Technical Note

Caffeine Content of Decaffeinated Coffee

Rachel R. McCusker[1], Brian Fuehrlein[2], Bruce A. Goldberger[1,3,*], Mark S. Gold[3], and Edward J. Cone[4]

[1]Department of Pathology, Immunology and Laboratory Medicine, [2]Department of Biomedical Engineering, [3]Department of Psychiatry, University of Florida College of Medicine, Gainesville, Florida and [4]ConeChem Research, LLC, 441 Fairtree Drive, Severna Park, Maryland 21146

Abstract

Caffeine is the most widely consumed drug in the world with coffee representing a major source of intake. Despite widespread availability, various medical conditions necessitate caffeine-restricted diets. Patients on certain prescription medications are advised to discontinue caffeine intake. Such admonition has implications for certain psychiatric patients because of pharmacokinetic interactions between caffeine and certain anti-anxiety drugs. In an effort to abstain from caffeine, patients may substitute decaffeinated for caffeinated coffee. However, decaffeinated beverages are known to contain caffeine in varying amounts. The present study determined the caffeine content in a variety of decaffeinated coffee drinks. In phase 1 of the study, 10 decaffeinated samples were collected from different coffee establishments. In phase 2 of the study, Starbucks® espresso decaffeinated ($N = 6$) and Starbucks brewed decaffeinated coffee ($N = 6$) samples were collected from the same outlet to evaluate variability of caffeine content of the same drink. The 10 decaffeinated coffee samples from different outlets contained caffeine in the range of 0–13.9 mg/16-oz serving. The caffeine content for the Starbucks espresso and the Starbucks brewed samples collected from the same outlet were 3.0–15.8 mg/shot and 12.0–13.4 mg/16-oz serving, respectively. Patients vulnerable to caffeine effects should be advised that caffeine may be present in coffees purported to be decaffeinated. Further research is warranted on the potential deleterious effects of consumption of "decaffeinated" coffee that contains caffeine on caffeine-restricted patients. Additionally, further exploration is merited for the possible physical dependence potential of low doses of caffeine such as those concentrations found in decaffeinated coffee.

Introduction

Caffeine (1,3,7-trimethylxanthine) is the most widely consumed psychostimulant in the world. Its physiological effects include diuresis, central nervous system stimulation, coronary vessel dilation, gastric acid secretion stimulation, and free fatty acids and glucose elevation (1). Caffeine containing beverages are popular, in part, due to decreased fatigue, increased mental acuity and improved cognitive functioning following the intake of moderate doses (2). Despite these desirable effects, various medical conditions including hypertension and arrhythmias call for health care professionals to recommend caffeine-free diets. Additionally, patients on certain prescription medications are also advised to discontinue their caffeine intake. The U.S. Food and Drug Administration has suggested the avoidance of the concomitant administration of caffeine with bronchodilators, anti-anxiety drugs, and quinolones (3). In those patients with autosomal dominant polycystic kidney disease, caffeine is a risk factor for the promotion of cyst enlargement. For this reason, the Polycystic Kidney Foundation recommends that these patients eliminate the use of caffeinated substances (4). In an effort to abstain from caffeine for the previously mentioned health concerns, many people substitute decaffeinated for caffeinated coffee, sometimes unaware that these beverages contain caffeine. In the present study, the caffeine content of decaffeinated coffee beverages was determined for beverages collected from a variety of coffee establishments.

Methods

Twenty-two decaffeinated coffee beverages were purchased and evaluated for caffeine content. In phase 1 of the study, six brewed decaffeinated coffee beverages (D1–D6) were purchased from various coffee shops in Severna Park and Bethesda, MD. In addition, four brewed decaffeinated beverages (D13–D16) were purchased from various restaurants in Gainesville, FL. In phase 2 of the study, six decaffeinated espresso coffee beverages (E1–E6) brewed from the same batch and six brewed decaffeinated coffee beverages (D7–D12) from the same batch were purchased from the same Starbucks coffee shop in Gainesville, FL on Day 1 and Day 2, respectively. Caffeine was quantitated in the coffee beverages utilizing a gas chromatographic technique previously reported (5). Quantitation of caffeine was based on a calibration curve prepared in a concentration range of 10–100 mg/L, with the limit of quantitation arbitrarily set at the concentration of the lowest standard.

* Author to whom correspondence should be addressed: Bruce A. Goldberger, Ph.D., Department of Pathology, Immunology and Laboratory Medicine, University of Florida College of Medicine, P.O. Box 100275, Gainesville, FL 32610-0275. E-mail: bruce-goldberger@ufl.edu.

Results

The results of the caffeine analyses of the various decaffeinated coffee samples purchased from various coffee shops and eating establishments (phase 1) are shown in Table I. The store, brand, and the country of origin, if known, along with the measured caffeine dose (mg) based on a 16-oz serving size are also listed. In phase 1 of the study, the 10 decaffeinated coffee samples had a caffeine concentration in the range of 0–13.9 mg per 16-oz serving.

The results of the caffeine analyses of the Starbucks espresso decaffeinated coffee samples purchased on Day 1 from the same outlet (phase 2) appear in Table II. The caffeine concentration of these specialty drinks are in the range of 3.0–15.8 mg per shot (1-oz). The intra-assay mean ($N = 6$), standard deviation, and % C.V. were 7.0 mg/serving, 5.7 and 81.5, respectively. The results of the caffeine analyses of the Starbucks brewed decaffeinated coffee purchased on Day 2 from the same outlet (also phase 2) appear in Table III. The caffeine concentrations of these drinks were in the range of 12.0–13.4 mg per 16-oz serving. The intra-assay mean ($N = 6$), standard deviation, and % C.V. were 12.9 mg/serving, 0.6, and 4.4, respectively.

Table I. Phase 1—Decaffeinated Coffee Samples

Sample #	Store	Type/Brand	Caffeine Dose (mg/16 oz)
D1	The Big Bean™	Brewed, blended	10.1
D2	The Big Bean	Brewed, Italian Roast, country origin, Columbia	10.6
D3	Starbucks	Brewed	8.6
D4	Royal Farms®	Brewed	8.6
D5	Dunkin' Donuts®	Brewed	10.1
D6	Hampden Café	Brewed, Antigua, Guatemala	10.6
D13	Krispy Kreme Doughnuts®	Brewed	13.9
D14	Krystal®	Folgers® Instant	none detected
D15	Gainesville Doughnuts	Brewed	10.1
D16	McDonald's®	Brewed	11.5

Table II. Phase 2—Starbucks Espresso Decaffeinated

Sample #	Caffeine Concentration (mg/shot)
E1	15.8
E2	3.3
E3	4.1
E4	3.0
E5	12.7
E6	3.2

Table III. Phase 2—Starbucks Brewed Decaffeinated

Sample #	Caffeine Concentration (mg/16 oz)
D7	12.0
D8	12.5
D9	13.0
D10	13.4
D11	13.4
D12	13.0

Discussion

The caffeine content of decaffeinated coffee obtained from different establishments was variable ranging from none detected to 13.9 mg per 16-oz serving. The six espresso decaffeinated samples demonstrated considerable variability ranging from 3.0 to 15.8 mg of caffeine per shot. The variability in the espresso beverage may more accurately be attributed to human manipulation involved in the production of the espresso extraction. In comparison, an earlier study found a caffeine concentration range of 18–48 mg/12-oz serving in a variety of popular caffeinated carbonated sodas (6). Further, in another study the average caffeine content of brewed caffeinated specialty coffees was found to be 188 mg/16-oz serving (5).

The finding that decaffeinated coffee contains caffeine has far-reaching clinical consequences. Clinicians and patients should be aware that decaffeinated coffee frequently contains caffeine. Ingestion of multiple servings of decaffeinated beverages could result in caffeine doses equivalent to a caffeinated beverage. In addition, one must be mindful of the potential for pharmacological interactions that exist between caffeine and prescription medications (7).

Caffeine fits the criteria for physical dependence potential. The literature lends support to the notion that caffeine exhibits reinforcing effects. Even low doses of caffeine have been found to exhibit these effects which are demonstrated by self-administration greater than that of a placebo. One double-blind study enlisting heavy coffee drinkers found evidence for the reinforcement of caffeine with doses as low as 25 mg/cup being consumed at a slightly higher rate than decaffeinated coffee containing 2 mg/cup (8).

One study reported the reinforcing effects of decaffeinated coffee, finding higher levels of self-administration of decaffeinated prepared capsules than placebo capsules (9). One possible explanation for a steady consumption of decaffeinated coffee might be due to the reinforcing effects of the low doses of caffeine present in decaffeinated coffee, concentrations comparable to those found in the current study.

Further evidence for the reinforcing effects of low doses of caffeine was found in another study among moderate caffeine consumers. More than half of the subjects discriminated 18 mg of caffeine, and one discriminated 10 mg of caffeine. All subjects based their discrimination on changes in mood, such as alertness, well-being, motivation, concentration, and energy (10). Another double-blind study found that coffee containing 25 mg of caffeine was repeatedly self-administered in two of the

six moderate coffee drinkers. The authors suggested that these reinforcing effects were not the result of a single dose, but a series of several caffeine doses (11).

One study also explored the relationship of caffeine tolerance to the reinforcing effects of caffeine. Subjects who had previously consumed caffeinated coffee for an average of 10 days were given the choice between caffeinated and decaffeinated coffee, all preferred the caffeinated, stating that it was more stimulating. These subjects also complained that the decaffeinated provided low stimulation. In contrast, another group was subjected to a decaffeinated background condition (i.e., subjects consumed decaffeinated coffee for one week or more before being given a choice) and upon given a choice, all found the decaffeinated to be satisfactory. However, when the decaffeinated group was given caffeinated beverages, all complained about the high stimulatory effects (12). It may be plausible based on this study that caffeine served as a reinforcer only for caffeine-tolerant subjects.

Another study evaluated the effects of substituting various doses of caffeine or placebo for a 300 mg/day maintenance dose. It was found that substituting lower doses of caffeine or placebo resulted in an increase in withdrawal symptoms, such as drowsiness, headache, impaired concentration and decreased sociability. When caffeine doses in the range of 25–100 mg/day were substituted for the 300 mg/day maintenance dose, such withdrawal symptoms were reported as mild. Ratings of severe withdrawal with the main symptom being headache were significantly increased only when placebo was substituted. Thus, it may be concluded that caffeine physical dependence can occur with lower caffeine doses than previously thought (13).

Although it may be concluded that caffeine is responsible for the reinforcing effects as seen in the previously mentioned studies, one study found the total amount of decaffeinated coffee and caffeinated coffee consumed did not differ greatly (8). Such evidence has led to the proposal that other substances in decaffeinated coffee might be responsible for its reinforcing effects. One explanation for such effects suggests that the decaffeinated coffee self-administration is due to the presence of one or more chemical components found in both decaffeinated and caffeinated coffee that exhibit opiate receptor binding activity (14).

As reflected by the data collected in the present study, low doses of caffeine are present in coffees purported to be decaffeinated. Therefore, substitution of decaffeinated coffee for caffeinated in an effort to eliminate caffeine consumption may not be effective for patients on a caffeine-restricted or abstinent diet. Further, it is possible that consumption of low doses of caffeine such as those found in decaffeinated coffee may demonstrate physical dependence through its reinforcing effects and avoidance of withdrawal symptoms. Consumption of multiple servings throughout the day of decaffeinated coffee with an average caffeine concentration as found in the current study may achieve concentrations supporting the physical dependence potential of caffeine. On the other hand, the steady consumption of decaffeinated coffee may be attributed merely to its pleasing taste or the desire for the ingestion of a warm beverage.

References

1. D.M. Graham. Caffeine—its identity, dietary sources, intake and biological effects. *Nutr. Rev.* **36:** 97–102 (1978).
2. G. Tanda and S.R. Goldberg. Alteration of the behavioral effects of nicotine by chronic caffeine exposure. *Pharmacol. Biochem. Behav.* **66:** 47–64 (2000).
3. FDA/National Consumers League-Pamphlet on Food and Drug Interactions, 1998; http://vm.cfsan.fda.gov/~lrd/fdinter.html (accessed July 2004).
4. F.A. Belibi, D.P. Wallace, T. Yamaguchi, M. Christensen, G. Reif, and J.J. Grantham. The effect of caffeine on renal epithelial cells from patients with autosomal dominant polycystic kidney disease. *J. Am. Soc. Nephrol.* **13:** 2723–2729 (2002).
5. R.R. McCusker, B.A. Goldberger, and E.J. Cone. Caffeine content of specialty coffees. *J. Anal. Toxicol.* **27:** 520–522 (2003).
6. R.R. McCusker, B.A. Goldberger, and E.J. Cone. Caffeine content of energy drinks, carbonated sodas, and other beverages. *J. Anal. Toxicol.* **30:** 112–114 (2006).
7. J.A. Carrillo and J. Benitez. Clinically significant pharmacokinetic interactions between dietary caffeine and medications. *Clin. Pharmacokinet.* **39:** 127–153 (2000).
8. R.R. Griffiths, G.E. Bigelow, I.A. Liebson, M. O'Keefe, D. O'Leary, and N. Russ. Human coffee drinking: manipulation of concentration and caffeine dose. *J. Exp. Anal. Behav.* **45:** 133–148 (1986).
9. R.R. Griffiths, G.E. Bigelow, and I.A. Liebson. Reinforcing effects of caffeine in coffee and capsules. *J. Exp. Anal. Behav.* **52:** 127–140 (1989).
10. R.R. Griffiths, S.M. Evans, S.J. Heishman, K.L. Preston, C.A. Sannerud, B. Wolf, and P.P. Woodson. Low-dose caffeine discrimination in humans. *J. Pharmacol. Exp. Ther.* **252:** 970–978 (1990).
11. J.R. Hughes, W.K. Hunt, S.T. Higgins, W.K. Bickel, J.W. Fenwick, and S.L. Pepper. Effect of dose on the ability of caffeine to serve as a reinforcer in humans. *Behav. Pharmacol.* **3:** 211–218 (1992).
12. R.R. Griffiths, G.E. Bigelow, and I.A. Liebson. Human coffee drinking: reinforcing and physical dependence producing effects of caffeine. *J. Pharmacol. Exp. Ther.* **239:** 416–425 (1986).
13. S.M. Evans and R.R. Griffiths. Caffeine withdrawal: a parametric analysis of caffeine dosing conditions. *J. Pharmacol. Exp. Ther.* **289:** 285–294 (1999).
14. J.H. Boublik, M.J. Quinn, J.A. Clements, A.C. Herington, K.N. Wynne, and J.W. Funder. Coffee contains potent opiate receptor binding activity. *Nature* **301:** 246–248 (1983).