Published in final edited form as:
Drug Alcohol Depend. 2011 July 1; 116(1-3): 37–44. doi:10.1016/j.drugalcdep.2010.11.017.

Smoking Outcome Expectancies in Young Adult Female Smokers: Individual Differences and Associations with Nicotine Dependence in a Genetically-informative Sample

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Abstract

Outcome expectancy is a central construct in models of addiction. Several outcome expectancies associated with smoking cigarettes have been identified, and studies suggest that individual differences in smoking expectancies are related to important aspects of tobacco use, including levels of smoking, nicotine dependence and smoking cessation. In the present study, we used a novel analytic method, exploratory structural equation modeling (ESEM), to quantify smoking expectancies from a subset of items adapted from the Smoking Consequences Questionnaire (SCQ; Brandon and Baker, 1991) and SCQ-Adult (Copeland et al., 1995). In our sample of 1262 monozygotic and dizygotic young adult, female twins who were regular smokers, we quantified six smoking expectancy factors similar to those reported in previous studies. These included Negative Affect Reduction, Boredom Reduction, Weight Control, Taste Manipulation, Craving/Addiction and Stimulation-State Enhancement. We used genetic model-fitting to examine the extent to which individual differences in the expectancies were influenced by latent genetic, shared environmental and non-shared environmental factors. We also examined the validity of the expectancy factors by examining their associations with nicotine dependence (ND) before and after adjusting for comorbid diagnoses of drug dependence and alcohol use disorder. Results of the validity analysis indicated that all of the expectancies were associated with ND after covariate adjustment. Although we lacked the statistical power to distinguish between genetic and shared environmental sources of variance, our results suggest that smoking outcome expectancies aggregate in families, but the majority of variance in these expectancies is due to environmental factors specific to the individual.

Keywords

Smoking expectancies; Smoking Consequences Questionnaire; Nicotine Dependence; Genetics; Female

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1. Introduction

Expectancy refers to the storage and recall of information about past events in order to anticipate future events (Goldman, 2002). Outcome expectancies are learned associations between specific behaviors and outcomes of engaging in that behavior (Jones et al., 2001). Functionally, outcome expectancies facilitate ongoing behavioral adjustment to ensure survival in a dynamic environment; however, there is evidence that expectancies about substance use outcomes can be maladaptive (Goldman, 2002; Goldman et al., 2006).

The outcome expectancy construct is central to cognitive and social learning models of addiction and relapse (Brandon et al., 2004; Donovan, 1988; Goldman, 2002; Jones et al., 2001; Marlatt, 1985; Marlatt and Gordon, 1985; Niaura et al., 1991; Stacy et al., 1990; Witkiewitz and Marlatt, 2004). These models theorize that an individual's decision about whether or not to use a substance is based, in part, on the anticipated positive and negative consequences associated with its use; positive outcome expectancies are thought to promote substance use and relapse, whereas negative outcome expectancies are thought to have the opposite effect.

Several types of smoking outcome expectancies have been identified. Most often, smoking expectancies have been assessed using the original (Brandon and Baker, 1991), abbreviated or revised versions (Cepeda-Benito and Reig Ferrer, 2000; Copeland et al., 1995; Myers et al., 2003; Rash and Copeland, 2008) of the Smoking Consequences Questionnaire (SCQ). In the original study, Brandon and Baker (1991) identified four types of smoking expectancies, Negative Consequences, Positive Reinforcement/Sensory Satisfaction, Negative Reinforcement/Negative Affect Reduction and Appetite-Weight control, which differentiated among never-smokers, daily smokers, and occasional smokers in a college student sample. Subsequent studies using the original or revised versions adapted for use with specific populations have found evidence for more refined sets of seven to ten expectancies, including Negative Affect Reduction, Boredom Reduction, Weight control, Stimulation-state Enhancement, Taste/Sensorimotor Manipulation, Health Risks/ Consequences, Craving/Addiction, Negative physical feelings, Social facilitation and Negative Social Impression (Buckley et al., 2005; Cepeda-Benito and Reig Ferrer, 2000; Copeland et al., 1995; Lewis-Esquerre et al., 2005; Rash and Copeland, 2008; Reig-Ferrer and Cepeda-Benito, 2007; Schleicher et al., 2008; Vidrine et al., 2009).

Studies show that individual differences in these smoking outcome expectancies relate to nicotine dependence (ND) and aspects of tobacco use in accord with predictions made by cognitive and social learning models. With few exceptions (Wetter et al., 1994), studies indicate that higher levels of current smoking and/or ND are associated with higher scores on most positive and negative reinforcement expectancies (Copeland et al., 1995; Jeffries et al., 2004; Rash and Copeland, 2008; Reig-Ferrer and Cepeda-Benito, 2007; Schleicher et al., 2008; Vidrine et al., 2009) and often higher Weight-Control expectancies (Copeland et al., 1995; Rash and Copeland, 2008; Reig-Ferrer and Cepeda-Benito, 2007; Schleicher et al., 2008; Vidrine et al., 2009). Studies also show that expectancies, measured at pretreatment, predict withdrawal symptom severity during cessation trials (Vidrine et al., 2009; Wetter et al., 1994) and predict outcomes such as smoking relapse and the number of cigarettes smoked during and after treatment (Copeland et al., 1995; Vidrine et al., 2009; Wetter et al., 1994). Further, expectancies have been shown to change over the course of cessation treatments. For example, Copeland et al. (1995) reported that 7 of 10 expectancies significantly decreased from pre- to post-treatment in smokers who received treatment compared to smokers in the control group, and abstainers tended to report the largest

decreases. Taken together, research suggests that smoking expectancies play significant roles in nicotine addiction, cessation treatments and treatment outcomes.

Given their roles in these key aspects of tobacco use, it is important to advance understanding of the factors that contribute to individual differences in expectancies. Specifically, it would be useful to examine the extent to which smoking expectancies are influenced by genetic and environmental factors. Goldman and colleagues (Goldman et al., 2006) suggest that expectancies are under significant evolutionary pressure, such that individual differences in expectancies are likely to have a genetic basis. In contrast, social learning theory (Bandura, 1977), emphasizes the role of the environment; expectancies develop through learning processes by engaging in the environment both directly (participating) and indirectly (observing others). Although these models suggest individual differences in expectancies are likely due to different sources, to our knowledge no prior studies have examined the extent to which smoking expectancies are influenced by genetic and/or environmental factors. This was one focus of the present study.

In the present study, the proportions of genetic, shared environmental and non-shared (i.e., individual-specific) environmental factors contributing to smoking expectancies were assessed in a sample of young adult female twins who were regular smokers. A novel analytic approach, exploratory structural equation modeling (ESEM; Asparouhov and Muthén, 2009) was used to model the smoking expectancies as latent variables using a subset of items adapted from the SCQ. We hypothesized that the smoking expectancies would be at least modestly genetically-influenced but also would be significantly influenced by environmental factors shared in common among members of the twin pairs.

Furthermore, given that the latent smoking expectancies were based only on a subset items adapted from the SCQ and were modeled using ESEM (most prior studies have used principal components analysis or confirmatory factor analysis), the second focus of the present study was to examine the validity of the smoking expectancies. To do so, we examined the relationships between the expectancies and ND after adjusting for demographic variables and comorbid substance use diagnoses. In line with prior research, we hypothesized that smoking expectancy scores would be significantly associated with diagnoses of ND. We further hypothesized that significant associations would remain after covariate adjustment.

2. Methods

2.1 Sample and Measures

Participants were from the Missouri Adolescent Female Twin Study (MOAFTS). The “MOAFTS” study (PI Andrew Heath) is a longitudinal study consisting of a cohort of female twin pairs born between 1 July 1975 and 30 June 1985. At baseline, twins, who were identified from birth records, were eligible to participate if both members of the twin pair had survived past infancy, were not adopted at birth and if their biological mother was a resident of the state at the time of their birth. Using a cohort sequential sampling design for initial recruitment, interviews were attempted with at least one biological parent (wherever possible, the biological mother) and both twins during 1994–1999, when the twins were 13, 15, 17 or 19 years old. Recruitment of the 13-year-olds continued over a two-year period as twins became age-eligible. After obtaining permission from parents, a telephone diagnostic interview was administered to the twins and their parents. Of the 2369 twin pairs identified as live-born, 95.6% were located. The final sample of twins interviewed at baseline for each cohort included 1633 pairs (72.5% of pairs targeted), including 579, 291, 367 and 373 pairs aged 13, 15, 17 and 19 years, respectively (n = 3446). Details of the study design,

recruitment and baseline assessments (which are not included in the present study) are given elsewhere (Heath et al., 2002; Heath et al., 1999; Knopik et al., 2005).

Subjects in the present study were those who participated in the MOAFTS wave four data collection conducted during 2002–2005. This sample includes 3060 women who had been interviewed at baseline (89% response rate), along with 728 women from the baseline sampling frame, who had not participated previously. Wave four data included a telephone diagnostic interview and a mailed questionnaire. The diagnostic interview was adapted from the Semi-Structured Interview for the Study of the Genetics of Alcoholism (SSAGA; Bucholz et al., 1994) that assessed lifetime DSM-IV psychopathology, and it also included a DSM-based nicotine dependency assessment adapted from the Composite International Diagnostic Interview (CIDI; Cottler et al., 1991). The mailed questionnaire included several assessments (not included in the present study) and a subset of 18 items adapted from the SCQ (Brandon and Baker, 1991) and the SCQ-A (Copeland et al., 1995).

Only a subset of 18 adapted items was included in the questionnaire, as opposed to a full-length version of the SCQ, to reduce participant burden. During the MOAFTs questionnaire development, three items were selected to represent each of six expectancies: Negative Affect Reduction, Boredom Reduction, Weight Control, Taste-Sensorimotor Manipulation, Craving / Addiction and Stimulation-state Enhancement (Copeland et al., 1995). Item selection was based primarily on two considerations: i) high factor loadings onto each expectancy as reported by Copeland et al. (1995); and ii) the extent to which the three items sampled each expectancy domain. The wording of the items was also changed slightly to be specific to cigarette smoking as opposed to some other form of substance use. For example, the item “smoking calms me down when I feel nervous” (Copeland et al., 1995) was changed to “smoking a *cigarette* calms me down when I feel nervous.” Lastly, one item from the SCQ-A was changed, inadvertently, by combining the stem of a Negative Affect Reduction item, “when I'm feeling down,” with a Stimulation-state Enhancement item, “a cigarette can really make me feel good.” The implication of this combined item is that it sampled components of both constructs. The 18 items are shown in the results section in Table 1.

The sample in the present study included 1262 women ages 18–29 (*Mn*=22.2), who participated in the telephone interviews, provided questionnaire data and were regular smokers. Following previous work (Madden et al., 1997), we defined regular smoking as having smoked 100 or more cigarettes lifetime or as having smoked 21–99 cigarettes but having smoked at least weekly for a period of two months or longer prior to the interview. DSM-IV lifetime diagnoses for Nicotine Dependence (ND), Alcohol Use Disorder (AUD) and Drug Dependence (DD) were taken from the wave four diagnostic interview data. The AUD diagnoses included alcohol abuse and alcohol dependence, and the drug dependence diagnoses included dependence on illicit substances (marijuana, cocaine, stimulants, opiates, sedatives, hallucinogens, PCP, solvents or inhalants). The majority of participants (93.2%) were of European American descent. The sample included 387 complete twin pairs and 488 singletons. 227 of the pairs were monozygotic (MZ) twins and 160 were dizygotic (DZ) twins. Of the singletons, 213 individuals were MZ and 275 were DZ.

2.2. Statistical Analyses

2.2.1. Exploratory Structural Equation Models (ESEMs)—We examined the factor structure of the smoking expectancy items and tested the associations of ND, DD and AUD with each smoking expectancy factor using ESEM (Asparouhov and Muthén, 2009). Briefly, ESEM uses exploratory factor analysis (EFA) to compute the measurement part of the factor model (e.g., rotated factor loadings, factor variances and covariances, residual variances), but expands traditional EFA by simultaneously incorporating tests of structural associations

(e.g, regressions of latent factors onto dependent variables). In contrast to CFA, ESEM does not impose the strict requirement that only certain items load onto certain factors; in ESEM, small but statistically significant cross-factor loadings do not have to be set to zero. Preliminary CFAs, which were fit to our 18 expectancy items using an a priori six-factor structure following the conceptual model outlined by (Copeland et al., 1995), were poor-fitting (e.g. six-factor CFA: CFI=.883, TLI=.959 and RMSEA=.174). Note that CFAs were poor fitting even when the item "when I'm feeling down, a cigarette can really make me feel good" was assigned to the Stimulation-state Enhancement factor, to the Negative Affect Reduction factor and also when it was excluded from the analysis. This contrasts with prior studies that used CFAs and reported good-fitting models using the original, adult, short, adolescent or spanish-speaking verions of the SCQ (Buckley et al., 2005; Cepeda-Benito and Reig Ferrer, 2000; Lewis-Esquerre et al., 2005; Myers et al., 2003; Reig-Ferrer and Cepeda-Benito, 2007; Vidrine et al., 2009). This issue is discussed in a subseqent section. Examination of the CFA modification indices and follow-up EFAs suggested that numerous small, but statistically significant cross-factor loadings caused the CFAs to fit poorly. Given evidence that such misspecification of the measurement part of a structural equation model can result in over-estimated factor correlations and biased structural associations (Asparouhov and Muthén, 2009), we chose the ESEM approach.

We fit four-, five-, six- and seven-factor ESEMs to the smoking expectancy items. The response options for the items were on a Likert-type scale (1 = Strongly Disagree, 2 = Disagree, 3 = Agree, 4 = Strongly Agree), and as appropriate for ordered categorical data (Lubke and Muthén, 2004; Muthén, 1984), the ESEMs (as were the preliminary CFAs and EFAs) were computed using mean and variance adjusted weighted least squares estimation (WLSMV). The models were computed in Mplus 5.2 (Muthén and Muthén, 1998–2007), accounted for missing observations, and the standard errors were adjusted for nonindependence of observations due to familial clustering. Each model used geomin rotation of the factor loadings. In each of the ESEMs, diagnostic variables (where diagnoses of ND, DD and AUD were coded 1 or coded 0 otherwise), were entered together as independent variables into separate regression equations where each smoking expectancy factor was the dependent variable.

Although theory considers expectancies to be causal (predictive of smoking behavior), the cross-sectional nature of our data do not allow for strong causal conclusions. Therefore, in the present study the diagnostic variables are treated as independent variables which, by entering all diagnostic variables into the regression equation simultaneously, allowed us to examine the unique associations of the expectancies with each diagnoses (i.e., association of each diagnosis after adjusting for diagnostic comorbidity). Age (grand-mean centered) and zygosity (MZ = 0, DZ = 1) were also entered as independent variables into the regression equations. The associations of ND, DD and AUD separately with each expectancy factor were also examined. The optimal number of factors was determined by using the Comparative Fit Index (CFI), Tucker-Lewis Index (TLI), and Root Mean Square Error of Approximation (RMSEA).

2.2.2. Genetic analyses—We used ESEM in Mplus to compute and output a score for each smoking expectancy factor from the best-fitting model for each participant. Here, we adjusted for age effects by regressing each expectancy factor onto the grand mean-centered age variable, but we did not include the diagnostic variables. Using the raw factor scores from MZ and DZ twin pairs reared together, we used Mx (Neale, 2004) to estimate univariate twin models to partition the variance in the expectancy scores into three sources: additive genetic (A), which is variance in expectancies due to latent genetic influences; shared environmental (C), which is variance due to facets of the environment shared by members of a twin pair (thus making them more alike); and non-shared environmental

which is variance due to environmental factors that are unique to each member of a twin pair (thus contributing to their dissimilarity). Models were fit to raw factor scores for MZ and DZ pairs and singleton twins, factor score means were equated across zygosity, and 95% confidence intervals were estimated. The significance of A or C parameters were tested by dropping these effects from the model separately or both simultaneously and testing the decrease in model fit using χ^2 difference tests.

3. Results

3.1. Prevalences

Among the regular smokers, the prevalences for lifetime DSM-IV diagnoses were 48.3% for nicotine dependence, 8.0% for drug dependence and 21.7% for alcohol use disorder. The scale score and standard deviation for each smoking expectancy are shown in Table 1. The scale scores for the expectancies each are the mean of the three items based on the model of Copeland et al. (1995).

3.2. ESEMs

3.2.1 Best fitting model—Consistent with recommendations for fit indices (Hu and Bentler, 1999), the ESEM with seven factors was better-fitting (CFI = .997, TLI=.998, RMSEA=.027) compared to the ESEMs with four (CFI = .880, TLI=.944, RMSEA=.152), five (CFI = .939, TLI=.970, RMSEA=.112) or six factors (CFI = .994, TLI=.997, RMSEA=.035). Although the seven-factor model was the best-fitting, the standardized loadings of all but one item onto seventh factor were low (−.151 to .210). This factor accounted for substantial variance primarily in only one item (a cigarette can satisfy my urge to smoke). In contrast, as evidenced by the pattern of standardized factor loadings in Table 1, the six-factor model recovered expectancy factors similar to those reported by Copeland et al. (1995). Given that the fit indices were well within range of recommendations made by Hu and Bentler (1999) and the factors represented the smoking expectancies selected during questionnaire development, we chose the more parsimonious six-factor model.

The correlations among the factors (adjusted for demographic and all diagnostic variables) are shown in Table 2. The correlations ranged from about 0.22 to 0.69 and all were significantly different from zero ($p < .001$).

3.2.2. Associations of ND, DD and AUD with the smoking expectancies—The standardized coefficients from the ESEM regression analyses are shown in Tables 3 and 4. Table 3 shows the coefficients from regressing each expectancy factor onto each diagnostic variable separately, and Table 4 shows the coefficients from regressing each expectancy factor onto the three diagnostic variables simultaneously. The standardized regression coefficients in each table are adjusted for age and zygosity.

The regression coefficients in Table 3, which are the univariate associations of ND, DD and AUD with each expectancy factor, indicate that ND was significantly associated with higher scores on all six expectancies, DD was associated with Negative Affect Reduction, Boredom Reduction, Weight Control, and Stimulation-State Enhancement, and AUD was significantly associated with Negative Affect Reduction and Boredom Reduction.

The regression coefficients in Table 4 indicate that after adjustment for the other diagnostic variables, only ND was significantly associated with higher scores on all six factors. DD was associated with higher scores on Negative Affect Reduction, Boredom Reduction and Weight control, but was not significantly associated with Taste Manipulation, Craving/

Addiction or Stimulation-State Enhancement. After adjusting for the other diagnostic variables, AUD was not significantly associated with any of the smoking expectancy factors.

3.3. Genetic analyses

The relative contribution of genetic and environmental influences on phenotypic variance of the smoking expectancies factors is shown in Table 5. The univariate models suggest that non-shared environmental factors were the largest contributors to phenotypic variance ranging from 62% for Boredom Reduction to 74% for Taste Manipulation. Modest heritability was found for Boredom Reduction (38%), where dropping the genetic component (A) significantly reduced model fit, $\chi^2(1)= 5.12$, $p < .05$, but dropping the shared environmental component (C) did not $\chi^2(1)= 1.0$, $p > .05$.

For all of the other expectancies, no significant decreases in the fit of the models were found when either the genetic or the shared environmental components were dropped (all $p > .05$). However, dropping both components simultaneously resulted in significant decreases in the fit of models for all of the expectancies (all $p < .001$). Together, the results indicate that familial influences (with total variance contribution, 1-E) accounted for a significant proportion of the phenotypic variance in the smoking expectancies; however, the separate contribution of additive genetic or shared environmental influences could not be distinguished for any of the expectancies except Boredom Reduction. This was most likely due to a combination of modest magnitudes of the genetic and shared environmental influences (i.e., small effect sizes) and relatively small sample size (i.e., low power) to detect these small effects.

4. Discussion

We used a subset of items adapted from the SCQ and a novel analytic approach, exploratory structural equation modeling (ESEM), to quantify six smoking expectancy factors similar those reported in previous studies that have used different versions of the SCQ (Buckley et al., 2005; Cepeda-Benito and Reig Ferrer, 2000; Copeland et al., 1995; Jeffries et al., 2004; Rash and Copeland, 2008; Reig-Ferrer and Cepeda-Benito, 2007; Schleicher et al., 2008; Vidrine et al., 2009). In our sample of young adult women who were lifetime regular smokers, we examined both the validity of the six expectancy factors as well as the extent to which individual differences in the expectancies were influenced by genetic and environmental factors.

As hypothesized, after adjusting for age, DD and AUD, each of the expectancies was significantly associated with ND. These results are in line with prior studies that have reported significant relationships between ND and these expectancies when assessed using the full-length (Copeland et al., 1995), Spanish (Cepeda-Benito and Reig Ferrer, 2000; Vidrine et al., 2009) and abbreviated (Myers et al., 2003; Schleicher et al., 2008) versions of the SCQ. Given that our ESEM was good-fitting and that each expectancy was significantly associated with ND even after covariate adjustment, the expectancies in our study appear to have good construct validity.

Other results are notable. The associations of ND with Negative Affect Reduction and Boredom Reduction were of somewhat greater magnitude than with Weight Control, Taste Manipulation, Craving/Addiction and Stimulation-State Enhancement. Negative Affect Reduction, Boredom Reduction and Weight Control were also significantly associated with DD. None of the expectancies were significantly associated with AUD after adjusting for diagnostic comorbity. This pattern of associations suggests that behaviors associated with negative affect regulation, boredom reduction and weight control (e.g., smoking when upset, smoking to relieve boredom, smoking to regulate weight) might be markers for higher risk

for ND and DD in young adult women. Importantly, however, our results indicate this effect does not generalize to AUD, suggesting that these expectancies have discriminant validity with respect to problematic alcohol use.

A novel finding of our research is that the majority of the phenotypic variance (about 70%) in the smoking expectancy factors in this sample of 18–29 year-old women was attributable to individual-specific or non-shared environmental influences with the remaining variance (about 30%) attributable to the combined effect of genetic and shared environmental influences. The strongest evidence for heritability was for Boredom Reduction (38%).

Overall, our results provide support to the hypothesis that smoking expectancies in young adult women run in families. However, we have insufficient power to determine the degree to which familial influences are due to genetic or to shared environmental influences, with the possible exception of Boredom Reduction. Nevertheless these results must be interpreted with certain caveats in mind. We restricted our analyses to those who are regular smokers, which limits the genetic variability of our sample by controlling for the genetic (and environmental) factors associated with becoming a regular smoker. For example, (Maes et al., 2004) reported that after controlling for genetic factors in common among tobacco initiation and regular tobacco use, the genetic factors specific to ND accounted for about 24% of the overall genetic influences on ND. In other words, because our sample included only regular smokers, the putative full continuum of genetic risk for expectancies was not represented, and heritability estimates must be interpreted with this in mind. Consequently, no strong conclusions can yet be made concerning the relative contributions of genetic and/or shared environmental to individual differences in these expectancies.

The results should also be interpreted with caution because the expectancies were assessed using only a subset of 18 items adapted from the SCQ. Full-length versions of the SCQ use more items to assess each expectancy and thus more thoroughly sample each expectancy domain; consequently, our expectancies might suffer somewhat from limited construct coverage (Messick, 1995). This is especially the case for Craving / Addiction and Stimulation-state enhancement where only two of the three items selected to represent these expectancies loaded highly onto these factors. Specifically, the items “I will become more addicted to nicotine if I continue smoking,” and “I will become more addicted the more cigarettes I smoke” loaded highly onto Craving / Addiction (.941 and .875, respectively), but the item, “a cigarette can satisfy my urge to smoke” only loaded at .253. Further, only two items, “smoking a cigarette energizes me” and “a cigarette can give me energy when I'm bored and tired,” loaded highly onto Stimulation-state Enhancement (.714 and 1.004, respectively). In practical terms, this means the results from our genetic analyses mostly likely pertain to only sub-components of these expectancy constructs as reported in other studies that used more items (e.g., Buckley et al., 2005; Cepeda-Benito and Reig-Ferrer, 2000; Copeland et al., 1995; Reig-Ferrer and Cepeda-Benito, 2007; Vidrine et al., 2009). Because we used only a subset of items, the range of phenotypic variance in each expectancy in our study is most likely restricted.

We also did not examine Gene X Environment interactions- the extent to which exposure to an environment (not shared among family members) alters genetic influences on the expectancies. It could be, for example, that exposure to a high-risk environment (i.e., a network of heavy-smoking peers) might substantially alter the heritabilities of the expectancies in individuals with certain genotypes but not in individuals with different genotypes. These caveats aside, our current results do suggest that individual differences in the ability to store information about past events in order to predict future events and to adjust behavior accordingly – the definition of outcome expectancy – are transmitted in families, but only weakly.

Our results generally are in line with those reported in previous studies on alcohol expectancies assessed in the MOAFTS twins. For example, genetic and environmental influences on alcohol expectancies were assessed in the twins when they were ages 14–22 (Slutske et al., 2002). Results indicated that familial influences (mostly shared environmental) accounted for approximately 26%– 31% of the variance in the individual differences. A follow-up study when the twins were ages 18–29 (Agrawal et al., 2008) reported that familial influences accounted for about 15% to 28% of the variance in alcohol expectancies. Thus, in both studies, there was little evidence for genetic influences in the overall sample.

Why are the estimates of familial influences on smoking expectancies modest? One explanation, as described above, is that we restricted our analyses to those who are regular smokers and, most likely, the full phenotypic range of each expectancy construct was not represented due to our use of a subset of items. A second explanation aligns with the work of Goldman and colleagues (Goldman et al., 2006) who suggest that outcome expectancies represent an integration of numerous information-processing, memory, learning and deliberative (when assessed via self-report) cognitive processes carried out by a diverse set of neural systems. Research shows that such cognitive processes are modestly heritable (working memory = 20%–30% (Swan et al., 1999); visual working memory = 27% (Kremen et al., 2007); visuospatial working memory =44% (van Leeuwen et al., 2009)), while others such as learning strategy have been shown to have no heritable influences and are largely influenced by individual-specific factors (Swan et al., 1999). It stands to reason that heritability would be low to the extent that the smoking expectancies represent some unknown admixture of these processes or mixtures of different cognitive processes in different individuals (which would increase measurement error, and therefore would be included in the estimate of non-shared environmental variance).

A third explanation is that, consistent with social learning models of expectancy development, individual differences in smoking expectancies are, in fact, influenced more by social / environmental factors than by genetic factors. Our results, which show little evidence of genetic influence, along with similar results from studies on alcohol expectancies (Agrawal et al., 2008; Slutske et al., 2002), appear to be more in line with the social learning perspective. In terms of implications for treatment and prevention, our findings that approximately 70% of variance in smoking expectancies is due to non-shared environmental factors, suggest that social learning-based interventions similar to those developed for alcohol expectancies (e.g., expectancy challenge, individualized feedback; Darkes and Goldman, 1993; Wiers et al., 2003) might be effective for altering smoking expectancies in young adult women smokers.

4.1 Limitations

Our results must be considered in light of several limitations. First, our sample is restricted to a cohort of young adult women smokers born in the Midwest, and our findings might not generalize to younger or older samples, those from other regions, to men or to samples consisting of more broad phenotypes (e.g., never-smokers or experimenters).

Second, generalizability of our results might be limited because our expectancy factors differ somewhat from those reported in previous studies. In contrast to prior research (e.g., (Buckley et al., 2005; Cepeda-Benito and Reig Ferrer, 2000; Lewis-Esquerre et al., 2005; Rash and Copeland, 2008; Reig-Ferrer and Cepeda-Benito, 2007; Schleicher et al., 2008; Vidrine et al., 2009), our data could not be characterized by a good-fitting CFA model due to the cross-factor loadings of some items. Most notably, the Craving / Addiction item, “a cigarette can satisfy my urge to smoke,” had relatively high cross-factor loadings on Negative affect reduction (.258), Boredom Reduction (.210) and Taste Manipulation (.219),

and the item, “When I'm feeling down, a cigarette can really make me feel good,” loaded at .378 onto Stimulation-State Enhancement and at .484 onto Negative Affect Reduction. Given that we could find no prior studies that reported such anomalies, we speculate that differences in our factor structure are likely due to factors specific to our sample, embedding of the items in the larger questionnaire, the use of an item subset, changes made to item wording, or the use of a combined item that sampled both Stimulation-state Enhancement and Negative Affect Reduction constructs.

However, we examined the robustness of our results by testing the ND-, DD- and AUD-expectancy associations using follow-up CFAs. In one CFA we assigned the combined item “When I'm feeling down, a cigarette can really make me feel good” to Stimulation-state Enhancement factor, in a second CFA we re-assigned this item to the Negative Affect Reduction factor, and in a third CFA we excluded this item from the analysis. Although fit indices suggested all of these models were ill-fitting, the results of the statistical tests of the associations of the factors with ND, DD and AUD were nearly identical to those from the ESEM.

The generalizability of our results could also be limited because the SCQ is a self-report measure that requires consistent interpretation of the items across participants, as well as introspection, conscious appraisal and deliberation. Thus, the SCQ likely does not assess expectancies that are automatic or outside of conscious awareness such as those measured using indirect or implicit methods (Albery et al., 2006). Further, evidence suggests that expectancies assessed via implicit methods provide additional (unique) information above and beyond that measured via self-report for alcohol (McCarthy and Thompsen, 2006; Wiers et al., 2002) and smoking (McCarthy and Thompsen, 2006). Thus, our results do not apply to expectancies measured via implicit methods.

4.2 Summary and future directions

Using a subset of items adapted from the SCQ, we found that six outcome expectancies, Negative Affect Reduction, Boredom Reduction, Weight-Control, Taste Manipulation, Craving/Addiction and Stimulation-State Enhancement, were significantly associated with ND in our sample of young adult women. These relationships persisted after adjusting for AUD and DD. Three expectancies, Negative Affect Reduction, Boredom Reduction and Weight-Control also were significantly associated with DD. However, none of the expectancies were associated with AUD providing evidence some evidence for discriminant validity. Although we were not able to definitively determine whether genetic versus shared environmental factors accounted for individual differences in the expectancies, our results suggest that among females, about 30% of variance in these expectancies are transmitted in families, with the remaining 70% of the variance being due to environmental factors specific to the individual. However, future studies should include additional phenotypes such as experimenters and never-smokers to ensure a more complete distribution of genetic risk is represented in the sample. Further, using a full-length version of the SCQ to more broadly represent each expectancy construct will ensure that future studies maximize the phenotypic variance in each expectancy. It will also be important to examine Gene X Environment interactions on smoking expectancies- this could reveal that expectancies might be more heritable in individuals with certain genotypes exposed to specific environments.

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Table 1

Scale Score Means and Standard Deviations, Smoking Expectancy Items, Expectancy Factors and Standardized Factor Loadings from the 6-factor ESEM.

		Negative Affect Reduction	Boredom Reduction	Weight Control	Taste Manipulation	Craving / Addiction	Stimulation-State Enhancement
	Scale scores (*SD*)	2.81 (.68)	2.61 (.74)	2.22 (.77)	2.34 (.79)	2.80 (.67)	2.12 (.63)
	Smoking Expectancy Item						
1	When I'm angry, a cigarette calms me down	**0.955** *	**− 0.049**	0.006	−0.020	−0.001	−0.021
2	When I'm upset with someone, a cig. helps me cope	**0.940** *	0.016	−0.033	−0.025	0.002	0.019
3	Smoking a cig. calms me down when I feel nervous	**0.736** *	**0.083**	0.034	**0.065**	**0.072**	0.037
4	When I'm feeling down, a cig. can really make me feel good.	**0.484**	0.030	0.011	**0.044**	**− 0.048**	**0.378** *
5	Cigarettes are good for dealing with boredom	**0.141**	**0.800** *	0.022	**− 0.061**	**− 0.065**	−0.002
6	If I have nothing to do, a smoke can help kill time	**− 0.061**	**1.006** *	0.002	**0.028**	**0.028**	0.006
7	When I'm alone, a cig. can help me pass the time	**0.037**	**0.895** *	−0.003	**0.030**	**0.025**	0.030
8	Smoking cigarettes keeps my weight down	**0.046**	**0.061**	**0.877** *	**− 0.048**	**− 0.049**	−0.007
9	Cigarettes keep me from eating more than I should	0.026	0.005	**0.829** *	0.026	**0.052**	**0.038**
10	Smoking cigarettes helps me control my weight	**− 0.055**	−0.022	**1.025** *	**0.021**	0.007	0.003
11	I enjoy the taste sensations while smoking a cigarette	**0.048**	0.011	−0.019	**0.884** *	−0.009	**0.048**
12	I enjoy the flavor of a cigarette	**− 0.033**	−0.018	0.005	**0.971** *	−0.005	0.005
13	When I smoke a cigarette, the taste is pleasant	0.006	0.014	0.012	**0.934** *	0.000	−0.019
14	A cigarette can satisfy my urge to smoke	**0.258**	**0.210**	**0.074**	**0.219**	**0.253** *	−0.003
15	I will become more addicted to nicotine if I continue smoking	−0.013	**− 0.038**	0.024	**− 0.041**	**0.941** *	−0.031
16	I will become more addicted the more cigarettes I smoke	0.019	**0.033**	**− 0.043**	0.018	**0.875** *	**0.044**

		Negative Affect Reduction	Boredom Reduction	Weight Control	Taste Manipulation	Craving / Addiction	Stimulation-State Enhancement
17	Smoking a cigarette energizes me	**0.096**	**− 0.091**	**0.101**	**0.056**	0.019	**0.714** *
18	A cig. can give me energy when I'm bored and tired	**− 0.041**	**0.061**	**− 0.027**	**− 0.045**	0.005	**1.004** *

Note:

* indicate the thee items used to compute the scale score for each expectancy, where each scale score is the mean of the observed scores for these items. ESEM factor loadings from in bold are statistically significant, $p < .05$.

Table 2

Intercorrelations Among the ESEM Smoking Expectancy Factors, adjusted for Zygosity, Age, ND, DD and AUD.

	Negative Affect Reduction	Boredom Reduction	Weight Control	Taste Manipulation	Craving / Addiction
Boredom Reduction	.694				
Weight Control	.541	.520			
Taste Manipulation	.473	.350	.380		
Craving / Addiction	.367	.298	.274	.219	
Stimulation-State Enhancement	.619	.603	.556	.548	.243

Note: all correlations are statistically significant, $p < .001$.

Table 3

Standardized Coefficients, Adjusted for Zygosity and Age, from the Univariate Regressions of Each Smoking Expectancy Factor onto ND, DD, and AUD Separately.

	Nicotine Dependence	Drug Dependence	Alcohol Use Disorder
Negative Affect Reduction	0.597***	0.598***	0.263**
Boredom Reduction	0.572***	0.591**	0.244**
Weight Control	0.380***	0.333**	0.117
Taste Manipulation	0.275***	0.203	0.024
Craving / Addiction	0.363***	0.189	0.116
Stimulation-state Enhancement	0.310***	0.244*	0.104

Note:

*** $p < .001$,

** $p < .01$,

* $p < .05$.

Table 4

Standardized Coefficients, Adjusted for Zygosity and Age, from the Multivariate Regressions of Each Smoking Expectancy Factor onto ND, DD, and AUD Simultaneously.

	Nicotine Dependence	Drug Dependence	Alcohol Use Disorder
Negative Affect Reduction	0.552***	0.407***	0.041
Boredom Reduction	0.530***	0.416**	0.031
Weight Control	0.363***	0.230*	−0.021
Taste Manipulation	0.277***	0.155	−0.076
Craving / Addiction	0.356***	0.080	0.008
Stimulation-state Enhancement	0.294***	0.155	−0.004

Note:

*** $p < .001$,

** $p < .01$,

* $p < .05$.

Table 5

Estimates and 95% CIs for Additive Genetic (A), Shared Environmental (C) and Non-shared Environmental (E) Influences on Individual Differences in the Smoking Expectancy Factors.

	Negative Affect Reduction	Boredom Reduction	Weight Control	Taste Manipulation	Craving / Addiction	Stimulation-state Enhancement
A	0.008 (0.00–0.38)	**0.38** (0.06–0.47)	0.14 (0.00–0.39)	0.03 (0.00–0.36)	0.23 (0.00–0.38)	0.32 (0.00–0.42)
C	0.29 (0.00–0.39)	0.00 (0.00–0.28)	0.15 (0.00–0.28)	0.22 (0.00–0.28)	0.05 (0.00–0.31)	0.004 (0.00–0.32)
E	**0.71** (0.60–0.81)	**0.62** (0.53–0.73)	**0.71** (0.61–0.83)	**0.74** (0.63–0.84)	**0.73** (0.62–0.85)	**0.68** (0.58–0.80)

Note: Estimates in bold font are significantly different from zero. Either A or C could be dropped with no statistically significant deterioration in the fit of the model for each expectancy except Boredom Reduction (all $p > .05$). However, dropping both A and C resulted in a significant deterioration in the fit of these models (all $p < .001$). For Boredom Reduction, dropping A resulted in a significant deterioration in model fit $\chi^2(1)= 5.12$, $p < .05$, but dropping C did not $\chi^2(1)= 1.0$, $p > .05$. For Boredom Reduction, the parameter estimates and 95% CIs for the best fitting model were A = 0.38 (0.27–0.47) and E = 0.62 (0.53–0.73).