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Marijuana Expectancies and Relationships with Adolescent and Adult Marijuana Use

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Abstract

Background—Outcome expectancy is a central construct in models of addiction and relapse. Much expectancy research has been conducted in the context of alcohol; however, less is known about the structure of expectancies for marijuana and their associations with marijuana use outcomes.

Methods—The data are taken from waves 3 and 4 of a longitudinal high-risk study of parents and adolescent offspring. Of those families who were retained at wave 3, 225 were high-risk and 205 were matched controls (low-risk). In the present study, we examine the factorial structure of marijuana expectancies (wave 3) in the offspring (using an instrument adapted from the alcohol literature) and test whether expectancies mediate the associations of familial risk for substance use, lifetime marijuana use in adolescence (wave 3) and current use in young adulthood (wave 4; reported approximately 5 years later).

Results—We quantified four marijuana expectancy factors similar to those identified in previous studies when the offspring were adolescents (*Mn* age=15.2) and results of our mediation models suggest that negative marijuana expectancies (but not positive expectancies) together with lifetime adolescent marijuana use completely mediated the association between familial risk and current use of marijuana during young adulthood (*Mn* age=20.2).

Conclusion—Familial risk for current marijuana use in young adulthood appears to be transmitted through two orthogonal, prospective pathways. One pathway involves marijuana use during adolescence, and the second pathway involves reduced expectancies that using marijuana will result in cognitive and behavioral impairments.

Keywords

Marijuana use; expectancy; adolescent; adult; mediation; familial risk

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1. Introduction

Substance-use expectancies play a key role in 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 outcomes associated with its use; positive expectancies are thought to promote substance use and relapse, whereas negative expectancies are thought to have the opposite effect.

The majority of expectancy research has focused on those for alcohol. Research shows that certain alcohol expectancies are seen prior to alcohol use onset (Casswell et al., 1988; Christiansen and Goldman, 1983; Christiansen et al., 1982; Dunn and Goldman, 1996; Kraus et al., 1994; Miller et al., 1990) and are associated with quantity and frequency of current drinking (Brown, 1985a, 1985b; Goldman et al., 1997; Ronnback et al. 1999). Further, expectancies prospectively predict alcohol use patterns (Christiansen et al., 1989) and identify adolescents at risk for alcohol involvement (Reese et al., 1994; Smith and Goldman, 1994). Given these associations, alcohol expectancies have been proposed as good targets for prevention and intervention efforts and have been found to be associated with treatment outcomes (Brown, 1985c; Young et al., 2011).

Marijuana expectancies are less well-researched but appear to cover similar domains and to function similarly as those for alcohol (Aarons et al., 2001; Alfonso and Dunn, 2007; Connor et al., 2011; Galen and Henderson, 1999; Schafer and Brown, 1991; Willner, 2001). For example, Shafer and Brown (1991) developed the Marijuana Effect Expectancy Questionnaire (MEEQ) in a college student sample and identified six classes of expectancies: Cognitive and Behavioral Impairment, Relaxation and Tension Reduction, Social and Sexual Facilitation, Perceptual and Cognitive Enhancement, Global Negative Effects, and Craving and Physical Effects. MEEQ scores significantly differed between users and non-users: negative expectancies were higher in non-users and certain positive expectancies were higher in users. These effects have been reported in clinical and community samples (Aarons et al., 2001), in treatment-seeking male polysubstance and alcohol users (Galen and Henderson, 1999), and in adolescents (Alphonso and Dunn 2007).

Similar findings were reported by Willner (2001), who adapted the adolescent form of the Alcohol Expectancies Questionnaire (AEQ-A; Brown et al, 1987) to assess marijuana expectancies in a large (n=4544) community sample of 11-16 year-olds. In addition to finding that non-users reported higher negative expectancies and lower positive expectancies, after adjusting for age, negative expectancies decreased and positive expectancies increased with the frequency of marijuana use. Taken together, studies suggest that negative expectancies deter or delay initiation of marijuana use, but once initiated, positive expectancies become more salient (and more predictive of frequency of use). Indeed, this pattern was noted by Schafer and Brown (1991) and has also been noted in studies of adult clinical samples (Connor et al., 2011; Hayaki et al., 2010).

Given the emerging evidence linking marijuana expectancies to marijuana use, refined analysis of the structure of expectancies and their relationships with longitudinal marijuana use outcomes is warranted. Further it would be useful to examine expectancy--marijuana use associations in a sample that includes individuals at high and low-risk for substance use. It is well known that risk for alcohol use disorder (AUD) is transmitted from parents to offspring via shared genes and shared environments (Heath et al., 1997; Kendler et al., 1994); children of alcoholic parents are two to five times more likely to develop AUDs as adults (Cotton, 1979; Russell et al., 1990). Importantly, studies have also shown that the risk transmitted is

not specific to alcohol use outcomes; such offspring also are at elevated risk for marijuana and other substance use during adolescence (Chassin et al., 1991; Sher et al., 1991) and in young adulthood (Braitman et al., 2009; Chassin et al., 1999). In the present study, we use marijuana expectancy and marijuana use data from a large, longitudinal study of high-risk families (originally ascertained for parental AUDs) and matched low-risk (control) families: i) to quantify the factorial architecture of marijuana expectancies in offspring during adolescence; and (ii) to examine whether expectancies are mechanisms that link familial risk and adolescent lifetime marijuana use with current (past three-month) marijuana use reported in young adulthood (approximately five years later). We hypothesized that marijuana expectancies are mechanisms that mediate the link between familial risk and marijuana use outcomes.

2. Methods

2.1 Participants and Procedures

Participants were from a longitudinal study of a community sample of children/adolescents ($n=454$) and their parents (Chassin et al., 1992, 1999). Of these, 246 adolescents were at high familial risk for substance use because of parental alcohol abuse or dependence (AUD), whereas 208 were offspring from matched non-alcoholic control families. High-risk families were recruited using telephone surveys, DUI records and surveys from a health maintenance organization. Inclusion criteria for high-risk families were Hispanic or non-Hispanic Caucasian ethnicity, English-speaking, Arizona residency, having a 10.5-15.5 year old adolescent, and lack of cognitive limitations precluding interview. Lifetime presence of DSM-III parent AUD was determined through diagnostic interviews with parents (Diagnostic Interview Schedule; Robins et al., 1982) or by spouse report on the Family History-- Research Diagnostic Criteria (FH-RDC; Andreasen et al., 1977). Potential control families were identified from reverse directories that listed households in the same neighborhood as high-risk families. Telephone interviews were used to recruit control families that matched high-risk families on ethnicity, family composition, target child's age (within 1 year), and socioeconomic status (using the property value code from reverse directories or by parents' report of income). Interview data obtained from controls confirmed their non-AUD status; neither biological nor custodial parents met DSM or FH-RDC criteria for lifetime AUD diagnoses. Families were initially interviewed when the offspring were aged 10-16 (Wave 1) and were re-interviewed annually when the offspring were ages 11-17 (Wave 2) and 12-18 (Wave 3). The fourth assessment was completed approximately five years after wave 3 when the offspring were ages 18-25. Sample retention was high with 449 (98.9%), 445 (98%) and 407 (89.6%) re-interviewed at the second, third and fourth waves, respectively. For reasons explained below, we used data from 430 participants (size of the sample with complete data at wave 3), and we imputed data for participants with missing data at wave four.

2.2. Measures

Adolescent marijuana expectancies were self-reported using an adaptation of an abbreviated form of the Alcohol Expectancies Questionnaire (Christiansen et al., 1982). The abbreviated questionnaire used 34 items to assess six expectancy domains previously identified using factor analysis (Christiansen and Goldman, 1983): Social Enhancement, Increased Arousal, Global Positive Effects, Relaxation-tension Reduction, Cognitive-motor Enhancement and Cognitive-behavioral Impairment. In the present study, the same 34 items were used, but the items were reworded by replacing "drinking alcohol" with "smoking marijuana." Item response options were on a five-point Likert-type scale (1= strongly agree, 2=agree, 3=neither agree or disagree, 4=disagree, 5=strongly disagree). Prior to analysis, responses were reverse coded so higher scores would represent higher levels of agreement.

Additionally, 24 of the retained participants did not complete the marijuana expectancy questionnaire at wave 3, reducing our sample size to 430.

Adolescent lifetime marijuana use (Mn age=15.2) was created from three interview questions, from waves 1-3, that asked participants to report the most they had ever used marijuana or hashish, as well as the number of times they had used these substances within the past year and past 3 months. For the lifetime and past year questions, response categories were: Never, 1 to 2 times, 3 to 5 times, more than 5 times, but less than once per month, 1 to 3 times per month, 1 to 2 times per week, 3 to 5 times per week and every day. For the past 3-month question, response categories were: Not at all, 1 to 2 times, once per month, 2-3 times per month, once per week, 2 to 3 times per week, 4 to 5 times per week, and Every day. The proportions of participants who reported any marijuana use at waves 1, 2 and 3 were 8.6%, 11.7% and 17.7%, respectively. To maximize power, participants reporting any use of marijuana at waves 1, 2 or 3 were classified as lifetime marijuana users. Further, of the participants ($n=76$) who reported ever using marijuana at wave 3, only 35.5% ($n=27$) reported using it more than 1 to 2 times, resulting in a highly skewed distribution. Consequently, we created a dichotomous variable that differentiated participants who reported using marijuana at least once in their lifetime (coded 1) from those who reported no use (coded 0).

Adult past three-month (current) marijuana use (Mn age=20.4) was created from a wave 4 interview item that asked participants to report the number of times they used marijuana or hashish in the past three months. Out of the 430 participants with complete data at wave 3, 393 (91.4%) were retained at wave 4. Similar to the adolescent lifetime use measure, the distribution of responses was highly skewed (the majority of retained participants, $n = 313$; 79.6%) reported not using marijuana in the past-three months (although lifetime marijuana use rates at wave 4 were higher). Consequently, we again created a dichotomous variable that differentiated those whom had used any amount (coded 1) from those whom had not used (coded 0). Further, in order to maximize power and to avoid potential biases in parameter estimates due to attrition, we imputed past three-month marijuana use data for the participants ($n = 37$) whom were not retained. To do so, we used Bayesian imputation (Enders, 2010) implemented in Mplus v6.1 (Muthén and Muthén, 1998-2010) to impute ten data sets. The imputation model used risk status (defined below) and adolescent lifetime, past year and past-three month marijuana use items at waves 1, 2 and 3 (dichotomized to represent ever use on each variable), as well as age, gender and ethnicity to specify the conditional distributions from which the imputed data were drawn.

Risk status was defined as having one or both parents with lifetime AUD *DSM-III* diagnoses. Risk status was coded “1” for offspring from families with a history of AUD (high-risk) and those from matched control families (low-risk) were coded “0.”

Covariates included age at interview, gender (1=female, 0=male) and ethnicity (1=Hispanic, 0=non-Hispanic).

2.3. Statistical Analyses

2.3.1. Preliminary Analyses—We attempted to recover the a priori six-factor structure (Christiansen and Goldman, 1983) with confirmatory factor analyses (CFA) using the marijuana expectancy items, but the model failed to converge. Therefore, we moved to the more flexible exploratory structural equation modeling framework (Asparouhov and Muthén, 2009) using Mplus v6.1 (Muthén and Muthén, 1998-2010).

2.3.2. Exploratory structural equation models (ESEMs)—We used ESEM to model the factor structure of the marijuana expectancy items and to extract factor scores for each

individual. 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 also providing standard errors for model parameters, overall tests of model fit and computes factor scores for individuals (Asparouhov and Muthen, 2009). Further, in contrast to CFA, ESEM does not impose the strict requirement that only certain items load onto certain factors; in ESEM, statistically significant cross-factor loadings do not have to be set to zero. Diagnostics from the CFA indicated that cross-loading of items onto the factors was problematic, and a CFA model was not appropriate.

We fit two-, three-, four-, five- and six-factor models to the expectancy items, and we determined the optimal number of factors by balancing parsimony and interpretability with measures of model fit including the Comparative Fit Index (CFI), Tucker-Lewis Index (TLI) and Root Mean Square Error of Approximation (RMSEA). The ESEMs (and the preliminary CFA) were computed using mean and variance adjusted weighted least squares (WLSMV), the expectancy items were treated as categorical, and factor loadings were rotated using geomin rotation. Expectancy factor scores for each individual ($n=430$) were used in the mediation models.

2.3.3 Mediation Models—We used path models to test our mediation hypotheses. To do so, we regressed, simultaneously: i) adult past three-month marijuana use onto the marijuana expectancy factor scores, onto adolescent lifetime marijuana use, and onto risk status; ii) the marijuana expectancy factor scores onto adolescent lifetime marijuana use; and iii) adolescent lifetime marijuana use onto risk status. Further, to adjust for potential confounding effects, we regressed the marijuana use variables at both waves and the marijuana expectancy factor scores onto age, gender, and ethnicity. After testing the regression coefficients for statistical significance using robust maximum likelihood estimation (MLR), we then dropped the model paths and covariates for which the coefficients were not statistically significant, and we re-ran the model. Indirect effects relevant to our hypotheses were tested for statistical significance using the product of coefficients approach (MacKinnon, 2000) with MLR in the reduced model.

3. Results

Descriptive statistics and results of tests for between-group differences for the offspring and parent high- and low-risk groups are shown in Table 1. Data for offspring are from waves 3 and 4 and data from parents are from wave 3. χ^2 tests were used for 2×2 contingency tables (offspring gender, ethnicity, lifetime marijuana use), but nonparametric tests (Kendall's Tau) were used for the larger contingency tables due to low or zero base rates in some of the cells. Of the 430 offspring, 225 (52.3%) were high-risk, and the high-risk and low-risk offspring groups did not significantly differ on age or gender. However, the high-risk group had significantly more participants who were Hispanic, were adolescent lifetime marijuana users, and were adult current marijuana users. We note that the ethnicity difference between risk groups was due to differential attrition; more non-Hispanic participants in the high-risk group were lost during follow-up. For parents, mothers' and fathers' ages were approximately equal across the high- and low-risk groups, but both mothers and fathers in the high-risk group had reported significantly higher rates of marijuana use. In contrast, both mothers and fathers in the high-risk group reported significantly lower education levels. Although defined on the basis of parental alcohol problems, family risk status was associated with offspring and parental marijuana use.

3.1. Best-fitting ESEM

Consistent with recommendations for fit indices (Hu and Bentler, 1999), the ESEM with six factors was better fitting (CFI=.991, TLI=.986, RMSEA=.049) compared to the ESEMs with two (CFI=.970, TLI=.966, RMSEA=.077), three (CFI=.979, TLI=.975, RMSEA=.066), four (CFI=.985, TLI=.981, RMSEA=.058) and five (CFI=.989, TLI=.984, RMSEA=.053) factors. Although the five- and six-factor models were better fitting, the results were suspect; either one or more of the factors lacked interpretability or the a priori factor structure was not well represented. In addition, examination of the scree plot suggested a four-factor solution. Given these considerations and that the fit indices for the four-factor model were well within the acceptable ranges, we selected the more parsimonious four-factor model. The pattern of factor loadings shown in Table 2 indicates the four-factor model recovered interpretable factors that represented, quite well, four of the six a priori expectancy constructs. For clarity of presentation, loadings less than .30 are not shown. In Table 2, we also report internal consistency reliability (Cronbach's alpha) for each of the expectancy factors (using items that loaded onto each factor at .30 or higher). Reliabilities ranged from .800 to .969 suggesting good internal consistency.

3.1.1. Marijuana Expectancy Factors—Results of the four-factor ESEM indicated that three factors represented positive expectancies (Global Positive Effects (GPE); Relaxation-tension Reduction (RTR), and Cognitive-motor Enhancement (CME)) and one factor represented negative expectancies (Cognitive-behavioral Impairment (CBI)). Table 2 shows that 20 of the 34 expectancy items (58.8%) loaded onto the GPE factor. Nine items loaded onto the RTR factor, and six of these cross-loaded onto the GPE factor. Ten items loaded onto the CME factor; four of these cross-loaded onto the GPE factor and one item cross loaded onto the CBI factor, which comprised a total of six items. Interpretation of the factors in ESEM is similar to other forms factor analysis; items that load highly onto a factor drive its interpretation. Cross-loadings onto that factor do not necessarily change its interpretation. Rather cross-loaded items, after controlling for other parameters in the model (factors, loadings, residuals, covariance among factors, etc.), share a component of variance with items that load onto one factor as well as a component of variance with items that load onto a second (or third) factor.

Table 3 shows that the factor scores from the three positive expectancies (GPE, RTR and CME) had moderate to high intercorrelations (.64-.86). In contrast, intercorrelations among the factor scores for the positive expectancies with the negative expectancy (CBI) were low (.00-.27). Further, the ESEM failed to provide evidence of Social Enhancement and Increased Arousal expectancies; with few exceptions, items originally selected to measure these expectancies loaded onto the GPE factor.

3.2. Mediation models

Table 3 also shows the zero-order correlations among all study variables. The correlations of the four expectancy factors with age and with adolescent lifetime marijuana use all were statistically significant, but CBI was the only expectancy that significantly correlated with adult past three-month marijuana use. All of the expectancies except CBI were significantly correlated with ethnicity. None of the correlations of the expectancies with risk status or with gender were statistically significant.

Given that the zero-order correlations indicated the expectancies were not associated with risk status, these paths were not included in the path model, which is shown in Figure 1. After adjusting for all variables in the model (including covariates and correlations among the expectancies, which are not shown in Figure 1), GPE was not significantly associated with either of the marijuana use variables. In contrast, RTR, CME and CBI were

significantly associated with adolescent lifetime marijuana use. However, only CBI was associated with adult past three-month marijuana use. Risk status was significantly associated with adolescent lifetime marijuana use, and adolescent lifetime marijuana use was associated with adult past three-month marijuana use. Importantly, after controlling for mediating variables and covariates, risk status was no longer associated with adult past three-month marijuana use, providing evidence that this association was fully mediated by adolescent lifetime marijuana use and the marijuana expectancies. To examine whether the high correlations (potential multicollinearity) among the positive marijuana expectancy factor scores were affecting estimation of the parameters in the path models, separate models were computed for each expectancy individually. Parameter estimates and results of statistical tests were essentially identical to the full model, suggesting parameter estimates were not affected by multicollinearity.

3.2.1. Reduced path model and tests of specific indirect effects—The results for the reduced path model, which excluded paths and covariates that were not statistically significant, are shown in Figure 2. This model provided stringent examination of mediation by testing for statistical significance of five specific indirect effects associated with the following paths: 1) risk status to adolescent lifetime marijuana use to adult past three month marijuana use; 2) risk status to adolescent lifetime marijuana use to CBI to adult past three-month marijuana use; 3) risk status to adolescent lifetime marijuana use to RTR; 4) risk status to adolescent lifetime marijuana use to CME; and 5) risk status to adolescent lifetime marijuana use to CBI. Results are displayed in Table 4, which shows that all the tests of specific indirect effects were statistically significant (all $ps < .05$).

Results suggest that the effect of risk status on adult past three-month marijuana use was fully mediated by adolescent lifetime marijuana use (indirect effect=1.513) and by adolescent lifetime marijuana use and CBI (indirect effect=0.343). Specifically, high-risk participants were significantly more likely to have used marijuana during adolescence, and those who had used marijuana during adolescence were more likely to have used marijuana within the past three months during adulthood (independent of the marijuana expectancies); in addition, those at high-risk were more likely to use marijuana in adolescence, and adolescent marijuana users were more likely to report lower CBI scores (less likely to expect marijuana causes cognitive and behavioral impairments, or alternatively, those who had not used marijuana were more likely to expect that marijuana causes cognitive-behavioral impairment). In turn, individuals with higher CBI scores were less likely to use marijuana in the past three-months as adults. Finally, although there was no direct association of risk status with the expectancies, the risk status-- adolescent lifetime marijuana use--expectancy indirect effects suggested that risk status influences expectancies, but only through adolescent lifetime marijuana use. Here, high-risk participants were more likely to report adolescent lifetime marijuana use and to report higher RTR (indirect effect=0.413) and CME (indirect effect=0.534) expectancies but lower CBI expectancies (indirect effect =-1.072).

3.2.2. Covariates—Results of the covariate analyses from the reduced path model indicated that age was significantly associated with adolescent and adult marijuana use and all three expectancies (all $ps < .05$). Older participants were more likely to report adolescent lifetime marijuana use (0.784, SE=0.199), less likely to report adult past three-month marijuana use (-0.217, SE=0.098), and more likely to report higher RTR (0.079, SE=0.033) and CME scores (0.097, SE=0.35) and lower CBI scores (-.106, SE=0.032, indicating less agreement that marijuana use causes impaired cognitive and behavioral functioning). Associations of gender and ethnicity with marijuana expectancies and use were not statistically significant (all $ps > .05$) after adjusting for all other variables in the model, with one exception. Gender was significantly associated with past three-month marijuana use

(-0.556, SE=0.259); here, females were less likely to report adult past three-month marijuana use.

4. Discussion

We used an adapted version of the Alcohol Expectancies Questionnaire-adolescent (Christiansen and Goldman, 1983) from a longitudinal study of participants who were at high- and low-risk for substance use, to examine the factor structure of marijuana expectancies and to investigate whether marijuana expectancies are mechanisms that link risk status and lifetime marijuana use in adolescence with past three-month marijuana use in young adulthood.

The ESEMs extracted three positive marijuana expectancy factors, Global Positive Effects, Relaxation-tension Reduction, Cognitive-motor Enhancement and one negative expectancy factor, Cognitive-behavioral Impairment. There was no evidence for Arousal or Social Enhancement expectancies; most items originally thought to measure these factors loaded onto the Global Positive Effects factor. There are several possible explanations for the lack of evidence for Arousal and Social Enhancement expectancies. First, expectancies tend to develop and become more salient as experience with a substance increases (Willner, 2001). It is possible that these expectancies do not emerge until experience with the effects of marijuana accumulates. This explanation seems likely given the majority of the sample (82.3%) reported never using marijuana at wave 3, when the expectancies were assessed. A second explanation is that these expectancies were originally found for alcohol (Christiansen and Goldman, 1983), and although some alcohol expectancy domains overlap with marijuana expectancy domains (Aarons et al., 2001; Schafer and Brown, 1991; Willner, 2001), these particular domains might not. Another possible explanation is that these factors might exist in the high-risk group but not in the low-risk group. However, the scree plots from exploratory factor analyses conducted in each group separately suggested four-factor solutions were appropriate for both groups, as did fit the fit statistics (low-risk: CFI=.985, TLI=.980, RMSEA=.059 versus high-risk: CFI=.985, TLI=.981, RMSEA=.062).

The expectancies for which we did find evidence, Global Positive Effects, Relaxation-tension Reduction, Cognitive-motor Enhancement and Cognitive-behavioral Impairment, have been identified in other samples using instruments specifically designed to assess marijuana expectancies or using instruments, similar to ours, which have been adapted from the alcohol expectancy literature. For example, Willner (2001) developed the adolescent Cannabis Expectancies Questionnaire (ACEQ) by adapting items from Alcohol Expectancy Questionnaire (Brown et al., 1987), and found evidence for a Global Positive Effects scale, similar to that identified by our ESEMs. The MEEQ (Schafer and Brown, 1991) includes Cognitive and Behavioral Impairment, Relaxation and Tension Reduction, and Perceptual and Cognitive Enhancement scales that also are similar to the factors identified in our study. That expectancies similar to ours have been identified by others using different instruments, supports the construct validity of our expectancies.

Results from our mediation models were generally in line with our hypotheses in that marijuana expectancies partially mediated the risk status -- adolescent marijuana use -- adult past three-month marijuana use pathway. Specifically, we found that participants who had not used marijuana during adolescence expected marijuana to cause greater cognitive and behavioral impairment and were less likely to use marijuana in the past three-months as adults (i.e., indirect effect from risk status to adolescent marijuana use to Cognitive-behavioral Impairment to adult past-three month marijuana). This is similar to prior research where negative expectancies have appeared to play a protective role for marijuana use (Alfonso and Dunn, 2007; Galen and Henderson, 1999; Schafer and Brown, 1991; Willner,

2001). Our results extend these findings, because they suggest negative expectancies are protective prospectively as adolescents (mean age = 15.2 years) make the transition to young adulthood (mean age 20.4 years).

Another interesting finding was the specific indirect effect from risk status to adolescent marijuana use to past-three month use in adulthood, which was independent of the marijuana expectancies. When controlling for this path and the path that included Cognitive-behavioral Impairment expectancies, the association between the risk status and adult marijuana use was no longer significant, which suggests the risk status -- adult past three-month association is fully mediated by adolescent marijuana use and by negative expectancies. This finding suggests that preventing marijuana use in adolescence and strengthening negative expectancies among nonusers could limit the use of marijuana in young adulthood.

Contrary to our hypotheses, we did not find evidence that positive expectancies mediated the risk status -- adult current marijuana use association. None of the positive expectancies were associated with adult marijuana use. The reasons for this are not clear, but one explanation is that given only 82 participants in the sample reported adolescent marijuana use and that prior research shows that positive expectancies increase as experience with the effects of marijuana increases, it might be that positive expectancies were somewhat “underdeveloped” in our sample, which limited their predictive validity. A second explanation is related to the dichotomous adult current marijuana use variable that was created due to low endorsement rates of higher frequencies of use. It is likely that the dichotomous measure lacked power to detect associations with positive expectancies as have been reported in other studies that used more refined measures of marijuana use frequency. A third explanation is that positive expectancies are predictive of marijuana use only when subsequent marijuana use is more proximal to the time when expectancies are assessed. Here, past three-month marijuana use was reported, on average, about 5.2 years after the expectancies were assessed. It is unknown whether positive expectancies would predict marijuana use at intermediate intervals; however, these data are not available in the present study and this should be examined in future research.

4.1. Limitations

We used an instrument to assess marijuana expectancies that was adapted from an instrument designed to assess alcohol expectancies. Although, this was done in a previous study (Willner, 2001) and research suggests substantial overlap between alcohol and marijuana expectancies (Aarons et al., 2001; Schafer and Brown, 1991; Willner, 2001), the full range of marijuana expectancy constructs might not have been fully represented in our data. For example, the MEEQ (Shafer and Brown, 1991) also assesses for social and sexual facilitation, global negative effects and craving and physical effects expectancies for marijuana.

There are other limitations of our findings. As stated above, the need to dichotomize the adolescent lifetime and adult current use variables may have limited our ability to detect associations between the positive expectancies and adult current marijuana use, and might limit the generalizability of our results. A more comprehensive assessment of marijuana use, potentially exploring both frequency of use and quantity smoked (especially ascertained at shorter time intervals) might allow for additional insights into how expectancies and marijuana use co-evolve. In addition, whether expectancies were interpreted, and consequently reported differently by marijuana users versus non-users is worth examining. For instance, non-users were more likely to report higher cognitive and behavioral impairment expectancies, which may be attributed to vicarious learning. Likewise, fewer reports of negative expectancies in experienced users may be attributable to drug tolerance.

Another limitation is these data were collected in the early 1990s when the prevalence of lifetime marijuana use combined across students in the eighth, tenth and twelfth grades was estimated to be relatively low (22.7%), especially when compared to the 31% prevalence estimate for 2011 (Johnston et al., 2011). This cohort effect likely explains the low prevalence of marijuana use in this sample. Thus, we cannot be sure how well our results generalize to the current population. Third, we do not consider other mechanisms that might mediate the relationships among familial risk, expectancies and marijuana use outcomes, such as various forms of psychopathology, comorbid use of alcohol, tobacco and other drugs, and current use of such drugs by parents. It is important that future studies address these issues.

4.2. Conclusion

Overall, our results indicate that marijuana expectancies, similar to those identified in previous studies, can be quantified in adolescents at high- and low-risk for substance use. Further, familial risk for marijuana use in young adulthood appears to be transmitted through two orthogonal, prospective pathways. One pathway involves marijuana use during adolescence, and the second pathway involves reduced expectancies that using marijuana will result in cognitive and behavioral impairments. Together, both pathways fully mediated the association of familial risk with adult past three-month marijuana use, indicating that prevention and intervention efforts should deter adolescent marijuana use onsets and focus on maintaining expectancies about marijuana's adverse cognitive and behavioral effects to minimize marijuana use in young adulthood.

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Figure 1.

Path model representing the associations among risk status, adolescent lifetime marijuana use, adolescent marijuana expectancies and adult past three-month marijuana use. The coefficients are across-data set averages and are unstandardized. Significance tests are based on standard errors across the 10 data sets. Correlations among the expectancies and paths to covariates are not shown. Note: * $p < .05$, *** $p < .001$.

Figure 2.

Reduced path model representing the associations among risk status, adolescent lifetime marijuana use, adolescent marijuana expectancies and adult past three-month marijuana. The coefficients are across-data set averages and are unstandardized. Significance tests are based on standard errors from across the 10 data sets. Correlations among the expectancies and paths to covariates are not shown. Note: * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 1

Sample descriptive statistics for offspring and parents at high- and low-risk for substance use and results of tests for significant between-group differences

Offspring					
Wave 3	High-risk (n=225)	Low-risk (n=205)	Tests of between-group differences		
	Mn (sd)	Mn (sd)	t	df	P
Age at Interview	15.1 (1.44) n (%)	15.2 (1.46) n (%)	.538 z	428 df	.59 p
Female Gender	105 (46.7)	94 (45.9)	0.05	1	.923
Hispanic Ethnicity	78 (34.7)	48 (23.4)	6.56	1	< .05
Lifetime Marijuana Use	62 (27.6)	19 (9.3)	23.46	1	<.001
Wave 4	(n=200) n (%)	(n=193) n (%)	K-Tau	SE	p
Current (past-3 month) MJ Use					
Never	149 (74.5%)	164 (85%)	.122	.047	< .05
1-2 Times	17 (8.5%)	9 (4.7%)			
3- 5 Times	2 (1.0%)	2 (1.0%)			
> 5 Times, but < Once per Mo.	6 (3.0%)	2 (1.0%)			
1-3 Times per Week	7 (3.5%)	3 (1.6%)			
1-2 Times per Week	6 (3.0%)	6 (3.1%)			
3-5 Times per Week	7 (3.5%)	2 (1.0%)			
Daily	6 (3.0%)	5 (2.6%)			
	(n=225) n (%)	(n=205) n (%)	Coeff	(95% CI)	
Dichotomized Current MJ Use ^①	57.7 (25.6)	31.6 (15.4)	0.365	.071-.660	
Parents					
	High-risk (Mothers n=214) (Fathers n=177)	Low-risk (Mothers n=202) (Fathers n=164)			
	Mn (sd)	Mn (sd)	t	df	p
Mother's Age	40.6(5.22)	41.4 (5.67)	1.51	414	0.13
Father's Age	42.5(5.97)	43.5 (6.01)	1.63	339	0.10
	n (%)	n (%)	K-Tau	SE	p
Mother's Education					
Did Not Graduate (HS)	15 (11.7%)	18 (8.9%)	-.144	.043	< 0.01
HS graduate	66 (30.8%)	44 (21.8%)			
Technical College	16 (7.5%)	19 (9.4%)			
Some College	74 (34.6%)	71 (35.1%)			
College Graduate	25 (11.7%)	40 (19.8%)			

Wave 3	Offspring		Tests of between-group differences		
	High-risk (n=225)	Low-risk (n=205)	<i>t</i>	<i>df</i>	<i>P</i>
	<i>Mn (sd)</i>	<i>Mn (sd)</i>			
Graduate/professional school	8 (3.7%)	10 (5.0%)			
<u>Mother's Lifetime MJ Use</u>					
Never	118 (52.4%)	157 (77.7%)	.238	.043	< 0.001
< 5 Times	46 (21.5%)	29 (14.4%)			
> 5 Times, but < Once per Month	14 (6.5%)	7 (3.5%)			
Once per Month	1 (0.5%)	0			
2-3 Times per Month	9 (4.2%)	0			
1 Time per Week or More	26 (12.1%)	9 (4.5%)			
<u>Father's Education</u>					
Did Not Graduate (HS)	27 (15.3%)	8 (4.9%)	-.219	.046	< 0.001
HS graduate	47 (26.6%)	25 (15.3%)			
Technical College	14 (7.9%)	11 (6.7%)			
Some College	55 (31.1%)	65 (39.9%)			
College Graduate	18 (10.2%)	30 (18.4%)			
Graduate/professional school	16 (9.0%)	24 (14.7%)			
<u>Father's Lifetime MJ Use</u>					
Never	78 (44.1%)	117 (71.3%)	.281	.047	<0.001
< 5 Times	31 (17.5%)	24 (14.6%)			
> 5 Times, but < Once per Month	21 (11.9%)	11 (6.7%)			
Once per Month	3 (1.7%)	0			
2-3 Times per Month	3 (1.7%)	0			
1 Time per Week or More	41 (23.2%)	12 (7.3%)			

^① Note: Average over 10 imputed data sets.

Table 2

Expectancy items, Factors, Cronbach's Alpha, Standardized Factor Loadings from four factor ESEM

	Global Positive Changes	Relaxation- tension Reduction	Cognitive- motor Enhancement	Cognitive- behavioral Impairment
Reliability (Cronbach's Alpha)	.969^a	.935^b	.907^c	.800^d
Expectancy Items				
1. allows me to join others who are having fun	0.980			
2. makes me feel more powerful than others	1.077			
3. makes parties more fun	0.873			
4. helps me celebrate social situations	0.767			
5. makes me feel stronger and more powerful	0.802			
6. makes me more creative and imaginative	0.736			
7. helps me when I am tense or nervous	0.651	0.305		
8. keeps my mind off of mistakes at school	0.601	0.307		
9. helps me feel less fearful	0.613			
10. helps me forget my worries	0.593	0.437		
11. helps me stand up to others	0.587			
12. makes the future seem brighter	0.580			
13. makes me feel in control of the situation	0.551		0.347	
14. helps cheer me up when I'm in a bad mood	0.532			
15. relaxes me	0.474	0.561		
16. lets me forget everything	0.417	0.379		
17. makes me feel more relaxed and less tense	0.335	0.621		
18. I do not worry as much about what other people think of me after smoking MJ		0.569		
19. gets rid of my feelings that I am not as good as other people		0.466		
20. it is easier to speak in front of a group of people after smoking MJ		0.304		
21. It is easier to play sports after I've been smoking MJ			0.891	
22. makes me feel more alert			0.789	
23. makes me feel healthier			0.712	
24. I can do things better after smoking MJ	0.375		0.643	
25. I feel more caring and giving after smoking MJ			0.616	
26. helps me understand things better	0.326		0.577	
27. I have stronger feelings when I've been smoking MJ			0.539	0.379
28. I often have trouble remembering what I did while I was smoking MJ				0.811
29. I sometimes lose control and run into things				0.790
30. I do stupid, strange or silly things when I've been smoking MJ				0.729
31. I'm harder to get along with after smoking MJ				0.667
32. causes hangovers				0.641
33. I can control my anger better when I'm smoking MJ	0.337		0.397	
34. allows me to be in any mood I want to be			0.364	

^aNote: Reliability analyses were based on 426,

b_{428} ,

c_{429} ,

d_{425} participants with complete data. Items that loaded at .30 or higher on each factor were included in computing reliabilities.

Table 3
Zero-order Correlations Among the Expectancies, Marijuana Use Variables and Covariates

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Global Positive Changes	--								
2. Relaxation -tension Reduction	.772 ***	--							
3. Cognitive-motor Enhancement	.855 ***	.644 ***	--						
4. Cognitive-behavioral Impairment	.240 *	.273 ***	-.001	--					
5. Adolescent Lifetime MJ use	.172 *	.216 **	.266 ***	-.481 ***	--				
6. Adult Past 3-month MJ use	-.005	.007	.015	-.193 *	.287 **	--			
7. Risk status (high risk =1)	.078	.071	.101	-.101	.427 ***	.224 **	--		
8. Gender (Female=1)	-.071	-.035	.022	.008	.054	-.194 *	.013	--	
9. Ethnicity (1=Hispanic)	.169 **	.132 *	.168 *	-.093	.104	.017	.205 **	.063	--
10. Age (wave 3)	.129 **	.158 **	.189 ***	-.257 ***	.489 ***	-.057	-.032	.061	.134 *

* Note: $p < .05$

** $p < .01$

*** $p < .001$.

Table 4

Results of Significance Tests of Indirect Effects

Path	Coefficient	SE	z	p
1. Risk status -- adolescent MJ use -- CBI-- adult MJ use	0.343	0.169	2.024	0.043
2. Risk status -- adolescent MJ use -- adult MJ use	1.513	0.587	2.576	0.010
3. Lifetime MJ use -- CBI-- adult MJ use	0.215	0.100	2.148	0.032
4. Risk status -- adolescent MJ use -- CBI	-1.072	0.246	-4.363	0.000
5. Risk status -- adolescent MJ use -- RTR	0.413	0.210	1.971	0.049
6. Risk status -- adolescent MJ use -- CME	0.534	0.227	2.351	0.019