

SHORT COMMUNICATION

---

# Coffee increases state anxiety in males but not in females

Paloma Botella and Andrés Parra\*

*Department of Psychobiology, Faculty of Psychology, University of Valencia, Valencia, Spain*

Coffee, reproducing the conditions under which caffeine is normally ingested, containing 3, 75, 150 or 300 mg of caffeine was given to healthy male and female volunteers. 25–30 min after drinking the beverage, they completed the Spanish version of the state-trait anxiety inventory (STAI). The beverage increased state anxiety, in a dose-dependent manner, in males but not in females. This could be due to a lesser sensitivity of females to coffee. Copyright © 2003 John Wiley & Sons, Ltd.

KEY WORDS — caffeine; STAI; mood; sex differences

## INTRODUCTION

Caffeine is perhaps the most consumed psychoactive drug in the world (D'Amicis and Viani, 1993). Some recent investigations suggest that giving coffee (or other caffeinated beverages) rather than pure caffeine to subjects is a more appropriate reproduction of the conditions under which caffeine is normally ingested (Hindmarch *et al.*, 1998, 2000). Results have often shown that caffeine increases alertness and attention (Smith *et al.*, 1999) but its effects on anxiety are under discussion.

In the present study, the effects of coffee on anxiety and cardiovascular functioning were measured. The caffeine content of the coffee given to participants was either 3, 75, 150 or 300 mg. These amounts fall within the range of caffeine amounts ingested by regular caffeine consumers. The results of the estimation of time intervals and the reaction time obtained in the same subjects were published separately (Botella *et al.*, 2001).

## METHOD

### *Participants*

Participants were healthy volunteer students at the University of Valencia, Spain; 39 males and 60 females, ranging in age from 18 to 31 years (mean  $\pm$  SD =  $22.6 \pm 2.9$  years). Subjects were not paid for participating. The inclusion criteria were: to be 18 years old or older; to be in good health as reported by the subjects themselves; and to have signed the informed consent form. The exclusion criteria were: being on any medication; having a history of mental disorders; having an irregular sleep pattern during the night before the experiment; and substance abuse. Moderate alcohol drinking and/or cigarette smoking were allowed. The mean daily caffeine intake for each subject was calculated according to Barone and Roberts (1996). As a result of this calculation, the placebo group of females appeared to consume more caffeine than their counterpart males [ $t_{(23)} = 3.12$ ;  $p < 0.01$ ], but this difference was not statistically significant in the remaining treatments. Other demographic characteristics for participants in this study can be seen in Table 1 of Botella *et al.* (2001).

### *Dose manipulation*

The beverage was prepared from espresso coffee kept frozen at  $-20^{\circ}\text{C}$  after boiling. The caffeine

---

\*Correspondence to: Dr A. Parra, Departamento de Psicobiología, Facultad de Psicología, Universidad de Valencia, Blasco Ibáñez 21, 46010 Valencia, Spain. Tel: +34 963 864 420.  
E-mail: andres.parra@uv.es

concentration of the coffee was determined with HPLC analysis. On the day of the test, the coffee was thawed and diluted with water to 75, 150 or 300 mg of caffeine in a volume of 100 ml. The placebo was decaffeinated coffee containing 3 mg of caffeine. The beverage was served in a disposable opaque glass with 10 g of sucrose at 3°–5°C.

### Procedure

Each subject arrived individually at the laboratory between 8:00 and 10:30 a.m. having had breakfast but no caffeine in the past 15 h. The groups were: placebo (decaffeinated coffee, P), 75, 150 or 300 mg of caffeine (C75, C150 and C300, respectively). All subjects were blind as to the caffeine content of their beverage until the end of their participation. Then 25–30 min after drinking the beverage, they completed the Spanish version of the state-trait anxiety inventory (STAI) (Spielberger *et al.*, 1970); and 30–40 min after drinking the beverage, blood pressure and heart rate were recorded. After all the tests were completed, and before knowing the actual dose of caffeine in the coffee ingested, the subjects were asked for their subjective level of increased alertness with none, low, medium or high as responses. Then the subjects were informed about the doses employed in the study and asked to guess which one they had been given.

### RESULTS

Males showed a higher state anxiety than females in the STAI test ( $F_{(1,91)} = 12.76$ ;  $p < 0.001$ ). This difference was due to the effect of the beverage because sex differences were not present in the placebo condition. The effect of the beverage was significant in males ( $F_{(3,35)} = 4.11$ ;  $p < 0.02$ ) but not in females ( $F_{(3,56)} = 0.32$ ;  $p = 0.81$ ). These and other state anxiety results are illustrated in Figure 1. As expected, the treatment was not statistically significant in the trait anxiety variable.

The effect of coffee on diastolic blood pressure was almost statistically significant ( $F_{(3,90)} = 2.63$ ;  $p < 0.06$ ). The effects of coffee on the remaining cardiovascular measures, i.e. systolic blood pressure and heart rate, were not statistically significant.

Positive correlations were found between the estimated and the actual dose ( $r = 0.34$ ;  $p < 0.001$ ), the actual dose and the subjective increased alertness ( $r = 0.40$ ;  $p < 0.0001$ ), and the subjective increased alertness and the estimated dose ( $r = 0.62$ ;  $p < 0.0001$ ).

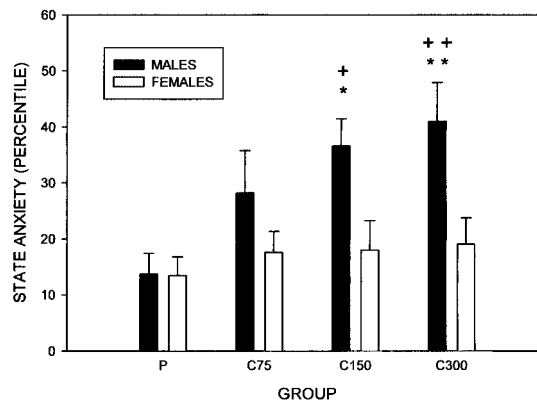


Figure 1. Percentile scores (mean ± SEM) in state anxiety (Spielberger *et al.*, 1970) of males and females who have had decaffeinated coffee (P) or caffeinated coffee containing 75, 150 or 300 mg of natural caffeine (C75, C150 and C300, respectively). \* $p < 0.03$ , \*\* $p < 0.02$  vs P male group, significances evaluated by Newman-Keuls posthoc tests. + $p < 0.03$ , ++ $p < 0.02$  vs females of the same group, significances evaluated by Student's *t*-tests,  $t_{(23)} = 2.45$ ,  $t_{(22)} = 2.72$ , respectively.

### DISCUSSION

The observed results in state anxiety, with males being dose-dependently sensitive to caffeinated coffee and with females showing no effect, seem to be well grounded. There are two reasons for this statement: first, the differences should not be attributed to differences in daily caffeine consumption because the reported ANOVAs were also performed as analyses of covariance, with daily caffeine intake as a covariate, and the results of such analyses showed no relevant discrepancy from the ANOVAs; second, the differences should not be attributed to differences in the availability of caffeine in the brain because the amounts of caffeine measured in saliva were almost identical in males and females (see Botella *et al.*, 2001). We think that the observed differences are due to differences in the sensitivity to caffeine or other substances contained in coffee. The mechanism of action of caffeine is not well known but it is assumed that it leads to an augmentation of dopamine neurotransmission (Fuxe *et al.*, 1998), a transmitter system in which females show a higher activity than males (Kaasinen *et al.*, 2001). On the other hand, several sources of evidence suggest that oestrogens have a neuroprotective role for dopamine functioning (for a review see Dluzen, 2000). It could be the case that the level of dopamine activity and the neuroprotective effects of oestrogens in females make their dopamine system less sensitive to changes induced by coffee.

The literature on the effects of caffeine on cardiovascular measures show no consistent results (Nurminen *et al.*, 1999). The present results, i.e. little or no effects, are in agreement with many of them.

Also, the results clearly show that the subjects were good estimators of the strength of the ingested coffee, i.e. the relative amount of caffeine in it, by simply looking at how much increased alertness they felt.

## ACKNOWLEDGEMENTS

We are very grateful to Amy Sevcik for revising the English of the manuscript.

## REFERENCES

- Barone JJ, Roberts HR. 1996. Caffeine consumption. *Food Chem Toxicol* **34**: 119–129.
- Botella P, Bosch F, Romero FJ, Parra A. 2001. Sex differences in estimation of time intervals and in reaction time are removed by moderate but not high doses of caffeine in coffee. *Hum Psychopharmacol Clin Exp* **16**: 533–540.
- D'Amicis A, Viani R. 1993. The consumption of coffee. In *Caffeine, Coffee and Health*, Garattini S (ed.). Raven Press: New York; 1–16.
- Dluzen DE. 2000. Neuroprotective effects of estrogen upon the nigrostriatal dopaminergic system. *J Neurocytol* **29**: 387–399.
- Fuxe K, Ferre S, Zoli M, Agnati LF. 1998. Integrated events in central dopamine transmission as analyzed at multiple levels. Evidence for intermembrane adenosine A<sub>2A</sub>/dopamine D<sub>2</sub> and adenosine A<sub>1</sub>/dopamine D<sub>1</sub> receptor interactions in the basal ganglia. *Brain Res Brain Res Rev* **26**: 258–273.
- Hindmarch I, Quinlan PT, Moore KL, Parkin C. 1998. The effects of black tea and other beverages on aspects of cognition and psychomotor performance. *Psychopharmacology (Berl)* **139**: 230–238.
- Hindmarch I, Rigney U, Stanley N, Quinlan P, Rycroft J, Lane J. 2000. A naturalistic investigation of the effects of day-long consumption of tea, coffee and water on alertness, sleep onset and sleep quality. *Psychopharmacology (Berl)* **149**: 203–216.
- Kaasinen V, Nagren K, Hietala J, Farde L, Rinne JO. 2001. Sex differences in extrastriatal dopamine d(2)-like receptors in the human brain. *Am J Psychiatry* **158**: 308–311.
- Nurminen ML, Niittynen L, Korpela R, Vapaatalo H. 1999. Coffee, caffeine and blood pressure: a critical review. *Eur J Clin Nutr* **53**: 831–839.
- Smith A, Sturgess W, Gallagher J. 1999. Effects of a low dose of caffeine given in different drinks on mood and performance. *Hum Psychopharmacol Clin Exp* **14**: 473–482.
- Spielberger CD, Gorsuch RL, Lushene RE. 1970. *Manual for the State-Trait Anxiety Inventory*. Consulting Psychologist Press: Palo Alto.