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Longitudinal Associations between Smoking and Affect among Cancer Patients Using Varenicline to Quit Smoking

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Abstract

During a quit attempt, high negative affect predicts relapse to smoking. In this study, we evaluated bidirectional longitudinal associations between smoking and negative affect among cancer patients treated with varenicline. Participants (N=119, 50% female, M_{age}=59 years) were smokers (≥5 cigarettes/week) who were diagnosed with cancer and were recruited for a 24-week trial of extended duration varenicline plus behavioral counseling; data for this secondary analyses were drawn from the 12-week open-label phase of the trial. Smoking was assessed via self-reported number of cigarettes in the past 24 hours. Negative affect was assessed using the Positive and Negative Affect Scale (PANAS). Data were collected at pre-quit (week 0), target quit day (week 1), week 4, and week 12. We evaluated cross-lagged panel models for negative affect and smoking using PROC CALIS in SAS. Models were run separately for participants who were adherent (≥80% of medication taken) or nonadherent to varenicline. Among adherent participants (n=96), smoking accounted for up to 22% of variance in subsequent negative affect throughout treatment. Cross-lagged associations were not observed between smoking and negative affect among non-adherent participants (n=23). Negative affect did not predict subsequent smoking among either adherent or nonadherent participants. These results suggest that varenicline may attenuate abstinence-induced negative affect among cancer patients treated for nicotine dependence.

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Conflict of interest: Dr. Schnoll receives medication and placebo free from Pfizer. Dr. Schnoll has provided consultation to Pfizer and GlaxoSmithKline, and consults with Curaleaf. All other authors declare that they have no conflicts of interest.

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Keywords

smoking cessation; affect; adherence; cancer; randomized controlled trial

1. Introduction

Cigarette smoking continues to be a great public health concern, contributing to an estimated three in ten cancer-related deaths in the U.S. (Jacobs et al., 2015). Continued smoking amongst cancer patients perpetuates adverse treatment outcomes, including a second primary cancer, worse side effects, and poorer survival (USDHHS, 2014), and yet nearly half of smokers continue to smoke after a cancer diagnosis (Cox et al., 2003). To provide effective smoking cessation treatments for this high-risk population, a better understanding of the reasons why cancer patients continue to smoke is critical.

In the general population, one proposed mechanism of relapse is that abstinence-related hedonic changes, including increased negative affect (NA) and decreased positive affect (PA), drive smokers to resume smoking (Cook et al., 2010; Copeland et al., 2009; Doran et al., 2006). While this mechanism has not been tested among smokers with cancer, cancer patients report high levels of psychological distress (Brown et al., 2013), and smokers with cancer and comorbid psychiatric conditions are less likely to quit (Blalock et al., 2011). However, the associations between successful smoking cessation and affect among cancer patients have not been evaluated.

First-line pharmacotherapies can improve the odds of successful cessation two- or threefold (Fiore et al., 2008). The seven FDA-approved smoking cessation pharmacotherapies, including five nicotine replacement therapies and two medications, are designed to ameliorate nicotine withdrawal symptoms, including affective changes (Fiore et al., 2008). Varenicline, a first-line smoking cessation pharmacotherapy that is safe and effective among cancer patients (Schnoll et al., 2019), may reduce psychological distress among those who achieve abstinence by improving NA and enhancing PA (Doran et al., 2018; Patterson et al., 2009). A better understanding of the impact of affective changes on quitting behavior amongst these smokers, while using first-line treatments, is crucial for supporting this high-risk population to quit smoking and thus improve their physical and psychological health.

The aim of this study was to better understand the relationship between smoking and affect among cancer patients using varenicline to quit smoking. Compared to previous studies that only evaluated NA and PA as baseline predictors or as outcome measures following abstinence, we assessed a bidirectional model to elucidate directionality between smoking and affect by assessing smoking, NA, and PA contemporaneously among cancer patients who were and were not adherent to varenicline during the 12-week, open-label phase of a clinical trial. This design allowed us to evaluate smoking level and affective changes within the context of an effective treatment that has demonstrated affective benefits during abstinence. Based on studies in the general population (Cook et al., 2010; Doran et al., 2018), we hypothesized that higher levels of NA and lower levels of PA would be associated with a greater likelihood of subsequent smoking, particularly among those who were adherent to varenicline.

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2. Materials and Methods

2.1 Participants

Eligible participants were men and women ≥18 years old who reported smoking ≥5 cigarettes per week and diagnosed with cancer within 5 years. Participants were excluded for: daily use of non-cigarette tobacco products; current smoking cessation treatment; medical contraindications for varenicline; or bipolar disorder, psychotic disorder, or current suicidality. Further inclusion/exclusion information can be found elsewhere (Crawford et al., 2018).

2.2 Study design and procedures

The parent trial was designed to evaluate the efficacy of extended (24-week) varenicline compared to standard (12-week) varenicline treatment, plus 24 weeks of behavioral counseling provided to all participants (NCT01756885). The present study is a secondary analysis from the 12-week open-label phase of the trial. Primary recruitment efforts focused on oncology clinics in Chicago, IL and Philadelphia, PA between May, 2013 and June, 2017. Potential participants were screened for initial eligibility via telephone, and final eligibility was confirmed at an in-person session where participants provided written informed consent and completed assessments. Eligible participants were then scheduled for a Pre-Quit (PQ) session, where they initiated varenicline following standard dosing: 0.5 mg once per day for days 1–3, 0.5 mg twice per day for days 4–7, and 1.0 mg twice per day for the remainder (through day 84 for the 12-week course). Participants were instructed to set their target quit date (TQD) for the day they started the full dose of varenicline. Smoking cessation counseling (based on PHS Guidelines; Fiore et al., 2008) was provided in-person at PQ (Week 0), TQD (Week 1), Week 4, and Week 12 and via telephone at Week 8. At each in-person session, participants completed assessments including medication adherence and side effects, smoking, and psychological symptoms. All procedures were approved by the institutional review boards at each site.

2.3 Measures

2.3.1 Sociodemographics and smoking history—At baseline, participants self-reported gender, race/ethnicity, and age; how long they had been smoking and how many cigarettes per day (CPD) they smoked on average; level of dependence as assessed by the Fagerström Test for Cigarette Dependence (FTCD; Fagerstrom, 2012; α=0.61).

2.3.2 Current smoking—At each in-person visit, participants were asked, “Have you smoked a cigarette, even a puff, in the past 24 hours?” and, if they answered yes, “How many cigarettes have you smoked in the past 24 hours?” Past 24-hour cigarette use (CPD) was used for the analyses, given that this value can be dynamic throughout a quit attempt.

2.3.3 Affect—Participants completed the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) at each in-person visit. The PANAS is a self-report measure on which participants rated their current state on a scale from 1 to 5 of 10 negative feelings (e.g., irritable, jittery; α=0.80, 0.86, 0.85, and 0.87 at PQ, TQD, W4, and W12 respectively) and 10 positive feelings (e.g., interested, enthusiastic; α=0.93, 0.90, 0.93, and 0.92 at PQ,

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TQD, W4, and W12 respectively). Total scores on each scale range from 10 to 50, with higher scores indicating higher levels of NA or PA.

2.3.4 Treatment adherence—We chose to stratify the analyses by varenicline adherence because participants who were nonadherent would be unlikely to experience the NA or PA benefits it offers. At each visit, participants self-reported varenicline use by pill count via timeline follow-back and confirmed by medication blister pack contents. Participants were categorized as adherent (took ≥80% of pills) consistent with previous studies (Carroll et al., 2018; Catz et al., 2011).

2.4 Data analysis

All analyses were conducted using SAS version 9.4. The primary analysis was a cross-lagged panel model, which allowed us to test bidirectional pathways while accounting for past and contemporaneous factors. We completed four models using PROC CALIS to evaluate associations between smoking (CPD) and NA or PA (PANAS scores) separately by adherence status (adherent or nonadherent). The models included data from: PQ, TQD, Week 4, and Week 12. Participants who attended all sessions were included because complete data are required for this analysis. We assessed autocorrelations (within CPD or within NA/PA; e.g., PQ CPD to TQD CPD) and cross-lagged associations (between CPD and affect over the course of treatment; e.g., PQ CPD to TQD NA). Error covariances within each session (e.g. PQ CPD and PQ PA) were included in each model. Model fit was described using χ^2, standardized root mean squared residual (SRMR), and comparative fit index (CFI) (Hu and Bentler, 1999).

3. Results

3.1 Sample characteristics

Demographic and cancer-related characteristics of the sample are presented in Table 1. A total of 119 participants attended all 4 in-person sessions during the open-label treatment phase. The 88 participants in the primary study who were excluded from this analysis did not differ from included participants on any of the baseline characteristics (demographics, cancer-related factors, smoking variables, or PANAS scores). Ninety-six participants (81%) were classified as adherent. No significant differences on baseline characteristics were observed between adherent and nonadherent participants.

3.2 Panel analysis

The panel models of smoking and affect represented an adequate fit of the data among adherent participants (NA: $\chi^2(df=12)=61.77$, $p<.001$; SRMR=0.100; CFI=0.791; PA: $\chi^2(df=12)=30.33$, $p=.003$; SRMR=0.078; CFI=0.946) and nonadherent participants, though potentially overfitting the data for PA (NA: $\chi^2(df=12)=14.52$, $p=0.269$; SRMR=0.084; CFI=0.963; PA: $\chi^2(df=12)=11.56$, $p=0.482$; SRMR=0.051; CFI=1.000).

The panel models of smoking and NA are presented in Figure 1 by adherent (Panel A) and nonadherent participants (Panel B). Among adherent participants, cross-lagged associations from smoking to NA increased over time, from PQ to TQD (0.13, $p=0.131$), TQD to W4

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(0.16, p=0.050), and W4 to W12 (0.23, p=0.007), indicating that higher levels of smoking predicted higher levels of NA at the subsequent visit. This pattern was not observed among nonadherent participants. Cross-lagged associations from NA to smoking were negligible throughout treatment (all ps>0.05). None of the cross-lagged associations between smoking and PA reached significance among either adherent or nonadherent participants (Supplemental Material).

The autocorrelations observed within smoking and affect provide face validity for these results. For example, smoking autocorrelations were moderate from PQ to TQD (range: 0.45–0.59; ps<.001), smaller between TQD and W4 (range: 0.21–0.24; ps<.05 for adherent, ps>.05 for nonadherent), and largest between W4 and W12 (range: 0.58–0.69; ps<.001). Within NA, adherent participants had moderate, significant effects throughout treatment (0.49–0.58, ps<.001) while nonadherent participants had large effects (0.66–0.81, ps<.001), suggesting limited variability across sessions among nonadherent participants. PA autocorrelations among both adherent and nonadherent participants were high (>0.70, all ps<.001), suggesting a potential ceiling effect of the PANAS PA scores, particularly among nonadherent participants.

4. Discussion

This study evaluated the bidirectional associations between smoking and affect among smokers with cancer during 12 weeks of varenicline. To our knowledge, this was the first study to explicitly evaluate the bidirectional associations between smoking and affect among cancer patients. Using an innovative application of cross-lagged panel analysis in a clinical trial, self-reported smoking predicted later NA, but not PA, amongst adherent participants; NA did not predict smoking in either model. A clearer understanding of this relationship may allow for more targeted smoking cessation interventions for smokers with cancer and reduce adverse treatment outcomes among this vulnerable population.

Specifically, only among participants who were adherent to varenicline, we observed that higher levels of smoking predicted higher levels of NA, while reducing or quitting smoking predicted lower levels of NA at the next visit, especially as participants progressed through treatment. On the other hand, these findings may be due to higher NA levels among those who are not successful in quitting (McClave et al., 2009) or because those with greater NA at baseline have more difficulty quitting smoking (Copeland et al., 2009). Thus, among cancer patients, varenicline adherence may ameliorate cessation-induced NA and support continued smoking abstinence. This is consistent with larger-scale trials that showed varenicline did not increase adverse neuropsychiatric events among smokers with and without psychiatric comorbidities (Anthenelli et al., 2016) and ameliorates abstinence-induced changes in affect (Doran et al., 2018; Patterson et al., 2009).

We did not find predictive associations between PA and smoking. This finding is in contrast with studies demonstrating that decreases in PA were predictive of relapse to smoking (Leventhal et al., 2014). Pre-existing deficits in PA has also been shown to predict further declines in PA upon smoking cessation (Cook et al., 2004). The lack of association between PA and smoking in this study suggests that varenicline may modulate PA, independent of

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smoking status. Other factors may influence the complicated relationships between affect and smoking cessation, such as whether the smoker's cancer was caused by smoking (e.g., lung cancer), which should be investigated in future studies.

Despite the novel application, there are limitations to this analysis. First, due to model constraints, only participants with complete data were included. Second, PANAS scores had limited variability: NA scores demonstrated a large positive skew (1.78), reaching a maximum of 33 of 50 points, while PA scores had a moderate negative skew (−0.86), potentially reaching a ceiling at 50 of 50 points. These distributions may have limited our ability to detect significant changes in scores. Finally, only 24 participants were included in the nonadherent group, and thus these results should be interpreted with caution.

Using a cross-lagged panel analysis we found that self-reported smoking predicted later NA, but not PA, through 12 weeks of smoking cessation treatment amongst cancer patients who were adherent to varenicline. These findings further our understanding of these interrelationships, which may support the development of targeted interventions for this vulnerable population of high-risk smokers.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Negative affect during a quit attempt predicted subsequent smoking
- Smoking did not predict later negative affect among cancer patients trying to quit
- Varenicline attenuated abstinence-induced negative affect among cancer patients

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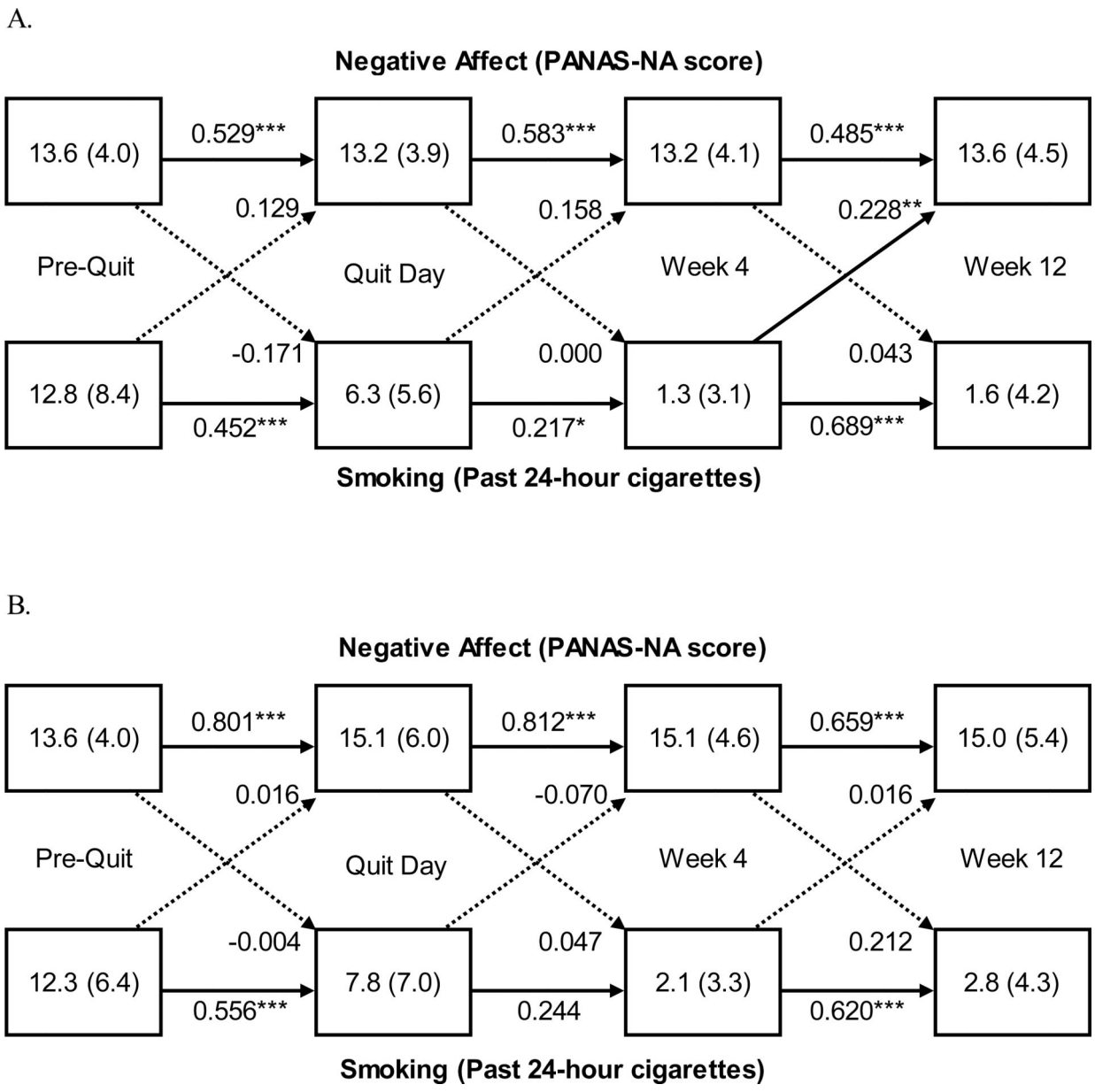

Figure 1.

Panel analysis of negative affect (NA) and smoking among adherent participants (n=96; Panel A) and among nonadherent participants (n=23; Panel B) during the 12-week, open-label phase of a smoking cessation clinical trial using varenicline plus behavior counseling among adults with a cancer diagnosis. Values in the boxes indicate the Mean (Standard Deviation) NA score or smoking level, respectively. Solid lines represent significant path coefficients. Dotted lines represent non-significant path coefficients. Values on the lines represent standardized path coefficients, which indicate the proportion of variance in Y (e.g., TQD NA) that is explained by X (e.g., PQ Smoking). Error covariances of endogenous variables (e.g., PQ NA and PQ Smoking) were included in the model. NA was assessed by

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the Positive and Negative Affect Scale (PANAS) negative affect score. Smoking was self-reported number of cigarettes smoked in the past 24 hours. *p<.05 **p<.01 ***p<.001

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Table 1.

Demographics and cancer-related characteristics by study medication adherence status

	Nonadherent (n=23)		Adherent (n=96)		
Variable	**N or M**	**(% or SD)**	**N or M**	**(% or SD)**	***p*-value**
Female, %	12	(52%)	47	(49%)	0.782
White, %	13	(57%)	72	(75%)	0.078
Age (years)	58.6	(9.0)	59.4	(8.4)	0.712
< College graduate, %	14	(61%)	61	(64%)	0.812
Employed, %	11	(48%)	47	(49%)	0.922
Years smoking	40.9	(8.9)	40.9	(10.4)	0.981
FTCD score	4.7	(2.5)	4.4	(2.1)	0.673
Tobacco-related cancer site,[a] %	18	(78%)	77	(80%)	0.834
Cancer stage					0.223
Stage 0–2	6	(26%)	18	(19%)	
Stage 3–4	6	(26%)	14	(15%)	
Remission/not reported	11	(48%)	64	(67%)	
Current cancer treatment,[b] %	7	(30%)	40	(42%)	0.375
Chemotherapy	3	(13%)	20	(21%)	
Radiation treatment	1	(4%)	7	(7%)	
Surgery	3	(13%)	12	(13%)	
Hormone therapy	2	(9%)	10	(10%)	

p-values indicate significance of differences between adherent vs. nonadherent sample by t-tests (continuous variables) or chi-square analyses (categorical variables).

[a]Participants' cancer types included: genitourinary (n=29), breast (n=20), skin (n=19), lung (n=16), hematological (n=15), head and neck (n=8), gastrointestinal (n=6), and kidney/pancreatic/liver (n=6).

[b]Cancer treatments (chemotherapy, radiation treatment, surgery, and hormone therapy) are not mutually exclusive.