

## Effects of Caffeine on Anxiety and Depression

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Normal persons were administered the Multiple Affect Adjective Checklist both before and one hour after double-blind administrations of 0 mg ( $n = 42$ ), 150 mg ( $n = 52$ ), or 300 mg ( $n = 63$ ) of caffeine per 45.36 kg of body weight and after controlling for caffeine tolerance. Caffeine was found to increase anxiety, depression, and hostility. Findings are related to previous literature, and implications for future research are discussed.

The perspective gained from the previous literature regarding the effects of caffeine on anxiety and mood is not a clear one. Most of the reports concerning caffeine are based on clinical impression. In two of the correlational studies, the relationship between caffeine consumption and anxiety is positive (Greden, Fontaine, Lubetsky, & Chamberlin, 1978; Winstead, 1976), and in two it is negative (Hire, 1978; Lynn, 1973). The two experimental studies did not control for body weight and usual consumption (DeFreitas & Schwartz, 1979; Goldstein, Warren, & Kaizer, 1965). Only two studies have related caffeine consumption to mood. One was a correlational study with psychiatric patients, which showed an inverse relationship (Greden et al., 1978), and the other was an experimental study that demonstrated no effects but did not control for body weight or usual consumption (Goldstein et al., 1965). Furthermore, there is evidence to suggest that the effect of caffeine in psychiatric patients could be an indirect one through the interference with antipsychotic drug effect (Kulhanek, Linde, & Meisenberg, 1979). The experimental research described here was double blind and controlled for body weight and usual consumption, and used three dosage levels.

### Method

#### Subjects

Subjects were nonpaid volunteers who were solicited from various colleges and businesses in the San Joaquin Valley of California. They were told that the experiment concerned the psychological effects of coffee. Two hundred and thirty-eight persons agreed to participate in the study, and 171 actually participated. Of these 171 subjects, 5 did not complete all of the experimental tasks and were

eliminated from the final subject pool. Dose levels were initially set at 0 mg, 200 mg, or 400 mg of caffeine per 45.36 kg of body weight. These levels were reduced after verbal reports from subjects who were administered the high caffeine dose indicated stomach distress. The 9 subjects who were administered these initial high dose levels were also eliminated from the final subject pool. A total of 157 subjects were included in the final statistical analysis. There were 64 males and 93 females with a mean age of 24.88 years and a standard deviation of 8.20 years. Of these subjects, 26 males and 34 females were students and 38 males and 59 females were from businesses.

#### Procedure

The subjects were twice administered the Multiple Affect Adjective Checklist (MAAC; Zuckerman, Lubin, Vogel & Valerious, 1964), which has three scales assessing current anxiety, depression, and hostility. In addition, they completed an information sheet that assessed age, sex, body weight, and average daily caffeine consumption, as determined by a check list of beverages and drugs that contained caffeine. The above were group administrations.

Participants were each notified of their assigned number, the time and place of the experimental session, and the general nature of the experimental design, including a request not to ingest anything prior to the morning session. To control for possible tolerance to caffeine, average daily caffeine consumption was computed for each subject from their respective information sheets. The following (caffeine per cup) estimates were used: brewed coffee, 125 mg; instant coffee, 92.5 mg; tea, 67.5 mg; decaffeinated coffee, 3 mg; herbal tea, 0 mg; and caffeinated soft drinks, 50 mg of caffeine per can or bottle. The dosages were averaged from the *Handbook of Nonprescription Drugs*, 5th ed (Kleinfeld, 1977), as was the caffeine content of over-the-counter drugs. The caffeine content of prescription medi-

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cation was obtained from the *Physicians' Desk Reference* (Medical Economics Company, 1980).

Fifty-four subjects were assigned to the low-consumption group (0–249 mg caffeine daily), 65 to the medium-consumption group (250–499 mg caffeine daily), and 38 to the high-consumption group (over 500 mg caffeine daily). This assignment is consistent with that of past caffeine research (Greden et al., 1978; Winstead, 1976). Within each of these groups, subjects were randomly assigned to one of the three dosages so that 14 low-consumption, 17 medium-consumption, and 11 high-consumption subjects received 0 mg of caffeine per 45.36 kg of body weight. The respective numbers of subjects were 20, 21, and 11 for the 150-mg dosage and 20, 27, and 16 for the 300-mg dosage. The unequal number of subjects in the three dosage groups resulted from subject dropout after initial randomization was determined. A private laboratory (Twining Laboratories, Fresno, California) was employed to randomly assign subjects to one of the experimental conditions and to measure the caffeine (U.S.P., Anhydrous, City Chemical Corporation) in each subject's numbered cup. Each 6-ounce (177.4 ml) styrofoam cup was numbered to correspond to each subject's previously assigned number. To insure double-blind conditions, lactose was added so that 1 g of white powder would be present in each cup. Decaffeinated coffee was added to the cup before serving. The MAAC was administered both just before and one hour after the caffeine consumption. The subjects engaged in their usual work activities in the intervening time. All of the subjects were later given more complete information about the study, including the double-blind procedures.

### Results

The range and mean of change scores on the pre and post administrations of the anxiety, depression,

and hostility scales of the MAAC and the percentage of subjects with change scores in the positive, negative, and no change directions are presented in Table 1.

A hierarchical multiple regression model was employed to examine the effects of each variable with the influences of preceding variables removed. The variables were entered into the equations in accordance with a priori ordering—with age and sex of subject entered in the first step and business versus student status in the second. The remaining variables were entered in the following order—prescores specific to criterion postscores, remaining prescores, dose, and consumption.

Pearson correlations between predictor and criterion variables demonstrated that age and occupation were not significantly correlated with the criterion measures. Sex was significantly correlated with postdepression ( $r = -.195, p < .01$ ) and posthostility ( $r = -.243, p < .01$ ), with males having the higher scores. Consumption was not correlated with the criterion measures and dose was significantly correlated with postanxiety ( $r = .335, p < .001$ ), postdepression ( $r = .235, p < .01$ ), and posthostility ( $r = .261, p < .01$ ).

The full model, consisting of age, sex, occupation, prescores, dose, and consumption, accounted for 32.3% of the variance associated with postanxiety scores,  $F(8, 148) = 8.81, p < .001$ , 53.7% of the variance associated with postdepression scores,  $F(8, 148) = 21.42, p < .001$ ; and 47.5% of the variance associated with posthostility scores,  $F(8, 148) = 16.71, p < .001$ . In all of the equations, age, sex, and occupation combined accounted for less than 8% of the variability. Prescores specific to criterion postscores were the most significant predictors for postdepression,  $F(8, 148) = 32.48, p < .001$ , and posthostility,  $F(8, 148) = 17.26, p < .001$ , respectively, accounting for 45.9% and 34.8% of the post-score variability. Only dose level reached statistical

Table 1  
*Range and Mean of Change Scores and Percentage of Subjects With Scores in Each Direction*

Scale and dose level	<i>M</i>	<i>SD</i>	Range	Subjects (%)		
				(-)	(0)	(+)
Anxiety						
Low	0.476	3.49	-6-14	40	17	43
Medium	1.500	4.51	-7-14	40	15	45
High	2.746	4.11	-5-13	21	14	65
Depression						
Low	0.095	4.11	-7-18	45	26	29
Medium	0.615	5.31	-11-13	42	10	48
High	1.794	5.12	-8-14	35	11	54
Hostility						
Low	0.143	2.56	-6-4	38	19	43
Medium	1.423	3.79	-9-16	27	15	58
High	1.937	3.96	-8-13	24	14	62

significance in the postanxiety equation,  $F(8, 148) = 15.16$ ,  $p < .001$ , accounting for 6.0% of the variability. Additionally, dose level was a significant predictor of postdepression scores,  $F(8, 148) = 6.24$ ,  $p < .01$ , and posthostility scores,  $F(8, 148) = 9.75$ ,  $p < .01$ , respectively, accounting for 4.0% and 3.6% of the variability, which made it the only consistently significant predictor across the three equations. Consumption accounted for less than 1% of the variability in all the equations

### Discussion

Finding a positive relationship between postanxiety scores and caffeine dosage was expected because of the previous literature and caffeine's known pharmacological actions. Symptoms referable to stimulation include nervousness, irritability, agitation, headache, tachypnea, tremulousness, reflex hyperexcitability, and occasional muscle twitchings (Ritchie, 1975; Truitt, 1971). However, the positive relationship between postdepression scores and caffeine dosage was less predictable. Although popular belief holds that caffeine raises mood, it is widely recognized that other central nervous system (CNS) stimulants, such as amphetamines and cocaine, produce depression following an initial mood elevation. In this study, if the posttest had been administered sooner, a positive relationship may not have been found.

The lack of demonstrated effects from prior consumption is noteworthy in view of evidence that caffeine tolerance does develop (Colton, Gosselin, & Smith, 1968). The reasons for the absence of consumption effects are not apparent. However, it is widely recognized that reported drug and dietary (Lansky & Brownell, 1982) consumption and actual consumption often differ appreciably. In one study, it was demonstrated that coffee consumption increased as caffeine content decreased, a phenomenon that conceivably could lead to an artificially low estimate of caffeine content for decaffeinated coffee users (Kozlowski, 1976).

However, we must caution against generalizing these results to the amount of caffeine in a typical cup of coffee. Such a high dosage is not ordinarily consumed at one sitting. On the other hand, the amount of caffeine consumed daily by the average coffee drinker usually equals or exceeds the amount administered during this experiment. Caution should also be used in differential inferences about anxiety, depression, and hostility, inasmuch as the

respective scales employed correlated significantly. Future research that delineates for whom caffeine is harmful and explores dosage and time-after-consumption parameters, appears to be warranted.

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Caffeine's Effects on Anxiety: Generalized Anxiety. Consuming caffeine can exacerbate the emotional symptoms of anxiety. The physical side effects can quickly become thoughts and feelings. The accelerated heart rate and other effects of caffeine can increase fear, worry, and dread. Anxiety sometimes makes it difficult to concentrate. It can make us feel tense, keyed-up, and on edge. We can become irritable. If you live with anxiety and seem to be sensitive to the effects of caffeine, monitoring your caffeine intake can make a positive difference in managing anxiety. But does that mean it's necessary to completely eliminate caffeine from your diet? Is a little bit okay? Depression. Tiredness. Headaches. Anxiety and nervousness. Concentration impairment. Muscle pain and stiffness. Anxiety and Nervousness. It is possible to have actual physical manifestations of anxiety. Your chest starts to get a little bit tighter and your breathing might become a little more labored and difficult. Concentration Impairment. Among the most common effects of caffeine withdrawal is insomnia. Some people cannot sleep through the process as their body is going through all these changes as a result of the absence of caffeine in the system. This is, ironically, as a result of the disruption of the sleep cycle due to the abuse of caffeine.