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# Increased caffeine and nicotine consumption in community-dwelling patients with schizophrenia

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**Abstract**

*Introduction:* It is known that people with schizophrenia make poor dietary choices and smoke at alarmingly high rates. There is also anecdotal evidence that they may ingest large amounts of caffeine. However, while smoking habits in this population have been examined, no recent study has quantified caffeine consumption taking into account various dietary caffeine sources unrelated to coffee including convenience foods such as candy bars, chocolate or soft drinks, and compared results to US population data.
*Methods:* We employed 24-h diet recalls to assess dietary habits in a sample of outpatients suffering from schizophrenia or schizoaffective disorder. Caloric intake and caffeine consumption were quantified and the relationship to various sociodemographic variables including body mass index (BMI) and dietary quality was examined.
*Results:* 146 patients were recruited. Mean BMI in the sample was 32.7±7.9. Patients ingested 3057±1132 cal on average. Patients smoked at higher rates (59.6% vs. 23.4%, $p \leq 0.001$), higher numbers of cigarettes/day (24±14.4 vs. 13.5±11.3, $t=8.549$, $p<0.001$) and ingested more caffeine (471.6±584.6 mg vs. 254.2±384.9 mg, $t=6.664$, $p<0.001$) than US population comparisons. Caffeine consumption was correlated to the number of cigarettes smoked daily ($r=0.299$, $p \leq 0.001$), but not to BMI ($r=0.134$, $p=0.107$) or dietary parameters such as caloric intake ($r=0.105$, $p=0.207$).
*Conclusion:* Community-dwelling schizophrenia patients consume significantly more caffeine and nicotine than US population comparisons. Clinicians should be aware that while a significant proportion of patients are overweight and have poor dietary quality – which merits lifestyle counseling on its own – there is a lack of correlation between those factors and smoking and caffeine intake. Thus, lifestyle modification counseling in all patients should address smoking and caffeine intake concurrently.


*Keywords:* Schizophrenia; Smoking; Nicotine; Addiction; Caffeine; Diet

## 1. Introduction

Nicotine and caffeine represent the two most widely self-administered mood-altering substances in western countries. Schizophrenia patients may consume large quantities of both substances (Rihs et al., 1996). Caffeine (1,3,7-trimethylxanthine) is the main psychoactive ingredient in coffee, soft drinks and tea, but is also increasingly found as an additive to a variety of liquid and solid foods, including soft- and energy drinks, iced tea, chocolate, dairy products, pies, cakes, brownies, breakfast bars, candy and syrup, to name

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but a few (Frary et al., 2005). Nicotine is the major active ingredient in tobacco, and the predominant reason to persistence of smoking and development of addiction (Benowitz, 1988). Caffeine interacts with nicotine's central pharmacologic action and increases subjective reports of positive effects of nicotine (Tanda and Goldberg, 2000), thereby acting as reinforcer further solidifying nicotine's addictive properties (Shoaib et al., 1999).

While smoking rates in the general population (currently at approximately 21%; Maurice et al., 2005) have been on a steady decline (Levy et al., 2005), similar changes have not been reported for patients with schizophrenia. De Leon et al. (2002a), for example, reported smoking rates between 74% and 83% in long-term hospitalized schizophrenia patients, and McCreadie et al. (2003) estimate that about 70% of community-dwelling individuals smoke, with half of them (54%) smoking heavily (20 cigarettes/day or more). Even compared to patients with other mental illnesses such as bipolar or mood disorders, schizophrenia patients seem to have higher proportions of smokers (Uck et al., 2004; De Leon et al., 2002b), a fact that has been linked to a proposed higher subjective reward value of smoking for the schizophrenic patient (Spring et al., 2003).

Caffeine has a variety of important psychotropic effects as well, and at low to moderate doses increases alertness and attention and reduces fatigue, while chronic use may produce withdrawal symptoms such as headaches, transient cognitive impairment and dysphoria if discontinued rapidly (Rogers et al., 2005). After high doses (>250 mg, at least 2–3 cups of brewed coffee), anxiety, agitation, irritability and insomnia may ensue; the symptoms may meet DSM-IV criteria for, i.e., caffeine intoxication, caffeine-induced anxiety disorder or caffeine-induced sleep disorder. There are observations that schizophrenic individuals may ingest large amounts of caffeine (Kruger, 1996), sometimes until they are acutely intoxicated (Koczapski et al., 1989). A few patients have even shown quite peculiar behavior when it comes to caffeine consumption such as eating ground coffee (Benson and David, 1986). It has been suggested that acute but not chronic intake of large amounts of coffee may significantly alter psychopathology and even exacerbate psychotic symptoms (Lucas et al., 1990; Mayo et al., 1993). Chronic intake of large quantities has been linked to more subtle changes such as interaction liabilities with psychoactive medications on CNS level (Donovan and DeVane, 2001) and on peripheral drug metabolisation in liver and kidneys (Carillo and Benitez, 2000). Chronic caffeine intake may, through competitive inhibition at the liver cytochrome system cytochrome P450 system, elevate blood levels of antipsychotic medication, thereby increasing the likelihood of unwanted side-effects. When paired with long-term nicotine exposure, some of these effects may be counteracted, since smoking byproducts, when chronically administered, induce liver cytochromes, especially isoenzyme CYP1A2 (Brown and Benowitz, 1989), and reduce both caffeine and antipsychotic blood levels.

There is much evidence that patients with Schizophrenia make poorer dietary choices than people in the population would make and that they have overall higher dietary intake (Strassnig et al., 2003). Whether and how this relates to caffeine and nicotine consumption is unclear. To our knowledge, the question whether schizophrenia patients have higher caffeine intakes than population controls incorporating the large amounts of caffeine derived from dietary sources other than coffee has not systematically been pursued, and relationships, if any, between smoking, caffeine intake and sociodemographic and clinical factors such as dietary quality, caloric intake or choice of antipsychotic medication have not been examined in schizophrenia patients before. For this cross-sectional study, we investigated those factors in a sample of stable community-dwelling patients in comparison to US population intake data for caffeine and nicotine, respectively.

## 2. Methods

### 2.1. Subjects

Patients with a DSM-IV TR (American Psychiatric Association, 2000) diagnosis of schizophrenia, schizoaffective disorder or psychotic disorder NOS were recruited from the outpatient clinic and partial hospital at Western Psychiatric Institute and Clinic (WPIC) Services and Research for Recovery in Severe Mental Illness outpatient program by their therapists, psychiatrists and nurses. The study subjects were a convenience sample. Subjects were referred by their clinicians or referred themselves to the study, in response to notices posted around the clinic, and all patients willing to participate in the study were invited to the interview. Diagnoses were confirmed by patient chart review. Assessments were carried out after obtaining written informed consent according to procedures approved by the University of Pittsburgh Biomedical Institutional Review Board. The procedure took about 45 min per participant and yielded data in three different domains: (i) sociodemographics and pertinent clinical information including tobacco and antipsychotic use; (ii) dietary

intake including caffeine intake; and (iii) anthropometric measures.

### 2.2. Data collection

Sociodemographic information (i) was obtained by open ended questions and, in case of missing values, data was completed by patient chart review. Smoking behavior was elicited by a semi-structured questionnaire and pts were asked (a) …"Are you currently using tobacco?", (b) "What are you using (i.e., cigarettes, chewing tobacco, pipe, cigars)?", (c) "If you are using cigarettes, how many cigarettes are you smoking per day" and (d) "How long have you been smoking?" Smoking was stratified into nonsmoker, 1–20, >21 cigarettes/day. A 24-h diet recall using standardized food models was used to collect the nutritional information, including caffeine intake (ii). This method is widely regarded as suitable for the purpose of gathering dietary intakes (Young et al., 1952; Madden et al., 1976) and has been employed throughout the NHANES I–IV (National Health and Diet Examination Surveys; Kohlmeier, 1992). To compute nutritional information gathered, we used the commercially available ESHA Food Processor Nutrition software 7.5 (Lee et al., 1995). Total caloric intake was calculated and saturated fat and trans-fat intakes were used as proxy measures of dietary quality (Kant, 1996). The software allowed for calculation of total caffeine intake which comprised not only intake of coffee but also caffeine derived from dietary sources other than coffee, such as tea, soft drinks, chocolate and energy drinks, iced tea, dairy products, pies, cakes, brownies, breakfast bars, candy and syrup, etc. Weight and height were recorded (iii) in kilograms (kg) and meters (m) and BMI was calculated ($kg/m^2$).

## 3. Data analysis

SPSS (for Macintosh) software was employed for data analysis. Descriptive analysis including mean, range and standard deviation for continuous variables was carried out to determine whether the variable(s) was (were) normally distributed and frequency counts for categorical data (for example, gender, race, etc.) were done to examine the proportions of various sociodemographic characteristics. The measures obtained through the 24-h diet recall were examined for the whole group and for groups defined by different demographic characteristics (for males and females, Caucasian and African–Americans). The participant's caffeine intake was compared to age-and gender-adjusted US population data as reported by Frary et al. (2005), who have recently established updated baseline caffeine intake information for the US population by analyzing data from the vast USDA (US Department of Agriculture, Food Survey and Research Group, 2000) CFSI database (2000). We also converted caffeine intake into standardized cup-sizes (85 mg caffeine/5 oz) according to Barone and Roberts (1996) and Hughes and Oliveto (1997), to give a more practical estimate of patients' caffeine consumption. Population smoking data was derived from 2004 CDC data (Trosclair et al., 2005) and from the U.S. Dept. of Health and Human Services, Substance Abuse and Mental Health Services Administration, NATIONAL SURVEY ON DRUG USE AND HEALTH (2003). Neither pipe nor cigar or chewing tobacco use was analyzed, as none of the participants indicated use of either form of tobacco. Student's *t*-tests and where appropriate chi-square and Fisher's exact tests, and ANOVA were employed to look for statistical differences between the means and proportions of two or more variables. Correlations were examined for statistical significance using Pearson's correlation coefficients.

## 4. Results

One hundred and forty-six subjects were recruited. Average age in the study sample was 43.3 (±8.9) years. Seventy-eight patients (53.4%) were male; one hundred and seven (73.3%) were single. DSM-IV diagnoses were schizophrenia ($n=69$, 47.3%), schizoaffective disorder ($n=53$, 36.3%) and psychotic disorder NOS ($n=24$, 16.4%). Mean caloric intake was 3056±1132 and mean body mass index was 32.8 (±7.8). Patients had a mean monthly income of 625±260 USD. There was a trend for males to consume more calories than females (3201.3±1084.2 vs. 2890.6±1171, $t=1.7$, $p=0.098$). Body mass index (BMI) of female subjects was significantly higher than that of males (35.1±8 vs. 30.8±7.3, $t=-3.133$, $p=0.002$). No differences in BMI (33.3±8.6 vs. 31.9±6.9, $t=1.1$, $p=0.268$) or caloric intake (3021.2±1169 vs. 3098.4±1094, $t=-0.409$, $p=0.683$) were found between Caucasians and African–Americans.

Patients had higher smoking rates and caffeine intake than population controls (Table 1).

There were no significant correlations between caffeine intake (c) or cigarette consumption (n) on dietary quantity (3056±1132 cal, $r=0.011$, $p=0.207$ (c); $r=0.09$, $p=0.277$ (n)), or dietary quality in terms of saturated fat intake (41.99±24.13 g, $r=-0.102$, $p=0.22$ (c); $r=-0.035$, $p=0.67$ (n)) and trans-fat intake

Table 1
Caffeine and nicotine consumption in patients vs. US population controls

| | Caffeine (mg, S.D.), 5 oz cups | | *t*-stats | Smoking (%, CI), 95% confidence interval | | $\kappa^2$, *p* | Cigarettes/day [a] | | *t*-stats |
|---|---|---|---|---|---|---|---|---|---|
| | Patients | Controls [b] | | Patients | Controls [c] | | Patients | Controls | |
| All | 471.6 (±584.6), 5.6 (±6.9) | 254.2 (±384.9), 3.0 (±4.5) | $p \leq 0.001$, $t=6.664$ | 59.6 (±8.1) | 23.4 (±0.9) | <0.001 | 24 (±14.4) | 13.5 (±11.3) | $p<0.001$, $t=8.549$ |
| Male | 450 (±529.2), 5.3 (±6.2) | 288.4 (±422.6), 3.4 (±4.8) | $p \leq 0.001$, $t=3.321$ | 58.9 (±11.2) | 18.5 (±0.7) | <0.001 | 23.8 (±14.2) | 13.6 (±11.7) | $p<0.001$, $t=5.888$ |
| Females | 496.3 (±645.4), 5.8 (±7.6) | 218.5 (±337.6), 2.6 (±4.0) | $p \leq 0.001$, $t=6.551$ | 60.3 (±10.8) | 20.9 (±0.6) | <0.001 | 24.2 (±14.9) | 12.2 (±10.8) | $p<0.001$, $t=6.993$ |

[a] Smokers in the sample ($n=86$) had smoked for 22.1(±10) years.
[b] Compared to US population caffeine intake data derived from Frary et al. (2005).
[c] Compared to age-adjusted data derived from the NATIONAL SURVEY ON DRUG USE AND HEALTH (2003).

(2.58±3.66 g, $r=-0.003$, $p=0.973$ (c); $r=-0.074$, $p=0.372$ (n)). Twelve people in our sample of schizophrenia patients had no exposure to caffeine (8.2%). One hundred and twelve patients (76.7%) had had exposure to more than one standardized cup of coffee and 19 patients had daily ingestion of more than 1000 mg of caffeine. The highest observed total caffeine intake was 2647.2 mg/day.

Longer smoking history was strongly correlated to number of cigarettes smoked ($r=0.688$, $p \leq 0.001$). Caffeine intake was correlated to number of cigarettes smoked ($r=0.242$, $p=0.025$). Non-smokers had a mean caffeine intake of 323.8±375.8 mg (3.8 standardized cups/coffee), light smokers 494.9±680 mg (5.8 cups) and heavy smokers 733.8±645.6 mg (8.6 cups). Income was not correlated with number of cigarettes smoked ($r=-0.075$, $p=0.372$) or caffeine intake ($r=-0.016$, $p=0.848$). More African–American patients (76%, $n=49$ of 67) than Caucasian–Americans (48%, $n=38$ of 79) smoked ($P2=9.4$, $p=0.002$). However, Caucasians were significantly heavier smokers (31.5±16.8 vs. 18.3±9 cigarettes/day, $t=4.7$, $p<0.001$). Similarly, caffeine intake was higher in Caucasian (618.8±707.8 mg) than African–American patients (298±319.7 mg, $t=3.4$, $p=0.001$). Males and females had similar caffeine intake (450±496.3 mg vs. 496.3±645.4 mg, $t=-0.475$, $p=0.635$) and smoked similar numbers of cigarettes per day (14.03±16 vs. 14.5±16.4, $t=-0.187$, $p=0.852$).

## 5. Discussion

This study shows that community-dwelling patients with schizophrenia smoke at much higher rates, smoke more cigarettes and consume more caffeine than comparable individuals in the US population. From our results, it appears that patients consume approximately twice the caffeine and twice as many cigarettes, and have two- to three-fold higher smoking rates. More than 90% of patients had at least some caffeine intake, and in quite a few (13%), caffeine intake exceeded 1000 mg/day (equivalent to approximately 12 cups). No evidence for associations between caffeine intake and cigarette smoking with sociodemographic and clinical factors such as dietary quality and caloric intake was found. The finding that Caucasians smoked even more and ingested more caffeine than African–Americans is consistent with previous population-based findings (Klesges et al., 1994). Yet, while the difference in caffeine intake likely represents different dietary preferences (Knight et al., 2004), lower nicotine intake in African–Americans may indicate underlying metabolic differences affecting tolerability (Perez-Stable et al., 1998). While acute ingestion of caffeine or nicotine in the quantities recorded in our sample is known to produce clinically changes in psychopathology such as increased anxiety or insomnia, a chronic ingestion pattern probably results in habituation and tolerance. However, abrupt reduction or cessation of intake is likely to produce withdrawal symptoms. Such a scenario may occur during inpatient admission, when access to both tobacco and caffeine is tightly regulated, and may contribute to behavioral problems in these settings. To our best knowledge, this is the only study assessing dietary intake including caffeine intake via a 24-h diet recall which enabled us to incorporate the large amounts of caffeine derived from other dietary sources than coffee, rather than simply basing caffeine intake on proxy estimates such as assessing the number of cups of coffee drunk during any given day or week.

In a western European sample of schizophrenia patients requiring acute hospitalization, Rihs et al. (1996) reported mean daily intakes of approximately 400 mg caffeine and 20 cigarettes. Caffeine intake was estimated by extrapolating from self-reported intake of cups of coffee per day, which, while serving well as a

rough assessment of coffee intake, may fail to account for different caffeine concentrations per cup, and does not incorporate other dietary caffeine sources (such as from soft drinks, tea or other caffeine-containing foods). There was a significant drop in cigarette and caffeine consumption once patients were hospitalized. As in our sample, no variations in caffeine or nicotine consumption by type of antipsychotic used were found, and a strong relation between smoking and caffeine intake was evident. Smoking rates in our sample were not much different from those reported from other surveys (De Leon et al., 2002a; McCreadie et al., 2003), our patients smoked roughly similar numbers of cigarettes to those reported by Rihs and colleagues and elsewhere (Herran et al., 2000). A more recent survey of caffeine intake in Spanish outpatients with schizophrenia (Gurpegui et al., 2004) also reported high rates of caffeine and nicotine consumption, yet used a different method to elicit caffeine data and thus did not report absolute mean daily caffeine intakes.

A variety of reasons may account for the high concurrent prevalence of caffeine and nicotine consumption in patients suffering from schizophrenia. It has been hypothesized, for example, that patients smoke to ameliorate anhedonia reduce medication side effects (Goff et al., 1992) or self-medicate to improve cognitive symptoms (Dervaux et al., 2004; Myers et al., 2004). Importantly, there are quite powerful pharmacokinetic effect of nicotine on caffeine, since smoking byproducts induce the liver cytochrome P450 isoenzyme CYP1A2 (Brown and Benowitz, 1989), the enzyme that is critically involved in degradation of caffeine. Patients, when smoking heavily, may thus need to ingest more caffeine than people in the general population to reach the same desirable positive mild psychostimulant effects, which may in part account for the strong correlation of use between both substances. For similar pharmacokinetic reasons, heavy smoking may also decrease the patient's serum load of a variety of psychotropic drugs which are metabolized through CYP1A2 including clozapine and olanzapine (De Leon, 2004). Alternatively, smoking may simply represent one of the few remaining activities schizophrenia patients experience as pleasurable (Gopalaswamy and Morgan, 1986). Centrally, nicotine stimulates the subcortical reward system and areas in the prefrontal cortex, which both appear hypofunctional in schizophrenia, thus ameliorating dopamine deficits in areas crucial to schizophrenia symptomatology. Indirectly, through actions on nicotinergic cholinergic receptors, nicotine increases firing of dopamine neurons in mesocorticolimbic pathways, enhancing dopamine release in both the nucleus accumbens and prefrontal cortex (Spring et al., 2003). Similarly, caffeine also has major pharmacological effects on the central nervous system and decreases presynaptic inhibition of dopamine release mediated by adenosine A2A receptors, or increases the release of especially glutamate through A1 receptor blockade (Cauli and Morelli, 2005). Caffeine and nicotine may thus synergistically exert psychostimulant effects through enhancement of dopaminergic transmission (Tanda and Goldberg, 2000); the major neurotransmitter system implicated in the pathophysiology of schizophrenia and also main target for antipsychotic treatment. Caffeine and nicotine also complexly interact on neurotransmitter level, and chronic low-dose caffeine administration potentiates nicotine's stimulatory effects through neurobiological changes in brain dopaminergic activity (Jones and Griffiths, 2003), which may also in part account for the strong interdependence between chronic use of both substances as found in our sample.

Our sample also showed that the longer patients smoked, the more they tended to smoke, thereby only increasing potential for prospective health problems in context of their already poor health status (Strassnig et al., 2005). Simply put, apart from the reasons discussed above, high smoking rates may at least partially indicate a lack of intervention from the health care providers as well (Himelhoch and Daumit, 2003), and high smoking rates in the schizophrenia population should not lead to the notion that smoking is inevitable and sometimes useful in psychiatric disorders (Srinivasan and Rhara, 2002). Unfortunately, quit rates among smokers with schizophrenia appear to be considerably lower than for other psychiatric diagnoses (Ziedonis and George, 1997; Ziedonis et al., 1994). Inquiry about caffeine and nicotine consumption is all but a first step towards identifying the problem and should be done routinely.

In summary, this study shows that community-dwelling patients with Schizophrenia smoke at much higher rates, smoke more cigarettes and consume more caffeine than US population comparisons. No evidence for associations between caffeine intake and cigarette smoking with sociodemographic and clinical factors such as dietary quality or caloric intake was found. The strong correlation between smoking and caffeine intake and concurrent lack of association with other dietary factors such as caloric intake and BMI, or dietary quality as expressed by saturated and trans-fat intakes highlights the need for lifestyle modification counseling which addresses smoking and caffeine intake concurrently.

## 6. Limitations

The study was a cross-sectional analysis of a stable outpatient population suffering from schizophrenia. While we believe that the reported dietary intakes of our subjects were stable at time of assessment, the validity of a dietary recall in schizophrenia particular population has not been established. The population sample data did not allow for stratification of caffeine use by smoking status. Patients were interviewed during weekdays, and while smoking rates may be constant throughout a full week, caffeine intake may differ between weekdays and weekends. Unfortunately, financial constraints associated with the study did not allow for measurement of plasma caffeine levels, which would have reflected much better the amount of caffeine ingested. Additionally, patients in our sample were predominantly overweight or obese. However, this was not a representative sample obtained by random selection, but rather a sample of convenience made up of volunteers from our outpatient program. Therefore, the results may not be applicable to the entire patient population, but may be appropriate for the large number of overweight or obese patients with Schizophrenia encountered in outpatient settings.

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