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Effects of smoking cues on caffeine urges in heavy smokers and caffeine consumers with and without schizophrenia

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ABSTRACT

Cigarette smoking and caffeine use are established and problematic drug-use behaviors in people with schizophrenia. Associative links between drugs of abuse may occur but the relationship between caffeine use and cigarette smoking has received little attention in schizophrenia. In this cross-cue reactivity laboratory study, we examined the effects of neutral and smoking cues on craving for caffeinated beverages in participants with schizophrenia or schizoaffective disorder (SS; $n=15$) and non-psychiatric controls (CS; $n=18$) all of whom were heavy smokers and daily caffeine users. Participants were tested under non-abstinent and 5-hour abstinent conditions. SS tended to report greater daily levels of caffeine use than CS. Although this difference was not significant, that may be due to the small sample sizes as the size of this effect was large. Daily caffeine intake was significantly correlated with daily smoking rate in SS but not CS. A significant interaction between group and cue type after controlling for caffeine intake indicated that exposure to smoking cues increased urge for caffeinated beverages in SS but not CS. These results indicate support for associative connections between cigarette smoking cues and craving for caffeine in smokers with schizophrenia.

1. Introduction

In people with schizophrenia, substance abuse is an established and problematic behavior that is related to poorer overall mental and physical health functioning (Winklbaur et al., 2006). For example, recent attention has pointed to increased rates of cigarette smoking (de Leon and Diaz, 2005; Kotov et al., in press), alcohol (Eriksson et al., 2007), cocaine (Shaner et al., 1995) and marijuana (Degenhardt and Hall, 2002) use in this population. Elevated caffeine use has also been reported (e.g., Benson and David, 1986; Zaslove et al., 1991). Cross-sectional studies have found that people with schizophrenia are more likely to be daily consumers of caffeinated beverages, or are more likely to consume higher caffeine doses, than those without psychiatric illness (Gurpegui et al., 2006; Hughes et al., 1998; Rihs et al., 1996; Schneier and Siris, 1987; Strassnig et al., 2006). High levels of caffeine use can be problematic for people with schizophrenia as caffeine can increase psychiatric symptoms, antipsychotic plasma levels, and medication side effects (de Leon 2004; Hughes et al., 1998).

People with schizophrenia may consume high levels of caffeine for a number of reasons. Caffeine indirectly increases dopamine (DA) neurotransmission (Tanda and Goldberg, 2000), and functional hyperactivity of the mesolimbic DA system associated with schizophrenia may predispose these patients toward experiencing stronger reinforcing effects of DA-releasing drugs and drug-related cues (Chambers et al., 2001). People with schizophrenia also may use caffeine for its beneficial effects on alertness and cognition (Haskell et al., 2005). Furthermore, schizophrenia is associated with polydipsia, which may non-specifically increase caffeine consumption (de Leon et al., 1994, 2002).

Another likely contributor is the high rate of cigarette smoking in this population, as caffeine and cigarette consumption are strongly associated in smokers with schizophrenia and in non-psychiatric smokers (Gurpegui et al., 2006; Istvan and Matarazzo, 1984; Strassnig et al., 2006).

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Several mechanisms may underlie this association. Although the genetic correlation between use of these substances appears to be modest (Kendler et al., 2008), impulsivity is associated with heavy use of both substances (Gurpegui et al., 2007). Smoking induces the cytochrome P450 isoenzyme CYP1A2, which metabolizes caffeine, so that smokers require more caffeine than non-smokers to obtain the desired effects of caffeine (de Leon et al., 2003). Furthermore, caffeine enhances the stimulating and reinforcing effects of nicotine in preclinical animal models (Shoaib et al., 1999; Tanda and Goldberg, 2000) and controlled human laboratory studies (Duka et al., 1998; Jones and Griffiths, 2003; Perkins et al., 1994), albeit not consistently (e.g., Chait and Griffiths, 1983; Perkins et al., 2005).

Naturalistic studies indicate that caffeine and nicotine are often used at the same time (Emurian et al., 1982; Nellis et al., 1982; Marshall et al., 1980). Through associative learning during their concurrent consumption, smoking-associated stimuli should come to trigger urges for caffeine use and vice versa, just as smoking cues increase craving for alcohol and vice versa in people who use those substances (e.g., Colby et al., 2004; Drobes, 2002). In this study, we examined the effects of smoking cues on urge to consume caffeinated beverages in smokers with schizophrenia (SS) and equally-heavy control smokers without psychiatric illness (CS), all of whom were daily caffeine users. We hypothesized that smoking cues would increase caffeine urges due to associative learning processes arising from concurrent self-administration of these substances. Based on the theory that schizophrenia is associated with increased responsivity to drugs and drug-related cues (Chambers et al., 2001), we further hypothesized that SS would have higher caffeine urges and would be more prone to experiencing effects of smoking cues on caffeine urges than CS. Participants were studied under smoking-abstinent and non-abstinent conditions to determine whether smoking cessation would exacerbate urges for caffeine in these smokers.

2. Methods

2.1. Participants

This study was conducted during a larger study that investigated the effects of a medication on smoking cue reactivity (Tidey et al., 2006); participants in the current study were tested under non-medicated conditions. Participants with schizophrenia or schizoaffective disorder (SS) and non-psychiatric control participants (CS) were recruited from the community using flyers and newspaper advertisements. All were at least 18 years old, had smoked between 20 and 50 cigarettes per day for the past year, had expired breath carbon monoxide levels of 18 ppm or more, were daily caffeine users, and had Fagerström Test for Nicotine Dependence (FTND) scores of 6 or more (Heatherton et al., 1991). The Structured Clinical Interview for DSM-IV (SCID; First et al., 1996) was used to confirm diagnoses of schizophrenia or schizoaffective disorder and rule out current Axis I disorders in control participants. Participants were excluded if they were pregnant or nursing, using alternative tobacco products; had breath alcohol levels of .005 g/l or more; had current substance abuse or dependence other than nicotine; were actively trying to quit smoking, were currently using medications known to affect cigarette smoking, or if the severity of their psychiatric symptoms would interfere with the completion of study procedures. Sixteen out of 18 (89%) of SS and 19 out of 25 (76%) of CS screened for this study met the daily caffeine use criterion and were enrolled; this difference was not significant (X^2 (1, N=43)=1.15, p=.28). All study procedures were approved by the institutional review boards of the medical center and university involved.

2.2. Individual difference measures

Information was collected on demographic variables, smoking histories and caffeine use histories. Current psychiatric symptoms for participants with schizophrenia or schizoaffective disorder were assessed using the Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987) and the Brief Psychiatric Rating Scale (BPRS; Overall and Gorham, 1962). Current depression was assessed in all participants, using the Hamilton Depression Rating Scale (HAM-D; Hamilton, 1960).

2.3. Cue reactivity assessment

Using a within-subjects design, responses to smoking cues were assessed under 5-h smoking abstinent and non-abstinent conditions, with two replications of each condition, for a total of 4 study sessions. Participants arrived at the laboratory for each session at approximately 8 a.m. They were instructed to follow their typical smoking and caffeine use patterns prior to arrival at the laboratory. Upon arrival, participants provided breath samples for the assessment of carbon monoxide levels (Smokerlyzer, Bedfont Scientific Ltd., Kent, UK). For the next 5 h, participants remained within the laboratory and were either permitted to smoke freely (non-abstinent condition) or were not allowed to smoke (abstinent condition). During this time, participants were allowed to read magazines and/or watch movies that were determined to contain no smoking cues. Participants were under continuous observation except for bathroom and lunch breaks, with CO monitoring to assure compliance with abstinence. Participants were not permitted to drink caffeinated beverages during lunch breaks.

At the end of this 5-h period, breath CO levels were assessed, and the cue reactivity trial commenced as described previously (Tidey et al., 2005, 2008). Following a 10-minute relaxation period, urge for smoking was assessed (data not shown), and urge for caffeine was assessed using the question "How strong is your urge for a caffeinated beverage such as coffee, tea or cola right now?", rated on a 100 mm visual analogue scale (VAS), with the anchors 0="no urge at all" and 100="strongest urge you've ever had". Participants were then presented with the neutral cues (pencil, 25 mm×65 mm eraser and a small pad of paper) on a covered tray. An audiotape instructed participants to lift the cover, look at and handle these cues. After 4 min had elapsed, participants were asked to complete the smoking and caffeine urge measures. The neutral cues were then replaced with the smoking cues (cigarette, lighter and ashtray) on a covered tray. Participants listened to audiotaped instructions to lift the cover and look at the cues. After 2 min elapsed, participants were asked to light the cigarette without inhaling. After 2 more minutes elapsed, participants were asked to extinguish the cigarette and to complete the smoking and caffeine urge measures. The