant dry weight and more than 50 compounds were identified. The major representative compounds were α-thujone (17.36 %), α-humulene (13.25 %), 1,8-cineole (12.73 %), β-caryophyllene (8.50 %) and bornol (8.29 %). To study EO toxic/protective effects in rat hepatocytes, we measured the cell viability (LDH leakage), lipid peroxidation and glutathione status in experiments undertaken with cells (suspensions of 1x10^6 viable cells per millilitre) exposed to EO alone (toxicity of the EO; tBHP as a positive control); and with cells exposed to EO and an oxidative compound (tBHP) together (in EO protection evaluation; quercetin as a positive control) for 30 min. Our results show that the EO is not toxic when present at a concentration below 0.2%; only at 2 μl EO/ml cell suspension occurred a significant LDH leakage and GSH decrease indicating cell damage. The EO toxicity may be due to GSH depletion or to a solvent effect on the membrane. In the range of concentrations tested the EO did not show protective effects.