Personality and Individual Differences xxx (2008) xxx–xxx

Contents lists available at ScienceDirect

Personality and Individual Differences

journal homepage: www.elsevier.com/locate/paid

Short Communication

Caffeine, stress, and proneness to psychosis-like experiences: A preliminary investigation

Simon R. Jones *, Charles Fernyhough

Department of Psychology, Durham University, South Road, Durham DH1 3LE, UK

ARTICLE INFO

Article history:
Received 8 July 2008
Received in revised form 15 October 2008
Accepted 31 October 2008
Available online xxxx

Keywords:
Coffee
Hallucination
Persecutory ideation
Psychosis
Schizophrenia
Tea

ABSTRACT

In diathesis–stress models of psychosis, cortisol released in response to stressors is proposed to play a role in the development of psychotic experiences. Individual differences in cortisol response to stressors are therefore likely to play a role in proneness to psychotic experiences. As caffeine has been found to increase cortisol response to a given stressor, we proposed that, when levels of stress were controlled for, caffeine intake would be related to hallucination-proneness and persecutory ideation. Caffeine intake, stress, hallucination-proneness and persecutory ideation were assessed by self-report questionnaires in a non-clinical sample ($N = 219$). Caffeine intake was positively related to stress levels and hallucination-proneness, but not persecutory ideation. When stress levels were controlled for, caffeine intake predicted levels of hallucination-proneness but not persecutory ideation. Implications of these findings are discussed and avenues for future research suggested.

1. Introduction

Diathesis–stress models of psychosis propose that stress may play a contributory role in the development of hallucinations and delusions, typically associated with the schizotypal/schizophrenia spectrum (Neuchterlein & Dawson, 1986). Cortisol has been highlighted as having a key role in translating the experience of psychosocial stressors into the biological factors associated with psychosis (Walker & Diforio, 1997). It therefore seems likely that factors causing individual differences in cortisol response to stressors will influence the likelihood of developing hallucinatory or delusional experiences. One such factor is caffeine, which has been found to cause an increase in the amount of cortisol released in response to a stressor (Lane, Adcock, Williams, & Kuhn, 1990). Although daily usage of caffeine creates a reduction in this effect, caffeine intake in habitual users still enhances the cortisol response to stress (Lovallo et al., 2005).

As such, caffeine intake may be associated with greater proneness to psychotic experiences due to its enhancement of the cortisol response to stress. We hence firstly hypothesised that, for a given level of stress, caffeine intake would be associated with greater levels of proneness to psychotic experiences. Controlling for stress levels is also necessary due to caffeine consumption increasing when individuals are stressed (Ratliff-Crain & Kane, 1995). We secondly hypothesised that caffeine intake would interact with stress, with caffeine intake being more strongly associated with proneness to psychotic experiences when levels of stress were high. No studies have yet tested such a hypothesis.

Existing studies of the relation between caffeine and the positive symptoms of schizophrenia (e.g., clinically relevant hallucinations and delusions) have produced mixed findings. Studies that have experimentally altered caffeine intake in patients with schizophrenia, by switching patients to decaffeinated coffee for a period of weeks, show conflicting results. Whilst some have found resultant decreases in positive symptoms (e.g., De Freitas & Schwartz, 1979), other studies have failed to replicate this (e.g., Mayo, Falkowski, & Jones, 1993). In the only study to systematically administer caffeine (10 mg/kg) directly to patients with schizophrenia, Lucas et al. (1990) found that suspiciousness/paranoia did not change, but that grandiosity and unusual thought content increased (data on hallucinations were not reported). However, such studies may be confounded by the interaction of caffeine with antipsychotic medication (Haslemo, Eikeseth, Tanum, Molden, & Refsum, 2006).

One way to avoid this confound is to assume that psychotic experiences exist on a continuum stretching into the healthy population (Johns & van Os, 2001) and examine the relation between caffeine and psychosis-like experiences in the general population. The only study to date to examine the relation between caffeine intake and schizotypy (Larrison, Briand, & Sereno, 1999) found no relation between the two. However, this study used a crude self-report measure of caffeine intake in which participants were simply asked to "circle the number of caffeinated. . .drinks they consumed

* Corresponding author. Tel.: +44 1913 343240; fax: +44 1913 343241.
E-mail address: s.r.jones@durham.ac.uk (S.R. Jones).

0191-8869/$ - see front matter © 2008 Published by Elsevier Ltd.
doi:10.1016/j.paid.2008.10.032

per day or week" (p. 103), and then split individuals into high, moderate and low intake groups, hence reducing the power of the study. Furthermore, it employed a general measure of schizotypy, with no focus on specific experiences such as hallucinations or feelings of persecution. We accordingly set out to test our hypotheses in a non-clinical population, with a methodology chosen to avoid the limitations of the Larrison et al. study.

2. Method

2.1. Participants

Students (N = 219, 154 women) at a United Kingdom university, with a mean age of 20.1 years (SD = 1.34, range = 18–28) were recruited through e-mail invitation. No financial incentive was offered and the likelihood of repeated participation was thus considered low. Answers were given anonymously, with only the age, gender and weight of the participants being recorded. Cigarette smokers were excluded from the study.

2.2. Measures

Participants completed the following questionnaires in the order stated below:

Durham Caffeine Inventory (DCI): Caffeine was assessed utilising a new tool designed to assess caffeine intake in a contemporary UK student population. The DCI presents specific types of drink and food containing caffeine (see Table 1), of which participants rate their typical intake over the past year, using a 12-point response scale ranging from "none/less than one per week" to "8+ per day". In addition to predefined categories (e.g., instant coffee, tea) participants are also asked about coffee purchased from coffee shops, other energy drinks, and caffeine tablets. Participants use the same response scale, but are free to enter the name of the beverage or make of tablets used. A full copy of the DCI is available upon request.

Hallucination-proneness: This was assessed using the modified Launay-Slade Hallucination Scale (LSHS-M: Laroi & van der Linden, 2005), a 16-item instrument designed to measure predisposition to hallucination-like experiences. Each item is scored on a five-point Likert's scale ranging from "Certainly applies to me" (4) to "Certainly does not apply to me" (0).

Proneness to persecutory delusion-like beliefs: The Persecutory Ideation Questionnaire (PIQ; McKay, Langdon, & Coltheart, 2006) is a 10-item questionnaire designed to measure persecutory ideation in both clinical and non-clinical samples. Items are rated on a five-point Likert's scale ranging from "Very True" (4) to "Very Untrue" (0).

Stress: This was determined by the Perceived Stress Questionnaire (Levenstein et al., 1993), a 30-item instrument which assesses the levels of stress an individual perceives themselves to have been under during the last year. Examples of items include "You feel tense" and "You have many worries". Items are scored on a four-point Likert's scale ranging from "Almost Never" (1) to "Usually" (4). A total score is derived using the formula ((raw score – 30)/90).

Table 1
Caffeine data utilized.

Beverage	Caffeine (mg)	Source
Tea (weak)	25	Food Standards Agency (2004)
Tea (medium)	42	FSA (2004)
Tea (strong)	51	FSA (2004)
Coffee (instant)	45	FSA (2004)
Coffee (brewed)	111	FSA (2004)
Coffee (e.g., Starbucks)	188	McCusker, Goldberger, and Cone (2003)
Cola-drinks	35	(details from the authors)
Red Bull	80	Alford, Cox, and Wescott (2001)
Solid milk chocolate	6	(details from the authors)
Solid dark chocolate	20	(details from the authors)
Caffeine tablets	100	Manufacturer website

3. Results

Cronbach's alphas for all scales employed were greater than 0.7, and hence satisfactory. Caffeine content for DCI items was calculated using the values presented in Table 1. The caffeine content of novel energy drinks and caffeine tablets was determined by reference to the relevant manufacturer's website. Descriptive statistics are presented in Table 2. Mean daily caffeine consumption of 141 mg was comparable to previous studies in student populations (e.g., Landrum, 1992). Bivariate non-parametric correlational analyses (using a Bonferroni corrected alpha of .05/3, α = .02) showed caffeine intake/kg to correlate significantly with stress, ρ = .17, p < .02, and LSHS-M, ρ = .22, p < .02, but not PIQ scores, ρ = .08, n.s. PIQ and LSHS-M scores were also significantly correlated, ρ = .40, p < .001.

Parametric statistical analysis was then performed using two hierarchical multiple linear regressions (MLRs) with LSHS-M and PIQ score as the respective dependent variables. Age, gender, and stress were entered in the first step, with caffeine/kg and the (mean-centered) interaction between stress and caffeine/kg entered in a second step. Results are presented in Table 3. Residuals were normally distributed (D = .05. p > .05, for both MLRs) and there was no evidence of multicollinearity in the data. In the first step, stress was found to predict LSHS-M and PIQ scores. After age, gender and stress levels had been controlled for in the first step, the second step was found to explain a significant amount of variance in LSHS-M but not PIQ scores (β values for the second step of the MLR of PIQ scores are hence not reported). Caffeine/kg predicted LSHS-M but not PIQ scores. The interaction between stress and caffeine/kg was not a significant predictor of either LSHS-M or PIQ scores.

Table 2
Descriptive statistics for variables under investigation.

	Mean (SD)	Range
Caffeine intake (mg/day)	143.48 (167.22)	1–951
Caffeine intake (mg/day/kg)	2.31 (2.88)	.01–20.15
LSHS-M	23.81 (12.18)	1–63
PIQ	8.71 (6.87)	0–33
Stress	.45 (.17)	.10–1.00

Note: LSHS-M = modified Launay-Slade Hallucination Scale; PIQ = Persecutory Ideation Questionnaire.

Table 3
Multiple linear regression analysis.

	LSHS-M	PIQ
R^2 (age, gender, stress)	.18	.28
F(3,214)	15.77, p < .001	28.07, p < .001
β (age)	−.11, n.s.	−.06, n.s.
β (gender)	−.01, n.s.	−.20, p < .001
β (stress)	.42, p < .001	.53, p < .001
R^2 (age, gender, stress, caffeine/kg, stress * caffeine/kg)	.21	.29
F(5,212)	11.29, p < .001	17.48, p < .001
ΔF(2,212)	3.89, p < .05	1.44, n.s.
β (age)	−.10, n.s.	–
β (gender)	−.01, n.s.	–
β (stress)	.39, p < .001	–
β (caffeine/kg)	.18, p < .01	–
β (stress * caffeine/kg)	−.04, n.s.	–

Please cite this article in press as: Jones, S. R., & Fernyhough, C. Caffeine, stress, and proneness to psychosis-like experiences: ... *Personality and Individual Differences* (2008), doi:10.1016/j.paid.2008.10.032

4. Discussion

The present study set out to examine the relations among caffeine intake, stress and proneness to psychosis-like experiences in a non-clinical population. Correlational analyses showed that caffeine intake was positively related to stress levels. This was in line with previous research (Ratliff-Crain & Kane, 1995). Caffeine intake was correlated with hallucination-proneness, but not persecutory ideation. The latter finding is consistent with the work of Lucas et al. (1990), which found that caffeine administration (in patients with schizophrenia) did not lead to increases in suspiciousness/paranoia.

When multiple linear regression analyses were performed, controlling for stress, caffeine intake per kg predicted hallucination-proneness but not persecutory ideation. The present study hence offers some support for our first hypothesis, namely that when levels of stress are controlled for, caffeine intake is positively related to levels of psychosis-like experiences. However, the effect was found to be weak and specific to hallucination-proneness, and not persecutory ideation. Our second hypothesis, that the interaction between caffeine intake and stress would predict hallucination-proneness and persecutory ideation, was not supported.

A number of limitations of this study need to be acknowledged. Firstly, we cannot eliminate the possibility that hallucination-proneness could be a cause rather than a result of increased caffeine intake. This would be consistent with the finding that caffeine intake can act as a coping mechanism to bring relief from problems (Ratliff-Crain & Kane, 1995). Secondly, the present study utilised a non-clinical sample, and it is unclear if the present results are likely to be generalizable to clinical populations. Finally, we used a retrospective self-report measure of caffeine that was not independently validated.

Our findings suggest a number of fruitful avenues for future research. Firstly, research may wish to address whether the relation found here between hallucination-proneness and caffeine intake is causal. For example, caffeine tablets could be administered to participants before undertaking signal detection tasks to determine if caffeine intake does lead to increased levels of hallucinatory experience. Secondly, research may wish to address the mechanisms which may lead to caffeine causing hallucinatory experiences. One interpretation of our finding of a relation between caffeine and hallucination-proneness, but not persecutory ideation, is that the former relation exists not because of potentiation of the cortisol response to stressors (which would presumably also lead to a link with persecutory ideation), but as a result of other mechanisms. It would also be profitable to revisit studies of caffeine intake in patients with schizophrenia. The present study offers preliminary evidence that, if levels of stress are controlled, caffeine intake in patients may relate to levels of hallucinatory experiences.

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Please cite this article in press as: Jones, S. R., & Fernyhough, C. Caffeine, stress, and proneness to psychosis-like experiences: ... *Personality and Individual Differences* (2008), doi:10.1016/j.paid.2008.10.032