

Does Coffee Drinking Influence Plasma Antioxidant Capacity?

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SUMMARY

Absorption, metabolic fate and availability for antioxidant protection in humans of dietary plant constituents, such as flavonoids and related polyphenols, are still not fully explained. Moreover, the definition “plant phenols” includes thousand of compounds with different chemical structures corresponding to different antioxidant activities. As chemical structure is an important determinant in their bioavailability, the profile of phenolic compounds in plasma can be quite different from that of the original dietary source due to metabolism and bio-transformation.

Recently, a number of beverages derived from vegetables have been tested for their in vitro and in vivo antioxidant activity (white and red wine, green and black tea, beer). A straightforward analysis of these studies confirm that (i) the capacity of a food to transfer its antioxidant activity is linked to several known and unknown chemical/biochemical/physiological characteristics, (ii) the effect of food phenols on the redox balance in vivo can not be a simple extrapolation of its activity in vitro.

Coffee contains several phenolic components, other than tocopherols, endowed with antioxidant capacity: chlorogenic acids (esters of some cinnamic acids with quinic acid), and caffeic, ferulic, p-coumaric acids in free form. Black tea contains catechins and thearubigins and theaflavins, which are oxidation products of catechins formed during enzymatic oxidation by polyphenol oxidase in fresh tea leaves.

In this study we concentrated on the capacity of coffee to affect the plasma redox homeostasis in humans, using tea as positive control.

In two different sessions, a standard amount (200 ml) of brewed coffee or black tea was administered in fasting conditions to 10 healthy non-smoker moderate-coffee drinkers. Beverages were taken within 10 min from brewing. Plasma at time 0, and 1 and 2 hours after coffee/tea administration was analysed for: uric acid; alpha-tocopherol and glutathione (reduced and oxidised); total antioxidant capacity, by measuring the competition with the bleaching of two different target molecules (r-phycoerythrin and crocin) triggered by the peroxyl radicals generated by thermal decomposition of 2,2 azo-bis (2 aminopropane)-chlorohydrate (AAPH).

The ingestion of 200 ml of coffee in bolus produced a 5.5% increase ($P < 0.05$) in the plasma antioxidant capacity (by the r-phycoerythrin test) at $t=1$, maintaining a 4% increase after two hours. The 4.7% increase 1 hour after the tea administration did not reach statistical significance, as a consequence of different individual responses. As for coffee, the antioxidant capacity measured by the crocin test gave a similar trend in the modulation of antioxidant activity, even if the differences were not statistically significant. No effect was seen in the case of tea drinking using this test.

The explanation of the discrepancy between the two methods employed can be found in the capacity of some molecules to affect the plasma concentration of uric acid, coupled with a different sensibility to uric acid of the two methods. In fact, only tea drinking produces a significant increase of plasma uric acid, a component of the group of molecules with antioxidant activity contributing to the AC as measured by the r-phycoerythrin test, but not by the crocin test. The other parameters of antioxidant status do not change significantly, except for a significant increase of alpha-tocopherol 2 h after tea drinking.

From all that, we can advance the hypothesis that the increase of plasma antioxidant capacity (by crocin test) induced by coffee is due to antioxidants derived from coffee, while in the case of tea the (although not statistically significant) increase is due to the increase of uric acid.

At the moment we are not able to justify why tea drinking induces an increase of uric acid and coffee drinking not. However, phenolic composition and quantitative distribution in different phenolic classes can be responsible for this phenomenon.