COFFEE SILVERSKIN EXTRACT PROTECTS AGAINST BENZO (A) PYRENE INDUCED DNA DAMAGE

Amaia Iriondo-DeHond[1], Ana Haza[2], Alicia Ávalos[2], María Dolores del Castillo[1] and Paloma Morales[2]

CIAL INSTITUTO DE INVESTIGACIÓN EN CIENCIAS DE LA ALIMENTACIÓN | CSIC CONSEJO SUPERIOR DE INVESTIGACIONES CIENTÍFICAS

[1]Department of Food Analysis and Bioactivity, Institute of Food Science Research (CIAL, CSIC-UAM), 28049 Madrid, Spain;
[2] Department of Nutrition, Food Science and Food Technology, Faculty of Veterinary, Universidad Complutense de Madrid, 28040 Madrid, Spain

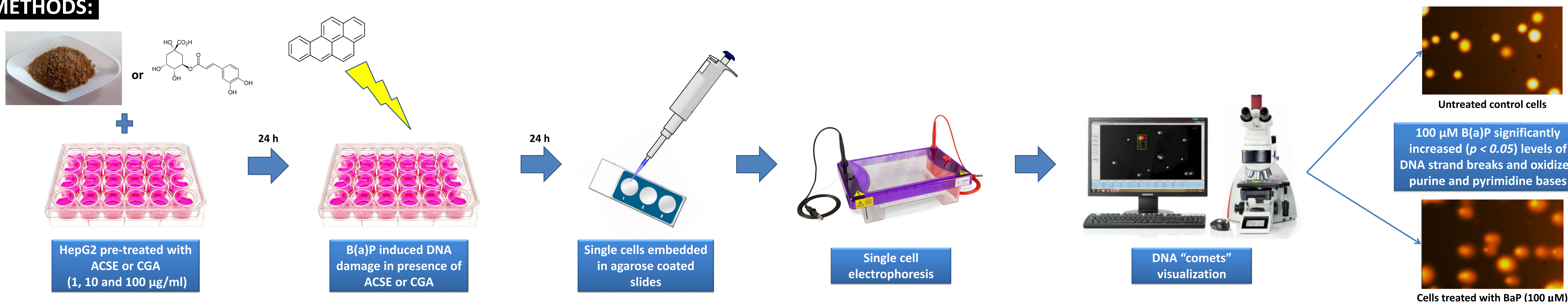
INTRODUCTION:

Benzo(a)pyrene (B(a)P) is a chemical carcinogen present in cigarette smoke and in thermally processed foods. B(a)P acts as a genotoxic carcinogen by forming DNA adducts. Several scientific reports have showed the usefulness of plant extracts in the prevention of B(a)P induced cancer in animals by various mechanisms including the prevention of DNA damage and the improvement of the antioxidant status. Coffee silverskin, the only by-product of coffee roasting, contains phytochemicals possessing antioxidant character such as chlorogenic acid (CGA). The extract (WO/2013/004873)[1] prepared from Arabica coffee silverskin (ACSE) is enriched in CGA and possesses high antioxidant power. ACSE may present chemoprotective potential against B(a)P preventing DNA damage.

AIM:

The aim of the present study was to investigate the chemoprotective potential of ACSE against B(a)P induced DNA damage in HepG2 cells and to find out the contribution of CGA in ACSE as a chemoprotective agent. Genotoxicity of ACSE and CGA in concentrations ranged from 1 to 100 µg/ml was previously confirmed.

METHODS:



RESULTS:

Strand breaks

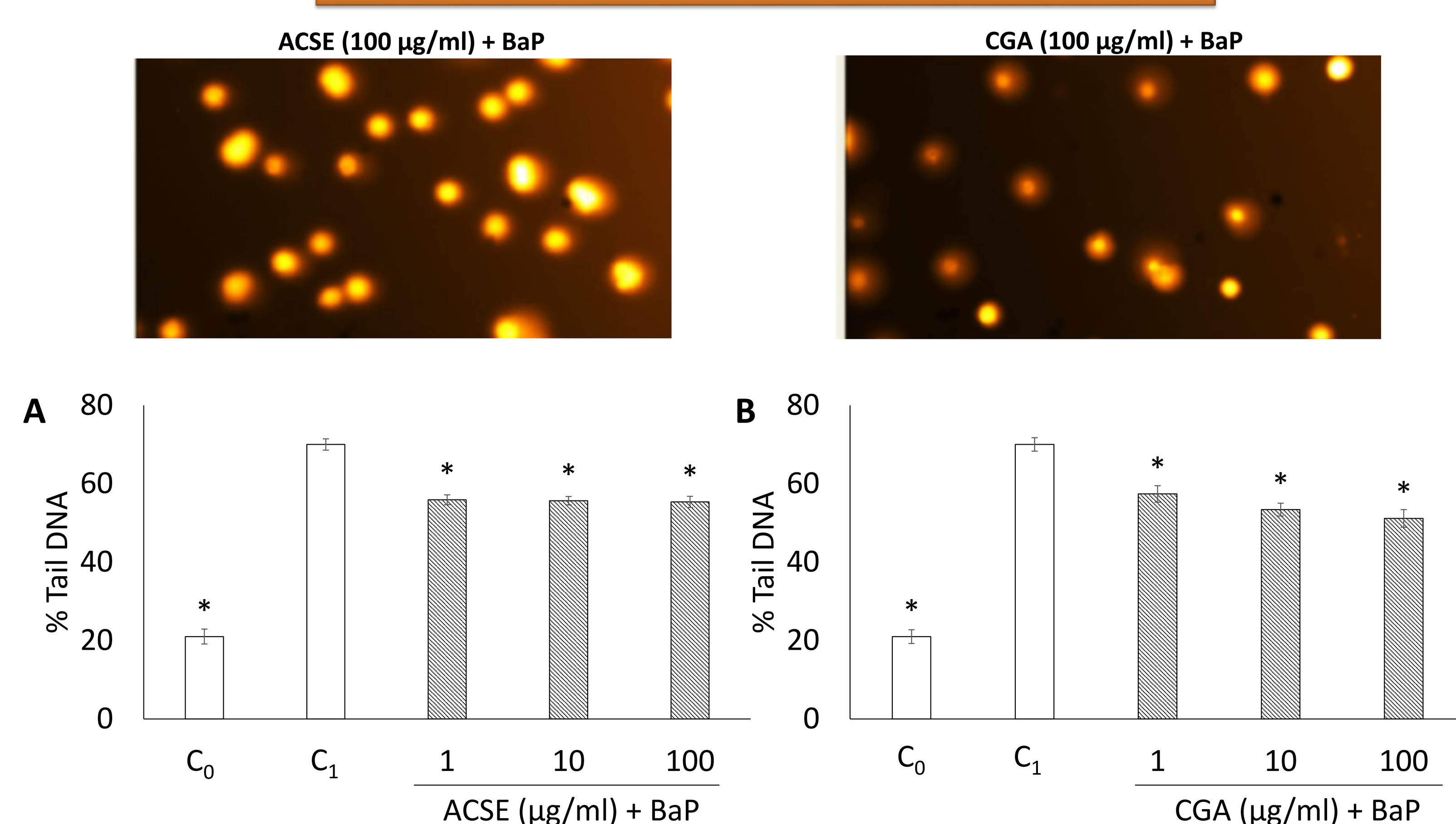


Figure 1. Effect of ACSE (A) or CGA (B) on BaP-induced DNA strand breaks in HepG2 cells. (C_0) Untreated cells without enzymes. (C_1) Cells treated with BaP (100 µM) and incubated without enzymes. (▨) Cells treated with BaP (100 µM) and ACSE or CGA (1, 10 and 100 µg/ml) and incubated without enzymes. Asterisks indicate significant difference from the control (C_1) (Tukey test, $p \leq 0.05$).

1. ACSE and CGA significantly inhibited strand breaks induced by B(a)P.
2. CGA alone at the concentration present in ACSE was effective against B(a)P.

Oxidation of DNA bases

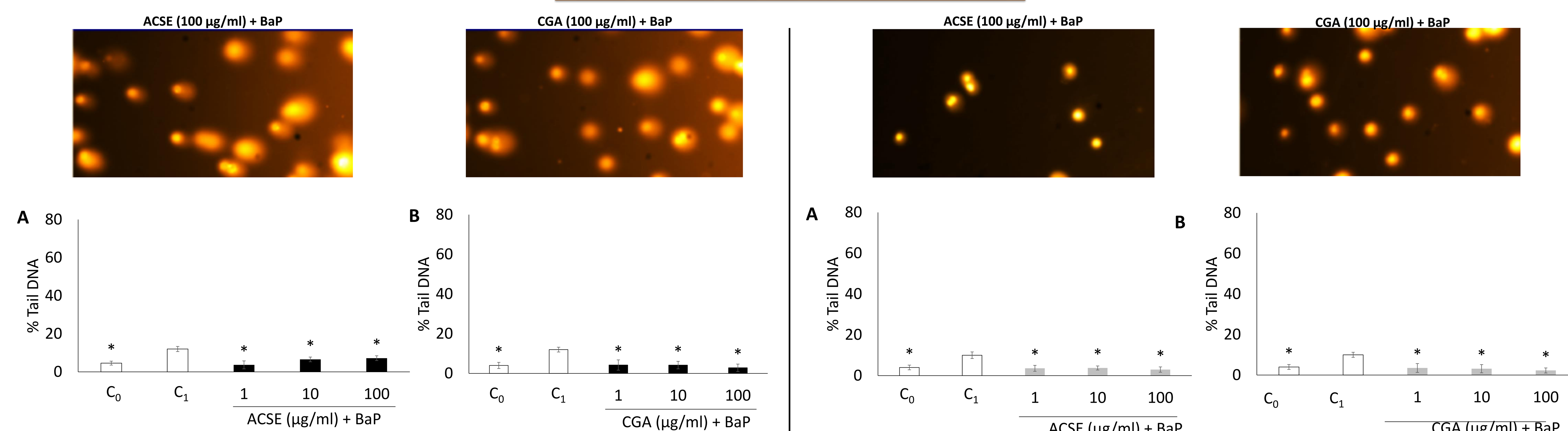


Figure 2. Effect of ACSE (A) or CGA (B) on BaP-induced oxidized purines in HepG2 cells. (C_0) Untreated cells with Fpg. (C_1) Cells treated with BaP (100 µM) and incubated with Fpg. (■) Cells treated with BaP (100 µM) and ACSE or CGA (1, 10 and 100 µg/ml) and incubated with Fpg. Asterisks indicate significant difference from the control (C_1) (Tukey test, $p \leq 0.05$).

Figure 3. Effect of ACSE (A) or CGA (B) on BaP-induced oxidized pyrimidines in HepG2 cells. (C_0) Untreated cells with Endo III. (C_1) Cells treated with BaP (100 µM) and incubated with Endo III. (▪) Cells treated with BaP (100 µM) and ACSE or CGA (1, 10 and 100 µg/ml) and incubated with Endo III. Asterisks indicate significant difference from the control (C_1) (Tukey test, $p \leq 0.05$).

1. ACSE and CGA chemoprotective effect may be associated to their antioxidant capacity.
2. CGA seems to be a contributor to the chemoprotective effect of ACSE against B(a)P induced DNA damage in HepG2 cells.

CONCLUSIONS:

ACSE presents potential as a natural and sustainable chemoprotective agent against the chemical carcinogen B(a)P being a promising candidate as functional food ingredient for cancer. Chemoprotection of cancer by dietary phytochemicals is a promising approach. Further investigation is needed to confirm these preliminary results.

References: [1] Del Castillo *et al.* (2013) WO2013/004873.
[2] Olive *et al.* (1992) *Exp. Cell. Res.* **198**(2):259-67

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