



Effect of coffee extracts submitted at different roasting levels on human prostate cancer cell line(DU-145)

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Keywords

Câncer, Coffee, roasting Coffee is an important source of compounds that may play bioactive functions in human body. However, these compounds can be transformed and degraded into other harmful substances, especially when exposed to the elevated temperature. This study aimed to observe the effect of coffee aqueous extracts (CAE) on and bioactive activity on human prostate cancer cells. Robusta coffee beans were selected, green beans (GB) were milled in an analytical grinder and the light (GL), medium (GM) and dark (GD) roasted beans were

roasted in a grain roaster (Gene Café®). CAE were lyophilized and stored until analysis. Antioxidant activity was measured by DPPH, FRAP and ABTS assays. Caffeine, sugars and asparagine were quantified by HPLC. Cell viability and apoptosis were performed using MTT method and flow cytometry, respectively, after 24h treatment with CAE (25-5000µg/mL). GB and GL extracts presented the highest antioxidant potential compared to GM and GD presenting values of 71.02 and 68.67% of reduction, 81.55 and 68.43 mmol of trolox/100g, 321.09 and 373.71 µmol of Fe²⁺/100g, in DPPH, ABTS, and FRAP assays, respectively. Only GB extract showed free sugars (11.56% of sucrose and 1.37% of fructose) as well as greater amount of asparagine. Lower amounts of (4.53 and 5.61) caffeine and (2.18 and 0.96) were observed in GM and GD, demonstrating that during roasting, these compounds were used in the formation of acrylamide, a high carcinogenic potential compound. CAE exerted in vitro cytotoxic effects on DU-145 cells with an IC₅₀ value of 2.14(GB), 5.20(GL), 7.57(GM) and 6.09(GD). All extracts promoted increase in apoptotic cells, being the highest increases observed with GB (6.71) and GL (12.32) extracts at the concentration of 5000µg/mL. Therefore, the data indicate that the compounds present GB and GL extracts had greatest potential to reduce cell proliferation and interfere in the mechanisms of death of tumor cells.