Smoking topography and outcome expectancies among individuals with schizotypy

Diana W. Stewart a,*, Christine Vinci b, Claire E. Adams a, Alex S. Cohen b, Amy L. Copeland b

a The University of Texas MD Anderson Cancer Center, Department of Health Disparities Research-Unit 1440, P.O. Box 301402, Houston, TX 77230-1402, USA
b Louisiana State University, Department of Psychology, Baton Rouge, LA, USA

Article info

Article history:
Received 9 February 2012
Received in revised form 12 November 2012
Accepted 20 November 2012

Keywords:
Cigarette smoking
Schizotypy
Schizophrenia

A B S T R A C T

Compared to smokers in the general population, smokers with schizophrenia smoke more cigarettes per day and have higher nicotine dependence and biochemical indicators of nicotine intake. They also have more intense smoking topography and greater positive smoking expectancies. Little is known about the relationship between smoking and schizotypy, defined as the personality organization reflecting a vulnerability to schizophrenia-spectrum pathology. This study assessed schizotypy symptoms, smoking characteristics and behaviors, and smoking expectancies in young adults with psychometrically defined schizotypy and demographically matched controls without schizotypy. Smokers with schizotypy had higher nicotine dependence and smoked more cigarettes per week compared to control smokers. They were also more likely to endorse greater positive consequences (i.e., improved state enhancement, stimulation, social facilitation, taste/sensorimotor manipulation, reduced negative affect and boredom) and fewer negative consequences of smoking. Smokers with schizotypy and control smokers did not differ on smoking topography or carbon monoxide levels. This is the first known study to investigate relationships between these smoking-related variables in smokers with schizotypy. Individuals with schizotypy possessed certain smoking-related characteristics and smoking expectancies similar to those with schizophrenia. This offers preliminary insight into unique smoking-related factors among individuals with schizotypy and highlights the importance of continued research in this area.

© 2012 Elsevier Ireland Ltd. All rights reserved.

1. Introduction

Cigarette smoking is the leading cause of preventable morbidity and mortality in the United States, accounting for approximately one-third of all cancer-related deaths (Mokdad et al., 2004; American Cancer Society, 2011). Although smoking prevalence has decreased over the past few decades in the general population, it remains disproportionately high for distinct groups such as those with severe mental illness (Lasser et al., 2000; Aubin et al., 2012). Smoking is particularly widespread in individuals with schizophrenia (Hughes et al., 1986; Workgroup on Substance Use Disorders, 2006). Up to 90% of those with schizophrenia smoke cigarettes (de Leon et al., 2002a; de Leon and Diaz, 2005). Accordingly, smokers with schizophrenia are 30% more likely than smokers in the general population to die from cardiovascular disease and 60% more likely to die from respiratory disease (Baxter, 1996; Dalack et al., 1998). The average life expectancy for individuals with schizophrenia is 20% lower than in the general population (Hennekens et al., 2005). Thus, understanding factors underlying poor health behaviors (particularly smoking) is critical toward reducing health disparities for this population. Perhaps subclinical symptomatology present prior to illness onset predisposes individuals to smoking. Knowledge on these issues might be gleaned by studying smoking characteristics of persons with schizotypy, who have a presumed genetic vulnerability to development of schizophrenia spectrum pathology but who do not yet manifest full-blown phenotypic expression of the disorder (e.g., Meehl, 1962). This is a particularly important area of research, as it avoids many confounds related to schizophrenia (e.g., illness chronicity, medication side effects) and might provide knowledge regarding particular motives and expectancies about the effects of smoking for individuals across the schizophrenia spectrum.

Compared to smokers without schizophrenia, smokers with schizophrenia smoke more cigarettes per day (CPD; de Leon et al., 1995; Ucok et al., 2004), endorse heavier smoking (≥25 CPD; Lasser et al., 2000), smoke stronger cigarettes (Olincy et al., 1997; Williams et al., 2007), are more nicotine dependent (Weinberger et al., 2007), and have lower cessation rates (Lasser et al., 2000). Further, they are efficient and intense smokers (Olincy et al., 1997;
Kelly and McCreadie, 1999; Strand and Nybäck, 2005; Tidey et al., 2005) who spend much of the day smoking, smoke their cigarettes down to the filter, and smoke discarded butts and filters, which contain greater concentrations of nicotine (Williams and Ziedonis, 2004). They also have higher nicotine and cotinine (an active metabolite of nicotine) levels (Olincy et al., 1997; Strand and Nybäck, 2005; Williams et al., 2005).

Moreover, smokers with schizophrenia differ from smokers without schizophrenia in smoking topography. Specifically, smokers with schizophrenia take more puffs per cigarette, have shorter inter-puff intervals, have larger puff volumes, and obtain higher carbon monoxide (CO) levels and boosts (Olincy et al., 1997; Hitsman et al., 2005; Tidey et al., 2005, 2008; Williams et al., 2006, 2010, 2011). A CO boost is the calculated difference between pre- and post-cigarette CO and is a biochemical indicator of smoke exposure (Zacyn et al., 1987).

Smokers with schizophrenia typically have more positive beliefs about the consequences of smoking, or smoking outcome expectancies (Glynn and Sussman, 1990; Lohr and Flynn, 1992; Glassman, 1993; Benowitz, 1999; McEvoy and Brown, 1999; Forchuk et al., 2002; Buckley et al., 2005; Esterberg and Compton, 2005; Barr et al., 2008; Solty et al., 2009; Tidey and Rohsenow, 2009). Smoking expectancies can be either positive (i.e., smoking facilitates social interactions, smoking reduces boredom and/or negative affect) or negative (i.e., smoking is harmful to health, others may disapprove of smoking). Higher negative expectancies predict greater intention to quit and successful smoking cessation (Brandon et al., 1999). Compared with smokers in the general population, smokers with schizophrenia typically report greater positive and fewer negative smoking expectancies. Data suggest that for smokers with schizophrenia, reduction of negative affect is the most predominant expectancy (Forchuk et al., 2002; Buckley et al., 2005; Esterberg and Compton, 2005; Solty et al., 2009; Tidey and Rohsenow, 2009). As positive expectancies are related to heavy and daily smoking in healthy adults (e.g., Brandon and Baker, 1991), it is not surprising that individuals with schizophrenia, who tend to be daily and heavy smokers, endorse strong positive expectancies.

Relations between smoking and schizophrenia are complex. Findings suggest that those with schizophrenia smoke to cope with unpleasant medication side effects and symptoms of schizophrenia, including cognitive deficits (e.g., attention, memory) and negative symptoms (e.g., anhedonia, boredom; see Adler et al., 1998; Dalack et al., 1998; George et al., 2006). Recent research (e.g., Galazyn et al., 2010) suggests that smokers with schizophrenia might have unique motivators for smoking such as increasing stimulation, reducing negative or aversive internal states (i.e., negative affect, nicotine withdrawal), and feeling a strong emotional attachment to their cigarettes. Further, they are likely to continue smoking despite environmental limitations, negative consequences, and/or the lack of alternative reinforcers. It is important to note that while smoking motives and smoking expectancies are similar constructs, motives refer to those reasons an individual endorses for why he/she continues to smoke (Fidler and West, 2009), whereas expectancies are the positive or negative consequences an individual expects to get from smoking (Copeland et al., 1995). As up to 86% of individuals with schizophrenia start smoking prior to illness onset (de Leon, 1996; Kelly and McCreadie, 1999; McEvoy and Brown, 1999; Beratis et al., 2001; de Leon et al., 2002a, 2002b; Riala et al., 2004), there is evidence that medication side effects and symptom severity are not to blame for increased smoking prevalence. This suggests a shared vulnerability between cigarette smoking and schizophrenia (e.g., de Leon and Diaz, 2005). Perhaps subclinical symptomatology existing prior to illness onset makes individuals more susceptible to smoking. Further knowledge on these issues might be gained by examining smoking behaviors and characteristics (e.g., smoking expectancies) in individuals with schizotypy.

Schizotypy is the putative genetic vulnerability to developing schizophrenia spectrum pathology without necessarily manifesting full-blown phenotypic expression (Meehl, 1962, 1990; Chapman et al., 1978; Lenzenweger, 2006). Those with schizotypy exhibit subclinical putatively genetic traits (e.g., unusual beliefs/experiences, anhedonia, social awkwardness) that are similar to symptoms of schizophrenia (Claridge, 1985; Siever et al., 1993). Approximately 10% of individuals in the general population display schizotypy traits (e.g., Meehl, 1962; Blanchard et al., 2000; but see Rawlings et al., 2008). However, schizotypy traits are also commonly observed in first-degree relatives of persons with schizophrenia (Schultz and Andreasen, 1999; Fanous et al., 2001), and have also been described as a prodromal phase of schizophrenia (Walker and Gale, 1995; Tscheslaski, 2008). Research investigating the relationship between smoking and schizotypy is germane, as it avoids many confounds associated with schizophrenia (e.g., medication interactions, illness chronicity, psychosocial decline; Estergberg et al., 2007).

Little research has investigated the association between smoking and schizotypy. However, findings indicate that schizotypy is positively correlated with smoking status (Allan et al., 1995; Larrison et al., 1999; Wiles et al., 2006; Estergberg et al., 2007; Stewart et al., 2010) and cigarettes smoked per day (Allan et al., 1995), and those with schizotypy are up to twice as likely as those without schizotypy to smoke (Allan et al., 1995; Estergberg et al., 2007; Stewart et al., 2010). Thus far, studies have not found significant differences in nicotine dependence based on schizotypy (Estergberg et al., 2007; Stewart et al., 2010). No known research has assessed smoking topography, biological indicators of smoking, or smoking expectancies among smokers with schizotypy. This area of research is critical, as it may shed light on potential distinctive motivators for smoking and/or expectancies about the anticipated effects of smoking among individuals prone to schizophrenia symptoms, but likely not affected by confounds such as medication side effects or illness chronicity. Identifying unique motivators for smoking as well as smoking expectancies in smokers with schizotypy might help to highlight areas of focus for smoking prevention and cessation interventions targeted for individuals with symptoms across the schizophrenia spectrum. If smokers with schizotypy report different motives and/or expectancies for smoking (e.g., social facilitation, boredom reduction), smoking cessation interventions targeting these factors (e.g., skills to facilitate social interaction without smoking) might be particularly effective for individuals across the schizophrenia spectrum.

This study compared smoking behaviors and smoking-related characteristics in smokers with schizotypy and controls. It was hypothesized that: (1) smokers with schizotypy would report more intense smoking-related characteristics (more cigarettes smoked per week, shorter time to first cigarette upon waking, daily smoking) and higher nicotine dependence, than control smokers; (2) compared to control smokers, smokers with schizotypy would report greater positive (e.g., improved stimulation and social facilitation, reduced negative affect and boredom) and fewer negative smoking expectancies; and (3) smokers with schizotypy would display more intense smoking behaviors (i.e., topography measures such as more puffs per cigarette, shorter inter-puff intervals, and larger total puff volume, and higher CO levels and boosts) than control smokers.

2. Method
2.1. Participants
2.1.1. Screening phase
Participants were undergraduate students enrolled at a large Southeastern university. Eligibility was determined by an online screening phase for which two means of recruitment were used. As part of a larger mental health study (see
Cohen et al., 2012), students were invited by email to complete an online screening survey and were entered into a lottery (10 prizes of $25 each) for compensation. Students were also recruited through a Psychology participant pool for course credit. The online survey included informed consent, the Schizotypal Personality Questionnaire-Brief Revised (SPQ-BR; Cohen et al., 2010), the Depression subscale of the Brief Symptom Inventory (BSI; Derogatis, 1975), and the Smoking Status Questionnaire (SSQ). A total of 1351 participants (1110 from the mental health study pool and 241 from the Psychology pool) completed the screening phase.

2.1.2. Laboratory phase
Participants were eligible to complete the laboratory phase if they were at least 18 years old, not currently pregnant, identified as current smokers, reported no severe depressive symptoms or suicidal ideation, and scored in specified ranges on the SPQ-BR. They were not required to be daily smokers, as most college student smokers are light or “occasional” smokers (Rigotti et al., 2000).

Research suggests that schizotypy is a categorical construct with an incidence of 10% (e.g., Mehl, 1962; Blanchard et al., 2000; but see Rawlings et al., 2008). Thus, this study examined schizotypy as a categorical rather than a dimensional construct. Categorization offers greater precision over employing dimensional definitions of schizotypy because schizotypal individuals are more easily separated from individuals with sub-threshold unconventional or eccentric traits. To help ensure that individuals in the schizotypy group had a high likelihood of actually having schizotypy, a conservative strategy was used and only the top 5% of scorers on the SPQ-BR were included in the schizotypy group. Like schizophrenia, schizotypy is heterogeneous, comprising positive, negative, and disorganization traits. Thus, those scoring at or above the 95th percentile (> 1.65 S.D. from gender and ethnicity-determined means) on the positive, disorganization, and/or negative SPQ-BR subscales were eligible for the laboratory phase. This comprehensive recruitment strategy ensures the representation of a diverse symptom set in our schizotypy sample and is consistent with a number of published studies on the subject (Stewart et al., 2010; Cohen et al., 2011).

Individuals who were unlikely to have schizotypy based on SPQ-BR scores (i.e., < 50th percentile based on gender and ethnicity-determined means) were eligible to participate as controls. Based on these criteria, 148 (69 smokers with schizotypy and 79 control smokers) were eligible to complete the laboratory phase, and 44 actually attended (18 controls and 26 with schizotypy).

2.2. Materials

2.2.1. Smoking Status Questionnaire (SSQ)
The SSQ assesses demographics (gender, age, and ethnicity) and smoking characteristics (e.g., smoking frequency, previous quit attempts). Daily smokers were asked to report how many cigarettes they smoke per day and occasional smokers reported how many cigarettes they smoke per week. All participants completed the Fagerström Test for Nicotine Dependence (FTND; Heatherton et al., 1991). The FTND is a widely used and valid measure of nicotine dependence, with higher scores indicating higher dependence. The FTND is positively correlated with smoking frequency, symptoms of nicotine withdrawal, and consequences of smoking are rated on a 10-point Likert scale (0 = “completely unlikely,” 9 = “completely likely”). The SSQ includes 10 subscales: Negative Affect Reduction, Stimulation/State Enhancement, Health Risks, Taste/Sensory Perception, Social Facilitation, Control, Craving/Addiction, Negative Physical Feelings, Boredom Reduction, and Negative Social Impression. Subscale scores were calculated as the mean value of each subscale. The subscales have good internal consistency and construct validity (Copeland et al., 1995).

2.2.2. Expired carbon monoxide (CO)
CO levels were measured using a portable Vitalograph ecosyler (Vitalograph Incorporation, Lenexa, KS, USA). According to the Society for Nicotine and Tobacco Subcommittee on Biochemical Verification (SRNT, 2002), a CO reading of 8–10 ppm (ppm) distinguishes nonsmokers from smokers. However, CO has a short half-life, ranging from 1 to 8 h depending on factors such as daily smoking rate and recency of smoking (SRNT, 2002). As college student smokers are typically light or occasional smokers (see Sutfin et al., 2009), CO levels were expected to be lower in this population compared to community samples.

2.2.3. Smoking topography
Smoking topography was assessed using the Clinical Research Support System (CReSS; Plowshare Technologies, Baltimore, MD, USA) micro portable smoking topography device, a battery-operated device that measures and stores data on a number of smoking parameters, including: number of puffs per cigarette, time to smoke each cigarette, interpuff interval, total and average puff volume, total and average puff duration, and maximum puff velocity. Participants smoke cigarettes through a plastic mouthpiece connected to an analog-digital converter with plastic tubing. The device detects cigarette insertion and removal and the aforementioned smoking variables. Following smoking, the data are transferred from the device to a desktop computer program. Data from measurements are averaged to obtain one value for each parameter. The CReSS smoking topography system has good reliability and validity and smoking behaviors do not appear to change as a function of smoking through a plastic mouthpiece (Lee et al., 2003).

2.3. Procedure
Eligible participants were invited to attend the laboratory session, which took approximately 45 min to complete. Participants were asked to bring a pack of their preferred brand of cigarettes with them to their appointment. They were asked to abstain from smoking for 6 h prior to their appointment to ensure a consistent level of nicotine deprivation. Non-smoking was verified using expired CO. A 6-hour time frame was chosen based on recent recommendations on the amount of time required for CO level to decrease below cut-off (about 8–10 ppm) for active smokers (SRNT, 2002). Upon arriving to the laboratory, participants provided their informed consent. Then, CO levels were assessed and participants completed the SSQ, the WISDM-30, and the SCQ-A. Following completion of questionnaires, participants were trained on the proper use of the CReSS micro portable smoking device. Then, each participant was brought to an outside area and smoked one of his or her own cigarettes using the topography device. Participants were instructed to smoke through the device in a similar manner to how they would smoke a cigarette without the device; they were also asked to limit distractions when smoking (e.g., not using their cell phone). Following smoking, CO was again assessed and CO boosts were calculated. Participants were compensated with either cash ($10 or $20) or course credit. All procedures were reviewed and approved by the university’s Institutional Review Board for Human Subjects.
2.4. Statistical analyses

Four sets of analyses were conducted. First, potential demographic differences between the schizotypy and control groups were assessed using Chi Square analyses and t-tests for categorical and continuous variables, respectively. Second, using a multivariate analysis of variance (MANOVA), schizotypy and control groups were compared on smoking-related characteristics (i.e., nicotine dependence, smoking frequency, daily smoking, and time to first cigarette). Third, a MANOVA was conducted to test for differences in smoking-related outcome expectations between schizotypy and controls groups. Fourth, to compare whether schizotypy and control groups differed on smoking behaviors (i.e., smoking topography measures, CO) a multivariate analysis of covariance (MANCOVA) was performed. Significant smoking behaviors and/or characteristics from the previous analyses were controlled for in these analyses.

Prior to conducting analyses, using the procedures recommended by Plowshare Technologies (Plowshare Technologies, Baltimore, MD, USA) smoking topography data were cleaned to identify erroneous puffs or cigarettes smoked. The criteria for false puffs includes: puff volume of greater than 150 milliliters (ml), average flow rate of less than 15 ml/second (s), peak flow rate of less than 16 ml/s, and puff duration of greater than 2800 milliseconds (msec). Further, puffs with an inter-puff interval of greater than 90 s were considered erroneous. Based on these criteria, two participants (with schizotypy) had unusable smoking topography data, as their cigarettes were too loosely inserted into the CReSS smoking topography device. They were excluded only from the final analyses.

Analyses were conducted using IBM SPSS version 19. Univariate outliers greater than 3.3 standard deviations from the means of each dependent variable were removed (Tabachnick and Fidell, 2007).

3. Results

3.1. Demographic characteristics

Participants (N=44) were predominantly female (n=24; 55%), Caucasian (n=36; 82%), and single (n=30; 68%), with a mean age of 20.09 years (±1.94). More than half (n=23; 52%) were non-daily smokers. Demographic characteristics (e.g., age, gender, ethnicity) were compared between smokers with versus without schizotypy using t-tests and Chi Square analyses. There were no significant group differences on any of these analyses (p’s > 0.05).

3.2. Smoking-related characteristics

A MANOVA was conducted with smoking characteristics (WISDM-30, FTND, cigarettes per week (CPW), time to first cigarette upon waking) as dependent variables and participant grouping (smokers with schizotypy versus control smokers) as the independent variable. The overall MANOVA was significant, Wilks’ Lambda = 0.74, F(5, 38) = 2.62, p = 0.04. As seen in Table 1, follow-up t-tests revealed significant differences between groups on the WISDM-30 total score, such that smokers with schizotypy had higher scores than control smokers, t(42) = 1.94, p = 0.01. Results also indicated a trend towards significance for CPW, as smokers with schizotypy reported smoking more cigarettes per week than control smokers, t(42) = 2.75, p = 0.06. Given that groups differed significantly on WISDM-30 total scores, posthoc follow-up t-tests were conducted to determine which subscales participants differed on. Smokers with schizotypy had significantly higher scores on the following subscales: Affiliative Attachment, t(42) = 2.13, p = 0.04; Loss of Control, t(42) = 2.02, p = 0.05; Cue Exposure, t(42) = 2.05, p = 0.05; Negative and Positive Reinforcement, t(42) = 2.52, p = 0.02; and Weight Control, t(42) = 2.48, p = 0.02.

3.3. Smoking outcome expectancies

A MANOVA was conducted with SCQ-A subscale means as dependent variables and participant grouping (smokers with versus without schizotypy) as the independent variable. Results revealed significant group differences, Wilks’ Lambda = 0.61, F(10,33) = 2.14, p = 0.05. As hypothesized, follow-up t-tests indicated that smokers with schizotypy reported significantly more positive smoking expectancies on the following SCQ-A subscales: Negative Affect Reduction, t(42) = 2.75, p = 0.02; Stimulation/State Enhancement, t(42) = 3.20, p = 0.03; Taste/Sensorimotor Manipulation, t(42) = 3.27, p = 0.002; Social Facilitation, t(42) = 4.31, p = 0.001; and Boredom Reduction, t(42) = 2.15, p = 0.04 (Table 2).

3.4. Smoking behaviors

A MANOVA was conducted with participant grouping (smokers with versus without schizotypy) as the independent variable and the following dependent variables: (1) average topography variables (puffs per cigarette, interpuff interval, maximum puff velocity, mean puff volume, and mean puff duration), (2) CO levels, and (3) CO boosts. As there were group differences on WISDM-30 total score and a strong trend towards significance for cigarettes smoked per week, these variables were entered as covariates. Results revealed no significant differences between smokers with schizotypy and control smokers, Wilks’ Lambda = 0.94, F(7, 32) = 0.76, p = 0.63 (Table 3).

4. Discussion

This is the first known study to assess smoking behaviors and smoking outcome expectancies in individuals with categorically-defined schizotypy. This is a rich area for research, as it avoids many confounds associated with schizophrenia (e.g., medication side effects, illness chronicity) and may offer unique knowledge about factors related to smoking among individuals across the

### Table 1

<table>
<thead>
<tr>
<th>Smoking characteristics of smokers with schizotypy (n=26) and smokers without schizotypy (n=18).</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Smoke with schizotypy</strong></td>
</tr>
<tr>
<td>Mean (S.D.)</td>
</tr>
<tr>
<td>Cigarettes smoked per week</td>
</tr>
<tr>
<td>Daily smoker</td>
</tr>
<tr>
<td>Nicotine dependence (FTND)</td>
</tr>
<tr>
<td>First cigarette of day (within 1 h)</td>
</tr>
<tr>
<td>WISDM total score</td>
</tr>
<tr>
<td>SPQ-BR positive score</td>
</tr>
<tr>
<td>SPQ-BR negative score</td>
</tr>
<tr>
<td>SPQ-BR disorganization score</td>
</tr>
</tbody>
</table>

*P < 0.05.

nt = nonsignificant trend (p = 0.06).

*P < 0.01.

*P < 0.001.
schizotypy have distinctive reasons for smoking. Research findings (i.e., Galazyn et al., 2010) and suggest that smokers with schizotypy tend to be heavy smokers (de Leon et al., 1995; Schuster and Johanson, 1974). There was a nonsignificant trend by which smokers with schizotypy reported smoking more CPW than smoking controls. However, just as with smoking controls, smokers with schizotypy were primarily light non-daily smokers.

It was expected that compared to control smokers, smokers with schizotypy would report more extreme smoking-related characteristics (i.e., daily smoking, higher smoking frequency, and time to first cigarette) and higher nicotine dependence (measured by the FTND and WISDM-30). This hypothesis was partially supported. Smokers with schizotypy reported significantly higher levels of nicotine dependence as measured by the WISDM-30 but not by the FTND. One potential reason for this discrepancy is that the WISDM-30 is a theoretically multidimensional measure of nicotine dependence motives, while the FTND is a measure of physical dependence that is strongly influenced by heaviness of smoking (i.e., TTF, CPD; see Schuster and Johanson, 1974). Forchuk et al., 2002; Tidey and Rohsenow, 2009). Thus, the present findings suggest that smokers with schizotypy on smoking behaviors (smoking topography) or a biochemical measure of nicotine intake (CO).

Note: There were no significant group differences on these variables.

It was expected that compared to control smokers, smokers with schizotypy would report more extreme smoking-related characteristics (i.e., daily smoking, higher smoking frequency, and time to first cigarette) and higher nicotine dependence (measured by the FTND and WISDM-30). This hypothesis was partially supported. Smokers with schizotypy reported significantly higher levels of nicotine dependence as measured by the WISDM-30 but not by the FTND. One potential reason for this discrepancy is that the WISDM-30 is a theoretically multidimensional measure of nicotine dependence motives, while the FTND is a measure of physical dependence that is strongly influenced by heaviness of smoking (i.e., TTF, CPD; see Schuster and Johanson, 1974). Forchuk et al., 2002; Tidey and Rohsenow, 2009). Thus, the present findings suggest that smokers with schizotypy on smoking behaviors (smoking topography) or a biochemical measure of nicotine intake (CO).

As hypothesized, compared with smoking controls, smokers with schizotypy reported greater positive and fewer negative smoking outcome expectancies. Smokers with schizotypy reported higher tendencies to smoke for stimulation or state enhancement, to improve taste and sensorimotor manipulation, to enhance social facilitation, sedation, and reduction of negative affect (i.e., relaxation, social facilitation, sedation, and reduction of negative affect) and fewer negative smoking expectancies (Glynn and Sussman, 1990; Lohr and Flynn, 1992; Forchuk et al., 2002; Tidley and Rohsenow, 2009). Thus, the present findings suggest that smokers with schizotypy smoke for many of the same reasons as those with schizophrenia.

### Table 2

<p>| Smoking behavioral measures for smokers with schizotypy (n=24) and smokers without schizotypy (n=18). |</p>
<table>
<thead>
<tr>
<th>Smokers with schizotypy</th>
<th>Smokers without schizotypy</th>
<th>Difference scores</th>
<th>t</th>
<th>Effect sizes (d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (S.D.)</td>
<td>95% CIs</td>
<td>Mean (S.D.)</td>
<td>95% CIs</td>
<td></td>
</tr>
<tr>
<td>Puffs per cigarette</td>
<td>13.65 (4.92)</td>
<td>11.48–15.80</td>
<td>12.17 (4.82)</td>
<td>9.72–14.69</td>
</tr>
<tr>
<td>Interpuff interval (s)</td>
<td>30.46 (28.76)</td>
<td>22.14–41.16</td>
<td>20.84 (7.21)</td>
<td>8.45–30.16</td>
</tr>
<tr>
<td>Maximum puff velocity (ml/s)</td>
<td>38.08 (11.45)</td>
<td>31.24–41.95</td>
<td>37.09 (14.17)</td>
<td>32.88–45.11</td>
</tr>
<tr>
<td>Mean puff duration (s)</td>
<td>40.57 (14.36)</td>
<td>33.19–47.39</td>
<td>43.23 (17.04)</td>
<td>35.47–51.45</td>
</tr>
<tr>
<td>Total puff volume</td>
<td>548.66 (243.66)</td>
<td>445.77–651.55</td>
<td>515.44 (290.97)</td>
<td>370.75–660.14</td>
</tr>
<tr>
<td>Time 1 CO (ppm)</td>
<td>2.78 (2.76)</td>
<td>1.61–3.19</td>
<td>2.50 (1.98)</td>
<td>2.09–3.89</td>
</tr>
<tr>
<td>Time 2 CO (ppm)</td>
<td>6.39 (3.99)</td>
<td>4.54–7.05</td>
<td>5.89 (3.10)</td>
<td>5.22–8.08</td>
</tr>
<tr>
<td>CO boost (ppm)</td>
<td>3.61 (2.10)</td>
<td>2.57–4.22</td>
<td>3.39 (1.72)</td>
<td>2.72–4.60</td>
</tr>
</tbody>
</table>

* a 0.05, b 0.01, c 0.001.
Addressing specific smoking expectancies might be a particularly effective area of intervention for smokers with symptoms across the schizophrenia spectrum. For example, those individuals who report the expectancy that smoking will reduce boredom, might benefit from cognitive restructuring and activity planning. As smoking expectancies are associated with intention to quit and predict smoking cessation (see Brandon et al., 1999), future research should explore how outcome expectancies relate to intention to quit smoking or stage of change in smokers with schizotypy.

It was expected that smokers with schizotypy would display more intense smoking behaviors (as measured by smoking topography and CO) than control smokers. Notably, there were no significant group differences on these measures. This is inconsistent with previous research conducted among smokers with schizophrenia (Olincy et al., 1997; Hitsman et al., 2005; Tidey et al., 2005, 2008; Williams et al., 2006, 2010, 2011). There are several potential explanations for this. First, in previous studies, participants were daily and heavier smokers, smoking at least 20 CPD. Most participants in the present study were non-daily or light smokers, which likely impacted the intensity of their smoking patterns. It is also possible that while smokers with schizotypy have elevated nicotine dependence and positive smoking outcome expectancies, they do not show the same smoking patterns evident in those with full-blown schizophrenia. Finally, this study had a small sample and this analysis was underpowered. Effect sizes for puffs per cigarette and interpuff interval were small, and negligible for all other variables of interest.

The present study has several limitations. First, it is limited by reliance upon self-report measures, which can be biased and unreliable. To supplement self-report measures, smoking topography and CO were assessed as objective indicators of smoking behavior. Notably, CO was assessed both before and after participants smoked a cigarette using the smoking topography device. Future research should assess cotinine (an active metabolite of nicotine) levels within this population, as studies have found that smokers with schizophrenia often have higher cotinine levels than smokers without schizophrenia (e.g., Strand and Nybäck, 2005). Another limitation is that smoking topography was assessed at one time point, rather than at multiple time points or over a longer time period. This might have contributed to the fact that smokers with schizotypy and controls did not differ on smoking topography variables. Future studies should investigate smoking topography over a longer time period. In addition, as this sample consisted of a relatively homogeneous sample of young adults who were primarily light smokers and Caucasian females, the generalizability of these results to the larger population is questionable. As the sample size was quite small due to recruitment difficulties, several of the analyses were underpowered. Studies are needed to replicate this research with larger and more heterogeneous samples (i.e., varying ethnicities, young adult non-students, daily versus non-daily smokers, and heavier smokers). Finally, we conceptualized schizotypy as a categorical construct. Dimensional models exist (Rawlings et al., 2008), and it is unclear whether the present findings would replicate using a non-categorical model.

There are several lines of research that might further elucidate the relationship between smoking and schizophrenia by utilizing smokers with schizotypy. First, researchers should examine smoking-related characteristics among non-daily and daily smokers with schizotypy, schizophrenia, and controls. Second, as smoking expectancies are associated with intention to quit smoking and smoking cessation (e.g., Brandon et al., 1999), studies should examine how outcome expectancies relate to intention to quit smoking or stage of change among non-daily and daily smokers with schizophrenia, schizotypy, and controls. Third, considerable findings have indicated that smokers with schizophrenia have more intense smoking topography and higher CO and cotinine levels (Olincy et al., 1997; Hitsman et al., 2005; Strand and Nybäck, 2005; Tidey et al., 2005, 2008; Williams et al., 2006, 2010, 2011). Thus, additional research is needed to investigate potential differences in smoking behaviors (i.e., smoking topography, CO, cotinine) following a period of short-term abstinence among non-daily and daily smokers with schizotypy, schizophrenia, and controls.

In conclusion, this study is the first to assess smoking behaviors (e.g., smoking topography, CO) and smoking outcome expectancies in individuals with categorically-defined schizotypy. Compared to smokers without schizotypy, those with schizotypy reported higher nicotine dependence, marginally higher smoking frequency, and greater positive and fewer negative smoking outcome expectancies. These findings are consistent with prior research conducted with smokers with schizophrenia and suggest that individuals with schizotypy might have the same expectancies about smoking as those with schizophrenia. Addressing these unique expectancies about smoking might be useful in tailoring smoking cessation programs and reducing tobacco-related health disparities for this population.

Funding

This research was conducted at Louisiana State University as part of Dr. Stewart's doctoral dissertation under the direction of Amy L. Copeland, Ph.D. Drs. Stewart and Adams are currently at the University of Texas MD Anderson Cancer Center. Therefore, this research was partially supported by the National Institutes of Health through MD Anderson’s Cancer Center Support Grant CA016672. Dr. Stewart was supported in part by a cancer prevention fellowship through the National Cancer Institute (R2ST CA57730: PI Shine Chang). Drs. Stewart and Adams were supported in part as Community Based Participatory Research Trainees from the Latinos Contra el Cancer Community Networks Program Center, National Cancer Institute (U54CA153505; PIs: David Wetter, Maria E. Fernandez, Lovell Jones). Dr. Adams was supported by a faculty fellowship from the University of Texas MD Anderson Cancer Center Duncan Family Institute for Cancer Prevention and Risk Assessment.

Acknowledgment

The authors would like to thank Alexa Thibodeaux and Christina Mueller for their assistance with data collection.

References


