

## Protective effect of *Ginkgo biloba* extract against genotoxic stress

**D. Oliveira<sup>1</sup>, L. Cadilhe<sup>1</sup>, A. Mendes<sup>1</sup> P. Parpot<sup>2</sup>, R. Oliveira<sup>1</sup>**

<sup>1</sup>CITAB - Centre for the Research and Technology of Agro-Environmental and Biological Sciences; Department of Biology, University of Minho, Campus de Gualtar, 4710-057, Braga, Portugal

<sup>2</sup>Centre of Chemistry, University of Minho, Campus de Gualtar, 4710-057, Braga, Portugal  
\*[danielasoliveira@outlook.pt](mailto:danielasoliveira@outlook.pt), \*[luiscadilhe@hotmail.com](mailto:luiscadilhe@hotmail.com), \*[ruipso@gmail.com](mailto:ruipso@gmail.com)

The *Ginkgo biloba* leaf extract (GBE) is intensively studied and sold all over the world due to its many health benefits. Although the antioxidant properties of GBE are well documented, studies on its antigenotoxicity are still scarce.

Genotoxic damage can be induced by the chemical agents camptothecin (CPT) and sodium nitroprusside (SNP), producing clastogenic and oxidizing effects, respectively. However, medicinal plant extracts rich in antioxidants may prevent or protect cells from genotoxic damage.

The chemical analysis of GBE allowed the identification of flavonoids derivatives, which are known for its strong antioxidant activity. The protective effect of GBE was evaluated in spot assays against CPT (15 mM), using the parental strain 972h<sup>-</sup> of *Schizosaccharomyces pombe* and the derived mutants *chk1* and *rad51*, affected in the homologous recombination (HR) DNA repair pathway. CPT induced total loss of viability, but in co-incubation with GBE the viability of 972h<sup>-</sup> was not affected, *chk1* was deeply affected and *rad51* still showed total loss of viability. It is suggested that GBE may be stimulating DNA repair by HR, since *rad51*, which lacks an essential gene in HR, was not protected. The protective effect of GBE was also assessed against SNP (1 mM), using the parental strain FO656 and the derived mutants affected in the base excision repair pathway (BER) *mag1*, *apn2*, *nth1*, and in the HR *rhp55*. Cell cycle progression analysis revealed that GBE slightly reduces the delay caused by exposure to SNP. The evidence suggests that GBE protects cells from SNP through a DNA-repair independent mechanism, which may involve the scavenging of NO and subsequent decrease in DNA modifications. Therefore, GBE shows a potential antigenotoxic effect against oxidative and clastogenic damage, probably due to antioxidant properties provided by the flavonoid fraction of GBE which is suggested by the results of the in vitro antioxidant assays (GBE showed DPPH and NO scavenging activity).