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Differences in negative mood-induced smoking reinforcement due to distress tolerance, anxiety sensitivity, and depression history

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Abstract

Rationale—Negative mood increases smoking reinforcement and may do so to a greater degree in smokers vulnerable to negative mood dysregulation.

Methods—Adult smokers (N = 71) without current depression were randomly assigned to one of two smoking conditions (nicotine or denic cigarettes, presented blind) maintained across all sessions. Subjects completed one neutral mood session and four negative mood induction sessions. Negative mood inductions included one each of the following: 1) overnight smoking abstinence, 2) challenging computer task, 3) public speech preparation, 4) watching negative mood slides. In each session, subjects took 4 puffs on their assigned cigarette, rated it for "liking" (reward), and then smoked those cigarettes ad libitum (reinforcement) during continued mood induction. Affect was assessed intermittently before and after smoking. Differences in responses were examined as functions of self-reported history of major depression and levels of distress tolerance and anxiety sensitivity.

Results—Smoking reinforcement, but not reward or negative affect relief, was greater in all sessions in those with a history of depression and greater after overnight abstinence in those with lower distress tolerance. Reward and affect relief, but not reinforcement, were greater during speech preparation among those high in anxiety sensitivity.

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Conclusions—Low distress tolerance may enhance acute smoking reinforcement due to abstinence, while depression history may broadly increase acute smoking reinforcement regardless of mood. Neither smoking reward nor affect help explain these individual differences in smoking reinforcement.

Keywords

Smoking; Reinforcement; Nicotine; Depression history; Distress tolerance; Anxiety sensitivity; Negative affect; Mood; Withdrawal

Clinical studies show that acute negative mood is a potent precipitator of relapse in smokers trying to quit (Shiffman and Waters 2004). Similarly, laboratory studies often demonstrate that negative mood induction procedures acutely increase smoking behavior (Kassel et al. 2003; Conklin and Perkins 2005; Perkins et al. 2008a), perhaps comparable to the influence of acute stressors on drug self-administration in rodents (Piazza and LeMoal 1998). However, not all smokers respond to negative mood with an increase in smoking, and some smokers may increase smoking more than others (Gilbert 1995). Identifying individual differences in negative mood-induced smoking may help us understand why some smokers are more vulnerable to smoking maintenance or relapse due to negative mood (Shiffman and Waters 2004).

Although individual differences in the relationship between negative mood and smoking reinforcement have been discussed (Gilbert 1995), virtually no controlled laboratory studies have examined such differences. However, several stable individual difference characteristics related to mood regulation are associated with withdrawal discomfort, relapse risk, or affective responses to smoking and may identify those prone to respond to negative mood by increasing smoking behavior. These characteristics include distress tolerance, anxiety sensitivity, and a history of major depression. Distress tolerance is a “tendency to continue to pursue a goal despite encountering various states of affective discomfort, which may be in response to perceived physical and/or psychological distress” (Brown et al. 2005, p. 718). Low distress tolerance, or inability to persist with difficult or frustrating tasks, may make the discomfort of withdrawal due to smoking abstinence harder to withstand (Abrantes et al. 2008). Greater withdrawal discomfort, which prominently includes negative mood (Piasecki et al. 2003), may explain the association of low distress tolerance with greater risks for cessation treatment drop-out (MacPherson et al. 2008) and relapse (Abrantes et al. 2008; Brandon et al. 2003; Brown et al. 2002, 2005, 2009). Inability to tolerate negative mood also may increase the likelihood that a smoker will smoke for relief of negative affect (i.e., negative reinforcement of smoking).

Similarly, anxiety sensitivity may be associated with greater smoking reward and reinforcement in response to negative mood (Brown et al. 2001). Separate from trait anxiety, anxiety sensitivity is a fairly stable cognitive characteristic that relates to risk of panic and related psychopathology (Vujanovic and Zvolensky 2009). Smokers high in anxiety sensitivity report greater affective responses to acute physical challenges (Vujanovic and Zvolensky 2009). Such smokers tend to believe that quitting smoking will be more difficult (Zvolensky et al. 2007), and they are in fact more likely to lapse early when trying to quit (Brown et al. 2001; Zvolensky et al. 2009). They also believe that smoking is more likely to relieve negative affect (Brown et al. 2001; Leyro et al. 2008) and do report greater relief from smoking after a stressful social challenge than under neutral mood conditions (Evatt and Kassel 2010), suggesting they may be likely to smoke more in response to negative mood-related challenges.

Finally, a history of major depression is much more common among smokers than nonsmokers (Hall et al. 1993; Hitsman et al. 2003). History of depression increases withdrawal severity (Niaura et al. 1999) but generally has not been linked to risk of early relapse (Hitsman et al.

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2003), with some exceptions (e.g., Japuntich et al. 2007). Spring et al. (2008) found that smokers with a history of depression experience greater enhancement of positive mood due to smoking a nicotine cigarette, but not a denicotinized (denic) cigarette, than do smokers without a history of depression. However, smoking a nicotine cigarette also worsened negative affect during negative mood induction in all subjects, contrary to the idea that nicotine per se relieves negative affect (see also Perkins et al. 2008a). Nevertheless, smokers with a history of depression report greater expectancy for negative affect relief due to smoking (Currie et al. 2001), consistent with the notion they may smoke more in response to negative mood (Gehricke et al. 2007). Recent research indicates that smoking-induced dopamine release is greater among smokers with a positive history of depression (Brody et al. 2009), perhaps helping to explain some of these individual differences in affective responses to smoking.

Despite studies linking these stable individual differences in mood regulation to withdrawal discomfort, relapse risk, or affective responses to smoking, we know of no controlled study of smoking reinforcement in response to negative mood as a function of these individual differences. The present study examined individual differences in smoking reinforcement in response to acute negative mood induction, focusing on the characteristics of distress tolerance, anxiety sensitivity, and depression history. In addition to overnight smoking abstinence, several different negative mood tasks were administered, along with a neutral mood control, in separate sessions to determine the generalizability of these individual differences across negative mood contexts (Bernstein et al. 2008). Subjects were randomized to nicotine versus denicotinized cigarette smoking to determine the influence of nicotine per se on smoking reinforcement due to negative mood (Rose 2006; Spring et al. 2008). We also assessed smoking reward (liking) and affect to determine whether they may relate to the individual differences in smoking reinforcement. Individual differences in smoking reinforcement in response to negative mood induction may stem from differences in severity of affective response to the mood induction, greater affective relief due to smoking, or to some other differential impact of negative mood such as an acute increase in smoking reward. Notably, greater acute smoking reward has been related to relapse risk (Shiffman et al. 2006).

Materials and methods

Participants

Participants (n = 71; 43 male, 28 female) were healthy adult smokers (≥10 cigarettes/day) from the surrounding university community. Excluded were those admitting to receiving psychiatric treatment within the past year, including medication or counseling, or who scored above 13 on the Beck Depression Inventory-II (Beck et al. 1996), indicating presence of depressive symptoms. This exclusion was done for two purposes: 1) to avoid exacerbating current depressive symptoms by exposure to the negative mood induction procedures and 2) to rule out current depression as a confounder with the other individual differences as the cause of increased smoking reward and reinforcement. Also excluded were those reporting chronic medical problems including hypertension, heart, lung, liver, or kidney diseases, and seizure history. Subjects were not screened for alcohol or substance abuse problems to maximize generalizability of results to the population of smokers. Most participants were Caucasian (87.3%), with 11.3% African-American, and 1.4% Asian. Mean ± SE sample characteristics were as follows: age of 26.2 ± 1.4 years, nicotine yield of preferred brand of 1.02 ± 0.03 mg, daily smoking rate of 19.5 ± 0.8 cigarettes/day for 10.1 ± 1.4 years, and Fagerstrom Test of Nicotine Dependence (FTND; Heatherton et al. 1991) score of 4.6 ± 0.3, indicating moderate dependence.

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Mood induction

Five different procedures were used to induce negative (four) or neutral (one) mood, with one procedure per session. Neutral mood was a control, by which to determine increases in responding due to negative mood. Overnight abstinence, which induces negative mood (Parrott et al. 1996), provided an assessment of the effects of tobacco deprivation on smoking reinforcement. The other three acute negative mood induction procedures were designed to produce a level of negative affect comparable to that due to overnight abstinence. These three procedures intentionally varied in active versus passive coping (i.e., whether or not there was an explicit behavior that subjects believed could reduce the degree of challenge posed by the task) and between immediate and clear versus anticipatory and vague response requirements (Gilbert 1995; Hasenfratz and Battig 1993).

1. Neutral mood control—Subjects watched slides from the International Affective Picture System (IAPS; Lang et al. 1988) that were neutral in emotional valence. Slides were shown in a dark room for 12 s each, one after the other, to sustain the intended mood. A similar procedure has been used as a neutral mood control in prior smoking research (Conklin and Perkins 2005).

2. Overnight abstinence—Subjects abstained overnight from smoking for at least 12 h before the session, verified by expired air CO≤ 10 ppm (SRNT Subcommittee 2002). To maintain subject attention during the session, they engaged in the neutral mood control task (above), but with different neutral slides to avoid repetition of stimuli.

3. Negative mood slides—This procedure involved high arousal negative affect pictorial slides from the IAPS (Lang et al. 1988) and was similar to the Neutral Mood procedure described above except for the mood valence of the slides. A similar procedure has been used to increase negative affect in prior smoking studies (Conklin and Perkins 2005; Perkins et al. 2008a).

   Subjects correctly identified 85.7 ± 0.9% of the content of the slides shown them during the negative and neutral mood slide procedures, with no differences due to mood valence or between nicotine and denic groups, verifying that subjects attended to the slides.

4. Computer challenge—Subjects were presented with a sequence of the digits 1–4 in random order on a computer monitor and were instructed to repeat the sequence in either the same or the reverse order using a keypad. Responding required the use of just one hand, allowing the other hand for smoking. Task difficulty (i.e., number of digits to remember) was continuously adjusted by the computer to ensure a comparable degree of challenge and task success (40% correct) for all subjects, regardless of ability. Correct responses earned $.50 and errors resulted in a loss of $. 25. This task increases negative affect in smokers and nonsmokers (Perkins et al. 1992).

5. Speech preparation—Subjects were told to prepare and then give two 3-min speeches, one at a time, on what they liked and disliked about their body. Subjects were not told they would have to give a second speech (dislike) when they received instructions for the first (like). Having to prepare two speeches allowed us to match the duration of this procedure with that of the other mood induction procedures. This task elicits robust negative affect in similar research (e.g. Kassel and Shiffman 1997; Sayette et al. 2001; Evatt and Kassel 2010).

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Individual difference measures

Distress tolerance—The Distress Tolerance Scale (DTS; Simons and Gaher 2005) contains 16 items assessing four factors: perceived ability to tolerate emotional distress, subjective appraisal of distress, attention being absorbed by negative emotions, and regulation of efforts to relieve distress. Each item provides 5 response options, from 1 ("strongly agree") to 5 ("strongly disagree"), with low scores indicating inability to tolerate distress. The general distress (i.e. total) score was used in analyses.

Anxiety sensitivity—The Anxiety Sensitivity Index (ASI; Reiss et al. 1986) is a 16-item Likert scale, with 5 response options ranging from 0 ("very little") to 4 ("very much"). Items assess the degree of fear about experiencing various anxiety symptoms. The psychometric properties of the ASI are outlined elsewhere (Peterson and Reiss 1992). The ASI total score, in which high scores indicate high sensitivity to anxiety symptoms, was used in analyses.

History of depression—The Inventory to Diagnose Depression-Lifetime (IDD; Zimmerman et al. 1986; Zimmerman and Coryell 1987) is a 22-item self-report scale designed to diagnose the lifetime history of major depression. Each IDD item provides 5 response options (scored 0–4) corresponding to a statement pertaining to the severity of a specific symptom, with an additional question about whether that symptom persisted for at least 2 weeks. The IDD produces a dichotomous "positive" or "negative" history of depression. IDD-determined depression history agrees reasonably well with history of major depression as determined by the Diagnostic Interview Schedule (Zimmerman and Coryell 1987). Psychometric properties of the IDD are presented by Zimmerman and Coryell (1987).

Self-report dependent measures

Smoking reward—Reward ("liking"), the hedonic value of a stimulus (Berridge and Robinson 2003), was assessed by an item from the Cigarette Evaluation Scale (Westman et al. 1996) which states, "How much do you like this cigarette?" It was rated on a 0 ("not at all") to 100 ("extremely") visual analog scale (VAS).

Negative and positive affect—Four measures of negative affect were used because of the lack of a single gold standard. These four, selected for their diversity of items and frequency of use in mood induction studies, were the Diener and Emmons (1984) Mood Form (converted from 0–6 to a 0–100 visual analog scale, or VAS), the Positive and Negative Affect Scale (PANAS; Watson et al. 1988), the "stress" items of the Stress–Arousal Checklist (SACL; Mackey 1980), and an abbreviated version of the State-Trait Anxiety Inventory-state (STAI; Spielberger et al. 1970) shown to be sensitive to acute laboratory challenges (Sayette et al. 2001). This brief STAI contains 6 of the 20 items, 3 negative affect items (upset, worried, and frightened) and 3 reverse-scored items (calm, secure, and self-confident). Positive affect was assessed with the positive affect subscales of the Mood Form and the PANAS.

Session procedures

These data are from a larger study aimed at examining the situation-specificity of negative affect relief due to smoking (Perkins et al. 2010). This study was approved by the University of Pittsburgh Institutional Review Board. Participants were randomly assigned to smoke nicotine cigarettes (Quest 1; 0.6 mg nicotine, 9 mg tar) (n = 37) or denic cigarettes (Quest 3; 0.05 mg nicotine, 9 mg tar) (n = 34) across the 5 sessions. The nicotine and denic groups did not differ in age, ethnicity, or smoking characteristics or in ASI or DTS scores, or in the proportion with a positive history of depression (n = 6 in each group). The cigarettes were obtained commercially from Vector Group, Ltd. (Miami, FL) and have been widely used in

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smoking research (e.g., Donny et al. 2006; Perkins et al. 2008a). Cigarette markings were covered over to blind subjects to brand.

Subjects participated in 5 experimental sessions, one per mood induction procedure (4 negative and 1 neutral). The order of mood induction procedures across sessions was randomized between subjects, but the same sequence of orders was used for each smoking group, stratified by sex. Except for the overnight abstinence session, participants smoked ad libitum prior to each session and smoked one cigarette of their own brand upon arrival. We wanted to control the time since their last cigarette and to ensure that the induction of negative mood was due to the specific mood procedure for that day and not to abstinence-induced withdrawal. Other than the specific mood induction task, each session followed the same timeline. After a period of quiet rest (BL, baseline), subjects were introduced to the mood procedure for that session and engaged in it for 5 min to induce mood (Time 1). Subjects then took four standard puffs on the designated cigarette and rated it for reward, followed by continuation of the mood induction procedure for another 3 min (Time 2). Mood induction then continued, and subjects were allowed to smoke their assigned cigarettes ad libitum over the last 10 min of the mood induction (Time 3). The smoking during this period was taken as the measure of smoking reinforcement. Affect was assessed at each time point (BL, Time 1–3).

All smoking was done via the Clinical Research Support System (CReSS; Borgwaldt KC, Inc., Richmond VA; www.plowshare.com), which assesses puff volume and has been used in numerous studies of acute smoking (e.g. Brody et al. 2009). The timing and amount of smoke inhalation during the four standard puffs prior to Time 2 was controlled by computer-administered instructions (Perkins et al. 1992, 2008a). Total volume from the four puffs did not differ between the nicotine and denic groups (238 ± 11 vs 243 ± 11 ml, respectively) or across the five mood procedures (ranging from 235 ± 8 to 248 ± 9 ml), indicating control of smoke intake prior to the reward rating. Smoking behavior during the ad libitum period (between Time 2 and Time 3) was done via the CReSS but not controlled by instructions.

Data analyses

No main effects or interactions involving sex were found in analyses, and so results were collapsed across men and women. We could not analyze the order of the five mood procedures across sessions because of the large number of possible orders, although orders were random and the order schedule was the same for each smoking group. Individual differences in smoking reinforcement (total puff volume during ad lib smoking), reward (liking), and affect were analyzed via analyses of variance (ANOVA). (FTND and cigarettes per day, indices of dependence, were not related to these individual differences or with the dependent measures and so were not used as covariates.) For the dichotomous individual difference of depression history, depression history (positive vs negative) and nicotine (nicotine vs denic cigarette) were between-subjects factors. For the continuous differences of ASI and DTS score, nicotine was a between-subjects factor and the individual difference (ASI or DTS score) was included as a covariate. In all analyses, a within-subject factor was mood procedure (neutral mood control and the four negative mood conditions). We hypothesized that smoking reinforcement would be greater due to positive depression history, higher ASI score, and lower DTS score. Interactions of these characteristics with mood procedure could indicate greater smoking reinforcement specific to negative mood context, while interactions of characteristics with nicotine could indicate greater reinforcement from nicotine intake per se. The pattern of effects on smoking reward was examined to determine whether the observed differences in smoking reinforcement may relate to differences in smoking reward. Effect sizes of particular effects of interest were presented by partial eta-squared values (η_p^2), which indicate the percent of variance explained.

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Negative and positive affect from Time 1 (during mood induction but before smoking) to Time 2 (after four puffs) and to Time 3 (after ad lib smoking) were analyzed to determine whether the individual differences in smoking reinforcement may relate to similar differences in affective responses to the mood procedures and to smoking. Because negative affect was assessed with four separate measures, the analyses of negative affect for each individual difference began with multivariate ANOVA (MANOVA), with follow-up univariate ANOVAs where the overall multivariate analyses were significant. Each of these analyses involved the same between- and within-subjects factors as in the reinforcement and reward analyses, plus the within-subjects factor of time. Interactions of the individual difference characteristics with mood procedure could indicate greater affective response to the negative mood induction contexts, while interactions involving time could indicate greater affective relief due to smoking.

Results

Distribution of individual differences

Mean ± SD scores for the sample were 15.5 ± 7.5 for ASI and 3.62 ± 0.64 for DTS. Men and women did not differ on either measure. The ASI mean is comparable to the means reported for some groups of smokers (e.g., MacPherson et al. 2008), but ASI may be higher in other smokers (Marshall et al. 2009; Evatt and Kassel 2010). The DTS is a newer measure, and fewer studies exist for comparison, but the mean DTS score in our sample is comparable to the mean observed in other research with young adults (Simons and Gaher 2005). For the IDD, 12 of the 71 subjects (6 in each smoking group), or 17%, gave responses indicative of a positive history of major depression. This percentage is comparable to the prevalence of lifetime depression history among less dependent smokers in a national survey of nearly 9,000 smokers (Manley et al. 2009) but is less than the prevalence seen in more dependent smokers (Manley et al. 2009) and in trials of quitting smokers (Hitsman et al. 2003). None of these individual difference characteristics was related to age or smoking history, including FTND score. History of depression was unrelated to ASI or DTS scores, $F(1,69)$'s < 1, but ASI and DTS were significantly correlated, $r(69) = -0.372$, $p < 0.001$, as higher anxiety sensitivity was related to lower tolerance of distress.

To simplify how the figures display findings involving the continuous characteristics of DTS and ASI scores, the figures present subjects divided by median split into high and low DTS and ASI subgroups, although analyses were done using continuous scores, as previously noted. A large number of subjects with DTS scores at the median were put in the “low” DTS subgroup. One subject did not complete the ASI and so is not included in those analyses.

Smoking reinforcement

Total puff volume during the ad libitum smoking period was increased by mood procedure, $F(4,256) = 14.21$, $p < 0.001$, as smoke reinforcement was greater after overnight abstinence and during speech preparation, compared to neutral mood. However, smoke reinforcement was not influenced by nicotine, $F(1,64) = 2.44$, $p > 0.10$, or the interaction of mood procedure × nicotine, $F(4,256) < 1$. Smoking reinforcement was also influenced by depression history and distress tolerance. As shown in Fig. 1, puff volume was greater in those with a positive versus negative depression history on the IDD, $F(1,65) = 13.95$, $p < 0.001$, $\eta_p^2 = 0.177$, but not by the interaction of IDD × mood procedure, as the difference due to IDD was apparent across all mood procedures. Smoke reinforcement was also influenced by the interaction of DTS × mood procedure, $F(4,260) = 3.54$, $p < 0.01$, $\eta_p^2 = 0.052$, as smoking was greater in those with low versus high DTS scores, but only after overnight abstinence (Fig. 1). No other effects were significant, and smoke reinforcement did not differ significantly by anxiety sensitivity (ASI score).

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Smoking reward and affect

Reward—Cigarette liking was increased by the main effects of mood procedure, $F(4,188) = 3.33$, $p < 0.02$, and nicotine, $F(1,47) = 5.97$, $p < 0.02$, but not by the interaction of mood procedure × nicotine, $F(4,188) = 1.14$, ns. Compared to liking during neutral mood, liking was greater after overnight abstinence but not the other negative mood procedures. Liking was also greater in response to the nicotine versus denic cigarette. In terms of individual differences, liking differed by anxiety sensitivity, as significant effects were seen for ASI score, $F(1,66) = 4.67$, $p < 0.05$, $\eta_p^2 = 0.066$, and by the interaction of ASI × mood procedure, $F(4,264) = 3.18$, $p < 0.02$, $\eta_p^2 = 0.046$. As shown in Fig. 2, cigarette liking was greater for those high versus low on the ASI but only during speech preparation. Cigarette liking was not related at all to depression history or distress tolerance.

Affect—MANOVA results showed significant main effects of mood procedure on negative affect, $F(16, 1054) = 7.73$, $p < 0.001$, confirming the robust efficacy of the four negative mood induction procedures, relative to neutral mood. Negative affect did not vary by distress tolerance or depression history. However, negative affect was influenced by the interaction of mood procedure × time × ASI, $F(32, 2094) = 1.55$, $p < 0.05$. Follow-up ANOVAs showed that this interaction was significant for the STAI and SACL measures, $F(8,528)$'s of 2.10, $p < 0.05$, $\eta_p^2 = 0.031$, and 3.07, $p < 0.005$, $\eta_p^2 = 0.044$, respectively. As also shown in Fig. 2, both negative affect measures decreased more over time (i.e. after smoking) in those with high versus low ASI, but only during speech preparation. We conducted exploratory correlations between these negative affect measures and reward during the speech preparation task, given the similar pattern of differences due to ASI. Reward was related to the SACL ($r = 0.27$, $p < 0.05$) but not STAI ($r = 0.18$).

Somewhat similar results were seen for positive affect, as the only significant finding involving individual differences was the interaction of mood procedure × time × ASI on responses to the Mood Form, $F(8, 528) = 2.97$, $p < 0.005$, $\eta_p^2 = 0.043$, but not the PANAS-PA subscale. Smoking increased positive affect more in those with high versus low ASI (Fig. 2). However, this difference was seen only after the initial four standard puffs and not ad lib smoking, and only after abstinence (rather than speech preparation).

Discussion

Results indicate that depression history and low distress tolerance, two stable individual difference characteristics related to mood regulation, may enhance acute smoking reinforcement, in general or in response to overnight smoking abstinence. However, neither characteristic was specifically related to greater smoking reinforcement in response to negative mood induction per se, and anxiety sensitivity was not related to smoking reinforcement at all. By contrast, only higher anxiety sensitivity was related to greater smoking reward and negative affect relief due to smoking, but only in response to the speech preparation task. Therefore, there is little evidence that these three individual difference characteristics are associated with greater smoking reinforcement specifically due to negative mood, or that the individual differences in smoking reinforcement that were observed stem from corresponding differences in affect. Nevertheless, greater smoking reinforcement in those with a depression history and low distress tolerance may help clarify why these characteristics are associated with smoking persistence or responses to smoking under some mood conditions.

Greater smoking reinforcement under all conditions, even during neutral mood, in smokers with a depression history indicates that negative mood is not necessary to elicit greater smoking reinforcement in this group. As suggested, the lack of individual differences in negative affect or reward does not provide directions for determining the mechanisms for this greater smoking reinforcement. Moreover, smoking history variables (including FTND score and cigarettes per

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day) were unrelated to the individual differences, ruling out differences in nicotine dependence as an explanation for the greater smoking reinforcement in those with a depression history. Yet, this observation is consistent with Brody et al. (2009), who found greater dopamine release after smoking a usual brand cigarette in smokers with a positive versus negative depression history in the absence of any mood induction, perhaps suggesting a possible mechanism to explain generally greater smoking reinforcement in such smokers.

Smoking reinforcement was also greater in those with low versus high distress tolerance, but specifically after overnight abstinence and not after the other negative (or neutral) mood conditions. Thus, low distress tolerance may increase acute smoking reinforcement in response to tobacco deprivation but may not broadly influence smoking reinforcement in response to other negative mood conditions. This finding is consistent with the greater rates of treatment drop-out and relapse in smokers low in distress tolerance (Abrantes et al. 2008; Brown et al. 2009). However, the lack of differences in affect does not support the notion that low distress tolerance is necessarily associated with greater withdrawal discomfort during abstinence or with greater relief of this discomfort after smoking.

The third individual difference characteristic examined, anxiety sensitivity, was associated with greater cigarette liking and greater decline in negative affect after smoking during speech preparation but not during the other negative or neutral mood procedures. Therefore, in contrast with depression history and distress tolerance, some evidence was found for those with high anxiety sensitivity being more sensitive to smoking reward and negative affect relief during a social stressor. This is consistent with other research showing that they are also more likely to believe that smoking will relieve negative affect (Brown et al. 2001; Leyro et al. 2008) and do report more relief due to smoking after a similar speech task than after neutral conditions (Evatt and Kassel 2010). However, because smoking reinforcement was not also greater among those high in anxiety sensitivity, it is difficult to argue that these responses reflect greater negative reinforcing effects of smoking in this particular mood context.

The nicotine content of the cigarettes did not interact with these individual differences to affect smoking reinforcement, reward, or affect. Our results are consistent with other findings showing little influence of nicotine on these and other acute responses to smoking, suggesting that non-nicotine, perhaps conditioned aspects of smoking are critical (Donny et al. 2006; Rose 2006; Perkins et al. 2008a). However, another study found that depression history is related to greater enhancement of positive affect from smoking a nicotine cigarette per se during positive mood induction (Spring et al. 2008). The conditions under which nicotine differentially influences affect depending on the smoker's characteristics warrants further study.

Strengths of the study include a neutral mood control condition and the several types of negative mood conditions, in addition to overnight abstinence, to determine the generalizability of individual differences in smoking reinforcement, reward, and affect across mood contexts. With this approach, we were able to show that anxiety sensitivity was related to greater smoking reward and affect relief specifically in response to a social stressor but not to the other negative mood procedures. Moreover, we were also able to show that the different types of negative mood procedures (other than smoking abstinence) failed to elicit differential reinforcement, reward, or affect as a function of depression history or distress tolerance; use of just one type could have limited the generalizability of our conclusions about the null effects of negative mood induction. Among other study strengths, the within-subjects comparison across mood procedures increased statistical power, and inclusion of a denic cigarette control group allowed us to examine individual differences specific to nicotine intake, which were generally not found.

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Limitations of the study include the reliance on self-report measures of individual differences, the small number of smokers with a history of depression, and the somewhat low ASI scores, suggesting that more robust effects may be seen in samples of smokers varying more broadly in the individual differences of interest. We did not screen smokers for alcohol or substance abuse to increase variability in these characteristics and enhance generalizability. However, the exclusion of smokers with current or recent depression or other psychiatric problems likely limited the proportion of those with a history of depression, low distress tolerance, or high anxiety sensitivity. Moreover, our subjects were not interested in quitting smoking, and these characteristics may be particularly potent influences on smoking reinforcement during negative mood among those attempting to quit permanently (Brandon et al. 2003; Brown et al. 2009). The relevance of our findings to smoking reinforcement in the natural environment is also unclear, given the brief durations of mood induction and smoking opportunity, as well as the uncertain generalizability of our acute negative mood induction procedures to negative mood contexts in the natural environment. Finally, the effect sizes for most of the significant effects were relatively modest, accounting for less than 10% of the variance in most cases. Consequently, limited statistical power is another concern, as our sample size provided power of approximately 0.7 to detect medium effects of individual differences in smoking in response to negative mood induction (Cohen 1988).

Further research of this kind with larger and more diverse samples of smokers, including those trying to quit, and over longer periods of observation may clarify the extent to which distress tolerance, anxiety sensitivity, and depression history influence smoking persistence or responses to smoking. Because these individual difference characteristics may be malleable by various interventions (e.g. Brown et al. 2008), it would be interesting to determine whether those same interventions could attenuate the greater smoking reinforcement observed here in smokers with depression history and during abstinence in smokers with low distress tolerance. Other individual difference factors, including genetics, should be examined for associations with smoking reinforcement during negative mood (e.g. Perkins et al. 2008b). Finally, the potential influence of these and other individual differences on reinforcement from other drug use, such as alcohol, during negative mood warrants similar examination (Wietkiewitz and Villarroel 2009).

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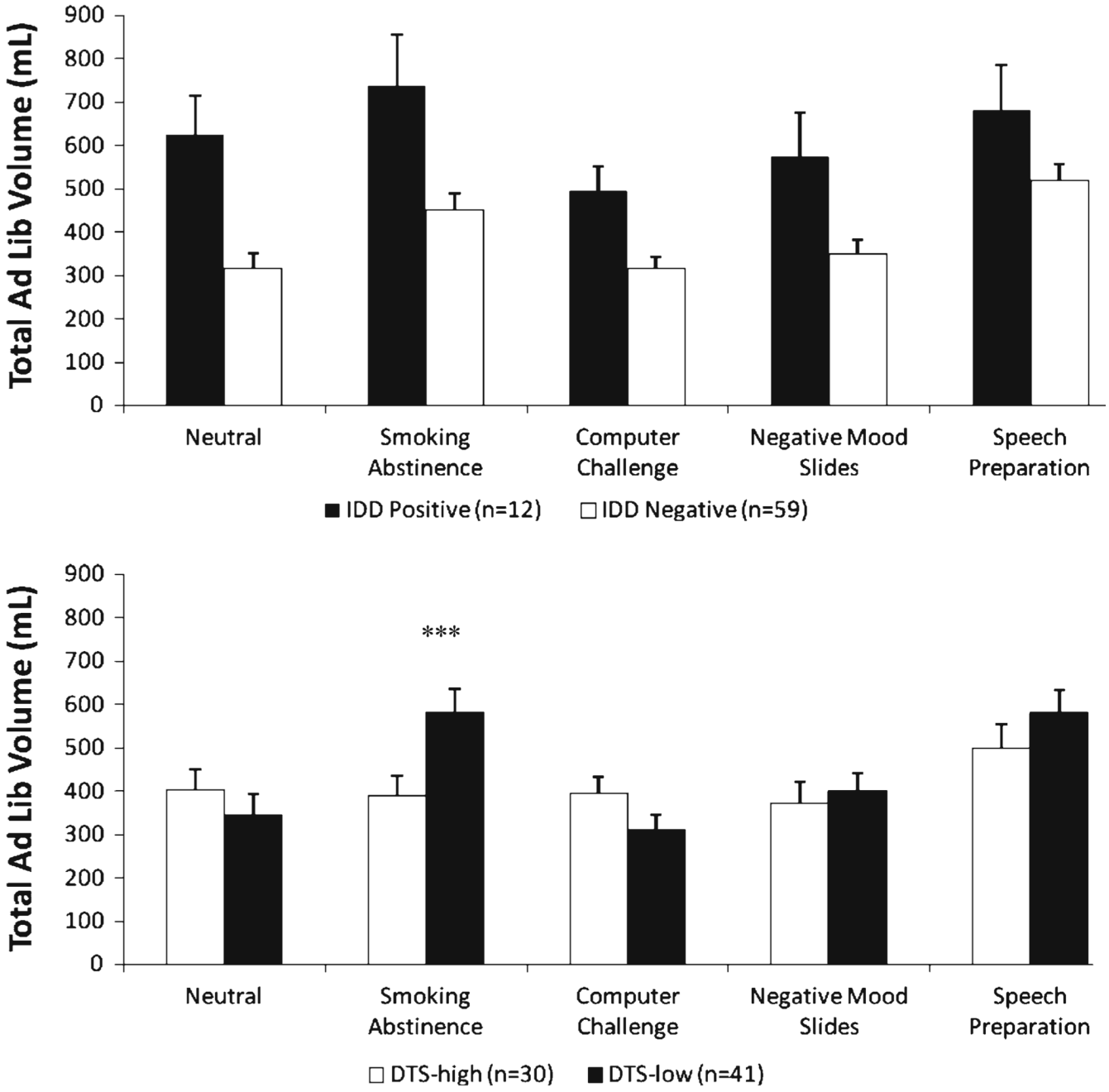

Fig. 1.

Mean ± SE puff volume (i.e. smoking reinforcement) during ad lib smoking across the neutral and four negative mood induction procedures by positive versus negative history of depression (IDD; top) and by high and low distress tolerance (DTS score; bottom). Results are collapsed across nicotine and denic cigarette groups because there was no effect of nicotine. Differences due to depression history were significant across all five mood procedures (i.e., main effect of depression history). *** $p < 0.001$ for the difference due to characteristic during specific context

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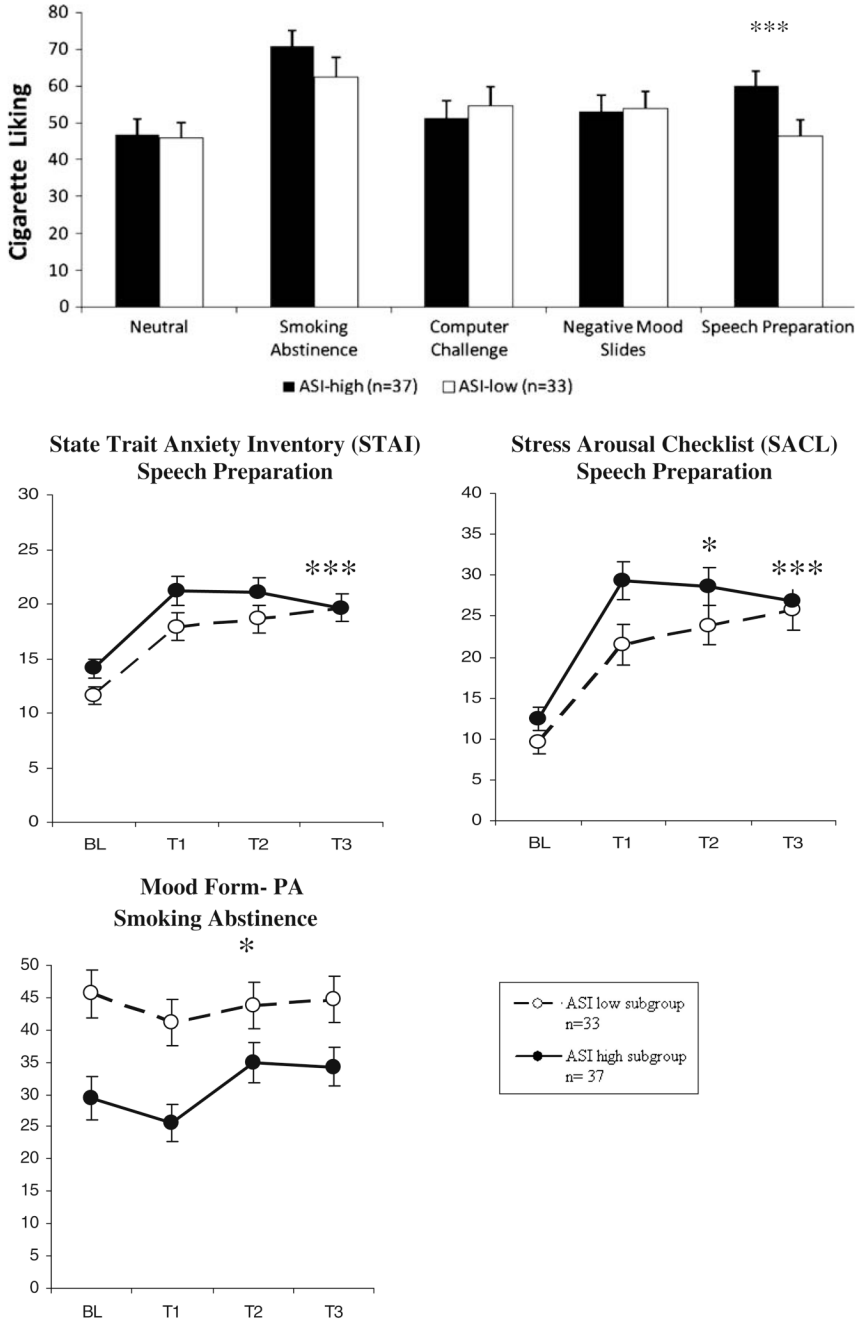

Fig. 2.

Mean ± SE cigarette liking (0–100) across the five mood induction procedures (top), and selected mean ± SE negative affect (STAI, SACL) and positive affect (Mood Form PA) responses across time (bottom), by high and low anxiety sensitivities (ASI score). Results are collapsed across nicotine and denic cigarette groups because there was no interaction involving nicotine. For liking, asterisks are as in Fig. 1. For negative and positive affect, * $p < 0.05$ and *** $p < 0.001$ for the difference due to ASI score in change in affect from T1 to T2 or from T1 to T3