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HIGHER SERUM CAFFEINE IN SMOKERS WITH SCHIZOPHRENIA COMPARED TO SMOKING CONTROLS

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Abstract

Previous studies of high dietary caffeine intake in individuals with schizophrenia have not demonstrated biological evidence of higher intake or controlled smoking behavior. This study aimed to examine differences in serum caffeine levels in 104 smokers with schizophrenia/schizoaffective disorder (SCZ/SA) and compare them to 63 smokers without any mental illness (CON). Since we were interested in measuring caffeine levels, we excluded all non caffeine users from the study. Blood draws were standardized to occur at midday on a usual smoking day. The mean serum caffeine level was significantly higher for SCZ/SA group compared to CON (2722 vs. 1122ng/mL; $p<0.001$). This trend persisted in subsets of smokers who smoked less than 20 cigarettes per day (CPD; 2052 vs. 587 ng/mL; $p<0.05$), 20 to 25 CPD (2743 vs. 1170 ng/mL; $p<0.001$) or more than 25 CPD (3430 vs. 1834 ng/mL; *NSS*). Linear backward stepwise regression analyses including demographic and smoking variables revealed that having a diagnosis of SCZ/SA (compared to CON) significantly predicted serum caffeine level ($B=1528.2$; $p<0.001$). In addition, SCZ/SA group had two times greater serum caffeine levels as compared to CON with similar smoking behavior. Clinical effects of smoking and caffeine intake are important and may complicate the interpretation of schizophrenia symptoms and antipsychotic medication side effects, thus warranting further research.

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Keywords

Caffeine; Nicotine; Smoking; Schizophrenia; Metabolism

1. Introduction

Caffeine is among the most widely used psychoactive substances in the world and does not seem to have adverse effects being classified by the FDA as a “generally recognized as safe” (GRAS) drug (Knight et al., 2004; USDA 2000). However little is known about its effects when consumed in higher doses. Numerous reports have shown that psychiatric patients drink large amounts of coffee (Mikkelsen, 1978; Furlong, 1975; Winstead, 1976), or consume more than 750 mg of caffeine a day (Greden et al., 1981; Koczapski et al., 1989; Rihs et al., 1996) and individuals with schizophrenia are reported to consume greater amounts caffeine (Strassnig et al., 2006, Gurpegui et al., 2006, Henderson et al., 2006).

One explanation is that individuals with schizophrenia may be consuming excess caffeine to compensate for the effects of antipsychotic medications which are often sedating or can slow cognitive processes (Deckert et al., 2003). Alternatively caffeine may be linked directly to the neurobiological and clinical features of schizophrenia. Caffeine’s antagonism of dysfunctional inhibitory adenosine pathways may provide clues for areas of new drug development (Ferre et al., 1994; Lara et al., 2006, Hughes et al., 1998). Tar from smoked tobacco is a powerful inducer of the liver CYP1A2 enzyme increasing caffeine metabolism by 60–70% (Zevin & Benowitz, 1999). Thus, some increased intake of caffeine among smokers is pharmacokinetic; i.e. smokers need to drink more coffee to achieve the desired concentrations/effects of caffeine (Benowitz et al. 1989; Benowitz et al. 2003).

A limitation of prior schizophrenia caffeine research studies is that they have not controlled for smoking, despite known pharmacokinetic effects. Given high rates of smoking in schizophrenia, studies need to examine caffeine intake and smoking simultaneously. The primary objective of this study was to measure serum caffeine levels in smokers with schizophrenia compared to controls without mental illness who smoked the same amount. We also examined the effect of dose and type of antipsychotic medication on caffeine level in the subsample of smokers with schizophrenia.

2. Methods

This study is a secondary data analysis of serum caffeine levels in outpatient smokers with schizophrenia/schizoaffective disorder (SCZ/SA) compared to control smokers without mental illness (CON). The study was approved by the UMDNJ Institutional Review Board.

2.1 Subjects

This study used data from smokers with and without schizophrenia who participated in quit smoking studies or studies of nicotine intake, that required them to have blood drawn for nicotine and are described elsewhere in greater detail (Williams et al 2005, Williams et al., 2007). We excluded subjects with a serum caffeine level of less than 1.0 ng/mL, who presumably were non-caffeine users. Thus, analyses for the purpose of this study were performed using data from the 167 eligible caffeine users (104 SCZ/SA; 63 CON).

2.2 Procedures

After signing the consent forms, subjects completed an assessment battery including a smoking history, tobacco use (measured as cigarettes per day; CPD), demographic and medication

questionnaire, and the Fagerstrom Test for Nicotine Dependence (FTND; Heatherton et al., 1991). Antipsychotic medication dose was converted to chlorpromazine equivalents (CPZ; Woods 2003). All subjects participated in an afternoon blood draw for nicotine, cotinine and caffeine to control for the time of the day effect. Participants also provided an expired carbon monoxide reading using an EC-50 Smokerlyzer (Bedfont Scientific, Williamsburg, VA, USA) on a usual smoking day. For subjects enrolled in the quit smoking study this was done prior to setting the quit date. Blood was centrifuged for 15 min and 5 mL of serum was drawn off and frozen at -20°C for later analysis. Specimens were numbered to protect the anonymity of subjects and sent to the Clinical Pharmacology Laboratory at the University of California, San Francisco for analysis of caffeine, nicotine and cotinine which were quantified by gas liquid chromatography technique using nitrogen phosphorus detection, modified from the assay of Jacob et al (1981).

2.3 Statistical analysis

Independent sample t-tests (for continuous variables) and Chi-square tests (for categorical variables) evaluated baseline differences between SCZ/SA and CON for their socio-demographic, smoking history and clinical variables. Mean serum caffeine levels were compared between the SCZ/SA and CON groups using independent sample t-tests. Analysis of variance (ANOVA) was used to compare group differences in the number of cigarettes smoked per day ($<20\text{CPD}$, $20\text{--}30\text{CPD}$, $>30\text{CPD}$). Two separate backward stepwise linear regression models were fitted to identify predictors of serum caffeine levels. The first model included the entire sample, whereas the second one included only smokers with SCZ/SA. The variables entered into the model included age, gender, race, education, employment status, marital status, cigarettes per day, age of onset of smoking, expired carbon monoxide (in ppm), Fagerstrom summary score, time to first cigarette of the day, number of past quit attempts, time of blood draw, and smoking mentholated cigarettes. In addition to the above variables, antipsychotic medication type (typical versus atypical), chlorpromazine equivalent (CPZ) and the specific diagnosis of schizophrenia (SCZ) compared with schizoaffective disorder (SA) were included only into the second regression model. We also analyzed caffeine levels in subgroups of patients who were taking antipsychotic medications (olanzapine, clozapine, haloperidol and fluphenazine) that were metabolized through the CYP1A2 enzyme to examine difference in dosing equivalents in these groups and determine possible caffeine-medication interactions. All analyses were performed using SPSS 17.0 (Chicago, IL, USA) and SAS 9.1 (Cary, NC, USA).

3. Results

3.1 Comparisons between Smokers with Schizophrenia or Schizoaffective Disorder versus Control Smokers

Smokers with SCZ/SA were slightly older and the group was comprised of more men than CON smokers (See Table 1). Smokers with SCZ/SA had higher mean serum cotinine level at baseline as compared to CON (289.2 vs. 232.1 ng/mL; $p=0.017$). No differences were detected between groups (SCZ/SA vs. CON) on CPD and expired CO at baseline.

3.2 Mean serum caffeine levels among SCZ/SA vs. CON

Serum caffeine levels were significantly higher in the SCZ/SA group as compared to CON (mean caffeine 2722 versus 1122 ng/mL; $t=-4.30$; $p<0.001$; Figure 1). There were no significant differences in serum caffeine levels between subjects with a diagnosis of schizophrenia vs. those with schizoaffective disorder (mean caffeine 2690 versus 2817 ng/mL; $t=-0.205$ $p<0.838$).

Since it is not known that induction of CYP1A2 by tobacco is dose-dependent (i.e. if heavier smokers have greater CYP1A2 induction than light smokers) we conducted additional analyses looking at serum caffeine levels among different CPD categories (<20CPD, 20-30CPD, >30CPD). We found significant group differences between the CPD categories ($F=4.803$, $DF=5$, $p<0.001$), thus suggesting a dose-dependent effect in both groups. In multiple comparison analysis using Bonferroni correction, however, mean caffeine levels in smokers with SCZ/SA of 20-30 CPD were only significantly different from CON who smoked the same amount (2743 vs. 1170 ng/mL; $p=0.013$). In addition, SCZ/SA who smoked more than 30 CPD, had significantly greater mean serum caffeine levels as compared to only the control groups who smoked <20 CPD (3430 vs. $p=0.01$) and those who smoked 20-30 CPD ($p=0.004$). This suggests that despite a trend, we observed no significant dose dependent effect for the amount of smoking on the serum caffeine level in our sample.

3.3 Summary of Backward Stepwise Linear Regression Analysis for Variables Predicting Caffeine Levels

Backward stepwise linear regression analyses showed that the diagnosis of schizophrenia/schizoaffective disorder (SCZ/SA; $B=1528.2$; $SE\ B=405.716$, $p<0.001$) was the only significant determinant of serum caffeine level. Repeated analysis limited to only the SCZ/SA group (excluding control smokers) showed that CPZ equivalents was a significant predictor of caffeine in these analyses ($B=1.470$; $SE\ B=0.653$; $p=0.027$), such that greater doses of antipsychotic drugs were associated with higher caffeine levels.

3.4 Other Effect of Antipsychotic Medication on Caffeine Level

We conducted additional analyses to examine the possible effects of antipsychotic medication type on caffeine levels in smokers with SCZ/SA. We divided subjects with SCZ/SA into 2 groups: those who were taking at least one antipsychotic medication primarily metabolized by CYP1A2 (olanzapine, clozapine, haloperidol and fluphenazine; $N=56$; Bozikas et al., 2004) and those who were taking antipsychotic medications metabolized through other pathways (i.e. all others; $N=48$). The mean CPZ equivalents for subjects on the CYP1A2 metabolized antipsychotic medications was higher than in the non-CYP1A2 group (749.2 vs. 444.1 CPZ; $t=3.01$, $p<0.01$). This is not surprising since smoking induces CYP1A2 therefore increasing the metabolism of these medications by up to 40%, resulting in a need for higher doses (Zevin & Benowitz, 1999; Bozikas et al., 2004; Williams & Ziedonis 2004). Since serum caffeine levels were not different in SCZ/SA on the CYP1A2 metabolized vs. non-CYP1A2 antipsychotic medications (2837.1 vs. 2588.7; $p=0.64$), this suggest that CYP1A2 metabolized antipsychotic medications are not interfering with caffeine metabolism.

Since it has been suggested that use of caffeine in schizophrenia may be compensatory to reduce sedation from antipsychotic medications, we also examined the possible effects of sedating antipsychotic medications on caffeine levels in smokers with SCZ/SA. We ranked antipsychotics based on their sedating properties into three groups (high sedation: clozapine, quetiapine, olanzapine, chlorpromazine, molindone, thioridazine; moderate sedation: thiothixene, perphenazine, ziprasidone; low sedation: haloperidol, fluphenazine, trifluoperazine, risperidone, aripiprazole; Lublin et al., 2005; Casey 1997). There were no differences in mean caffeine levels among the three sedation subgroups (F -statistic=0.204; $DF=2$; $p=0.816$), even after adjusting for CPZ equivalents (F -statistic=0.019; $DF=2$; $p=0.891$).

DISCUSSION

This study expands upon previous research by documenting higher levels of serum caffeine in SCZ/SA compared to individuals without schizophrenia, and after adjusting for the metabolic

effects of smoking. We also observed a trend for dose dependence between the amount of cigarettes smoked and serum caffeine levels, and may have been limited in our ability to detect differences based on sample size and relatively few smokers in the low smoking groups (<15 CPD).

Strengths of our study include standardized conditions for sampling caffeine in conjunction with a larger study of smoking and nicotine intake that controlled for time of day. Diagnoses (or lack of diagnoses) were confirmed using a structured clinical interview (SCID-IV). Limitations of this study included inability to measure dietary intake of caffeine or quantification of caffeine metabolites.

Possible explanations for why caffeine levels are higher among SCZ/SA include: 1) a metabolic interaction with psychiatric medications; 2) an attempt to reduce the sedating side effects of antipsychotic drugs; and 3) a disease-related difference in reinforcing effects of caffeine. Although smokers with SCZ/SA in this study were taking antipsychotics and other psychotropic medications, it is unlikely that drug interactions impacted on these results. Additionally, we did not observe higher caffeine levels in individuals taking sedating medications, arguing against the idea that individuals with schizophrenia consume more caffeine to compensate for this medication side effect. We also did not find that taking older “typical” antipsychotic medication impacted caffeine level although one study did (Gurpegui et al., 2004). Of note, the majority of our sample were taking atypical antipsychotics (79%), consistent with national data (Leslie & Rosenheck 2003).

Although we did not measure illness severity in this study, other investigators have found an association between higher caffeine consumption and increased symptom level severity in schizophrenia (Lucas et al., 1990). It is possible that high amounts of caffeine consumed by smokers with SCZ/SA in our study made them symptomatically worse, but we had no evidence to support this premise.

Caffeine exerts its effects for the most part by competing for binding at the high-affinity adenosine receptor sites, acting as a non-specific adenosine receptor antagonist. Since adenosine is a neuromodulator with sedative, anxiolytic and anticonvulsant actions, caffeine’s activating effects can be conceptualized as antagonizing these endogenous inhibitory pathways. It has been hypothesized that adenosine dysfunction may contribute to the neurobiological and clinical features of schizophrenia (Lara et al., 2006; Ferre et al., 1994, Hughes et al., 1998). This would provide a self-medication rationale linking the illness to heavy use of caffeine.

Some of the most important effects of nicotine and caffeine come from their co-occurrence. Nicotine and caffeine are among the most commonly used substances in schizophrenia and their consumption is often characterized by concurrent use. Animal studies indicate that caffeine enhances the discriminative-stimulus effects of nicotine in rats trained to discriminate nicotine from saline (Gasior et al., 2002). This suggests that caffeine consumption may be a contributing factor in the onset, maintenance of or relapse to tobacco dependence. Caffeine also increases the reinforcement from nicotine (Jones & Griffiths, 2003; Shoaib et al., 1999). Smokers also consume more caffeine due to interacting metabolic effects described previously. Clinical effects of nicotine and caffeine are important and may complicate the interpretation of schizophrenia symptoms and antipsychotic medication side effects. Both substances are linked to altered inhibitory mechanisms in brain functioning yet few studies have examined both simultaneously.

Given their metabolic and symptomatic interactions, there is a need for future studies of schizophrenia to examine the effects of nicotine and caffeine simultaneously. Smoking studies in schizophrenia should consider the role of caffeine in the interpretation and measurement of

nicotine withdrawal symptoms since both could cause non-specific symptoms of agitation, irritability and poor concentration. Caffeine effects could also potentially confound tests of cognitive function in acutely abstinent smokers with schizophrenia. Finally, studies of diet, weight gain and obesity in schizophrenia should also include examination of both cigarette smoking and caffeine intake.

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Figure 1.

a. Mean serum caffeine levels (ng/mL) among smokers with SCZ/SA compared to CON. b.

Mean serum caffeine levels (ng/mL) among smokers with SCZ/SA by sedating properties of antipsychotic medications and adjusted for CPZ equivalents

*** $P < 0.001$

■ SCZ/SA □ CON

Table 1

Baseline characteristics of smokers with SCZ/SA compared to CON smokers (N=167)

	SCZ/SA (n=104) Mean (SD)	CON (n=63) Mean (SD)	P-value
Cigarettes per day	25.8 (13.2)	22.7 (8.6)	
Baseline CO (ppm)	23.3 (11.2)	22.0 (8.6)	
Serum Cotinine (ng/mL)	289.2 (161.2)	232.1 (112.8)	*
FTND	7.1 (2.0)	6.1 (2.5)	**
Years smoked	22.8 (10.2)	20.9 (11.8)	
Age of first smoking	15.1 (5.5)	15.0 (3.7)	
Age	44.2 (8.9)	39.6 (12.3)	**
Race/Ethnicity	Count (%)	Count (%)	*
African-American	18 (17.3)	19 (30.2)	
Caucasian	78 (75.0)	32 (50.8)	
Hispanic	4 (3.8)	8 (12.7)	
Asian	4 (3.8)	3 (4.8)	
Other	0 (0.0)	1 (1.6)	
Gender	Count (%)	Count (%)	**
Male	67 (64.4)	25 (39.7)	
Female	37 (35.6)	38 (60.3)	
Education	Count (%)	Count (%)	
No High school	27 (26.0)	9 (14.3)	
High school/GED	61 (58.7)	42 (66.7)	
Associate's degree	3 (2.9)	3 (4.8)	
Bachelors degree or higher	11 (10.6)	9 (14.3)	
Unknown	2 (1.9)	0 (0.0)	
Current Smoking Goal ^a	2.3 (1.5)	2.4 (1.4)	
Past quit attempts	3.76 (4.0)	2.54 (2.8)	*
Brand of cigarette mentholated	Count (%)	Count (%)	
No	45 (48.9)	23 (38.3)	
Yes	47 (51.1)	37 (61.7)	
Time of Day of Blood Draw	12:38pm (1:51)	12:45pm (1:58)	
CPZ Equivalent	609.1 (528.3)	-	
Antipsychotic type	Count (%)	-	
Typical	9 (8.7)		
Atypical	82 (78.8)		
Both	13 (12.5)		

p<0.001;**
p<0.01;*
p<0.05;

All others NSS

^a 1=to quit and stay off forever; 2=to not smoke for a limited time; 3=to be able to control how much I smoke; 4=to quit someday but not now; 5=to continue to smoke.