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Cue-Evoked Positive Affect, Depression Vulnerability and Smoking Years

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Abstract

Objectives—To evaluate whether cue-evoked affective response would moderate the relationship between depression-proneness and smoking years.

Methods—Depression-proneness profiles were derived using clinician diagnosed personal and family histories of major depression, recurrent depression, trait-anhedonia, and ruminative coping styles (n=70). Affective distress was produced by idiographic, guided negative mood imageries in the presence of an in vivo cigarette exposure.

Results—Contrary to expectations, results showed that individuals less vulnerable to depression reported longer smoking histories. Stress-induced decreases in positive affect bolstered the association between depression vulnerability and smoking years.

Conclusion—Depression-proneness assumptions are challenged and implications to affective influences on smoking behavior are discussed.

Keywords

depression; vulnerability; affect; smoking

Vulnerability to depression has long been touted as a strong motivator for chronic cigarette smoking.[1-3] The association between depression and smoking is especially important given evidence that depression-prone smokers have heightened susceptibility to smoking-related mortality and cancers not related to smoking when compared with non-prone smokers and depression-prone nonsmokers.[4] Among those who enter smoking treatment, there is a debate about whether a positive depression history serves as an obstacle to prolonged abstinence.[5-7] Moreover, little is known about whether and to what extent depression-prone smokers might be less motivated to enter smoking treatment,[8] potentially extending the years a person smokes beyond those smokers who are less vulnerable to depression. The primary aim of the present paper was to test the hypothesis that depression-proneness would be associated with self-reported number of years smoking. The secondary aim was to test affective mechanisms

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that could enhance the probability that depression-proneness would be related to extensive smoking years.

The proposition that chronic mood disregulation binds depression-prone individuals to cigarettes assumes that, over time, repeated pairing of chronic negative affect with the acute reduction of dysphoria after smoking creates contingencies that promote regular patterns of use. Despite this commonly held assumption about what drives smoking among depression-prone individuals, little is known about nicotine's effects on negative moods that are not initiated by nicotine withdrawal. Some evidence suggests that prolonged nicotine patch administration produces clinically significant reductions (as much as 43.9%) in depressive symptoms among non-smokers,[9-10] suggesting that chronic nicotine administration might have antidepressant effects in clinically depressed individuals.[9] In addition, much research examining nicotine's effects on negative mood states has shown that older, heavy smokers report substantial relief from mild to moderate stress and anxiety following nicotine administration,[11-12] with higher doses of nicotine producing the greatest mood relief.[11,13,14] In most cases, however, it is difficult to rule out the possibility that benefits were derived from reversal of withdrawal-associated negative mood.

To the extent that low levels of positive affect by itself or in combination with elevated negative affect is an essential component for clinically significant depression,[15,16] the ability of nicotine to ameliorate such deficiency may be an important source of reinforcement for depression-prone smokers. For example, recent studies have supported the idea that nicotine directly increases basal levels of positive affect.[17,18] Positive affect also declines substantially during nicotine abstinence,[19] particularly among those more vulnerable to depression (eg, anhedonic smokers).[20] Clinically significant decreases in post-quit positive affect explains withdrawal-related urges to smoke.[20] Such decreases are also ameliorated by a high dose of antidepressant medication (60mg Fluoxetine).[19] Recent data suggest that smokers with low levels of positive affect have greater difficulty maintaining abstinence after cessation unless they are taking antidepressant medication.[21] Young depression-prone individuals are more likely to report cigarette smoking and higher rates of nicotine dependence.[22,23] Their propensity for smoking and for progression toward dependence on nicotine was explained by expectations about nicotine's ability to improve positive affect[22] and by lower state levels of positive affect,[23] respectively.

Overall, research has yet to fully elucidate the influence of depressive vulnerability on smoking patterns. Given some question about how precisely smoking research has assessed depression vulnerability,[5] vulnerability was conceptualized, within the present study, as a cluster of stable, endogenous, and latent characteristics and/or factors.[24] Specific variables that were combined to create a composite depression-proneness measure included clinician diagnosed personal history of Major Depressive Disorder (MDD), number of prior MDD episodes, family history of MDD, trait level of anhedonia, and ruminative response style. This composite measure of depressive vulnerability incorporated multiple factors beyond the presence/absence of a past MDD episode. For example, recurrent episodes of MDD reflect a stable pattern of mood, given that the predictive validity of future MDD strengthens as the number of episodes increases.[25] Both family history of depression and trait-anhedonia have been associated with heri-table/endogenous disturbances.[26-28] Ruminative response styles

represent thought processes that lock a person's attention onto upsetting preoccupations.[29] Such processes are latent in the sense that they are not always present but are exacerbated during negative mood states.[30]

The present study was designed to test the hypothesis that depressive vulnerability would be directly associated with the number of years a person has smoked. We predicted that increased vulnerability to depression would predict more extensive smoking years. We further sought to examine whether affective responses to a mood and cigarette craving induction would enhance the association between depression-proneness and years smoked. Specifically, we assessed non-deprived smokers' affective reactions to an idio-graphic negative mood induction coupled with in vivo cigarette exposure. We predicted that smokers' affective responses to the negative mood induction with in vivo cigarette cue exposure would moderate the proposed relationship between depression-proneness and years smoked. Given evidence that negative and positive affect are influenced by distressing states,[19,20] we expected that both negative and positive affect would moderate the relationship between depression-proneness and smoking years.

METHODS

Participants

The present study was a secondary analysis of a larger cue reactivity study designed to examine differences in affective and craving reactivity among smokers with and without a history of major depression. Participants included 70 regular smokers (39.7% female) from a large, Midwestern city. All were between the ages of 18 and 62 (M=40.5, SD=10.6). They smoked, on average, 20.4 (SD= 6.7) cigarettes per day and were moderately dependent on nicotine (M=5.8 SD= 2.2).[31] Forty-four (62.9%) participants were men. Forty-five (64.3%) participants identified themselves as African-American, 21 (30%) European-American, 2 (2.9%) Latino-American, one (1.4%) Asian-American and one (1.4%) multi-ethnic. Eighty-three percent of the sample reported an education level below an Associate's degree with the greatest portion of the sample reporting either a high school diploma (37%) or some college (40%).

Eligibility requirements for the parent study included being between the ages of 18 and 65 and smoking at least 15 cigarettes per day over the past year. Participants also either had at least one prior experience with major depression or never experienced clinically significant depression. Potential participants were excluded for medically unstable conditions (eg, recent myocardial infarction), scores of 14 or higher on the Hamilton Depression Rating Scale,[32] pregnancy, current use of antidepressant medication, current psychiatric illness, and current use of nicotine replacement therapy. Women with late luteal phase disorder or severe premenstrual disturbances were also excluded because emotional instability related to the late luteal phase disorder or severe premenstrual disturbances may unduly alter mood responses (see review).[33]

Participant Flow

Initially, 231 individuals responded to the advertisements and were screened via telephone. Sixty-six participants were found ineligible from the telephone screening for the following reasons: 39% currently medicated, 27% uninterested in participating, 15% not meeting stratification requirements, 5% high blood pressure, 3% recent cessation attempts, and 3% self-report psychiatric illness. The remaining individuals found ineligible due to smoking less than what was required by the study (< 15 cigarettes per day), age requirements, current treatment, pregnancy, and disconnected telephone, each contributing less than 2%. After the telephone screening, 165 individuals were deemed initially eligible and scheduled for a screening visit: 72 did not attend. Accordingly, 93 participants consented to the study although 23 did not complete for the following reasons: 61% did not complete experimental sessions, 13% attempted to participate in the study more than once after being found ineligible, 9% met criteria for a current psychiatric illness, 9% gave inaccurate information on telephone screening, 4% did not meet stratification requirements and 4% were referred to a physician due to high carbon monoxide readings. In total, 70 participants completed the study.

Measures

Affect—Positive and negative affect were assessed at each visit using the positive affect and negative affect subscales of the Positive and Negative Affect Scale (PANAS).[34] The PANAS consists of 20 adjectives rated on a 1 to 5 point scale ranging from very slightly or not at all (1) to extremely (5). The positive affect subscale consists of ten words measuring feelings of activation, elation, enthusiasm, and enjoyment, with scores ranging from 10 to 50. The negative affect subscale consists of ten words measuring feelings of distress, hostility, nervousness, scorn, and gloominess. Scores on the negative affect scale range from 10 to 50. The PANAS scale possesses strong internal consistency.[34] Within the present study, the α coefficients ranged from .86 to .92.

Axis I disorders—Participants were screened for current and past psychiatric illness using the Structured Clinical Interview for DSM-IV Axis I Disorders, Non-Patient Edition (SCID-NP).[35] The SCID-NP was used in addition to telephone screening interview to assess for mood disorders and current alcoholism and drug use. Personal history of depression was coded dichotomously as absent or present. Number of episodes was coded as 0, 1, 2, 3, 4 or 5+.

Anhedonia—The Fawcett-Clark Pleasure Scale (FCPS)[26] measured anhedonia by asking how subjects usually react to 36 situations typically experienced as enjoyable. Using 5-point Likert-type scales, respondents rated how pleasurable they would find events like embracing a loved one or witnessing their grown children's success. Scores on the FCPS potentially range between 36 and 180, with lower scores indicating diminished hedonic capacity. Internal consistency from the present data for the FCPS is very high: $\alpha = .92$.

Caffeine and alcohol intake—Given evidence that suggests daily consumption of alcohol and caffeine may influence cue reactivity,[36,37] a beverage score was assessed. The beverage intake form comprised 6 questions that assessed the amount of coffee, tea, soda,

spirits, wine and beer that was consumed within the last 24 hours prior to the exposure session. A total beverage consumption score was derived by summing the number from each question. Scores ranged from 0 to 20.

Depression-Proneness Inventory—The Depression-Proneness Inventory (DPI)[38] was used to assess proneness to depression. The ten items were answered on a 7-point Likert scale, with scores ranging between 7 and 70. Higher scores indicate elevated proneness to depression. The DPI has been shown to have construct validity[38] and be internally consistent.[39] The DPI exhibited strong internal consistency in the present study: $\alpha = 92$.

Family history of depression—Family history of depression was assessed using a scale designed specifically for the present study. Participants were asked, “Have any of your relatives had what you would call a significant drinking, drug use or psychological problem; one that did or should have led to treatment?” A list of potential disorders given to the participants included depression, alcohol problems, drug problems, bipolar disorder, anxiety problems, psychotic problems, and smoking behavior. The participant responded yes or no to the questions of specific issues. For yes responses, the participant was asked whether the person was a first or second degree relative. Answers were coded 0, 1 or 2 (no, yes-2nd degree, yes-1st degree). For those who had both first and second degree relatives, answers were coded as a first degree.

Ruminative response style—The Response Style Questionnaire (RSQ)[29] was used to measure dispositional responses to dysphoric moods. On the RSQ, participants were asked what they generally do when they feel sad or down. The 21 items of the Ruminative Response subscale are answered using 4-point Likert-type scales. The Ruminative Response subscale has demonstrated excellent internal consistency[30-40] and fair test-retest reliability over 1 year.[30] In the current study, the Ruminative Response subscale showed adequate internal consistency: $\alpha = .83$.

Nicotine dependence—Nicotine dependence was assessed at baseline using the 6-item Fagerstrom Test for Nicotine Dependence (FTND).[31] Scores ranged between 0 and 11, with higher scores indicating greater nicotine dependence. Comparisons between FTND scores and measures of nicotine intake support the construct validity of the scale.[41]

Guided imagery script—Based on a procedure developed by Litt, Cooney, Kadden and Gaupp,[42] a trained interviewer asked each participant to describe several events within the past year that made him/her “upset - very anxious, angry or sad,” and several that did not make him/her “upset or happy.” Participants were encouraged to describe each event in their own words, indicating what led up to the situation, what occurred, how they felt about it, and what the outcome was. After generating the events, participants rated each incident on ten point Likert scales to indicate the degree to which it made them feel anxious, angry, or sad, and the vividness of the mental image generated. Only events scoring > 7 on all scales were scripted for the negative mood induction procedure, and only scores of 0 or 1 were scripted for the neutral mood induction procedure. Mentally reliving an emotional autobiographical event has been found to be an effective mood induction technique.[43]

Procedure

Screening—During the screening session, participants were told about the details of the study and written informed consent was obtained. Consenting participants were administered the SCIDNP[35] to assess whether they met DSM-IV criteria for a lifetime history of major depressive disorder and for current nicotine dependence. In addition, the SCIDNP was used to rule out alcohol or drug abuse/dependence problems. Participants were then asked to give a sample of expired carbon monoxide (CO) and to complete demographic, smoking, DPI, and affect questionnaires. Finally, subjects were interviewed about recent upsetting experiences in order to generate incidents that were used to create guided imagery scripts.

Baseline exposure procedure—Prior to experiencing each cue exposure condition, participants sat in a comfortable chair, provided a CO sample, completed the caffeine and alcohol intake form, and smoked one cigarette of their regular brand in order to standardize nicotine exposure. Then, the experimenter removed the pack of cigarettes, ashtray and lighter from the room. Participants were instructed to sit and relax for 30 minutes. During the relaxation period, mood was assessed using the PANAS.

Negative mood induction procedure—After baseline self-report measurements, an audiotape of music was played over headphones to induce a negative mood state. Participants were given the following instructions and prompted to recall one of the sad memories they described during screening. Participants were instructed:

> As you can hear, there is music playing in the background. The music is to help you attain an upsetting mood state. I'd like you to listen to the music and try to remember the time when.... Try to really intensely get into the mood you felt during this situation. It is very important that you try to develop a bad mood that is as intense and real as you can possibly make it. I want to remind you that we have a procedure to bring your mood back to normal at the end of this experiment. So don't be afraid to really intensely get into this bad mood.

The music used in the negative mood exposure presented excerpts from classical pieces, including Russia Under the Mongolian Yoke by Prokofiev and Adagio Pour Cordes by Barber, which have been shown to induce dysphoric moods.[44,45] The induction lasted approximately 10 minutes and was followed by a manipulation check via the PANAS.

Environmental cue: in vivo cigarette condition—The cue condition was employed to enhance the ecological validity of the stressor by coupling stress in the presence of smoking paraphernalia. Participants were shown their brand of cigarettes, a lighter and an ashtray positioned on the tray. Participants were instructed to light one cigarette without putting the cigarette to their mouths. Participants were also instructed to hold the cigarette in the flame of the lighter until it burned. Participants then held the cigarette comfortably in their dominant hand until the research assistant asked them to extinguish the cigarette and place the remains in the ashtray.

Analytic plan—All hypotheses were tested via hierarchical linear regression. A depression-proneness profile (predictor variable) was regressed on self-reported number of years smoked (criterion variable). Covariates were chosen theoretically, based on their potential for influencing affective responses to exposure sessions. Covariates included age and a composite score of alcohol and caffeine consumption on the testing day. In each analysis described below, all covariates were entered on the first step, and the variables of interest were entered on subsequent steps.

A depression-proneness profile (DPP) score was derived before testing the influence of depression vulnerability on years smoked. The DPP score comprised summing a clinician diagnosed history of depression (0-absent,1-present), number of episodes (0,1,2,3,4,5+), family history of depression (0-absent,1-present), trait-anhedonia (0-low,1-high), and rumination (0-low,1-high). Trait-anhedonia and rumination were coded as a dichotomous variable using median split cutoffs. DPP scores ranged from 0 to 9 (M=2.87; SD=2.5). The score was utilized to reflect stable, endogenous, and latent characteristics/factors that are associated with a person who is vulnerable to depression. The number of episodes variable is the strongest predictor of depression-proneness[25] and thus carried more weight in the score. All variables are moderately related to depression vulnerability.[25-30]

After testing the influence of DPP scores on years smoked, we tested whether the relationship was moderated by affective responses to the exposure conditions. Moderators were tested by entering both the potential moderator and the predictor on the second step, following the covariates, and entering the product of the 2 on the third step. Two different moderators were tested: post-exposure positive affect (PA) and post-exposure negative affect (NA). In each model used to test moderation, pre-exposure affect was entered as a covariate on the first step. Finally, when analyses indicated that a variable was a significant moderator of the association between depression proneness and years smoked, simple effects analyses were conducted to determine the direction of the relationship by performing a median split on the moderating variable and testing the effect of depression proneness on years smoking within each half of the sample.

RESULTS

Manipulation Check

To test the validity of the negative mood induction compared with the neutral mood induction, we conducted a within-subjects, repeated measures, 2 (condition) by 2 (time) ANOVA. PANAS negative affect increased significantly from pre- to post-exposure during the negative mood induction + in vivo cigarette condition, [$F(1,68)=53.63$, $P=.0001$]. In addition, examination of test-retest reliability showed that baseline PANAS negative affect was significantly correlated with post-exposure PANAS negative affect ($r=.254$, $P=.035$).

Primary Analyses

The primary regression analysis showed that depressive vulnerability did not initially predict smoking years [$\beta = -.477$, R^2 change = .317, $P = .138$]. However, moderation analyses are particularly important when an expected relationship is not significant; thus moderation was

subsequently tested. We tested whether post-exposure negative affect (NA) moderated the association between vulnerability to depression and years smoked. We found that post-exposure NA did not moderate this relationship [β = −.007, R^2 change = .030, P = .825]. We then tested whether post-exposure positive affect (PA) moderated the association between depressive vulnerability and years smoked. As illustrated in Table 1, results indicated that PA was a significant moderator [β = .083, R^2 change = .028, P = .021]. PA was split by the median score with individuals with below median scores labeled as “low” PA responders and individuals on or above the median labeled as “high” PA responders. Simple effects analyses indicated that there was no relationship between DPP scores and years smoked for the high PA responders [β = −.224, R^2 change = .002, P = .620]; however, among low PA responders, the relationship was significant [β = −1.01, R^2 change = .057, P = .026]. In other words, the link between DPP scores and years smoked was stronger for low PA responders than for high PA responders.

DISCUSSION

The present study tested the association between vulnerability to depression and number of years smoking. Our secondary aim was to evaluate the extent to which affective responses during non-deprived distress would moderate the presumed depression vulnerability-years smoked relationship. Contrary to our expectations, there was a negative relationship between depression vulnerability and smoking years. This relationship appeared to be enhanced when those with lower DPP scores reported greater reductions in positive affect following the mood induction + in vivo smoking cue challenge.

The present findings evoke interesting questions about the role of vulnerability to depression in smoking behavior. Because depression assessment issues convolute results from prior smoking research, the present study attempted to improve the assessment of depression vulnerability by using multiple indicator variables that reflected stable, endogenous and latent characteristics of depression. Results from the derived DPP scores supported the notion that depression vulnerability is not strongly associated with the number of years a person smokes. In fact, there appeared to be a negative relationship where lower DPP scores were associated with longer smoking years.

To date, more is known about the motivational influences of affective responses associated with nicotine abstinence than is known about pre-quit distress. Abstinence-induced changes in negative and positive affect reinforce smoking, in general.[3,11,17,19,20,23] For those potentially prone to depression, withdrawal-related decreases in positive affect may produce more motivation to smoke than post-quit elevations in negative affect.[20] Our findings are relatively mixed in their support of current knowledge. Decreases in stress-induced positive affect do enhance DPP scores association with the years a person smokes. However, the moderating effect was among an unexpected negative relationship (ie, lower DPP scores predicting longer smoking years).

Despite the promising findings, there are a few limitations that warrant discussion. First, the present sample was relatively small (n=70) and our ability to generalize the findings to other cultural groups outside of African-Americans and European-Americans was limited.

Second, the influence of temporary withdrawal effects cannot be completely ruled out, given that participants had not smoked for forty minutes in the present protocol (baseline cigarette to post-exposure affect assessment). Nonetheless, research showing that depression-prone smokers experience greater affective withdrawal responses during acute abstinence[48] lends support to the idea that the present results were not tainted by temporary withdrawal effects, because the moderating effect of mood was primarily observed among smokers less prone to depression. Third, the DPP scores were derived by theoretical means and are in need of analyses with a larger sample to verify a unified construct among the variables.

In conclusion, there remain many unresolved issues in understanding the influence of depression proneness on smoking behavior. The present study provides support for the premise that DPP scores relationship with smoking years is contingent on a person's positive affective responses to non-deprived stressors. Because of the cross-sectional nature of the data, unresolved questions are related to why depression-prone smokers might smoke for a shorter period than their less prone counterparts and why distressing affective experiences independent of nicotine abstinence would not influence the length of their smoking history. Future research may need to examine whether depression-prone smokers have greater tolerance for distressing situations as a result of the presumably increased frequency with which they experience such situations. Further, might the frequency of distress experienced by depression-prone smokers motivate them to seek treatment more quickly than smokers not as prone to depression?

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Table 1

Hierarchical Regression Analysis of the Interaction Between PANAS PA and DPP Scores on Smoking Years

Criterion Variable: Smoking Years					
Step	**Variable**	**Beta**	**R^2 Change**	**F change**	**P**
1.	**Covariates**		**.642**	**60.18**	**.000**
	Age	.790			.000
	Alcohol/Caffeine Intake	.880			.032
2.	**Predictors**		**.015**	**1.39**	**.256**
	DPP Scores	−2.25			.008
	PANAS PA	−.143			.240
3.	**Interaction Term**	**.083**	**.028**	**5.60**	**.021**

Note.

PANAS PA = Post-exposure PANAS positive affect scores