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Reward Expectations Lead to Smoking Uptake among Depressed Adolescents

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Abstract

Aims—While the comorbidity between adolescent depression and smoking has been well documented, less is known about why smoking is disproportionately higher among depressed adolescents. Emerging research suggests that reward-related mechanisms may be important to consider. This study sought to determine whether adolescents with higher depression symptoms have greater smoking reward expectations, which in turn, influence smoking progression.

Participants—The sample was composed of 1,393 adolescents participating in a longitudinal survey study of adolescent health behaviors.

Design and Measurements—In this prospective cohort study, variables were measured via self-report every six months from age 14 to age 17 resulting in six waves of data.

Findings—Parallel processes latent growth curve modeling indicated that higher depression symptoms across mid to late adolescence predicted a 17% increase in smoking reward expectations ($\beta = 3.50$, $z=2.85$, $p=.004$), which in turn predicted a 23% increase in the odds of smoking progression ($\beta = .206$, $z=3.29$, $p=.001$). The indirect effect was significant with delta method ($\beta_{\text{indirect}} = .72$, $z = 3.09$, $p=.002$; 95% CI= .26, 1.18) and bootstrap ($\beta_{\text{indirect}} = .72$, $z = 2.10$, $p=.03$; 95% CI= .05, 1.39) standard errors.

Conclusions—The study provides novel evidence that expectations of smoking reward facilitate smoking uptake among depressed adolescents. Smoking reward expectations may identify depressed adolescents at risk of smoking. Addressing alternative ways to meet the reward expectations rather than smoking may be an important component to consider in the preventing smoking and promoting smoking cessation among adolescents with elevated depression symptoms.

Keywords

adolescents; depression; reward expectations

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1. Introduction

Smoking and depression typically have an adolescent onset (Rohde et al., 2004). Over 20% of adolescents regularly smoke cigarettes (CDC, 2008), with the percentage of regular smokers doubling from mid to late adolescence (CDC, 2008; Johnston et al., 2008). Over 25% of adolescents experience elevated depression symptoms during this developmental period (Fergusson et al., 2005; Georgiades et al., 2006; Lewinsohn et al., 2004) and about 20% have at least one major depressive episode by 18 years of age (SAMHSA, 2005). Given that smoking is considered a rewarding behavior and depression has been conceptualized as a disorder of disrupted reward processing (Forbes, 2009), it is not surprising that smoking is over-represented among adolescents with clinical episodes and subclinical symptoms of depression (Brown et al., 1996; Fergusson et al., 2003; Goodman and Capitman, 2000; Rohde et al., 2004; Windle and Windle, 2001).

While the comorbidity between adolescent depression and smoking (onset, persistence and cessation) has been well documented (Audrain-McGovern et al., 2009), less is known about why smoking is disproportionately higher among depressed adolescents compared to adolescents without depression. Research provides support for different directional causal relationships between adolescent depression and smoking (Fergusson et al., 2003; Goodman and Capitman, 2000; Prinstein and La Greca, 2009). However, the mechanisms linking depression and smoking may lend clarity to common and unique etiological pathways for the comorbidity. Recent research suggests that depression symptoms and smoking reciprocally influence one another across adolescence, and that peer smoking behavior accounts, in part for this relationship (Audrain-McGovern et al., 2009). Adolescents with elevated depression symptoms may be more vulnerable to or attracted by peers who smoke, highlighting the role of social contingencies in smoking uptake.

Converging research offers further insight into reward related mechanisms that may increase our understanding of how depression may influence adolescent smoking. Depression, even at the level of elevated symptoms, is often accompanied by withdrawal, less involvement in activities and diminished reward from usual activities (Jacobson et al., 1996; Lewinsohn et al., 1998; MacPherson et al., 2010). Behavioral theory suggests that this decline in overall reinforcement creates a vulnerability for substance use, especially when substances are readily available (Higgins et al., 2004; Jacobson et al., 1996; Rogers et al., 2008). Thus, as elevated depression symptoms precipitate declines in involvement in other reinforcers or the pleasure derived from those reinforcers, adolescents may develop expectations that other activities, such as smoking confer reward.

Depressive symptoms may also decrease adolescents' perceptions of their personal risks associated with smoking (Rodriguez et al., 2007) and alter neurocognitive processes involved in planning and decision making that favor positive expectations of smoking and a greater willingness to use cigarettes (Maalouf et al., 2011). For adolescents with depression, these expectations may also be shaped by environmental factors such as cigarette advertisements (Tercyak et al., 2002) and the benefits that they perceive their peers to be deriving from smoking (Audrain-McGovern et al., 2009). Previous research has shown that positive smoking expectations predict adolescent smoking status (Spruijt-Metz et al., 2005) and smoking initiation (Simons-Morton, 2004), but their role in smoking progression has received little attention (Wahl et al., 2005). Expectations of smoking reward have been shown to correlate with a past history of major depression and young adult smoking status (McChargue et al., 2004). Based upon the literature, it is possible that adolescents with elevated depression symptoms develop greater smoking reward expectations that then influence smoking progression.

This prospective cohort study sought to determine whether adolescents with higher depression symptoms have greater smoking reward expectations, which in turn, influence smoking uptake. We were interested in one causal direction whereby depression confers vulnerability to smoking, yet also examined whether smoking contributed to depression. Since depression symptoms are episodic, with individual variation in chronicity, severity and recurrence across time, we chose an analytic model that would capture the risk associated with having significant depression symptoms at six time points across three years. As smoking is over-represented among individuals prone to depression (Brown et al., 1996; Fergusson et al., 2003; Goodman and Capitman, 2000; Rohde et al., 2004; Windle and Windle, 2001), elucidating mechanisms responsible for the comorbidity may shed light on novel smoking prevention approaches for this at risk population.

2. METHODS

2.1 Participants and procedures

Participants were high school students (50% female and 74% White) taking part in a longitudinal study of the relationship between adolescent physical activity and adolescent smoking adoption. Participants were enrolled in one of four public high schools outside Philadelphia, PA. This cohort was drawn from the 1,517 students identified through class rosters at the beginning of ninth grade. Students were ineligible to participate in this study if they had a special classroom placement (e.g., severe learning disability) or if they did not speak fluent English. Based on the selection criteria, a total of 1,487 (98%) students were eligible to participate. Of these 1,487 eligible teens, 1,478 (99%) had a parent's consent to participate. Thirty adolescents were absent on the assent/survey days and 19 adolescents did not provide assent due to lack of interest in the study. Thus, 1,429 of 1,478 teens with parental consent (97%) provided their assent to participate and completed a baseline survey.

The adolescent cohort was formed in the 9th grade and is being followed until the end of 12th grade. A self-report survey is administered every six months (fall and spring) on-site during compulsory classes each year of high school for a total of eight surveys. The surveys take approximately 40 minutes to complete. The current analyses span the first six of eight waves (~ age 14-15 to 16-17 years of age). Participants were individuals (N=1,393) with complete data on the covariates. University Institutional Review Board approval of the study protocol was obtained.

2.2 Measures

2.2.1. Smoking—Smoking was assessed at each wave with 13 standard epidemiological questions, such as “Have you tried or experimented with cigarette smoking, even a few puffs?” and “When was the last time you smoked a cigarette?” (Audrain-McGovern et al., 2009; Eaton et al., 2006). A 6-level ordered categorical variable was created reflecting smoking progression from: 0=never smoked; 1= puff to one cigarette in lifetime; 2=smoked at least one cigarette, but none in the past 30 days; 3=smoked at least one cigarette lifetime and at least one in the past 30 days; 4=smoked at least one cigarette a week; and 5=smoked at least 1 cigarette a day.

2.2.2. Depression symptoms—Depression symptoms were assessed at each wave with the Center for Epidemiological Studies Depression (CES-D) inventory. The CES-D is a valid and reliable 20-item self-report measure designed to assess depression symptoms in the general population (Radloff, 1991; Roberts et al., 1990). Scores range from 0 to 60. The 20-items were linearly summated to form a single depression symptoms score at each wave. As we sought to compare depressed adolescents to nondepressed adolescents, we used a clinical cut-off of 22 to distinguish adolescents with (CESD > 22, higher depression) and

adolescents without ($CESD \leq 22$, lower depression) clinically significant levels of depression symptoms (Roberts et al., 1991).

2.2.3. Smoking Reward Expectations—Positive expectations of smoking were measured with a 7-item Likert-style scale (Cronbach's $\alpha > .75$) (Spruijt-Metz et al., 2005; Wahl et al., 2005). Statements included, "I think I would enjoy smoking", "I think smoking would be relaxing". Statements covered smoking as a source of pleasure, something to do, a way to handle problems, a method to stay thin and look mature, and a way to relax. Responses ranged from 1= disagree to 4=agree with higher scores indicating more positive expectations.

2.3 Covariates

Demographic variables included gender and race. Household smoking was assessed with a binary variable (0=nobody living in the household smokes, 1=at least one household member smokes). Peer smoking was measured by summing responses to three items asking whether the adolescent's best friend smokes, and whether and, if so, how many of his or her other four best male and four best female friends smoke (range 0 to 9 friends smoking) (Audrain-McGovern et al., 2009). Marijuana use and alcohol use were each assessed with one item asking "During your lifetime, how many times have you used marijuana?" and "During your lifetime, how many days did you have at least one drink (not just a sip) of alcohol?" Response choices ranged from 0 to 100 or more days for alcohol use and 0 to 100 or more times for marijuana use (Audrain-McGovern et al., 2004; Eaton et al., 2006). Perceived parental monitoring was measured with a 5-item Likert-style scale that evaluated adolescent perceptions of parental knowledge of whereabouts, activities, and friendships (DiClemente et al., 2001; Kodl and Mermelstein, 2004; Simons-Morton, 2004). Response options range from 1=almost nothing to 3=a lot. Higher scores indicated greater monitoring (Cronbach's $\alpha = .72$). We controlled for these covariates in the model as potential confounding variables given their associations with smoking and depression symptoms (Audrain-McGovern et al., 2004).

2.4 Data analysis

Univariate statistics were generated to describe the study population in terms of demographics, smoking practices, smoking reward expectations, and depression symptoms. Univariate estimates were generated with SAS 9.1.3 software.

2.4.1. Latent Curve Growth Modeling (LCGM)—Associated-processes LCGM was conducted to assess the direct and indirect effects of depression symptoms on adolescent smoking through smoking reward expectations across time. LCGM is a Structural Equation Modeling (SEM) method that models repeated observed measures on factors (latent variables) representing random effects (η s). A level factor represents baseline level and a trend factor represents growth or rate of change across time from baseline (i.e., each unit change in time is associated with a η change in a given process). Latent variables define the form of growth (e.g., linear, quadratic, cubic) across time by way of factor loadings (i.e., correlations between the observed and latent variables). Factor loadings were fixed to define baseline or level (factor loadings are restricted to equal 1 from the level/intercept factor to each observed measure) and trend after testing for a non-linear (quadratic) growth form. For a linear trend, factor loadings are set so they increase uniformly with each unit increase in time (six months in the present study). In the present model, the factor loading from the trend factor to the first observed measure was constrained to equal zero as the first observed measure was the baseline level. The second was constrained to equal one, indicating a unit increase in the rate of change in each process (i.e., smoking, smoking expectancies, and depression symptoms) for a unit increase in time. The remaining loadings for the trend

factor were constrained similarly to define a linear growth form (i.e., 2, 3, 4, and 5, for waves 3 through 6, respectively).

In the present analysis, we conducted associated processes LCGM. Associated processes LCGM is a multivariate method that allows testing paths among random effects (i.e., levels [η_0] and trends [$\eta_{1,2,\dots}$]) from two or more LCGMs (Duncan et al., 1999). Three associated processes were modeled in the present study, one each for the repeated observed measures of depression symptoms (binary), adolescent smoking (ordered categorical), and smoking reward expectations (continuous). We were interested in whether depression symptoms at baseline (level) or the rate of change in depression symptoms across time (trend) affected smoking progression (trend) directly and indirectly through changes in smoking reward expectations (trend) above and beyond the effects of change in time (wave) on smoking.

To control for the effects of prior smoking on change in smoking expectations and depression symptoms, we included and tested paths from smoking baseline level to smoking reward expectations trend and depression symptoms trend. Similarly, to control for the effects of prior smoking reward expectations on change in smoking and depression symptoms, we included and tested paths from smoking expectations baseline level to depression symptoms trend and smoking trend. Finally, to control for the effects of prior depression symptoms on change in smoking and smoking expectations, we included and tested paths from depression symptoms baseline level to smoking trend and smoking reward expectations trend. As smoking and depression symptoms are categorical, we estimated model parameters with a Weighted Least Squares estimation technique (WLSMV) in which the diagonal weight matrix uses robust standard errors, and the chi-square test statistic is Mean and Variance adjusted (Muthén and Muthén, 1998-2004). Suggested criteria for model fit are non-significant model chi-square, CFI above .95, RMSEA below .05–.08, and a WRMR value below .9 (Loehlin, 2004). LCGM was conducted using *Mplus* version 6.0 software (Muthén and Muthén, 1998-2010). As we were interested in testing hypothesized mediated paths from depression symptoms trend to smoking trend through smoking expectations trend, we also assessed specific indirect effects for significance using delta method and bootstrap standard errors. Specific indirect effects are significant if the product of the individual path coefficients is significant, and if the confidence interval for the indirect effect estimate does not include zero (Shrout and Bolger, 2002).

2.4.2 Missing data—There were 1,429 adolescents in the baseline sample. Due to wave non-response and loss to follow-up, the number of adolescents who completed a survey in the subsequent waves were 1,331 (wave 2), 1,122 (wave 3), 1,136 (wave 4), 1,110 (wave 5), and 1,092 (wave 6). To account for missing data, multivariate modeling used all available data. *Mplus* allows modeling with missing data using maximum likelihood estimation of the mean, variance, and covariance parameters, when requested, employing the Expectation Maximization (EM) algorithm, assuming data are missing at random (Muthén and Muthén, 1998-2004). We only accounted for missing data on the repeated measure of smoking, smoking reward expectations and depression symptoms. Cases with missing data on the covariates were not included in the analysis. An analysis of the difference between the participants with and without missing data and those retained and lost to follow-up revealed no significant differences in either the predictor variables or the repeated measures of the dependent variables smoking, smoking expectancies, and depression symptoms.

3. Results

3.1. Descriptive statistics

Table 1 presents the means and standard deviations for continuous model variables, along with the proportions for the categorical model variables. The proportion of participants with

high depressive symptoms (scores above 22) was 26% at baseline (wave 1, 9th grade fall) and 21% at wave 6 (11th grade spring). Cross-sectional averages for smoking indicated that the proportion of never smokers decreased from 71% at baseline to 67% at wave six, whereas the proportion of weekly and daily smokers increased from 7% to 9% over the same period. At wave two through six, 13%, 13%, 9%, 13%, 8%, of the sample progressed along the uptake continuum, respectively. Cross-sectional averages for smoking reward expectations ranged from 10.19 to 12.33. Figures 1 - 3 depict serial cross-sectional average changes among depression, smoking reward expectations and smoking progression.

3.2. Model fit

The SEM fit the data fairly well, χ^2 (253, 1393) = 454.97, $p < .0001$; CFI = .99; RMSEA = .02. The significant chi-square is the likely result of the large sample size as larger sample sizes are associated with an increased likelihood of detecting even minor model misspecifications (Saris et al., 2009). Figure 4 presents the model tested with standardized regression coefficients for significant paths only.

3.3. Direct effects on depression symptoms, smoking reward expectations and smoking

Table 2 presents the non-standardized path coefficients, standard errors, test statistics, and p -values for all paths tested in the LCGM. Below we report significant, direct (unmediated) paths to depression symptoms, smoking reward expectations and smoking.

3.3.1. Depression symptoms—Being female (OR = 1.22, 95% CI = 1.07, 1.39), of black race (OR = 1.35, 95% CI = 1.14, 1.59), lifetime alcohol use (OR = 1.07, 95% CI = 1.02, 1.12), peer smoking (OR = 1.06, 95% CI = 1.03, 1.10) and household smoking (OR = 1.25, 95% CI = 1.09, 1.43) increased the odds of having high depression symptoms at baseline, whereas a standard deviation (SD = 2.67) increase in parental monitoring was associated with a 12% decrease (OR = .88, 95% CI = .82, .93) in the odds of having high depression symptoms at baseline. A standard deviation (SD = 3.33) change in baseline smoking expectations was associated with a 4% decrease (OR = .96, 95% CI = .93, .99) in the odds of having higher depression symptoms across time. Baseline peers smoking was associated with an increased likelihood of higher depression symptoms across time, although the effect was very small (OR = 1.01, 95% CI = 1.001, 1.02).

3.3.2. Smoking reward expectations—Lifetime alcohol use ($\beta = .50$, $z = 8.42$, $p < .0001$), lifetime marijuana use ($\beta = .55$, $z = 8.72$, $p < .0001$) and peers smoking ($\beta = .42$, $z = 10.65$, $p < .0001$) each had a significant and positive effect on smoking expectations at baseline (level factor). Parental monitoring had a significant and negative effect ($\beta = -.14$, $z = -4.50$, $p < .0001$) on baseline smoking reward expectations. Depression symptoms trend had the only significant and positive effect on smoking reward expectations trend ($\beta = 3.50$, $z = 2.85$, $p = .004$). This indicated that progression from low to high depression (binary variable) over time was associated with a three and a half fold increase in the rate of change in smoking reward expectations over time.

3.3.3. Smoking—Lifetime alcohol use (OR = 1.24; 95% CI = 1.18, 1.30) and marijuana use (OR = 1.24; 95% CI = 1.18, 1.30), peer smoking (OR = 1.23; 95% CI = 1.19, 1.26) and household smoking (OR = 1.56; 95% CI = 1.34, 1.81) each increased the likelihood of smoking at a higher level at baseline (level factor). A standard deviation (SD = 2.67) increase in parental monitoring decreased the odds of smoking at a higher level at baseline by 7% (OR = .93, 95% CI = .87, .996). Smoking reward expectations trend had a significant and positive effect on smoking progression trend ($\beta = .206$, $z = 3.29$, $p = .001$). The smoking progression variable is ordered categorical and therefore the level (η_0) and trend (η_1) factors are modeled as log odds. When the β is exponentiated (e^β), log odds are converted to odds.

As such, rate of acceleration in smoking expectations was associated with a ($e^{.206} = 1.23$) 23% increase (OR=1.23; 95% CI=1.09, 1.38) in the odds of smoking progression.

3.4. Indirect or mediated effects: Do adolescents with higher depression symptoms develop greater smoking reward expectations, which in turn, influence smoking uptake?

As noted above, progression from low to high depression over time was associated with a three and a half fold increase in the rate of change in smoking reward expectations over time. Also, rate of acceleration in smoking reward expectations was associated with a 23% increase in the odds of smoking progression. We assessed whether this specific indirect effect of depression symptoms trend on smoking trend through smoking reward expectations trend was significant, using delta method standard errors and then bootstrap standard errors to confirm the results (Shrout and Bolger, 2002). The indirect effect was significant with delta method ($\beta_{\text{indirect}} = .72$, $z = 3.09$, $p = .002$; 95% CI= .26, 1.18) and bootstrap ($\beta_{\text{indirect}} = .72$, $z = 2.10$, $p = .03$; 95% CI= .05, 1.39) standard errors. This indicated that higher depression symptoms across time predicted a 17% (smoking reward expectancies scores could range 21 points from 7-28 points, with a 3.50 unit increase in score for an increase from low to high depression $3.50/21 = .167$ or 17%) increase in smoking reward expectations, which in turn predicted a 23% increase in the odds of smoking progression. Thus, smoking reward expectations is one mechanism by which adolescent depression influences smoking uptake.

3.5. Alternative model

In order to account for the possibility that the directional effect was reciprocal (i.e., a significant path from smoking progression to depression symptoms through smoking reward expectations), we ran an alternative model with the between trend paths reversed. The indirect effect with delta method standard errors was significant ($\beta_{\text{indirect}} = .29$, $z = 2.46$, $p = .01$; 95% CI= .06, .52), such that smoking trend had a significant effect on smoking expectations trend ($\beta = 2.09$, $z = 7.30$, $p < .0001$). The rate of smoking progression was associated with a doubling in the rate of change in smoking expectations over time, which translated to a 10% increase in smoking reward expectations for each unit change in time. Further, increases in smoking expectations was associated with a .14 increase ($\beta = .14$, $z = 2.45$, $p = .01$) in the log odds (logit) rate of change in depression over time, which translated to an $e^{.14} = 1.15$ or 15% increase (OR=1.15, 95%CI=1.03, 1.29) in the odds of having high depression with each unit change in time. However, the indirect effect with bootstrap standard errors was not significant ($\beta_{\text{indirect}} = .29$, $z = .63$, $p = .53$; 95% CI= .61, 1.20), suggesting that the validity of the indirect effect was dependent on the use of less stringent criteria.

4. Discussion

The study provides novel evidence that expectations of smoking reward facilitate smoking uptake among depressed adolescents. Higher depression symptoms across mid to late adolescence predicted a 17% increase in smoking reward expectations, which in turn predicted a 23% increase in the odds of smoking progression. These findings offer an initial glimpse at reward-related processes that may occur early in the smoking acquisition process for youth with elevated depression symptoms. Expectations that smoking offers several benefits highlight depressed adolescents who are vulnerable to smoking. These expectations of reward are an important mechanism to target in smoking prevention programs for depressed youth.

The influence of smoking reward expectations on adolescent smoking escalation has been largely unexplored. Research has shown that expectations that smoking would remove displeasure, such as reducing negative affect, predicts smoking escalation in adolescents

(Heinz et al., 2010). The present study adds to the sparse literature by showing that expectations that smoking will provide pleasure, motivates smoking among depressed youth. Expectations that smoking confers rewards (e.g., enjoyable, something to do when bored, stay thin) increases the likelihood that depressed adolescents will choose to smoke. As nicotine can ameliorate the affective and reward processing deficits associated with depression (Barr et al., 2008; Gilbert et al., 2008; Kenny and Markou, 2006; Spring et al., 2008; Warburton and Mancuso, 1998), and impact neurocognitive processes that are typically diminished in depression (Evans and Drobos, 2009; Gilbert et al., 2008; Heishman et al., 2010), these adolescents may indeed learn that smoking is rewarding. Thus, their smoking experience may then serve to validate their expectations.

Converging research supports the notion that depressed adolescents may find smoking more rewarding than nondepressed adolescents. Depression-prone smokers have greater smoking-induced dopamine release than smokers not prone to depression (Brody et al., 2009). It is possible that nicotine's primary reinforcing effects are amplified for youth either prone to depression or during periods of elevated depression symptoms. In addition, individuals prone to depression tend to have fewer reinforcers (Jacobson et al., 1996; Lewinsohn and Amenson, 1978; MacPhillamy and Lewinsohn, 1974) and derive less reward from natural reinforcers in their environment (Forbes et al., 2009; Shankman et al., 2007; Wichers et al., 2009). Recent research has shown that declines in the frequency and enjoyability of typical reinforcers leads to smoking escalation among young adults with elevated depression symptoms (Audrain-McGovern et al., 2011). Pre-clinical models suggest that nicotine potentiates reward from available reinforcers by increasing the sensitivity of brain reward systems or the ability to derive pleasure from available reinforcers (Kenny and Markou, 2006). Thus, nicotine has relevant secondary reinforcing effects by increasing the pleasure derived from reinforcers in the context of limited reinforcers (Spring et al., 2008). Further investigation of reward related mechanisms may identify unique biological and behavioral functions of smoking and nicotine in adolescents with elevated depression symptoms.

From a clinical perspective, these findings provide further support for mid to late adolescence as an etiologically important developmental period for the comorbidity between adolescent smoking and depression. On average, about 25% of adolescents had clinically significant levels of depression symptoms across three years. Assessing smoking reward expectations in this subgroup may identify those adolescents at risk for smoking. As almost 30% - 60% of adults entering smoking cessation programs have a history of major depression (Cinciripini et al., 2005; Ginsberg et al., 1995; Glassman et al., 1988; Haas et al., 2004) and 50% have elevated depression symptoms (Cinciripini et al., 2005; Lerman et al., 1996; Niaura et al., 2001), addressing this issue during adolescence could have a significant impact on the excess smoking burden incurred by individuals prone to depression. Addressing alternative ways to meet the rewards (e.g., source of pleasure, something to do) expected of smoking may prove to be an important part of adolescent smoking prevention and cessation interventions for depressed youth. Smoking reward expectations may also highlight areas where a depressed adolescent has weak skills such that smoking is perceived as a helpful option, but in reality is an ineffective solution (e.g., a way to handle problems). Although relatively less attention has been paid to these features in adolescent smoking prevention programs, these components may be critical to preventing smoking uptake among youth who have elevated depression symptoms.

The present study also considered the possibility that smoking influenced the development of depression symptoms through smoking reward expectations (e.g., reciprocal directional effect). The rate of smoking progression was associated with a doubling in the rate of change in smoking expectations over time, which translated to a 10% increase in smoking reward expectations for every six months that passed. Further, increases in smoking

expectations were associated with a 15% increase in the odds of having high depression across time. Thus, smoking uptake shaped and indeed increased smoking expectations across time. Of note, as smoking expectations increased so did the likelihood of depression. Although the indirect effect failed more stringent tests of significance, the findings raise questions about whether unrealistic or unmet smoking reward expectations (e.g., way to handle problems, a method to stay thin) could contribute to the development of depression in adolescent smokers. This would certainly be consistent with social and cognitive vulnerability models of the depression (Prinstein and Aikins, 2004; Prinstein et al., 2005). Addressing alternative and effective ways to meet the rewards expected of smoking and educating youth on unrealistic expectations for smoking may impact adolescent smoking irrespective of the directional pathway.

While this is the first study to examine the link between depression symptoms, smoking reward expectations and adolescent smoking progression, the study has strengths along with potential limitations. Study strengths include an excellent participation rate, a large sample, repeated measures of key variables, smaller measurement intervals than previous adolescent smoking studies, and control for many confounding influences in the statistical models. Although the clinical significance of depression symptoms or “subthreshold depression” among adolescents has been well established (Lewinsohn et al., 2000), we are not able to measure the extent of depression as diagnostic assessments were not completed. Higher levels of depression as measured by the CES-D may have been reflective of clinical episodes rather than subclinical depression symptoms.

It is also possible that expectations that smoking will reduce displeasure, such as negative mood influences smoking among depressed youth. Unfortunately, we did not measure these expectations in the present study. A broader understanding of the expectations that depressed adolescents have for smoking may help highlight other smoking prevention intervention targets. Finally, we controlled for many confounding influences at baseline, but time-varying influences and the role of psychological comorbidity were not considered in the already complex model. Peer smoking effects, externalizing and anxiety symptoms stand out as important variables to consider across time.

Although not necessarily a limitation, it is important to point out that not all depressed adolescents initiated or progressed in smoking. It is possible that those adolescents who have more consistently elevated symptoms are those most at risk for smoking progression. Identifying which depressed adolescents find smoking rewarding may pin-point those adolescents most vulnerable to progressing to regular smoking and illuminate novel nicotine/smoking actions in this vulnerable population. Our model accounted for 46% of the variance in smoking progression emphasizing that other mechanism warrant investigation.

In summary, this study highlights a novel connection between high depression symptoms, expectations of smoking reward and adolescent smoking progression. As such, this study represents a shift away from research focusing primarily on the role of negative affect in adolescent smoking to reward-related processes. Further research is warranted to increase our understanding of how smoking and nicotine affect adolescents prone to depression, ultimately informing efficacious smoking and possibly depression treatments for this population. Earlier interventions may mitigate the comorbidity that appears to track well into adulthood thereby decreasing the disproportionate smoking attributable morbidity and mortality in a population prone to depression (Prochaska, 2010).

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Figure 1.
Smoking progression among adolescents with high and low depression symptoms.

Figure 2.
Increase in smoking reward expectations among adolescents with high and low depression symptoms.

Figure 3.
Increase in smoking reward expectations among adolescents who did and did not progress in smoking.

Figure 4.

The indirect effect of depression on smoking progression through smoking reward expectations.

Note: Only significant paths are shown in the model.

Table 1

Characteristics of the Sample

Variable	Level	N	(%)
Gender	Female	697	50
	Male	696	50
Race	Black	209	15
	White	1033	74
	Other race	153	11
Household smoking	At least one member smokes	613	44
	No household members smoke	780	56
Lifetime alcohol use	0 days	724	52
	1 or 2 days	223	16
	3 to 9 days	167	12
	10 or more days	279	20
Lifetime marijuana use	0 times	1142	82
	1 or 2 times	84	6
	3 to 9 times	56	4
	10 or more times	111	8
Smoking at baseline	Never smoked	989	71
	Puffed to one cigarette total	97	7
	Did not smoke in the past month	153	11
	Puff to 1 cigarette in past month	56	4
	One cigarette a week	28	2
	Smokes daily	70	5
Smoking progression	Wave 2	171	13
	Wave 3	145	13
	Wave 4	101	9
	Wave 5	138	13
	Wave 6	87	8
Depression symptoms (> 22 = high)	Wave 1	362	26
	Wave 2	342	26
	Wave 3	293	26
	Wave 4	261	23
	Wave 5	243	23
	Wave 6	229	21
		Mean	SD
Peers smoking		1.55	2.34
Parental monitoring		12.33	2.67
Smoking reward expectations	Wave 1	10.19	4.00
	Wave 2	10.62	4.35
	Wave 3	10.55	4.32
	Wave 4	10.71	4.54

Variable	Level	N	(%)
	Wave 5	11.02	4.54
	Wave 6	10.84	4.46

Table 2

Unstandardized regression coefficients, standard errors, z-statistics, and p-values for all model regressions.

	Smoking level				Smoking trend			
	Coefficient	SE	z	p-value	Coefficient	SE	z	p-value
Depression level					-0.02	0.02	-0.74	0.46
Depression trend					0.14	0.46	0.31	0.75
Expectations level					.01	.01	.84	.40
Expectations trend					0.21	0.06	3.30	0.001
Female	0.03	0.07	0.42	0.67	0.04	0.02	1.47	0.14
Black	0.11	0.10	1.11	0.27	-0.03	0.04	-0.88	0.38
Other race	-0.10	0.13	-0.75	0.45	-0.04	0.05	-0.92	0.36
Parental monitoring	-0.03	0.01	-2.18	0.03	-0.01	0.005	-1.92	0.05
Lifetime alcohol use	0.21	0.02	9.05	0.00	0.01	0.01	.84	0.40
Lifetime marijuana use	0.21	0.02	8.75	0.00	-0.01	0.01	-0.58	0.56
Household smoking	0.44	0.08	5.80	0.00	-0.01	0.03	-0.21	0.83
Peers smoking	0.20	0.01	13.84	0.00	0.002	0.01	0.25	0.80

	Expectations level				Expectations trend			
	Beta	SE	z	p-value	Beta	SE	z	p-value
Depression level					0.07	0.06	1.13	0.26
Depression trend					3.50	1.22	2.85	0.004
Smoking level					0.01	0.10	0.06	0.95
Female	-0.08	0.08	-0.46	0.002	-0.07	0.08	-0.84	0.40
Black	-0.15	0.24	-0.62	0.001	-0.03	0.12	-0.24	0.81
Other race	0.26	0.25	1.06	0.18	-0.10	0.15	-0.66	0.51
Parental monitoring	-0.14	0.03	-4.50	0.00	0.00	0.02	-0.002	1.00
Lifetime alcohol use	0.50	0.06	8.42	0.005	0.04	0.03	1.24	0.21
Lifetime marijuana use	0.55	0.06	8.72	0.39	-0.05	0.05	-1.12	0.26
Household smoking	0.10	0.19	0.55	0.58	0.15	0.09	1.73	0.08
Peers smoking	0.42	0.04	10.65	0.00	-0.04	0.02	-1.61	0.11

	Depressive symptoms level				Depressive symptoms trend			
	Beta	SE	z	p-value	Beta	SE	z	p-value
Smoking level					-0.02	0.02	-1.27	0.20
Expectations level					-0.01	.005	-2.88	0.004
Female	0.20	0.07	3.03	0.002	-0.004	0.02	-0.19	0.85
Black	0.30	0.09	3.43	0.001	-0.04	0.03	-1.51	0.13
Other race	0.14	0.10	1.35	0.18	-0.05	0.03	-1.54	0.12
Parental monitoring	-0.05	0.01	-4.05	0.00	-0.002	0.004	-0.55	0.58
Lifetime alcohol use	0.07	0.02	2.81	0.005	-0.02	0.01	-0.26	0.80
Lifetime marijuana use	-0.02	0.03	-0.86	0.39	0.01	0.01	1.34	0.18
Household smoking	0.22	0.07	3.19	0.001	0.01	0.02	0.55	0.58
Peers smoking	0.06	0.01	4.12	0.00	0.01	0.01	1.99	0.05