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What Are the Effects of Caffeine on Mental Health?

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ABSTRACT

In this paper, we sought to understand how excessive caffeine consumption can lead to an increase in anxiety and depression resulting from disturbed sleep so we could learn to avoid these side effects while still enjoying caffeinated beverages. Previous research has found that caffeine consumption is associated with anxiety, decreases in serotonin levels and mental health effects to the extent to which caffeine disrupts sleep. In our first (correlational) study, we tested the strength of these relationships by examining naturalistic daily changes in their variables longitudinally over a period of twelve days. We measured anxiety and depression levels each by using a 10-point Likert scale, serotonin levels by filling out a daily Athens Insomnia Scale, and caffeine consumption by filling out a daily Caffeine Consumption Questionnaire (CC-Q). Based on the strength of correlation found between caffeine and serotonin in our correlational study, we then conducted a second (experimental) study to test for a causal relationship between these two variables. Over an 11-day period, we randomly assigned a participant each day to either a caffeinated condition or a decaffeinated condition using a double blind procedure and had the participant fill out the Athens Insomnia Scale each following morning While our correlational study failed to find statistically significant correlations of caffeine intake with anxiety, depression, or serotonin, the manipulation of caffeine in our experimental study produced a significant increase in insomnia. The results of our experimental study suggest that lowering the amount of caffeine you drink in a day can increase your sleep quality.

1. Introduction

1.1 Research Problem

Caffeine consumption can negatively impact mental health which, in turn, diminishes the quality of life. Caffeine consumption also appears to be a normal routine of many college students but its effects on the human body aren’t as widely known. We want to understand how caffeine affects mental health and if there is a way to limit its negative effects. For example, as one of the authors of this paper suffers from caffeine addiction as well as anxiety, we have noticed a big difference in mental health when drinking caffeine or not. With this research, we hope to understand not only how caffeine directly affects the nervous system but also find solutions for those who suffer from it. Caffeine can also reduce sleep quality and/or quantity, lowering energy and mood the next day. Our hope is to understand how caffeine induces anxiety and sleep-related depression to minimize side effects through strategic dosing. This will allow us to continue enjoying caffeinated beverages without the negative consequences.

1.2 Literature Review

One previously found mental health effect of caffeine consumption is an increase of anxiety symptoms. For example, in an experimental study done by O'Neill et al. (2016), researchers used adolescent male Sprague-Dawley rats and had them consume caffeine for 28 days while age-matched control rats consumed only water. After seven days of withdrawal from caffeine, the brain tissue of the rats was tested. Based on these results, the researchers suggested that adolescents who drink caffeine may have dysregulation of the neuroendocrine stress response system. It also found that brain areas involved in anxiety, such as the hypothalamus and the amygdala, showed a significant increase in basal corticotropin-releasing factor mRNA during caffeine withdrawal, suggestive of increased stress.

Another previously found mental health effect of caffeine consumption is depression from sleep disruption. For example, in a double-blind controlled trial study with California University graduate-level students, Distelberg et al. (2017) had participants randomly assigned to either a caffeine-treatment condition (710mL of caffeinated coffee containing 450 mg of caffeine) or a decaf-control condition (710 mL of decaf coffee containing 12 mg of caffeine). Subjective questionnaires were administered on day 5 of the pre-treatment abstinence phase, as well as day 3 and 5 of the treatment and post-treatment abstinence phases. The Insomnia Severity Index (ISI) was used to measure sleep issues, including difficulties falling asleep, staying asleep, and waking too early, while a 7-point Likert scale was used to access sleep quality (0 = "Between 0 and 1 hours"...7 = "More than 10 hours"). Finally, subscales from the Brief Symptom Inventory (BSI) and the Duke Health Profile (DHP) were used to measure depression. The BSI is a 53-item questionnaire, and the DHP is a 17-item questionnaire, 4 of which measure depression, anxiety, pain, and disability. The ISI and sleep quality measures showed a significant difference between groups, with the caffeine condition reporting more sleep problems and fewer hours of sleep as compared to the decaf control condition. Depression results were more complex, with participants reporting the most depressive symptoms at the beginning and end of the treatment phase, and as many as 3 days into the post-treatment phase. Based on these results, researchers suggest that caffeine's negative impact on sleep contributes to depressive symptoms the next day.

A third previously found mental health effect of caffeine consumption is a decrease of serotonin levels. For example, in an experimental study done by Arnold et al.(2019), researchers used male rats in late, middle, and early adolescence and had them consume caffeine for 28 days and then water for 28 days. Researchers then dissected the brains of the rats and tested their tissue. The researchers found that chronic caffeine consumption in adolescences lead to long-term decreasing in the functioning of serotonergic brain areas therefore may contribute to anxiety-like symptoms.

1.3 Hypotheses

Based on the above literature review, we predicted the following hypotheses:
Hypothesis #1: If caffeine consumption increases then anxiety will increase.
Hypothesis #2: If caffeine consumption increases then depression will increase.

Hypothesis #3: If caffeine consumption increases then serotonin levels will decrease.

2. Methods

2.1 Participants

The 2 authors of this paper served as the participants in its studies. The participants ranged in age from 28 to 34 years old, with an average age of 31 years, and included 2 cisgender women. The participants were both undergraduate students at Camosun College who completed the current studies as an assignment for Psyc 245 ("Drugs & Behavior") and were grouped together due to their mutual interest in the effects of caffeine on mental health. Both participants were regular caffeine users with a moderate-high daily intake and one participant was diagnosed with anxiety and severe depression.

2.2 Correlational Study Methods

We first performed a correlational study to test concurrently all of our hypotheses by examining naturalistic daily changes in their variables longitudinally. Each participant always kept a study journal with them over this study's one-week period in order to record self-observations of daily caffeine intake as the predictor variable, and anxiety, depression, and serotonin levels as outcome variables.

2.2.1 Anxiety

To measure anxiety, a 10-point Likert scale: (0= absence of symptoms, 5= moderate worry, physical agitation; 10= out of control behaviour, hitting, rhyming voices) was used (see Appendix B). Beginning on day 2 of the 12- day study, participants recorded their anxiety levels at three separate occasions (waking, afternoon, and before bed) using their preferred method (phone, notebook, or laptop) for each day of the study. Based on these calculations, the average anxiety level of each participant for each following day was recorded and analyzed.

2.2.2 Depression

To measure depression, a 10-point Likert scale (0 = absence of symptoms; 5 = definite malaise, insomnia; 10 = despair, suicidal feelings) was used (see Appendix B). Beginning on day 2 of the 12-day study, participants rated and recorded their depression levels at 3 separate times (upon waking, midday, and before bed) in either a notebook or cellphone for each day of the study. From these records, the average daily depression level of each participant for each following day was calculated and analyzed.

2.2.3 Serotonin Levels

To measure serotonin levels, participants completed the Athens Insomnia Scale (See Appendix C). The scale consisted of 8 insomnia symptoms where each item is scored on a scale of 0 (none) to 3 (severe) with a total score 0-24 where <12 is the participant may not be suffering from insomnia and 12-24 participants are likely to be suffering from insomnia. Participants completed this scale daily (preferably after waking) for two weeks. Higher levels of insomnia on this scale were interpreted as indicating lower levels of serotonin, since accumulation of this neurotransmitter is responsible for promoting sleep.

2.2.4 Caffeine

To measure caffeine consumption, all the participants filled out a Caffeine Consumption Questionnaire (CC-Q) on a daily basis (See Appendix A). The questionnaire includes 9 broad items that contain caffeine: for instance, one is coffee while another is a chocolate bar. Participants had to pick one or more of the nine options as well as fill out the amount consumed that day using milligrams (mg). This was offset by the average number of ounces per day as well as the average total per day. Based on the sum of each of these numerical answers, one’s level of caffeine consumption was assessed.

2.3 Correlational Study Planned Analyses

To assess the strength and statistical significance of associations between variables predicted by our 3 hypotheses, we performed Pearson product moment correlations of their predictor variable daily caffeine intake with their outcome variables anxiety, depression, and serotonin levels. For testing Hypothesis #1, we correlated each participant's average daily anxiety score on each following day by the total amount of daily caffeine consumed. For testing Hypothesis #2, we correlated each participant’s average daily depression score on each following day by the total amount of daily caffeine consumed. For testing Hypothesis #3, we correlated each participant’s average insomnia score each following morning upon waking by the total amount of daily caffeine consumed. We performed all of the above correlations separately for each participant as well as using data pooled across all of the participants. For the correlations using pooled data, in addition to using the raw data, we also performed correlations after we had first transformed the data from each participant into *z*-scores in order to standardize differences in averages and variability seen between the participants in their data and thus make them more comparable. A correlation coefficient was considered statistically significant if the probability of its random occurrence (p) was $< .05$ (i.e., less than 5% of the time expected by chance alone).

2.4 Experimental Study Methods

Based on the strength of the correlation between caffeine consumption and serotonin levels found in our correlational study, we then chose to conduct an experimental study to test for a causal relationship between these two variables from Hypothesis # 3: If caffeine consumption increases then serotonin levels will tend to decrease.

We manipulated the independent variable, caffeine consumption, over an 11-day period by randomly assigning (through coin flip) a participant each day to either consume a regular caffeinated coffee or a decaffeinated coffee using in a double blind procedure. The participant’s partners, friends, or family members flipped a coin each day when they were to consume caffeine. When the coin was flipped to heads, the participants drank caffeinated coffee and when the coin was flipped to tails, the participants drank a decaffeinated coffee. The next morning, participants would record serotonin levels by filling out an Athens Insomnia Scale (See Appendix D). Higher levels of insomnia on this scale were interpreted as indicating lower levels of serotonin, since accumulation of this neurotransmitter is responsible for promoting sleep.Once the data was recorded, the partner, friend, or family member who assisted with the experimental condition, then told the participant what method was assigned to them (caffeinated coffee or decaffeinated coffee) and data was analyzed correspondingly.

On caffeine experimental days, participants drank 16oz of Tim Horton’s Dark Roast coffee, containing 260mg of caffeine. On decaf control days, participants drank 16oz of Tim Horton’s Dark Roast decaf coffee, containing 12mg of caffeine. In both conditions, the participant added 2 tablespoons of cream to their coffee and 2 tablespoons of sugar. The experimental caffeine amount was based on the approximate average the participant usually drank. The coffee was consumed shortly after waking, within a 1-hour period.

To ensure the participant remained unaware of their daily assigned conditions, the participant stayed out of the kitchen while their partner acted as an assistant who prepared each day's coffee. Following administration, the assistant recorded the assigned condition in a private journal. The following day, the participant took dependent variable measurements and then collected the identity of the previous day's condition from their assistant. To avoid potential taste differences between conditions, the participant chose a decaf coffee that had the same roast level as their regular caffeinated coffee. Creamer and/or sugar were also added to minimize any potential taste differences.

2.5 Experimental Study Planned Analyses

To assess the statistical significance of differences seen in serotonin levels on decaffeinated coffee vs caffeinated coffee, Student's *t*-tests were performed. We performed *t*-tests separately for each participant as well as using data pooled across all the participants. For the *t*-tests using pooled data, in addition to using the raw data, we also performed *t*-tests after we had first transformed the data from each participant into *z*-scores to standardize differences in averages and variability seen between the participants in their data and thus make them more comparable. An average difference between conditions was considered statistically significant if, using a one-tailed distribution (i.e., to determine if there is a difference between groups in a specific direction), the probability of its random occurrence (p) was $< .05$ (i.e., less than 5% of the time expected by chance alone).

3. Results

3.1 Correlational Study Results

As shown in Table 1, none of our correlations resulted in any statistical significance ($p < .05$). For hypothesis #1, there was no significant correlation found between caffeine consumption anxiety using either pooled raw data (r = 0.04, p = 0.81; see Figure 3) or pooled standardized data (r = 0.07, p = 0.70; see Figure 4). For hypothesis #2, there was no significant correlation found between caffeine consumption and depression using either pooled raw data (r = 0.04, p = 0.83; see Figure 5) or pooled standardized data (r = 0.10, p = 0.57; see Figure 6). For hypothesis #3, there was no significant correlation found between caffeine consumption and insomnia scores using either pooled raw data (r = 0.08, p = 0.65; see Figure 1) or pooled standardized data (r = -0.13, p = 49; see Figure 2). Based on a comparison of the pooled standardized data for each of the three hypotheses, caffeine consumption was found to be most strongly correlated with insomnia scores (r = -0.13).

3.2 Experimental Study Results

As shown in Table 2, there was a significant difference in insomnia scores between the caffeinated coffee and decaffeinated coffee conditions (p = 7.59E-07 using both raw and standardized data). Using the raw data, insomnia scores following the caffeinated coffee condition showed a mean of 16.45 (S.D.= 3.45) while following the decaffeinated coffee condition the mean was 5.40 (S.D.= 3.89) (see Figure 7). Using the standardized data, the z-scores for insomnia levels following the caffeinated coffee condition the mean was 0.79 (S.D.= 0.52) while following the decaffeinated coffee condition the mean was -0.87 (S.D.=

0.58) (see Figure 8). Assuming that increases in insomnia are associated with decreases in serotonin, these results suggest that our caffeinated coffee condition decreased serotonin levels in comparison to the decaffeinated coffee condition.

4. Discussion

4.1 Summary of Results

Based on previous research, we hypothesized that an increase in caffeine consumption would be followed by increases in two variables and a decrease in another variable: anxiety increase (Hypothesis #1), depression increase (Hypothesis #2), and serotonin decrease (Hypothesis #3). Data pooled across participants in our correlational study did not support any of the hypotheses. However, our experimental study showed significant differences in insomnia scores between its caffeinated coffee and decaffeinated coffee conditions, which supported our Hypothesis #3.

4.2 Relation of Results to Past Research

Our correlation study indicated that caffeine and anxiety did not have a significant correlation, which is not consistent with previous research. According to the study conducted by O'Neill et al. (2016), caffeine and anxiety should be positively correlated. A difference in methodology between our study and the O'Neill et al. (2016) study that could possibly account for their discrepancy in results is that we did not use the same type of participants: female college students in our study versus male Sprague-Dawley rats in their study. Further studies should be done on the study of human subjects instead of non-human subjects to provide a more relevant correlation between caffeine and anxiety for humans.

Our correlational study failed to show that caffeine increases depression as reported by previous research. In addition to increased insomnia, Distelberg et al. (2017) found that depression, measured using subscales from the BSI and the DHP, increased as a result of caffeine consumption. Based on this research, we hypothesized that caffeine's negative impact on sleep would contribute to an increase in depressive symptoms the following day, but the data failed to confirm this hypothesis. Differences in methodology may account for the discrepant results. Distelberg et al. (2017) blindly experimentally administered set amounts of caffeine to participants while in our nonexperimental study participants' caffeine consumption in our study varied from day-to-day. Future studies should test to confirm that varied caffeine intake and participant knowledge don't distort subjective depression results. In addition, while Distelberg et al. (2017) had participants with no history of mental health conditions or chronic physical health limitations, our study consisted of participants with known mental health issues. Future studies should examine the differences of caffeine's impact on participants with, and without, mental health conditions.

Our correlation study indicated that caffeine and serotonin are not significantly correlated, which is not consistent with previous research. According to the study conducted by Arnold et al. (2019), caffeine and serotonin levels are negatively correlated. A difference in methodology between this study and the Arnold et al. (2019) study that could possibly account for their discrepancy in results include differences in participant types: female college students in our study versus adolescent male rats in their study. In addition, we measured serotonin indirectly by the inverse of scores on the Athens Insomnia Scale while Arnold et al. (2019) measured serotonin levels directly. However, our experimental study indicated that caffeine did significantly decrease serotonin levels, which is consistent with previous research. Further studies should verify whether the caffeine-induced increases insomnia are indeed associated with decreases in serotonin and whether this has any effect upon mental health, such as anxiety and depression.

4.3 Implications of Results

Our study could be a step in the right direction at helping the mental well-being of college students as well as young adolescents on the side effects of too much caffeine. As a result of our study, we discovered just how much caffeine can affect our insomnia, with lowering the amount of caffeine you drink in a day improving sleep quality. It remains to be seen whether these effects also produce changes in mental health.

References

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Table 1

Correlations for Study Variables

Variables	Participant #1		Participant #2		Pooled raw data		Pooled standardized data	
	r	*n*	*r*	*n*	*r*	*n*	*r*	*n*
Serotonin Decrease & Caffeine Increase	-0.05	16	-0.20	16	-0.08	32	-0.13	32
Anxiety Increase & Caffeine Increase	-0.10	16	0.24	16	0.04	32	0.07	32
Depression Increase & Caffeine Increase	-0.02	16	0.23	16	0.04	32	0.10	32

* $p < .05$.

Table 2

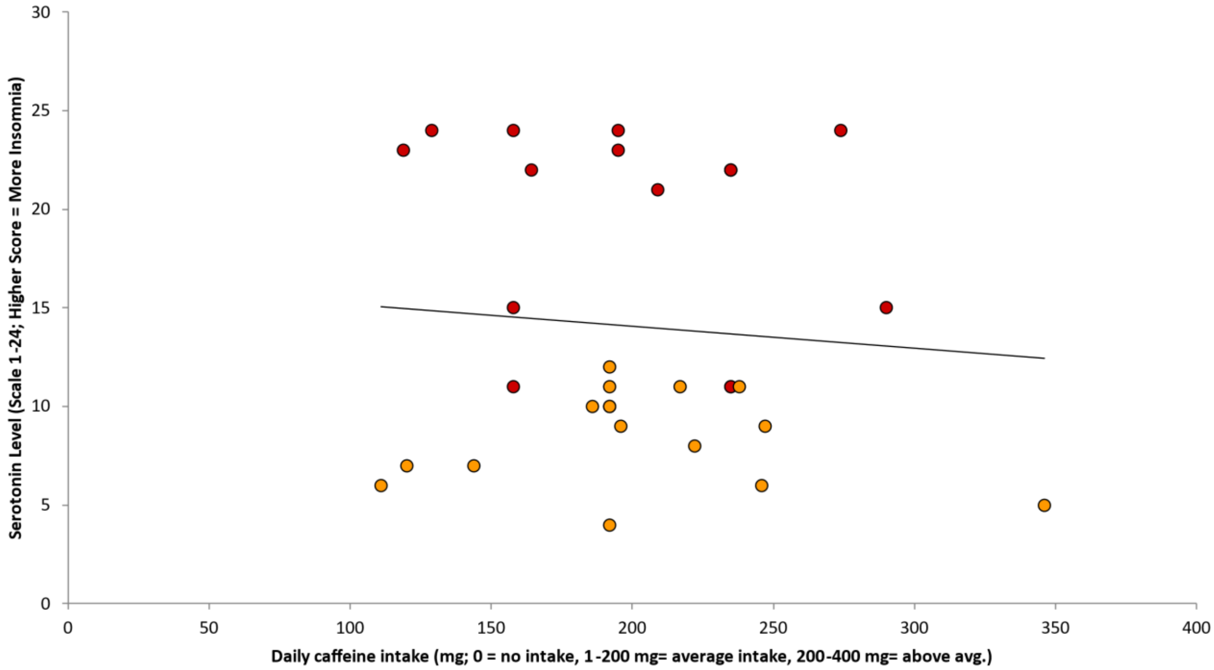
Descriptive Statistics for Insomnia Scores Across Different Caffeine Intake Conditions

Condition	Statistic	Participant #1
Caffeinated Coffee	*M*	16.45*
	SD	3.45
	n	11
Decaffeinated Coffee	*M*	5.49
	SD	3.89
	n	10

Note. *M*, *SD*, and *n*, represent mean, standard deviation, and sample size, respectively. Insomnia was rated on a scale of 1-24, where higher score = more insomnia. * $p < .05$ for comparison of Caffeinated Coffee condition with its respective Decaffeinated Coffee condition.

Figure 1

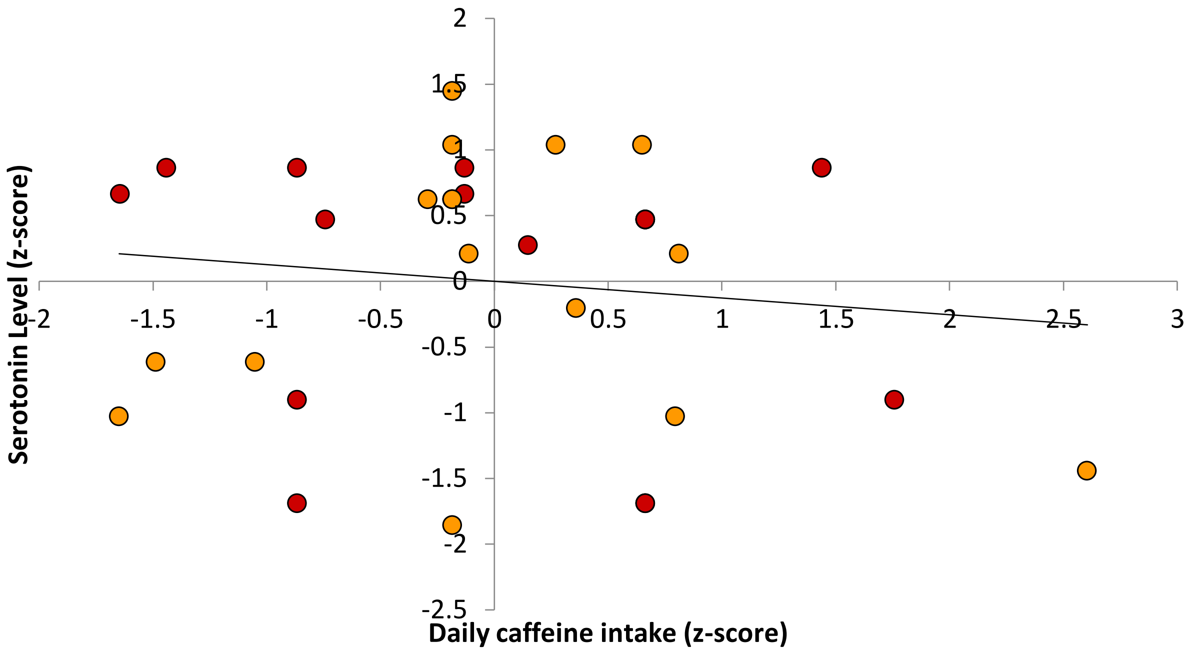
Association Between Caffeine and Insomnia Scores Using Pooled Raw Data



Notes. Marker colour differentiates participants: red = participant #1, orange = participant #2. Some data might not be visible in the figure due to overlapping markers. Higher insomnia scores are interpreted to mean lower serotonin levels.

Figure 2

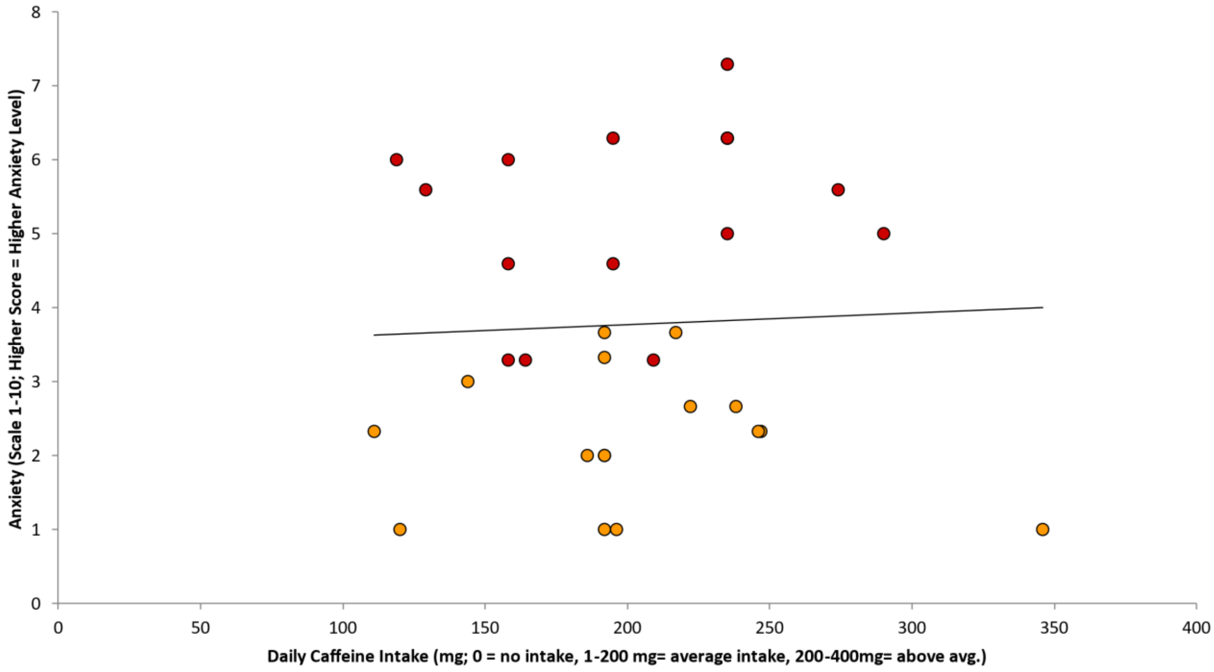
Association Between Caffeine and Insomnia Scores Using Pooled Standardized Data



Notes. Marker colour differentiates participants: red = participant #1, orange = participant #2. Some data might not be visible in the figure due to overlapping markers. Higher insomnia scores are interpreted to mean lower serotonin levels.

Figure 3

Association Between Caffeine and Anxiety Using Pooled Raw Data

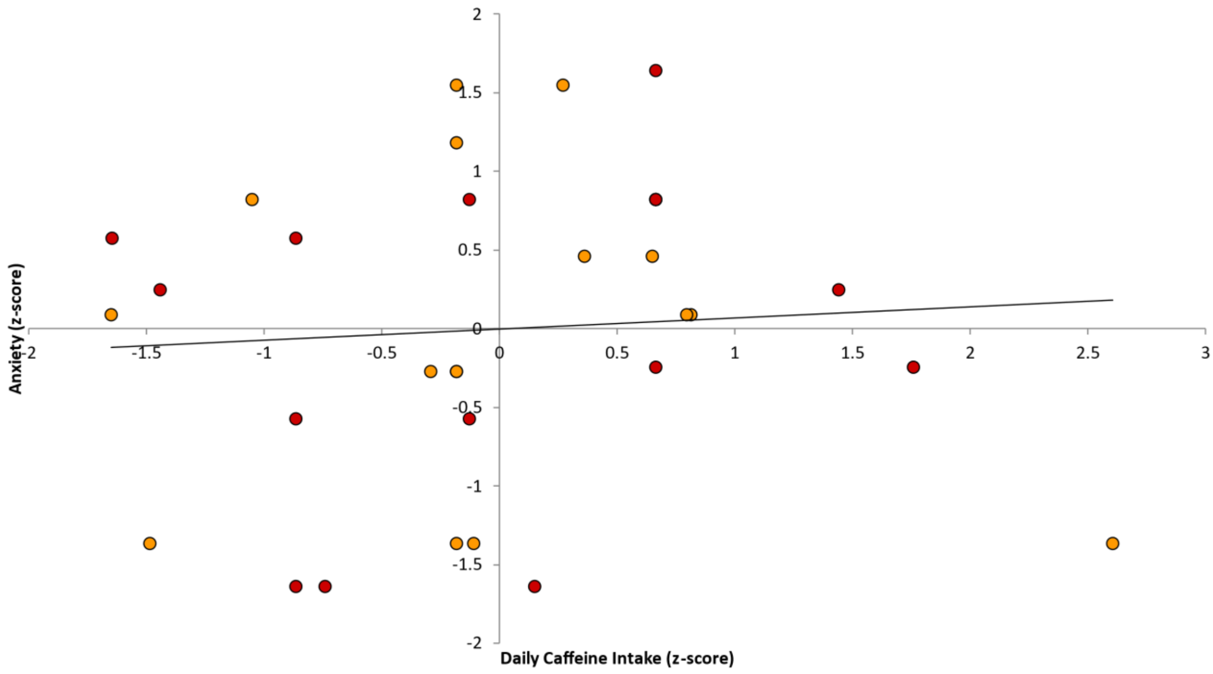


Notes. Marker colour differentiates participants: red = participant #1, orange = participant #2.

Some data might not be visible in the figure due to overlapping markers.

Figure 4

Association Between Caffeine and Anxiety Using Pooled Standardized Data

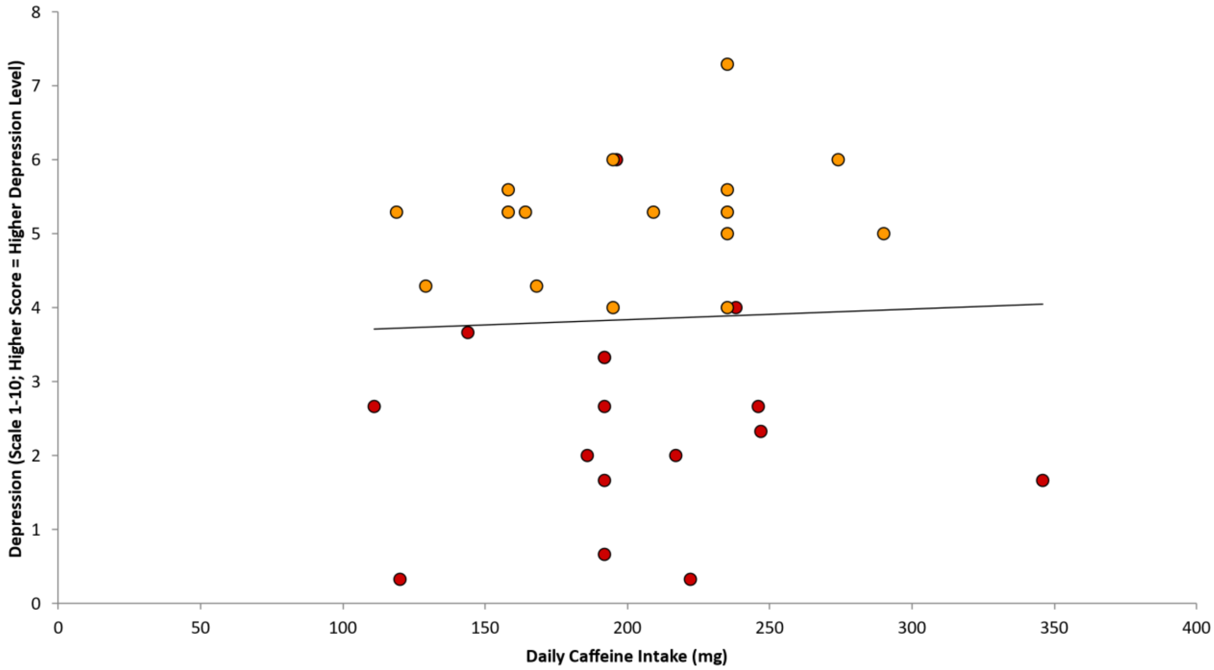


Notes. Marker colour differentiates participants: red = participant #1, orange = participant #2.

Some data might not be visible in the figure due to overlapping markers.

Figure 5

Association Between Caffeine and Depression Using Pooled Raw Data

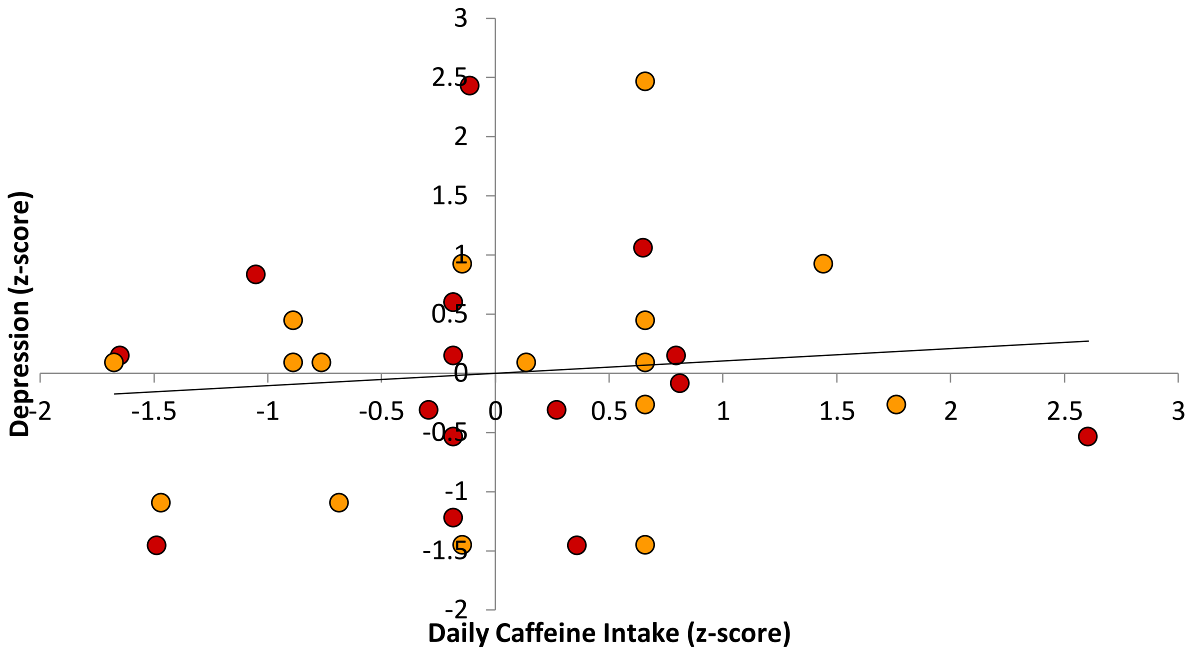


Notes. Marker colour differentiates participants: red = participant #1, orange = participant #2.

Some data might not be visible in the figure due to overlapping markers.

Figure 6

Association Between Caffeine and Depression Using Pooled Standardized Data

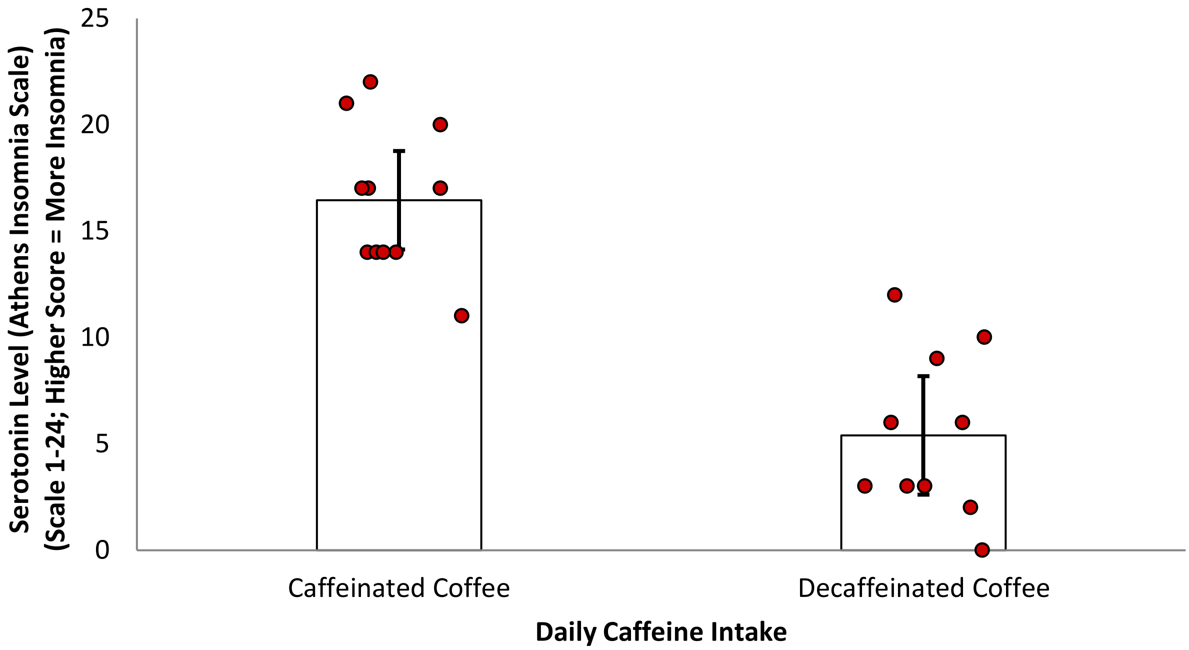


Notes. Marker colour differentiates participants: red = participant #1, orange = participant #2.

Some data might not be visible in the figure due to overlapping markers.

Figure 7

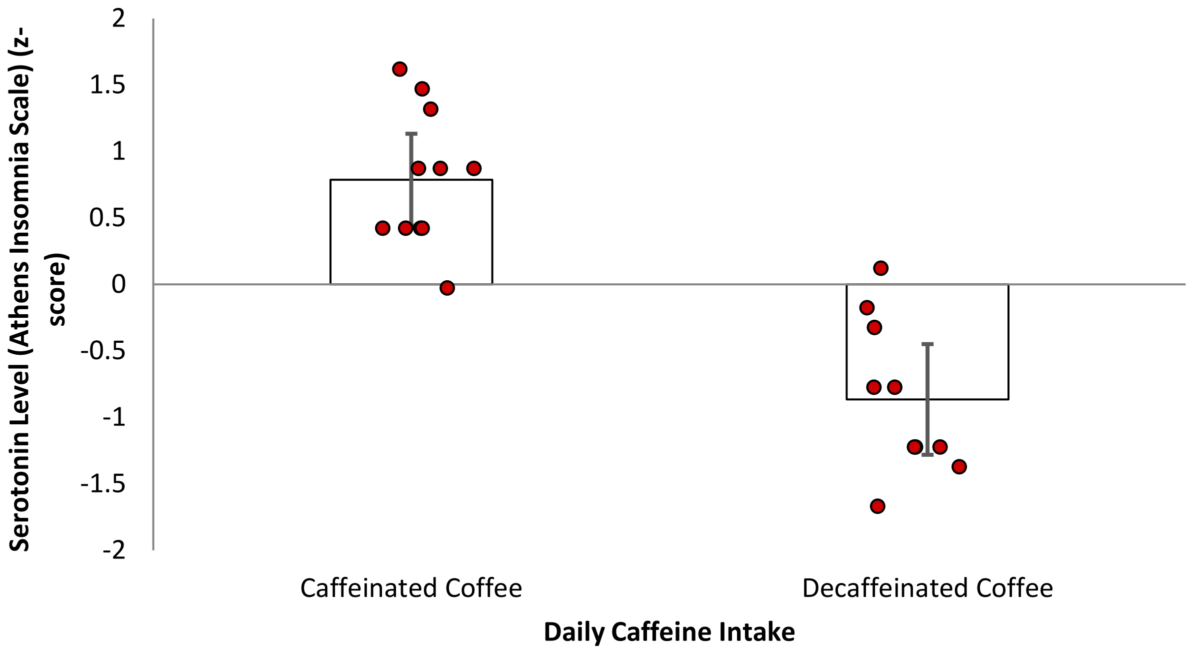
Average Insomnia Scores Across Different Caffeine Intake Conditions Using Raw Data



Notes. Insomnia scores are shown for caffeinated coffee and decaffeinated coffee conditions. Errors bars show ± 95% confidence levels. Overlapping scatterplot show individual data points.

Figure 8

Average Insomnia Scores Across Different Caffeine Intake Conditions Using Standardized Data



Notes. Insomnia scores are shown for caffeinated coffee and decaffeinated coffee conditions. Errors bars show ± 95% confidence levels. Overlapping scatterplot show individual data points.

Appendix A

Caffeine Consumption Questionnaire

Beverages			Average number of ounces/doses/tablets per day	Average total per day
Coffee (6 oz.)	125mg	X	_______	_______
Decaf Coffee (6 oz.)	5 mg	X	_______	_______
Espresso (1 oz.)	50 mg	X	_______	_______
Tea (6 oz.) Green	50 mg	X	_______	_______
Tea (6 oz) Black	50 mg	X	_______	_______
Cocoa (6 oz.)	15 mg	X	_______	_______
Energy drinks (12 oz.)	*equivalent 200 mg	X	_______	_______
Caffeinated Soft Drinks (12 oz.)	40-60 mg	X	_______	_______
Chocolate candy bar	20 mg	X	_______	_______

Appendix B

Daily Rating Scale for Anxiety and Depression

1-10 Depression Scale

8-10 despair, suicidal feelings

6-7 feeling really bad, at the edge

5 definite malaise, insomnia

3-4 depression slightly stronger

1-2 minorly depressed mood

0 absence of symptoms

1-10 Anxiety Scale

8-10 out-of-control behavior, hitting, rhyming voices

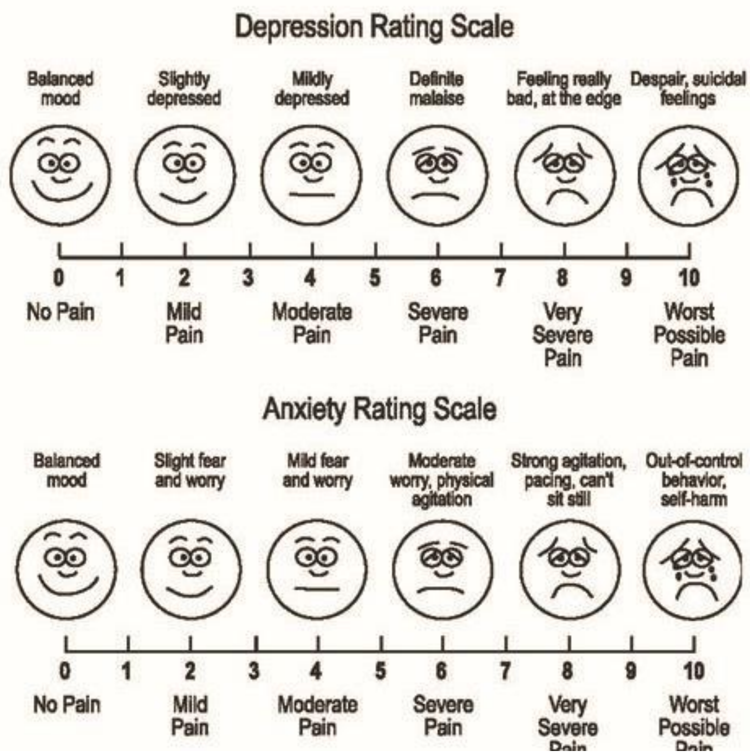
6-7 strong agitation, pacing

5 moderate worry, physical agitation

3-4 mild fear and worry

1-2 slight fear and worry

0 absence of symptoms



Appendix C

ATHENS INSOMNIA SCALE

This scale is intended to record your own assessment of any sleep difficulty you might have experienced. Please, check (by circling the appropriate number) the items below to indicate your estimate of any difficulty.

Sleep factors	**Athens insomnia scale**			
Sleep induction	**0: No problem**	**1: Slightly delayed**	**2: Markedly delayed**	**3: Very delayed or did not sleep at all**
Awakenings during the night	**0: No problem**	**1: Minor problem**	**2: Considerable problem**	**3: Serious problem or did not sleep at all**
Final awakening	**0: Not earlier**	**1: A little earlier**	**2: Markedly earlier**	**3: Much earlier or did not sleep at all**
Total sleep duration	**0: Sufficient**	**1: Slightly insufficient**	**2: Markedly insufficient**	**3: Very insufficient or did not sleep at all**
Sleep quality	**0: Satisfactory**	**1: Slightly unsatisfactory**	**2: Markedly unsatisfactory**	**3: Very unsatisfactory or did not sleep at all**

Well-being during the day	**0: Normal**	**1: Slightly decreased**	**2: Markedly decreased**	**3: Very decreased**
Functioning capacity during the day	**0: Normal**	**1: Slightly decreased**	**2: Markedly decreased**	**3: Very decreased**
Sleepiness during the day	**0: None**	**1: Mild**	**2: Considerable**	**3: Intense**