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Depressive Symptoms and Cigarette Demand as a Function of Induced Stress

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Abstract

Introduction: Depressed smokers may disproportionately value cigarettes as compared to other reinforcers in the context of increases in negative affect (NA). Thus, cigarette demand may be an important construct for understanding the relationship between depression, NA change, and tobacco use. The aim of the current study was to examine the interaction between depressive symptoms and change in NA as a function of induced mood as a predictor of cigarette demand.
Methods: Participants included 73 young adult daily smokers (41.70% female, 73.60% White, age *M* (SD) = 19.70 (1.15)) who attended two experimental sessions: one stress and one neutral. During each session, participants completed ratings of depressive symptoms, NA, and cigarette demand.
Results: We examined the predictive utility of depressive symptoms, change in NA as a result of a stressor, and the interaction between depressive symptoms and NA change on demand indices. Separate models were constructed by session. Results indicated significant interactive effects between depressive symptoms and change in NA for predicting intensity, breakpoint, and P_{max} during the stress session. Specifically, change in NA moderated the relationship between depression and demand indices such that among individuals high in NA change, depressive symptoms were positively related to P_{max} and breakpoint, whereas among individuals low in NA change, depressive symptoms were positively related to intensity.
Conclusions: When exposed to stress, cigarettes may become more valuable for individuals with depressive symptoms.
Implications: This study contributes to the literature attempting to understand the complex relationships between depression, stress-related changes in NA, and tobacco use. This study suggests that one mechanism that may be important to the relationship between depression and tobacco use is cigarette demand. Specifically, for individuals with elevated depressive symptoms, certain aspects of cigarette demand may be higher (intensity, breakpoint, and P_{max}) when exposed to stress, which may contribute to tobacco use being maintained over time.

Depressive symptomatology constitutes a robust risk factor for the etiology of tobacco use across the life span.[1–5] Depression is characterized by high, but variable, levels of negative affect (NA),[6] and considerable work has been conducted to understand the nature of the relationship between depressive symptoms and cigarette smoking. One hypothesis suggests that cigarette smoking may serve to regulate NA among those with elevated depressive symptoms.[5,7] Specifically, tobacco use is maintained in part via a negative reinforcement process whereby cigarette smoking functions to modulate NA in the context of depression.[8,9] Despite this hypothesized relationship, ecological momentary assessment studies examining NA modulation as a function of smoking have yielded equivocal results,[10–12] suggesting that this relationship may be most pronounced for certain groups who are more susceptible to smoking for NA reduction. Following

from this framework, tobacco use may serve the greatest functional role among individuals with elevated depressive symptoms who are also experiencing acute increases in NA.[10]

There is support for the relationship among depressive symptoms, NA change, and cigarette smoking, and a number of studies have examined the specific effect of NA induction on smoking-related constructs[13–16]; however, few laboratory studies have investigated these relationships in aggregate. One such study by Fucito and Juliano[17] demonstrated that current depressive symptoms moderated the relationship between mood and smoking. Specifically, in response to a sad mood induction, as compared to a neutral mood induction, smokers with elevated current depressive symptoms took more cigarette puffs, smoked longer, and experienced a greater increase in expired air carbon monoxide from baseline to post-smoking. In contrast, utilizing a similar mood induction paradigm, Perkins and colleagues[18] found that the relationship between depression history and smoking reinforcement ("total puff volume"), reward ("cigarette liking"), and NA relief did not vary as a function of mood induction. Thus, the experimental literature to date is largely inconclusive regarding the interaction between depression and change in NA as they relate to cigarette smoking.

One important smoking-related construct that may further our understanding of the intersection of depressive symptoms and NA in tobacco use is cigarette demand. Based on an operant framework of addictive behavior, cigarette demand, also termed *relative reinforcing efficacy* or the relative value of a drug, reflects the behavior-strengthening or behavior-maintaining properties of cigarettes.[19] Cigarette demand is a distinct construct from other assessments of cigarette-related reward such as in vivo smoking behavior (ie, smoking topography) and self-reported liking of a cigarette. Within a negative reinforcement framework, smokers with depressive symptomatology may place greater value on cigarettes as compared to other reinforcers when experiencing an increase in NA as cigarette smoking may serve to regulate NA. Thus, to the extent that depressed smokers may disproportionately value cigarettes as compared to other reinforcers as a result of increases in NA,[20] cigarette demand may be an especially useful construct for understanding the complex relationship between depression, NA change, and cigarette smoking. For example, recent literature suggests that smokers with elevated anhedonic symptoms may value cigarettes as reinforcers comparatively higher than nondrug (monetary) reinforcers and that the relationship between this facet of depressive symptoms and in vivo smoking behavior is partially mediated by acute mood state prior to a smoking task.[20] In light of this imbalanced incentive reward for smoking as compared to nondrug reinforcers among depressed smokers, a crucial next step is to understand the interactive effect of depressed mood and change in NA as they predict the relative reinforcing efficacy of cigarettes.

Cigarette demand is fairly efficient to assess using a cigarette purchase task (CPT).[19] Through use of a CPT, individual demand curves can be created that reflect established dimensions of the relative value of cigarettes under progressively increasing financial costs. Dimensions include intensity of demand (cigarette consumption at zero cost), maximum expenditure on cigarettes (O_{max}), the price associated with the maximum cigarette expenditure (P_{max}), the price at which consumption is entirely suppressed (breakpoint), and price sensitivity (elasticity). Importantly, the CPT is reliable[21] and exhibits a high degree of correspondence with actual cigarette consumption and cigarette purchases.[22] Moreover, the CPT demonstrates convergent validity with theoretically related constructs including cigarettes per day and nicotine dependence, with intensity and O_{max} having the strongest relationships with these variables.[19] No studies to date have examined demand curve analysis through the use of a CPT in smokers with depressive symptomatology or how the relationship between depression and cigarette demand may vary as a function of induced mood. However, one study found that heavy drinkers with symptoms of depression and post-traumatic stress disorder reported elevated alcohol demand relative to heavy drinkers without those comorbid symptoms,[23] and another study found that acute stress increased several facets of alcohol demand (intensity, O_{max}, and breakpoint).[24]

In order to address this gap in the literature, the aim of the current study was to examine the interaction between depressive symptoms and change in NA as a function of induced mood (neutral and negative) as a predictor of cigarette demand. Given that there are individual differences in response to NA induction paradigms,[25] examining change in NA as a moderator was important for understanding the effects of depressive symptoms on cigarette demand among individuals who were more responsive (ie, higher NA change) and who were less responsive (ie, lower NA change) to the experimental stressor. We hypothesized that the relationship between baseline depressive symptoms and cigarette demand indices would vary as a function of induced mood such that depressive symptoms would be predictive of heightened cigarette demand but only in the context of negative mood induction. We also hypothesized that the interaction between depressive symptoms and NA change for predicting cigarette demand would similarly vary as a function of mood induction condition, such that in the context of negative mood induction, NA change would moderate the relationship between depressive symptoms and cigarette demand. Specifically, we hypothesized that individuals who exhibit both higher depressive symptoms and greater change in NA in the context of negative mood induction would report the largest cigarette demand.

Method

Participants

Participants were recruited from a large mid-Atlantic college campus using flyers and postings on Internet message boards and Web sites (eg, campus listservs, Craigslist, and Facebook). Interested individuals were advised to contact the study by phone or e-mail to complete an online screening to determine eligibility. Participants were eligible for the study if they met the following inclusion criteria: (1) between the ages of 18 and 21 and (2) current regular smokers defined as smoking not less than 5 cigarettes/smoking day for the past 6 months and smoking on not less than 20 of the last 30 days. The present data were collected as part of a larger study with the aim of examining the relationship between social phobia (SP) and cigarette smoking.[26] As such, an additional inclusion criterion was scoring either greater than 35 (comprising a high SP group) or between 9 and 24 (comprising an average SP group) on the Social Interaction Anxiety Scale (SIAS).[27]

Measures

Smoking History and Current Smoking Information

Smoking history was assessed using the smoking history and current status indices agreed upon by a National Cancer Institute (NCI) consensus panel[28] including information such as smoking rate, age of onset of any tobacco use, and age of onset of regular smoking. Nicotine dependence was assessed using the modified version of the

Fagerström Tolerance Questionnaire (mFTQ).[29] Timeline Follow-Back (TLFB)[30] procedures were used to index the number of cigarettes smoked. Participants reported on the number of cigarettes smoked during the last 30 days at baseline and on the number of cigarettes smoked in the time between sessions during their second session.

Depression

Depressive symptomatology was assessed using the Center for Epidemiologic Studies Depression Scale (CES-D).[31] The items primarily measure affective and somatic aspects of depression, including depressed mood, feelings of guilt and worthlessness, feelings of helplessness and hopelessness, psychomotor retardation, loss of appetite, and sleep disturbance. High internal consistency has been demonstrated in both general (α = 0.85) and patient (α = 0.90) populations and the measure exhibits adequate 6-month test–retest reliability (r = 0.54).[31] Participants completed the CES-D twice: once at the beginning of the neutral session and once at the beginning of the stress session. Internal consistency for the CES-D for each administration was high (CES-D neutral α = 0.91; CES-D stress α = 0.93).

Affect

The 10-item NA subscale of the Positive and Negative Affect Scale (PANAS)[32] was used to measure NA. The PANAS is commonly used to detect changes in emotional reactions to stimuli in the manner used here. The NA score was calculated by taking the sum of ratings for the 10 NA items. The PANAS was administered for both pre- and post-mood manipulations.

Cigarette demand

Cigarette demand was assessed using a CPT.[19,33] Participants completed the CPT twice: once following the neutral mood induction and once following the stress induction. The following instructions were used: "Imagine a TYPICAL DAY during which you smoke. The following questions ask how many cigarettes you would consume if they cost various amounts of money. The available cigarettes are your favorite brand. Assume that you have the same income/savings that you have now and NO ACCESS to any cigarettes or nicotine products other than those offered at these prices. In addition, assume that you would consume cigarettes that you request on that day; that is, you cannot save or stockpile cigarettes for a later date. Be sure to consider each price increment carefully." This was a hypothetical CPT and participants did not purchase real cigarettes.[34,35] Estimated cigarette consumption was assessed at 48 prices from $0 to $9. Prices increased in 5¢ increments between $0 and $2 and in $1 increments thereafter. These prices were intended to provide maximum resolution at prices around the current market price of cigarettes. Equivalent prices per pack were provided to the right of prices per cigarette. Four observed indices (intensity, breakpoint, O_{max}, and P_{max}) and one derived index (elasticity) were generated from the CPT. Intensity was defined as consumption at $0.00. Breakpoint was defined as the first price that suppressed consumption to 0 (participants who never reported zero consumption were assigned a breakpoint of $9). O_{max} was defined as participant's maximum expenditure on cigarettes. P_{max} was defined as the price associated with O_{max}.

Elasticity was derived using GraphPad Prism v. 5.04 for Windows (GraphPad Software, San Diego, CA, www.graphpad.com) and the macro available online through the Institute for Behavioral Resources website (www.ibrinc.org). Elasticity was generated from Hursh and Silberberg's[36] exponential equation: $\log Q = 1\log Q_0 + k\,(e^{-\alpha P} - 1)$. In this equation, Q = quantity consumed, Q_0 = consumption at $0.00, k = range of cigarette consumption in logarithmic units, P = price, and α = elasticity. Hursh and Silberberg[36] recommend holding k constant to allow for individual differences in elasticity to be scaled with a single parameter (α) which is standardized and independent of the magnitude of the reinforcer. In the present study, k was held constant across curve fits at 4. Consumption values of 0, which cannot be log transformed, were eliminated prior to calculating elasticity, as were participant data in which less than five consumption values were provided and/or where missing data occurred for more than one price on the CPT[37] (this resulted in the removal of three participants from the elasticity analyses for the neutral session and three participants from the elasticity analyses for the stress session). Using a criterion of not less than 30% of variance in consumption accounted for by the exponential demand curve equation,[23,38] an additional two participants were excluded from the elasticity analyses for the stress session, one participant was excluded from elasticity analyses for the neutral session, and two participants were excluded from elasticity analyses for both sessions (total sample size for elasticity analyses for the stress session was 64 and total sample size for elasticity analyses for the neutral session was 66). The exponential demand curve equation provided a good fit for most participants' data (mean R^2 stress session = 0.83, SD = 0.11; Mean R^2 neutral session = 0.81, SD = 0.12).

Procedure

The study consisted of two sessions held at the Center for Addictions, Personality and Emotion Research at the University of Maryland College Park. All procedures were approved by the University of Maryland's Institutional Review Board.

Screening

The online screening included questions about smoking behavior and completion of the SIAS. Demographic information (eg, date of birth, gender, and ethnicity/race) was collected and reconfirmed at session 1. If eligible for the study, participants were contacted via e-mail or phone for scheduling.

Experimental Sessions

Condition order was counterbalanced and, with the exception of video content, the sessions followed identical procedures. In order to standardize time since last cigarette smoked, following completion of consent procedures, participants were escorted outside and given the option to smoke a cigarette of their own brand. Although smoking a cigarette was optional, all participants chose to smoke in both sessions. Participants then completed self-report measures (smoking history or mFTQ, CES-D) in a separate room. During session 1, participants completed the TLFB for cigarettes smoked in the past month while, during session 2, participants completed the TLFB for the time between sessions 1 and 2. Because the mFTQ and NCI smoking history indices are stable over short time periods, they were counterbalanced between sessions 1 and 2. Participants were instructed to return to the lab for their second session 1 week following their first session. Average number of days between sessions was M (SD) = 5.86 (5.70) days.

Following measure completion, participants watched a control video (nature video) or a social stressor video previously used in similar social stress mood induction experimental paradigms.[39,40] The control video was validated by Rottenberg and colleagues[41] to

induce a neutral mood and has been used in other experimental studies implementing affect manipulation to study cigarette smoking.[17] For the stress manipulation video, we utilized a previously validated social stressor video adaptation of Trier Social Stress Test[40,42] that utilized video anxiety induction procedures.[43] Briefly, participants were told that they would be giving a speech to a panel of judges who would judge the quality of their speech and that they would then watch an example video of past participants giving their speeches. After watching the video, all participants were told that the speech topic that had been randomly selected for that day was to give a speech about the parts of their body they liked the least and why they liked them the least. After video presentation, participants completed the CPT. At the end of the session, participants were debriefed and compensated for participation.

Data Analytic Plan

All data were analyzed in SPSS version 22. Baseline NA was defined as score on the NA subscale of the first administration of the PANAS, and change in NA was defined as the difference on the NA subscale of the PANAS between the second administration of the PANAS and the first administration of the PANAS in each session. Zero-order correlations were obtained to examine associations between depression, change in NA, demand indices (intensity, breakpoint, O_{max}, P_{max}, and elasticity), and theoretically related constructs (income, age, race, education, cigarettes per smoking day (CPSD), and nicotine dependence). Subsequently, we used hierarchical multiple regression[44] to examine the predictive utility of depressive symptoms, change in NA, and the interaction between depressive symptoms and change in NA for demand indices above and beyond that of baseline NA and relevant covariates. In order to examine the within-session relationship between depressive symptoms, change in NA, and cigarette demand within each experimental session, separate models were constructed by session (stress and neutral). Covariates were entered as a block in step 1, the grand mean-centered main effects of depressive symptoms and change in NA were entered as a block in step 2, and the interactive effect of depressive symptoms and change in NA was entered in step 3 in order to evaluate the relative contribution of the interaction between depressive symptoms and NA change above and beyond the main effects of each variable and the effects of relevant covariates. Simple slope analyses were utilized in order to understand the nature of significant interactions.

Results

Descriptive Statistics

Of the 104 individuals who screened eligible for the study, 73 (41.7% female, age *M* (SD) = 19.70 (1.15)) attended at least one experimental session and were included in analyses. Two participants attended only one experimental session. Average CPSD for the sample was *M* (SD) = 7.43 (4.72), while average cigarettes per day was *M* (SD) = 7.25 (4.82). On average, participants first smoked at age 15.83 (2.45), began smoking weekly at age 17.57 (1.54), began smoking daily at age 18.28 (1.31), and smoked on 28.56 (3.00) days of the last 30 days. Nicotine dependence levels were relatively low (mFTQ *M* (SD) = 3.90 (1.41)). As intended, the stress manipulation resulted in a significant increase in NA ($t(70) = 4.61$, $P < .001$) across participants. See Table 1 for descriptive data.

Table 1. Sample Demographics

	Full sample
n	73
Age (*M* (SD))	19.70 (1.15)
Female (%)	41.70%
Race (%)	
Non-Hispanic White	73.60%
African American	6.90%
Asian American	15.30%
Hispanic	2.80%
Other	1.40%
CPSD (*M* (SD))	7.43 (4.72)
CPD (*M* (SD))	7.25 (4.82)
mFTQ (*M* (SD))	3.90 (1.41)
Depressive symptoms (*M* (SD))	
CES-D neutral	16.35 (10.07)
CES-D stress	17.00 (10.91)
Change in NA (*M* (SD))	
Change in NA neutral	–2.15 (3.94)
Change in NA stress	4.70 (7.39)
Demand metrics (*M* (SD))	
Intensity neutral	11.63 (7.12)
Breakpoint neutral	1.85 (2.32)
O_{max} neutral	4.25 (3.13)
P_{max} Neutral	0.93 (1.47)
Elasticity neutral (α)	2.33×10^{-4} (2.36×10^{-4})
Intensity stress	11.03 (6.25)
Breakpoint stress	1.77 (2.33)
O_{max} stress	4.50 (4.61)
P_{max} stress	1.06 (2.03)
Elasticity stress (α)	2.37×10^{-4} (2.45×10^{-4})

CPSD = cigarettes per smoking day; CPD = cigarettes per day. There were no significant differences in each of the five demand indices between sessions.

Bivariate Correlations Between Independent and Dependent Variables

See Table 2 for bivariate correlations between studied variables. Regarding demographic factors, male gender was significantly associated with higher annual family income and greater depressive symptoms on both stress and neutral experimental days. Consistent with predictions, depressive symptoms on the CES-D on the day of the stress session were significantly correlated with demand indices following the stress induction procedure (*r*s = .26–.46, *P*s < .05). Depressive symptoms on the CES-D on the day of the neutral session were not significantly correlated with demand indices following the neutral mood induction procedure. CPSD was significantly associated with demand indices on both the stress and neutral days and, as such, was included as a covariate in subsequent analyses. SP group was significantly correlated with intensity and elasticity during both the neutral and stress sessions and with O_{max} during the neutral session and thus was included as a covariate. Other demographic factors including age, gender, race, and annual family income were not significantly correlated with demand indices and were not included as covariates in subsequent analyses.

Regressions Predicting Cigarette Demand as a Function of Session

Prior to conducting the following regression analyses, we examined the overall distribution of our data to determine whether it fulfilled

Table 2. Bivariate Correlations Between Studied Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
Demographics																							
1. Age	—	−.02	.21	−.14	.05	−.11	.14	−.09	−.02	.05	.05	.05	.09	.09	**−.28***	−.01	.09	.06	.09	.02	.15	.12	**−.25***
2. Gender[a]		—	.09	**.27***	−.01	−.11	.03	**−.32****	−.17	−.09	.02	−.04	−.12	−.13	−.08	**−.31****	−.11	.02	−.04	.03	.09	.18	−.07
3. Race			—	−.14	−.14	−.23	.02	.09	.12	−.12	−.13	.13	.09	.12	.02	.07	−.06	−.002	−.16	−.01	−.14	−.05	.07
4. Income				—	.04	−.01	−.06	−.03	−.14	.11	.03	−.02	.03	−.04	.14	−.03	−.17	.10	.07	.07	.11	.10	.10
5. CPSD					—	**.65****	**.28***	**.35****	.09	.13	**.79****	.16	**.48****	.17	**−.38****	.18	−.02	.00	**.77****	.12	**.57****	−.07	**−.41****
6. mFTQ						—	.14	**.36****	**.31****	.09	**.57****	**.35****	**.52****	**.33****	**−.30***	.20	.09	−.13	**.56****	**.26***	**.55****	.07	**−.31***
7. SP group[b]							—	**.36****	.13	**.29***	**.38****	.14	.19	.02	**−.25***	**.37****	.10	−.16	**.24***	.21	**.26***	.05	**−.35****
Stress																							
8. CES-D								—	**.42****	.05	**.46****	**.33****	**.43****	**.28***	**−.26***	**.82****	.16	−.12	**.37****	.18	.22	−.06	−.22
9. Baseline NA									—	**.41****	**.26***	.20	.22	.20	−.24	**.24***	.16	−.05	.12	.07	.10	−.04	−.21
10. NA change										—	.01	**.35****	**.34****	**.28***	−.16	.12	−.05	−.10	.10	**.43****	**.33****	**.26***	**−.27***
11. Intensity											—	.13	**.38****	.08	**−.44****	.22	−.01	.06	**.88****	.08	**.48****	−.09	**−.50****
12. Breakpoint												—	**.89****	**.88****	**−.35****	**.27***	−.04	−.07	.11	**.75****	**.55****	**.59****	**−.29***
13. Omax													—	**.82****	**−.46****	**.32****	−.05	−.04	**.38****	**.69****	**.67****	**.47****	**−.41****
14. Pmax														—	**−.27***	**.30***	−.06	−.08	.11	**.58****	**.45****	**.51****	−.21
15. Elasticity															—	−.09	−.15	.11	**−.38****	**−.31***	**−.44****	−.20	**.86****
Neutral																							
16. CES-D																—	**.24***	−.17	.18	.16	.07	−.02	−.03
17. Baseline NA																	—	**−.66****	.08	−.01	−.04	−.11	−.04
18. NA change																		—	−.02	−.04	−.01	.04	.04
19. Intensity																			—	**.06**	**.49****	−.10	**−.50****
20. Breakpoint																				—	**.69****	**.80****	**−.35****
21. Omax																					—	**.54****	**−.60****
22. Pmax																						—	**−.25***
23. Elasticity																							—

Significant values are given with boldface emphasis. CPSD = cigarettes per smoking day, mFTQ = modified Fagerström Tolerance Questionnaire, SP = social phobia, CES-D = Center for Epidemiologic Studies Depression Scale, NA = negative affect.

[a]Gender was dummy coded with 0 = *female*, 1 = *male*.

[b]SP group was coded 0 = *average SP symptoms*, 1 = *high SP symptoms*.

*$P < .05$; **$P < .01$.

the required regression assumptions. Results revealed substantial skewness and kurtosis for the demand indices. In order to correct for this skewness, we log 10 transformed our cigarette demand dependent variables, which resulted in acceptable skew values. See Supplemental Materials for graphical depiction of cigarette demand curves by session.

Neutral Session

Separate regressions were conducted for each demand index and indicated that, above and beyond CPSD and SP group, depressive symptoms on the day of the neutral session, NA change as a result of the neutral mood induction, as well as the interaction between depressive symptoms and change in NA did not significantly predict any of the demand indices (all *P*s > .05; see Supplementary Appendix).

Stress Session

For intensity, breakpoint, and P_{max}, results indicated that the third steps of the individual models predicted a significant proportion of variance in each of the individual demand indices (intensity: $\Delta R^2 = 0.04$, $P = .02$; breakpoint: $\Delta R^2 = 0.05$, $P = .04$; P_{max}: $\Delta R^2 = 0.07$, $P = .01$). In the third step of each of these models, the two-way interaction between depressive symptoms and change in NA significantly predicted intensity, breakpoint, and P_{max}, respectively (see Table 3). These models accounted for 63.7%, 32.7%, and 27.5% of the variance in intensity, breakpoint, and P_{max}, respectively (see Table 3).

For O_{max} and elasticity, the third step of each model did not predict a significant percentage of variance above and beyond the second step of each model (O_{max}: $\Delta R^2 = 0.01$, $P = .43$; elasticity: $\Delta R^2 < 0.0001$, $P = .85$). In the third step of each model, neither the two-way interaction between depressive symptoms and change in NA nor the main effect of depressive symptoms significantly predicted O_{max} or elasticity (see Table 3). However, in each model, the main effects of baseline NA, change in NA, and CPSD all significantly predicted O_{max} and elasticity such that a greater number of CPSD, higher baseline NA, and greater NA change were positively related to O_{max} and were predictive of more inelastic demand (see Table 3). These models accounted for 48.2% and 43.2% of the variance in O_{max} and elasticity respectively.

The significant two-way interactions between depressive symptoms and change in NA for predicting intensity, breakpoint, and P_{max} were explored in line with procedures outlined by Aiken and colleagues.[45] Specifically, consistent with our study hypotheses, we examined the association between depressive symptoms and intensity, breakpoint, and P_{max} at high (+1 SD) and low (−1 SD) levels of NA change. CPSD, SP group, and baseline NA were grand mean centered and included as covariates in these analyses. The resulting *t* tests for the betas indicated the following: For intensity, at low levels of NA change, depressive symptoms were significantly positively related to intensity ($B = 0.01$, $t(69) = 2.67$, $P = .01$), whereas at high levels of NA change, depressive symptoms were not significantly related to intensity ($B = -0.002$, $t(69) = -0.55$, $P = .58$). For breakpoint, at high levels of NA change, depressive symptoms were significantly positively related to breakpoint ($B = 0.01$, $t(69) = 2.43$, $P = .02$), whereas at low levels of NA change, depressive symptoms were not significantly related to breakpoint ($B = -0.002$, $t(69) = -0.32$, $P = .75$). Finally, for P_{max}, at high levels of NA change, depressive symptoms were significantly positively related to P_{max} ($B = 0.02$, $t(69) = 2.56$, $P = .01$), whereas at low levels of NA change, depressive symptoms were not significantly related to P_{max} ($B = -0.01$, $t(69) = -0.72$, $P = .47$).

Discussion

We examined the main and interactive effects of current depressive symptoms and change in NA in the context of a mood induction on cigarette demand indices. Results suggest unique relationships between depression, NA change, and individual aspects of cigarette demand. We hypothesized that there would be a significant main effect of depressive symptoms on demand indices during stress but not neutral mood inductions. This hypothesis was not supported. When accounting for covariates as well as change in NA as a result of the stressor and the interactive effect of change in NA and depressive symptoms, there were no significant main effects of depressive symptoms on the day of the stress session on cigarette demand.

We also hypothesized that there would be significant interactive effects between depressive symptoms and change in NA for predicting demand indices under conditions of negative but not neutral mood induction. This hypothesis was partially supported such that for three of the four observed cigarette demand indices (intensity, breakpoint, and P_{max}) there were significant interactions between depressive symptoms and change in NA for predicting the demand indices during the stress session but not during the neutral session. For breakpoint and P_{max}, depression was significantly positively related to breakpoint and P_{max} at *high* levels of NA change. In contrast, for intensity, depression was significantly positively related to intensity at *low* levels of NA change.

These findings are consistent with a similar interaction observed by Rousseau and colleagues[46] who found that depressive symptoms in conjunction with acute negative affect were predictive of increased demand, specifically heightened price sensitivity. Regarding the results for intensity as compared to breakpoint and P_{max}, to our knowledge, these are the first results to suggest different relations between predictor variables (ie, depression was positively related to intensity at low levels of NA change, whereas depression was positively related to breakpoint and P_{max} at high levels of NA change) for demand outcomes. In the data presented here, the correlations between intensity and the other observed demand indices were, in general, lower in magnitude than the correlations of O_{max}, P_{max}, and breakpoint among themselves. Thus, the similar relationships for the interaction between depression and NA change for predicting breakpoint and P_{max} may, in part, reflect that these demand indices possess more conceptual overlap (both measure price sensitivity). In light of the interactions presented here, in order to understand why depression, NA change, and tobacco use are related, breakpoint and P_{max} may be the most relevant individual facets of relative reinforcing efficacy, although replication of these results is certainly necessitated. In contrast, among individuals less responsive to stressors, tobacco use may be maintained in part due to the positive relationship between depression and intensity (ie, smoking large amounts when cigarettes are free or inexpensive). Previous studies examining alcohol demand have also found different relationships between predictor variables and alcohol demand intensity and other demand indices. For example, Yurasek and colleagues[47] found that as compared to heavy drinking nonsmokers, heavy drinking smokers had significantly higher O_{max}, P_{max}, and breakpoint but not higher intensity.

Although there were not significant interactive effects between depressive symptoms and change in NA for predicting O_{max} and elasticity during the stress session, there was a significant main effect

Table 3. Hierarchical Regressions Predicting Demand Indices for the Stress Session

Stress session	*B*	SE (*B*)	*T* score	*P*	95% CI for *B*		ΔR^2
Intensity (*n* = 72)							
Step 1							0.59
CPSD	0.04	0.01	8.52	<.001	0.03	0.05	
SP group	0.03	0.04	0.62	.54	−0.06	0.11	
NA baseline	0.01	0.004	2.30	.03	0.001	0.02	
Step 2							0.01
CPSD	0.04	0.01	7.88	<.001	0.03	0.05	
SP group	0.02	0.05	0.42	.68	−0.07	0.11	
NA baseline	0.01	0.01	1.10	.28	−0.004	0.01	
CES-D	0.003	0.002	1.36	.18	−0.001	0.01	
NA change	-0.002	0.003	-0.55	.58	−0.01	0.01	
Step 3							0.04
CPSD	0.04	0.01	8.42	<.001	0.03	0.05	
SP group	0.01	0.04	0.15	.88	−0.08	0.10	
NA baseline	0.01	0.004	1.52	.13	−0.002	0.02	
CES-D	0.003	0.002	1.36	.18	−0.001	0.01	
NA change	0.001	0.003	0.31	.76	−0.01	0.01	
CES-D × NA	−0.001	0.00	−2.45	.02	−0.001	0.00	
Breakpoint (*n* = 72)							
Step 1							0.10
CPSD	0.01	0.01	0.68	.50	−0.01	0.03	
SP group	0.14	0.09	1.48	.14	−0.05	0.32	
NA baseline	0.01	0.01	1.68	.10	−0.003	0.03	
Step 2							0.18
CPSD	0.001	0.01	0.09	.93	−0.02	0.02	
SP group	−0.002	0.09	−0.02	−.99	−0.18	0.18	
NA baseline	0.02	0.01	2.52	.01	0.01	0.04	
CES-D	0.01	0.004	1.33	.19	−0.003	0.02	
NA change	0.02	0.01	3.45	.001	0.01	0.04	
Step 3							0.05
CPSD	−0.002	0.01	−0.17	.87	−0.02	0.02	
SP group	0.02	0.09	0.22	.82	−0.16	0.20	
NA baseline	0.02	0.01	2.21	.03	0.002	0.04	
CES-D	0.01	0.004	1.40	.17	−0.003	0.02	
NA change	0.02	0.01	2.62	.01	0.004	0.03	
CES-D × NA	0.001	0.00	2.13	.04	0.00	0.002	
O_{max} (*n* = 72)							
Step 1							0.39
CPSD	0.04	0.01	5.37	<.001	0.03	0.06	
SP group	0.04	0.07	0.60	.55	−0.10	0.19	
NA baseline	0.01	0.01	2.19	.03	0.001	0.03	
Step 2							0.08
CPSD	0.04	0.01	5.06	<.001	0.02	0.06	
SP group	−0.05	0.08	−0.62	.54	−0.20	0.10	
NA baseline	0.02	0.01	2.88	.01	0.01	0.04	
CES-D	0.003	0.003	0.75	.45	−0.01	0.01	
NA change	0.02	0.01	2.97	.004	0.01	0.03	
Step 3							0.01
CPSD	0.04	0.01	4.91	<.001	0.02	0.05	
SP group	−0.04	0.08	−0.52	.61	−0.19	0.11	
NA baseline	0.02	0.01	2.70	.01	0.01	0.04	
CES-D	0.003	0.003	0.77	.45	−0.004	0.01	
NA change	0.01	0.01	2.50	.02	0.003	0.03	
CES-D × NA	0.00	0.00	0.79	.43	−0.001	0.001	
P_{max} (*n* = 72)							
Step 1							0.05
CPSD	0.01	0.01	0.91	.36	−0.01	0.03	
SP group	0.02	0.10	0.18	.86	−0.19	0.23	
NA baseline	0.01	0.01	1.43	.16	−0.01	0.03	

Table 3. *Continued*

Stress session	*B*	SE (*B*)	*T* score	*P*	95% CI for *B*		ΔR^2
Step 2							0.15
CPSD	0.004	0.01	0.41	.69	−0.02	0.03	
SP group	−0.12	0.11	−1.15	.25	−0.33	0.09	
NA baseline	0.02	0.01	2.16	.03	0.002	0.04	
CES-D	0.01	0.01	1.13	.26	−0.004	0.02	
NA change	0.02	0.01	3.07	.003	0.01	0.04	
Step 3							0.07
CPSD	0.001	0.01	0.11	.91	−0.02	0.02	
SP group	−0.09	0.10	−0.91	.37	−0.29	0.11	
NA baseline	0.02	0.01	1.81	.08	−0.002	0.04	
CES-D	0.01	0.01	1.23	.22	−0.004	0.02	
NA change	0.02	0.01	2.12	.04	0.001	0.03	
CES-D × NA	0.001	0.001	2.54	.01	0.00	0.003	
Elasticity (n = 64)							
Step 1							0.32
CPSD	−0.04	0.01	−4.37	<.001	−0.06	−0.02	
SP group	−0.02	0.08	−0.23	.82	−0.18	0.14	
NA baseline	−0.01	0.01	−1.71	.09	−0.03	0.002	
Step 2							0.11
CPSD	−0.04	0.01	−4.35	<.001	−0.05	−0.02	
SP group	0.09	0.08	1.07	.29	−0.08	0.26	
NA baseline	−0.02	0.01	−2.85	.01	−0.04	−0.01	
CES-D	−0.001	0.004	−0.38	.71	−0.01	0.01	
NA change	−0.02	0.01	−3.22	.002	−0.03	−0.01	
Step 3							0.000
CPSD	−0.04	0.01	−4.19	<.001	−0.05	−0.02	
SP group	0.09	0.09	1.00	.32	−0.09	0.26	
NA baseline	−0.02	0.01	−2.78	.01	−0.04	−0.01	
CES-D	−0.002	0.004	−0.39	.70	−0.01	0.01	
NA change	−0.02	0.01	−2.83	.01	−0.03	−0.01	
CES-D × NA	0.00	0.00	−0.19	.85	−0.001	0.001	

CPSD = cigarettes per smoking day. SP = social phobia, SP group coded 0 = average SP, 1 = high SP. CES-D = depressive symptoms on the CES-D. NA baseline = negative affect on the first administration of the PANAS. NA change = negative affect change between the second and first administrations of the PANAS. CES-D × NA = the interaction between depressive symptoms and negative affect change. CPSD, SP group, NA baseline, CES-D, and NA change were all grand mean centered. Demand indices were log10 transformed.

of change in NA for predicting these demand indices. These results suggest that in the context of the NA induction, individuals who experienced greater increases in NA as a result of the experimental manipulation as compared to individuals who experienced lower increases in NA: (1) spent a greater amount of hypothetical money in total on cigarettes and (2) were less responsive to increases in the cost of cigarettes. Thus, the positive relationship between NA and tobacco use may be due, in part, to heightened cigarette demand when experiencing acute increases in NA. These findings are consistent with previous experimental studies that have found that NA induction as compared to neutral mood induction is predictive of cigarette smoking. Specifically, NA induction as compared to neutral mood induction is predictive of both a decreased latency to smoke and an increased number of puffs taken.[25,48] The current study suggests that one mechanism through which acute increases in NA may influence ad libitum smoking is through increases in the relative reinforcing efficacy of cigarettes when experiencing NA.

Taken together, the present study suggests that the relationship between depression and cigarette smoking may be, at least in part, explained by differences in the relative reinforcing efficacy of cigarettes when experiencing negative mood as compared to when experiencing neutral mood. Although we did not examine these relationships in the context of a quit attempt, considering that current depressive symptoms are associated with poor cessation outcomes[49,50] and that elevated alcohol demand is associated with poor alcohol intervention response,[51] our results suggest that one factor that could complicate a smoking cessation attempt for an individual with current elevated depressive symptoms is the heightened value of a cigarette to a smoker when experiencing increases in NA. For example, recent smoking cessation trials utilizing ecological momentary assessment throughout the course of a quit attempt have found that change in NA both prior to[52] and following initiation[53] of a quit attempt is predictive of success in quitting smoking. Although these studies did not examine the interactive effects of depressive symptoms and change in NA for predicting cessation, the relationship between change in NA and cessation may be due at least in part to changes in cigarette demand. For example, depressed mood coupled with increases in NA over time may contribute to a heightening of the value of a cigarette that may contribute to lapsing after initiating a cessation attempt. Future work is needed to examine these hypothesized relationships among individuals actively engaging in efforts to quit smoking. Moreover, this study suggests that coping skills training particularly for regulating NA may be especially useful for smoking cessation programs in order to target the relationship between depression and cigarette demand.

Results from the present study should be interpreted with the following limitations in mind. First, participants in the study were relatively light smokers with low levels of nicotine dependence. They were also all undergraduate students between the ages of 18 and

21 who were recruited for the original study based on self-reported symptoms of SP. Future research should examine the relationships between depressive symptomatology, NA change, and cigarette demand among heavier, more dependent smokers as well as among smokers of different ages. Second, a diagnostic interview was not administered as part of the current study and, as such, we cannot draw conclusions regarding the relationship between diagnostic-level depression, mood, and cigarette demand. However, across both administrations of the CES-D, participants on average scored above the clinical cutoff for the measure, suggesting risk for clinical depression. Third, we did not assess the use of alternative tobacco products such as e-cigarettes and hookah that are popular among this age-group.[54] Fourth, the mood manipulation employed in the present study was a social stressor. Although this stressor has been utilized previously in the manner presented here for inducing NA, it is unclear how other NA manipulations, such as those designed to induce lower arousal NA (eg, sadness), may be related to cigarette demand.

With these limitations in mind, the present study highlights the importance of depression and NA change for predicting cigarette demand. A number of future directions within this line of research would be valuable. There are several candidate moderators that we were unable to explore, including gender, nicotine dependence, age, and severity of depressive symptoms that may help to better understand the relationship between depression, negative mood, and cigarette demand. Additionally, the present study focuses on the relationship between depression and change in NA as a predictor of cigarette demand. As diminished positive affect has also been linked to cigarette smoking,[55] it will be important for future studies to examine the relationship between depression, changes in positive mood, and cigarette demand. Finally, the present study results fit well within existing public policy research regarding the role of taxation in influencing tobacco use.[56] Specifically, recent research highlights that tobacco taxation may indirectly affect tobacco use via changes in cigarette demand.[57] Although different research groups have focused on individual demand indices rather than the aggregate approach used in the present study, studies to date suggest that groups of hardened or marginalized smokers, such as individuals with depressive symptomatology and those who experience high levels of stress, may be less responsive to taxation, resulting in a disproportionate number of individuals with depressive symptoms who are current smokers.[58] Thus, in the future, additional population-level interventions may be necessary in order to influence tobacco use among this high-risk group of smokers.

Supplementary Material

Supplementary Materials can be found online at http://www.ntr.oxfordjournals.org

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Declaration of Interests

The authors have no conflicts of interest to declare. The sponsor had no role in the design and conduct of the study or in the preparation, review, or approval of the manuscript.

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