INVESTIGATION OF *IN VITRO* INHIBITION OF CYP1A1 MONOOXYGENASE BY PHENOLIC COMPOUNDS FROM COFFEE

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Dietary phenolic compounds can prevent chronic pathologies such as cancer and cardiovascular diseases, mostly due to their antioxidant and anti-inflammatory activities. Chlorogenic acids (CGA) are abundant phenolic compounds in coffee, one of the most appreciated drinks in the world. Despite the recent advancements in the knowledge of CGA metabolism, it is unclear the involvement of liver microsomal enzymes of the cytochrome P450 system, catalysts of phase I in the biotransformation of xenobiotics. The inhibitory effects of CGA and metabolites on the activities of ethoxyresorufin-O-deethylase (EROD), a selective marker for CYP1A1, were determined in a pool of hepatic microsomes from β-naphthoflavone-treated rats and from untreated rats. Besides α-naphthoflavone, a direct CYP1A inhibitor, other phenolic compounds like hesperetin and quercetin were used as positive controls. For the uninducible OK model, α-naphthoflavone 10 μM inhibited EROD activity by 38.2 % and, for the inducible OK model, by 78.6 %. The latter model clearly provided a better analysis of the inhibition of the specific isoform CYP1A1. As confirmed by the substances hesperetin and quercetin 100 μM, which inhibited EROD activity by 84.9 % and 93.7 % respectively. However, CGA (5-caffeoylquinic, dihydrocaffeic, ferulic, isoferulic, and p-coumaric acids) and metabolites (gallic and vanillic acids) 100 μM did not induce significant reduction of EROD activity in both models. This finding can indicate that CGA and the evaluated metabolites are not metabolized by CYP1A1.

Financial support: FAPERJ, CNPq