Community Ment Health J (2017) 53:383–393
DOI 10.1007/s10597-017-0110-2

ORIGINAL PAPER

Interactive Effect of Parent and Adolescent Psychiatric Symptoms on Substance Use among Adolescents in Community Treatment

Lourah M. Kelly[1] · Sara J. Becker[2,3] · Jennifer C. Wolff[3] · Hannah Graves[3] · Anthony Spirito[3]

Received: 19 February 2016 / Accepted: 27 January 2017 / Published online: 16 March 2017

Abstract Both adolescent and parent psychiatric symptoms are well-established risk factors for adolescent substance use (SU), but the ways that these symptoms interact are not well understood. This study examined the interactive effects of parent and adolescent psychiatric symptoms on adolescent frequency of alcohol and marijuana use, over and above the effects of parental SU. Seventy adolescents presenting to a community mental health center (CMHC) participated. Parent and adolescent psychiatric symptoms were measured with the brief symptom inventory (BSI) and child behavior checklist (CBCL), respectively. Hierarchical regressions revealed different patterns for adolescent alcohol and marijuana use. For alcohol, the BSI parent phobic anxiety subscale predicted increased adolescent use while the parent interpersonal sensitivity subscale predicted decreased use: the effects of these parental symptoms were strongest among adolescents with higher levels of externalizing problems on the CBCL. For marijuana, the BSI parent psychoticism subscale predicted increased adolescent use, whereas paranoid ideation predicted decreased use. Results suggest that adolescent SU treatment and assessment should attend to both adolescent and parent psychiatric symptoms.

✉ Lourah M. Kelly
lkelly2@suffolk.edu

[1] Psychology Department, Suffolk University, 73 Tremont St, 8th Floor, Boston, MA 02114, USA

[2] Department of Behavioral and Social Sciences, Center for Alcohol and Addiction Studies, Brown School of Public Health, 121 South Main Street, Providence, RI 02912, USA

[3] Department of Psychiatry and Human Behavior, Alpert Medical School of Brown University, Box G-BH, Providence, RI 02912, USA

Keywords Adolescent · Substance use · Psychiatric symptoms · Community behavioral health clinic

Introduction

Adolescent substance use (SU) is a major public health concern. According to the 2014 National Household Survey on Drug Use and Health (Center for Behavioral Health Statistics and Quality 2015), 11.5% of adolescents in the U.S. age 12–17 reported using alcohol in the past month and 7.4% reported using marijuana, which corresponds to roughly 2.9 and 1.8 million adolescents, respectively. In this age cohort, SU is associated with a range of negative outcomes including family conflict, problems with peers, truancy, sexual risk taking, and legal involvement (e.g., Crowe and United States 1998; Hallfors et al. 2002; Henry and Thornberry 2010). Approximately 80% of adolescents who receive publicly funded treatment for SU-related problems first present to outpatient settings (Substance Abuse and Mental Health Services Administration [SAMHSA], 2015). As such, community mental health clinics (CMHCs) offer an ideal opportunity to study factors associated with adolescent SU.

Adolescents who present for SU treatment in CMHCs typically have high rates of co-occurring mental health disorders (Weaver et al. 2003; Kaminer and Bukstein 2007), which contribute to greater functional impairment and worse treatment outcomes (Shane et al. 2003; Mertens et al. 2007). In the Cannabis Youth Trial (Dennis et al. 2004), the largest randomized trial of adolescents presenting to outpatient community SU treatment to date, 33% of the 600 youth met criteria for an internalizing psychiatric disorder (i.e., a disorder associated with "internalizing" symptoms such as depression, anxiety, suicidal thoughts or behaviors)

and 61% met criteria for an externalizing mental health disorder (i.e., a disorder associated with "externalizing" symptoms such as disruptive behavior or attention deficit hyperactivity disorder). Such high co-occurrence rates in large community-based samples have led some researchers to suggest that adolescent SU and psychiatric symptoms are two related outcomes that share common underlying risk factors. One such risk factor is family or parent dysfunction (Fergusson and Horwood 1997; Kamon et al. 2006). Consistent with this view, prior research has consistently found that problems with parenting processes such as monitoring, communication, and closeness with the child are important risk factors for the development and maintenance of adolescent SU (see Vakalahi 2001; Cordova et al. 2014; Marceau et al. 2015).

One potentially important parenting factor that has received far less attention in the adolescent SU literature is parental psychiatric symptoms. To date, the relatively few studies that have examined the effects of parent psychiatric symptoms on adolescent SU have predominantly focused on parents who meet criteria for an alcohol use disorder (Hussong et al. 2010, 2012) or another drug use disorder (Kirillova et al. 2001; Li et al. 2002). Results of these investigations demonstrate that when parents have a substance use disorder, higher levels of parent psychiatric symptoms are associated with higher rates of adolescent SU (Clark et al. 2004). However, the findings from this work may not generalize to all parents of adolescents in SU treatment.

A second concern with prior literature is that it has typically focused on the main effects of parent and adolescent psychiatric symptoms on adolescent SU (i.e., the independent effect of each risk factor) without examining the interactive effect of the two risk factors (i.e., the combined effect of the two risk factors). Studying interactions can inform clinical interventions by revealing if the total effect of two risk factors is different than what would be expected based on the sum of their separate effects. For instance, if the combination of adolescent and parental depression had a large effect on adolescent drinking, even if each risk factor only had a small or non-significant effect, then the two risk factors would be said to have an interactive effect. In this example, the significant interaction would suggest that the combination of parental and adolescent depressed mood was a particularly important risk factor for adolescent SU, which could be targeted in treatment. Due to the focus on main effects in prior research (e.g., Marmorstein et al. 2012), the interactive effects of parental and adolescent psychiatric symptoms are not well understood, but are potentially important for treating adolescent SU.

A third limitation of previous investigations has been failure to consider the unique effects of adolescent and parent psychiatric symptoms on the two most commonly used substances among adolescents presenting to treatment: alcohol and marijuana (Swendsen et al. 2012). Eighty-three to 89% of all adolescents presenting to SU treatment primarily use alcohol or marijuana (SAMHSA 2015). Most prior investigations of adolescent SU that have examined parent and/or teen risk factors have measured only adolescent marijuana use (Miller et al. 2013), only adolescent alcohol use (Hussong et al. 2010, 2012), or assessed a combined indicator of marijuana and other illicit drug use (Kamon et al. 2006). This approach is not consistent with several cross-sectional and longitudinal studies indicating that alcohol and marijuana have different associations with co-occurring mental health problems (see Armstrong and Costello 2002 for a review) as well as with common negative consequences (e.g., risky behaviors, driving, emotional consequences; Palamar et al. 2014).

Finally, previous research on the link between parent psychiatric symptoms and adolescent SU has not examined multiple dimensions of parent psychiatric symptoms simultaneously (see Mowbray and Oyserman 2003 for a review). An early study by Merikangas et al. (1998) found that adolescents whose parents met criteria for an anxiety disorder (i.e., generalized anxiety, phobia, panic disorder, separation anxiety) had significantly higher rates of alcohol use and alcohol use disorders than adolescents whose parents did not meet criteria for an anxiety disorder Another early longitudinal study by Weissman et al. (1997) found that parental depression was associated with a five-fold increase in risk of adolescent alcohol dependence. Fewer studies have examined the effects of parent psychiatric symptoms on adolescent marijuana use. However, a recent study suggests that a genetic predisposition to schizophrenia, an illness characterized by psychotic symptoms, may be related to increased risk of marijuana use in adolescents (Power et al. 2014). Kamon et al. (2006) conducted one of the only studies that has examined the effect of more than one type of parent psychiatric symptomology on adolescent SU and, counter to expectations, the study did not find an association between parent symptoms and adolescent SU. Of note, however, this study did not examine alcohol and marijuana use separately or account for psychotic symptoms. To disentangle the effects of parent psychiatric symptoms on adolescent alcohol and marijuana use, studies need to simultaneously control for multiple symptom dimensions such as depression, anxiety, and psychosis.

The goal of the current study was to evaluate the joint effect of parent and adolescent psychiatric symptoms on adolescent SU in a sample of adolescent-parent dyads presenting for treatment in a CMHC. We aimed to identify the joint effect of these risk factors above and beyond the influence of problematic parental substance use. To address the limitations of prior research, the current study examined multiple dimensions of parent psychiatric symptoms and explored risk factors separately for

marijuana and alcohol. Parent psychiatric symptoms were measured using the well-validated Brief Symptom Inventory (BSI; see Derogatis 1993), which examines concerns across nine dimensions: depression, anxiety, phobic anxiety, somatization, obsessive compulsive, interpersonal sensitivity, hostility, psychoticism, and paranoid ideation. Adolescent psychiatric symptoms were measured using the child behavior checklist (CBCL), which examined symptoms of two broad symptom classes: internalizing and externalizing problems.

Due to the lack of prior research on the interactive effects of parent and adolescent psychiatric symptoms, our analyses were primarily intended to be exploratory. However, we had two specific hypotheses. First, we expected there to be significant interactions between similar types of parent and adolescent psychiatric symptoms (i.e., adolescent depression × parent depression, adolescent psychoticism × parent psychoticism), even when controlling for the effects of parental problematic substance use. We expected these interactions to reveal that the joint effect of parent and adolescent symptoms was greater than their additive effects. Second, we expected there to be different associations between parent psychiatric symptoms and the two primary substances of adolescent use. Based on previous research, we expected parental symptom types commonly associated with anxiety (i.e., BSI anxiety, somatization, phobic anxiety) and depression (i.e., BSI depression, interpersonal sensitivity) to be associated with adolescent alcohol use (Weissman et al. 1997; Merikangas et al. 1998), and for parental symptoms commonly associated with psychosis (i.e., BSI psychoticism, paranoid ideation) to be associated with adolescent marijuana use (Power et al. 2014).

Methods

Participants and Procedures

Study procedures were approved by the University institutional review board. Participants were recruited from an outpatient program for adolescents aged 13–18 with comorbid mental health and substance use problems in a CMHC in the northeast region of the United States. All adolescents who began treatment in the program and their parent or legal guardian were invited to participate in the study. After written informed consent and assent were obtained from parents and adolescents, respectively, each participant completed a battery of questionnaires. Seventy-four out of 88 adolescent-parent dyads (84%) agreed to participate. All 74 adolescents and 70 of the parents completed the full assessment battery. The 70 parent-adolescent dyads who completed the assessment were included in the analysis.

Measures

Adolescent Psychiatric Symptoms

The Child Behavior Checklist – 6/18 (CBCL; Achenbach and Rescola 2001) is a parent-report measure of adolescent internalizing and externalizing problems. The 119 items of the CBCL measure emotional and behavioral difficulties over the past 6 months using a 3-point Likert scale (0 = Not True, 1 = Somewhat or Sometimes True, 2 = Very True or Often True). The delinquency/rule breaking behavior and aggressive behavior sub-scales were summed to create the overall externalizing problems scale. The withdrawn/depressed, anxious/depressed, and somatic complaints subscales were similarly summed to create the overall internalizing problems scale. CBCL T-scores of 70 or greater indicate clinical levels of internalizing and externalizing problems. The internalizing and externalizing problems scales have demonstrated excellent internal consistency in treatment-seeking samples ($\alpha = 0.88$; Van Meter et al. 2014 and $\alpha = 0.88$; Nakamura et al. 2009, respectively). In the current sample, internal consistency was 0.89 for the internalizing scale and 0.93 for the externalizing scale.

Parent Psychiatric Symptoms

The 53-item Brief Symptom Inventory (BSI; Derogatis and Melisaratos 1983; Derogatis 1993) is a self-report measure that assesses nine dimensions of parent psychiatric symptomatology: depression, anxiety, phobic anxiety, somatization, obsessive compulsive, interpersonal sensitivity, hostility, paranoid ideation, and psychoticism. Items are answered on a five-point scale from 0 (Not at all) to 5 (Extremely). BSI t-scores ≥ 65 indicate clinical levels of psychiatric symptoms. In outpatient samples, internal consistency of subscales range from 0.71 to 0.89 (Derogatis and Melisaratos 1983; Boulet and Boss 1991): this range was highly consistent with the alpha values found in the current study, which ranged from 0.69 to 0.90. We simultaneously controlled for all nine scales in the analyses, though as noted in our hypotheses, we did not expect all of the scales to be significantly associated with adolescent SU.

Parent Problematic Substance Use

Adolescent report was used as an indicator of problematic parental substance use. Adolescents were asked a series of questions about parental abuse and neglect: one of the questions asked how often their parent was unable to care for them due to being drunk or high. If the adolescent

responded "often" or "very often," their parent was categorized as having problematic substance use.

Adolescent Substance Use

Adolescent alcohol and marijuana use were both measured using self-report of recent frequency of use, which is the most common approach to measuring adolescent SU in the treatment literature (Waldron and Turner 2008; Hogue et al. 2014; Tanner-Smith and Lipsey 2015). The first item from the Adolescent Drinking Questionnaire (ADQ; Jessor et al. 1989), a well validated adolescent self-report measure, was used to measure frequency of alcohol use over the past three months. Responses ranged from 1 (no alcohol use) to 8 (everyday). Frequency of marijuana use over the past 3 months was measured using the Drug Use Questionnaire (Spirito 1999). Adolescents were asked to estimate the total number of days over the past 90 days that they had smoked any marijuana, with possible answers ranging from 0 to 90 days. Responses to this item were recoded to match the alcohol use item, with options ranging from 1 (no marijuana use) to 8 (daily use).

Data Analytic Plan

Three sets of preliminary analyses were conducted prior to testing our hypotheses. First, we examined whether the key study variables (i.e., frequency of adolescent alcohol and marijuana use, adolescent CBCL scales, and parent BSI scales) were associated with adolescent demographic variables (i.e., gender, age, race, ethnicity) to determine which of these variables to include in final models. Second, we examined variable distributions to ensure that they met the modeling requirements (i.e., skew and kurtosis fell within the acceptable ranges of ± 1.5 as recommended by Tabachnick and Fidell 2013). Finally, we accounted for missing scale-level data. Five of the 70 parents (7%) were missing data on one or more of the BSI scales but had available data on other scales and/or assessment measures. We used multiple imputation methods, a common statistical approach that uses all available data to generate missing values, to estimate missing data on the BSI scales for these parents. To ensure that our results were robust to the effects of these imputations, we replicated the analyses by excluding any participants with missing data and confirmed an identical pattern of results.

We used hierarchical regression analysis, an analytical approach that enters data in sequential steps, to test our hypotheses. Two hierarchical regressions (one for alcohol frequency and one for marijuana frequency) were conducted to explore the main and interactive effects of adolescent internalizing problems, adolescent externalizing problems, and nine dimensions of parent psychiatric symptoms. In step one, the key covariates—any significant demographic variables, adolescent internalizing symptoms, adolescent externalizing symptoms, and parent problematic substance use—were entered. These variables were entered first so that we could control for their effects in all subsequent steps. In step two, the nine parent psychiatric symptom types from the BSI were entered to determine their main effects on frequency of drinking and marijuana. We entered the parent symptom types in a separate regression step so that we could determine which symptom types were most important. This allowed us to drop non-significant variables and simplify the model. We used backwards elimination, a statistical method that removes one non-significant variable at a time until all variables meet a predetermined criterion. We used a criterion of $p < 0.10$ for this process. In the third and final step, interaction variables between the remaining BSI subscales and the adolescent internalizing as well as adolescent externalizing symptom scales (e.g., BSI subscale × CBCL internalizing, BSI subscale × CBCL externalizing) were entered. Model comparisons using the F-change test were used to obtain the simplest model in the third and final step.

As recommended by Holmbeck (2002), all variables were centered around their means. Significant interactions were explored and graphed using simple slopes, consistent with the recommendations of Aiken and West (1991). Specifically, significant interactions predicting drinking and marijuana frequency were interpreted by plotting simple regression lines at one standard deviation above (e.g., clinically significant symptoms) and below (e.g., not clinically significant symptoms) the mean of the moderator. Version 22 of the Statistical Package for the Social Sciences (SPSS) was used for analyses.

Results

Table 1 presents the demographics and clinical characteristics of the sample. More than half of the sample identified as male. Participants ranged in age from 13 to 18 years with a mean of about 15 years. Racial composition of the adolescents was about two-thirds White, Non-Hispanic, with modest representation of Hispanic White, Non-Hispanic Black, and Hispanic Black. This racial/ethnic distribution was representative of patient demographics in the CMHC.

Marijuana and alcohol were the most commonly used substances by adolescents in the sample. Over three-quarters of participants reported any marijuana use (77.1%, $n = 54$) and over half reported any alcohol use (58.6%, $n = 41$) in the past 3 months. Most participants who reported use of either alcohol or marijuana reported use of both substances. Only 16% of adolescents reported use of any other drug; cocaine and prescription drugs (taken not

Table 1 Adolescent and parent demographic and clinical characteristics

Variables	Mean (SD)	N (%)
Adolescent variables		
Male		42 (60%)
Age	15.63 (1.13)	
Race		
White, Non-Hispanic		44 (62.9%)
Black, Non-Hispanic		5 (7.1%)
Hispanic White		13 (18.6%)
Hispanic Black		2 (2.9%)
Mixed or Other		4 (5.8%)
Not reported		2 (2.9%)
CBCL internalizing	65.0 (10.7)	27 (38.6%)
CBCL externalizing	70.0 (10.4)	41 (58.6%)
Alcohol use frequency		
Never		29 (41.4%)
1–2 times		16 (22.9%)
Once per month		6 (8.6%)
Less than weekly, more than once per month		9 (12.9%)
Weekly		5 (7.1%)
2–3 times per week		2 (2.9%)
4 + times per week		3 (4.3%)
Daily		0 (0%)
Marijuana use frequency		
Never		16 (22.9%)
1–2 times		9 (12.9%)
Once per month		2 (2.9%)
Less than weekly, more than once per month		4 (5.7%)
Weekly		3 (4.3%)
2–3 times per week		14 (20.0%)
4 + times per week		10 (14.3%)
Daily		12 (17.1%)
History of Other Drug Use		
Cocaine		6 (8.6%)
Hallucinogens		1 (1.4%)
Inhalants		2 (2.9%)
Prescription drugs		6 (8.6%)
Other drug use		3 (4.3%)
Parent variables		
Problematic substance use		8 (11.4%)
BSI psychiatric symptom scales		
Somatization	57.7 (11.0)	21 (30%)
Obsessive–Compulsive	61.0 (12.7)	31 (44%)
Interpersonal sensitivity	57.5 (9.5)	14 (20%)
Depression	59.9 (10.2)	28 (40%)
Anxiety	57.8 (10.9)	23 (33%)
Hostility	58.0 (11.2)	26 (37%)
Phobic anxiety	55.9 (11.6)	17 (24%)
Paranoid ideation	59.6 (10.5)	25 (36%)
Psychoticism	60.0 (11.3)	26 (37%)

For psychiatric symptom variables (CBCL, BSI), the number and percentages refer to the adolescent and parent participants who scored in the clinical range

as prescribed) were the most commonly reported other drugs (each used by 9% of the sample).

Regarding psychiatric symptoms, more adolescents had externalizing than internalizing mental health problems. About 60% of adolescents had externalizing problem scores in the clinical range (58.6%, $n = 41$), while only about 40% had internalizing problem scores in the clinically significant range (38.6%, $n = 27$). Although mean parent psychiatric symptom scores on the BSI fell in the non-clinical range (all M's < 65), the proportion of parents scoring in the clinically significant range on each of the BSI symptom subscales ranged from 20 to 44%. Overall, 67% of parents scored in the clinical range on at least one of the BSI symptom subscales.

Adolescents who identified as female ($M = 3.1$, $SD = 2.1$) had significantly higher levels of alcohol frequency than those who identified as male ($M = 2.1$, $SD = 1.4$, $t(42) = 2.26$, $p < 0.05$). There were no other gender differences and none of the other demographic factors were associated with any of the primary variables of interest. Thus, gender was the only demographic variable included as a covariate in the analyses.

Correlations among the primary variables of interest were also examined. Alcohol and marijuana use frequency were not significantly related ($r = 0.18$, $p = 0.18$), further supporting our choice to examine these substances separately. Adolescent internalizing problems were significantly and positively related to alcohol use frequency ($r = 0.27$, $p < 0.05$) but not to marijuana use frequency. All nine parent psychiatric symptom dimensions were significantly related to adolescent internalizing problems with r's ranging from 0.36 (paranoid ideation) to 0.58 (psychoticism). Only parent obsessive–compulsive ($r = 0.25$, $p < 0.05$), depression ($r = 0.34$, $p < 0.05$), and hostility ($r = 0.34$, $p < 0.05$) were significantly related to adolescent externalizing problems. Parent domains were all significantly related to each other, but correlations were not high enough to preclude regression analyses (r's < 0.77; Mason and Perreault 1991).

Hierarchical Linear Regressions Predicting Alcohol Use

The first hierarchical regression examined the main and interactive effects of parent and adolescent psychiatric symptoms on adolescent alcohol use. In Step 1, there were

significant effects of adolescent gender (β = 0.28, $t = 2.52$, $p = 0.01$) and internalizing problems (β = 0.27, $t = 2.09$, $p = 0.04$). Furthermore, there was a non-significant trend for problematic parental alcohol use to be associated with increased adolescent drinking (β = 0.19, $t = 1.71$, $p = 0.09$). In Step 2, two of the nine BSI variables remained after backwards elimination with our $p < 0.10$ criterion: interpersonal sensitivity (β = −0.33, $t = -1.99$, $p = 0.05$) and phobic anxiety (β = 0.28, $t = 1.80$, $p = 0.08$). These two variables had small to moderate effects on adolescent drinking: parent interpersonal sensitivity was associated with lower levels of adolescent alcohol use, while parent phobic anxiety was associated with higher levels of adolescent alcohol use. In the third step of the model, there were two significant interaction terms: parent interpersonal sensitivity × adolescent externalizing problems, and parent phobic anxiety × adolescent externalizing problems. The interactive effects of adolescent internalizing problems with these parenting dimensions were not significant and model comparisons indicated that removing these terms improved model fit. In the final model, the parent interpersonal sensitivity × adolescent externalizing problems (β = −0.38, $t = -2.53$, $p = 0.01$) and parent phobic anxiety × adolescent externalizing problems (β = 0.43, $t = 2.72$, $p < 0.01$) terms were moderate in size and remained significant. Table 2 presents the final model, which accounted for 33% of the variance in alcohol use frequency.

Figure 1 depicts the simple slope analysis of the parent interpersonal sensitivity × adolescent externalizing problems interaction. As noted previously, there was a main effect such that as parents' interpersonal sensitivity increased, adolescents reported lower levels of alcohol use; the simple slope analysis indicated that this main effect was driven primarily by adolescents with higher levels of externalizing problems (β = −0.02, $p = 0.29$). For adolescents with lower levels of externalizing problems, the relationship was in the opposite direction such that increased parent interpersonal sensitivity was associated with higher levels of adolescent alcohol use (β = 0.01, $p = 0.41$). The simple slope p values indicate that neither of the simple slopes were significantly different than zero; however, the significant interaction terms indicate that the slopes were significantly different from each other. In other words, the effect of interpersonal sensitivity was significantly different at high and low levels of externalizing problems.

Figure 2 depicts the simple slope analysis of the interaction between parent phobic anxiety and adolescent externalizing problems. The effect of parent phobic anxiety on the adolescent's alcohol use varied based on the adolescent's level of externalizing problems. As parent phobic anxiety increased, adolescents with higher levels of externalizing problems exhibited more alcohol use (β = 0.07, $p < 0.01$), whereas adolescents with lower externalizing problems exhibited less alcohol use (β = −0.01, $p = 0.36$). This analysis and the statistical significance of the slope at higher levels of externalizing problems suggests that the positive main effect of parent phobic anxiety on adolescent alcohol use was driven primarily by adolescents with high levels of externalizing problems.

Hierarchical Linear Regressions Predicting Marijuana Use

The second hierarchical regression examined the main and interactive effects of parent and adolescent psychiatric symptoms on adolescent marijuana use. In Step 1, there

Table 2 Hierarchical multiple regressions predicting adolescent alcohol use from adolescent and parent psychiatric symptoms

Variable	B	SE(B)	β	R^2	$R^2\Delta$
Step 1: adolescent demographics and psychiatric symptoms					
Gender	0.98	0.40	0.28*		
Internalizing	0.04	0.02	0.27*		
Externalizing	−0.001	0.02	−0.007		
				0.15	0.15*
Step 2: parent psychiatric symptoms variables					
Interpersonal sensitivity	−0.07	0.03	−0.38*		
Phobic anxiety	0.05	0.02	0.31*		
				0.22	0.07†
Step 3: parent-adolescent interactions					
Interpersonal sensitivity × Externalizing	−0.01	0.002	−0.40**		
Phobic anxiety × Externalizing	0.01	0.002	0.45*		
				0.33	0.11**

Parent psychiatric symptoms were measured using the Brief Symptom Inventory and adolescent psychiatric symptoms were measured using the Child Behavior Checklist

†$p < 0.10$; *$p < 0.05$, **$p < 0.01$

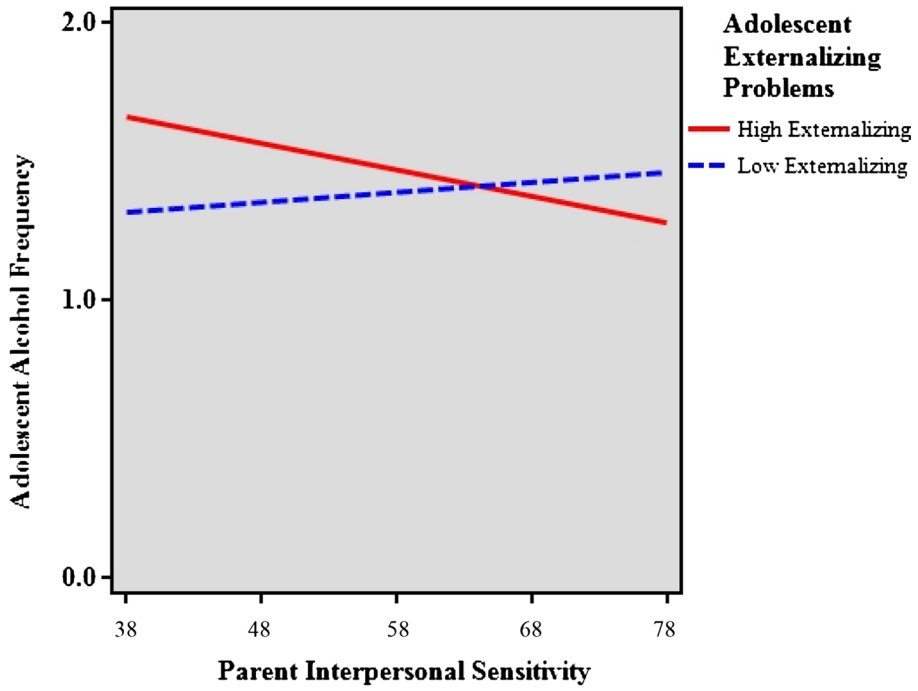

Fig. 1 Interaction between parent interpersonal sensitivity and adolescent externalizing symptoms on adolescent alcohol use. *Note*: Continuous variables were centered in the analyses but then converted back to their original scales for the purposes of visual depiction of the interaction

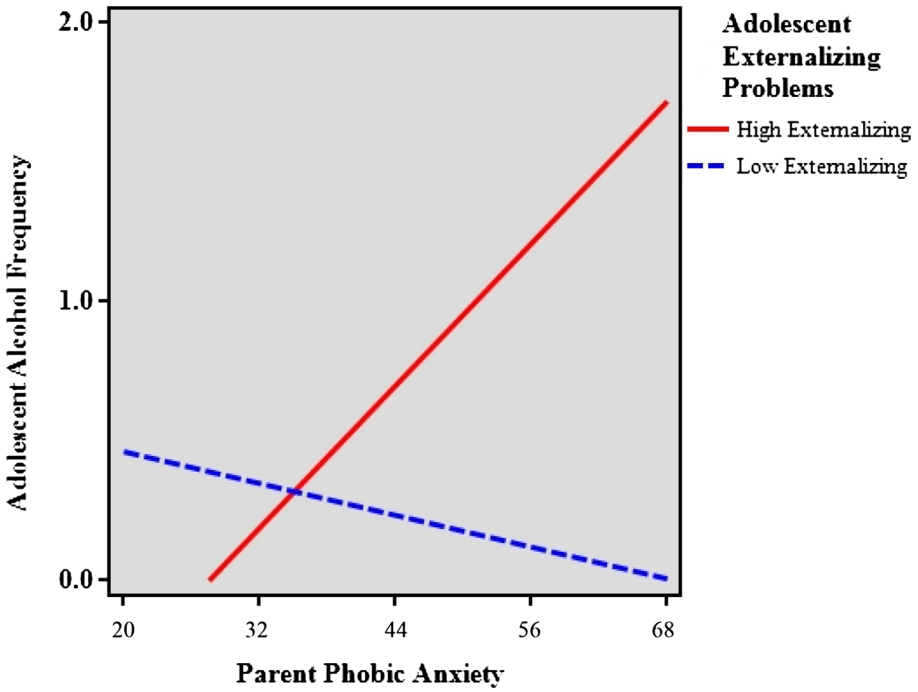

Fig. 2 Interaction between parent phobic anxiety and adolescent externalizing symptoms on adolescent alcohol use. *Note*: Continuous variables were centered in the analyses but then converted back to their original scales for the purposes of visual depiction of the interaction

were no significant main effects of gender, adolescent internalizing or externalizing problems, or parental problematic substance use. In Step 2, two of the nine parent BSI variables remained after backwards elimination and both had significant moderate size main effects on marijuana use: psychoticism ($\beta = 0.44$, $t = 2.67$, $p = 0.01$) and paranoid ideation ($\beta = -0.52$, $t = -3.59$, $p < 0.001$). These main effects indicate that increased parent scores on the BSI psychoticism subscale were associated with higher levels of adolescent marijuana use, whereas increased scores on the paranoid ideation subscale were associated with lower levels of adolescent marijuana use.

In the third and final step of the model, four possible interactions (i.e., parent psychoticism × adolescent

internalizing, parent psychoticism × adolescent externalizing, parent paranoid ideation × adolescent internalizing, parent paranoid ideation × adolescent externalizing) were entered in the model. Only the interaction between parent paranoid ideation and adolescent internalizing problems was significant (β = 0.42, $t = 2.25$, $p = 0.03$). The interactive effects of parent paranoid ideation and adolescent externalizing problems, and between parent psychoticism and both adolescent internalizing and externalizing problems were not significant; model comparisons indicated that removing these three terms improved model fit. However, with only the parent paranoid ideation and adolescent internalizing problems interaction in the third step, the interaction effect became a non-significant trend (β = 0.20, $t = 1.72$, $p = 0.09$) indicating that some shared variance with the psychoticism interactions may have contributed to this significant effect. Due to the lack of significance and small effect size, we removed the interaction from the model. Table 3 shows the final model, which accounted for 28% of the variance in frequency of marijuana use.

Discussion

The purpose of this paper was to investigate the interactive effects of parent and adolescent psychiatric symptoms on substance use among treatment-seeking adolescents in a CMHC. Due to literature showing the strong link between parental and adolescent substance use, we controlled for problematic parental substance use in the analyses predicting both adolescent marijuana and alcohol use frequency. The pattern of results differed significantly in the alcohol and marijuana analyses, suggesting important differences in the effects of parent and adolescent psychiatric symptoms on use of these substances.

Table 3 Hierarchical multiple regressions predicting marijuana use from adolescent and parent psychiatric symptoms

Variable	B	SE(B)	β	R^2	$R^2\Delta$
Step 1: adolescent demographics and psychiatric symptoms					
Gender	−0.19	0.65	−0.03		
Internalizing	0.03	0.03	0.11		
Externalizing	0.05	0.04	0.19		
				0.07	0.07
Step 2: parent psychiatric symptoms variables					
Paranoid ideation	−0.14	0.04	−0.55**		
Psychoticism	0.11	0.04	0.45**		
				0.25	0.18**
Step 3 parent-adolescent interactions					
Paranoid Ideation × Internalizing	0.005	0.003	0.20†		
				0.29	0.04†

Parent psychiatric symptoms was measured using the Brief Symptom Inventory and adolescent psychiatric symptoms was measured using the Child Behavior Checklist

*$p < 0.05$; **$p < 0.01$; † $p < 0.10$

For adolescent alcohol use, adolescent internalizing problems were significantly related to baseline frequency of use and to parent psychiatric symptoms. However, adolescent internalizing problems did not interact with any of parent symptom types, suggesting that its main effect on adolescent alcohol use was persistent regardless of level of parent symptomatology. By contrast, adolescent externalizing problems did not have a main effect on alcohol use, but did have an interactive effect with two types of parent psychiatric symptoms: parent phobic anxiety and parent interpersonal sensitivity. Combined, these results suggest that adolescent internalizing problems was a robust risk factor for adolescent alcohol use, while adolescent externalizing problems only had effects on adolescent alcohol use when in combination with specific types of parent psychiatric symptoms. These results were contrary to prior studies indicating that adolescent externalizing problems are a robust predictor of adolescent SU (e.g., King et al. 2004) and may reflect the characteristics of our adolescent sample, which had clinically significant levels of externalizing problems. High levels of adolescent externalizing problems may have resulted in ceiling effects, which could have limited our ability to detect significant associations and interactions.

One of the two significant interactions been parent and adolescent symptomatology provided partial support for our hypotheses, while the other was unexpected. We had expected parent psychiatric symptoms—and particularly those symptom types related to anxiety and depression (i.e., BSI subscales of depression, interpersonal sensitivity, anxiety, phobic anxiety, and somatization)—to interact with adolescent symptoms in a synergistic manner: in other words, we expected the combination of the two risk factors to be worse than their additive effects. The significant interaction between the parent phobic anxiety subscale (a subscale that assesses specific phobic fears of things like crowds, open spaces, public places or means of transportation), and adolescent externalizing symptoms was partially consistent with this hypothesis. Parent phobic anxiety had a significant moderately sized main effect: increased parent phobic anxiety was associated with increased adolescent alcohol use. In addition, parent phobic anxiety and adolescent externalizing problems had a negative joint effect on adolescent alcohol use (i.e., above and beyond their additive effects), but only among teens with higher levels of externalizing problems. Counter to our expectations that parental anxiety symptoms would be most detrimental for youth who also had anxiety (i.e., internalizing) problems,

this interaction suggested that parental anxiety symptoms were most detrimental for youth who were acting out. Although not expected, these findings are at least partially consistent with prior literature suggesting that parental anxiety is a risk factor for adolescent alcohol use (Merikangas et al. 1998; Mowbray and Oyserman 2003), and suggests that the main effects of parental anxiety observed in prior studies may be exist primarily in the context of specific types of adolescent and parent symptomatology.

The interactive effect of parent interpersonal sensitivity (a subscale that assesses feelings of inadequacy and uneasiness during interpersonal interactions) and adolescent externalizing problems was not expected and ran directly counter to our predictions. We hypothesized that parent and adolescent psychiatric symptoms would have a negative, synergistic effect. What we found was that parent interpersonal sensitivity had a moderately sized, significant protective effect against adolescent alcohol use in this sample. The interaction term suggested that this protective effect was attributable to adolescents with higher levels of externalizing problems. Adolescents with lower levels of externalizing problems did not experience this protective effect and, in fact, parent interpersonal sensitivity was associated with increased alcohol use. Given the lack of prior research in this area, it is difficult to conjecture why parental interpersonal sensitivity was associated with lower rates of adolescent alcohol use in this sample and why this relationship was strongest among adolescents with externalizing problems. It is well established that parent processes such as parental monitoring, discipline, and closeness to the child are protective against adolescent substance use, especially for youth with externalizing disorders (see Vakalahi 2001; Cordova et al. 2014; Marceau et al. 2015). One possible explanation of our findings is that parents who were high in interpersonal sensitivity and felt inadequate, may have had been more likely to have high levels of these parenting processes (i.e., monitoring, discipline, and closeness with the child). Our results highlight a need for future research on the effects of parental interpersonal sensitivity on adolescent SU.

For marijuana, results of our analysis revealed two significant main effects of parent psychiatric symptoms on frequency of use but no significant interactions. Consistent with our hypotheses, the parent psychoticism subscale (which assesses symptoms ranging from feelings of social alienation to unusual perceptual experiences) was associated with increased frequency of marijuana use. Put differently, adolescents whose parents reported psychotic symptoms engaged in more frequent marijuana use. This finding is congruent with prior literature suggesting that a family history of psychosis may be a risk factor for marijuana use (Power et al. 2014) and may be associated with increased sensitivity to marijuana (Kuepper et al. 2013). It is possible that those adolescents whose parents reported psychiatric symptoms were more susceptible to the beneficial effects of marijuana and were therefore likely to use more often. Additional research is needed to explore this issue.

An unexpected finding was that higher scores on the parent paranoid ideation subscale (which assesses symptoms associated with patterns of thinking such as projection, hostility, suspiciousness, centrality, and fear of loss of autonomy) were associated with lower frequency of adolescent marijuana use. In other words, we found that adolescents whose parents reported paranoid ideation engaged in less frequent marijuana use. Clinically, the phenomenon of individuals finding marijuana aversive due to paranoid ideation has been well documented on patient websites and in the popular press (Szalavitz 2011; TruthOnPot.com 2013). However, prior studies examining the relationship between adolescent marijuana use and psychosis have not teased apart the unique effects of paranoid ideation, and few studies have examined the influence of parent psychosis or paranoid ideation. A distinction between psychotic symptoms and paranoid ideation may be warranted in future research on adolescent marijuana use.

Study findings should be considered within the limitations of the sample and the design employed. The sample size was relatively small, which may have affected the ability to detect differences and interactions particularly in multivariate analyses. The fact that we found significant associations in a small sample is a testament the strength of the observed associations, but the small sample increases the likelihood that our results are not reliable. Another consideration is that this sample reported sub-clinical levels of adolescent internalizing problems and clinical levels of externalizing problems. As a result, our ability to detect both main and interactive effects of adolescent externalizing problems may have been constrained by ceiling effects. In addition, parent SU was measured categorically based on the adolescent's knowledge and report of their parent's SU. We therefore may not have fully captured the influence of parental SU on adolescent drinking and marijuana use. Lastly, results should not be generalized beyond the characteristics of the adolescents in this sample, who were predominantly White/European American, primarily used alcohol and marijuana, and who had modest levels of SU, sub-clinical levels of internalizing problems, and clinical levels of externalizing problems. Future studies are needed to determine if these findings generalize to other samples and treatment settings.

Notwithstanding these limitations, our results highlight at least two directions for future investigations. Studies would benefit from examining specific mechanisms underlying the positive associations between specific types of parent psychiatric symptoms and adolescent SU: such reasons might include parental modeling of SU, access to

substances in the home, adolescent perceptions of SU, and genetic risk. Conversely, research is needed to explore reasons why some specific parent symptom types may have protective effects on adolescent SU. Potential explanations could include dolescents' physiological response to specific substances, adolescents' beliefs of genetic risk for negative side effects of SU, and positive parenting practices.

Results of this study also have potential clinical implications. Therapists at CMHCs would likely benefit from attending to adolescents' co-occurring mental health symptoms during adolescent SU treatment; adolescent internalizing symptoms in particular appeared to be associated with more frequent alcohol use, suggesting that these symptoms should be addressed throughout the assessment and treatment process. Our results also suggest that CMHCs working with adolescent substance users might benefit from assessing and monitoring parent psychiatric symptoms. Specific types of parent anxiety and psychotic symptoms in particular may differentially interact with adolescent psychiatric symptoms to contribute to alcohol and marijuana use: parental depression and anxiety symptom types were associated with alcohol use, whereas parental psychotic symptom types were associated with marijuana use. Overall, our results are consistent with a wealth of literature indicating the value of addressing adolescent SU using a family systems' approach (see Tanner-Smith et al. 2013) and suggest that attending to both parent and adolescent psychiatric symptoms may be helpful in developing targeted treatment plans and monitoring ongoing treatment progress.

Compliance with Ethical Standards

Conflict of interest Authors have no conflicts of interest. This study was approved by the University's IRB.

References

Achenbach, T. M., & Rescorla, L. A. (2001). *ASEBA school age forms profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth & Families.

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park: Sage.

Armstrong, T. D., & Costello, E. J. (2002). Community studies on adolescent substance use, abuse, or dependence and psychiatric comorbidity. *Journal of Consulting and Clinical Psychology, 70*, 1224–1239.

Boulet, J., & Boss, M. W. (1991). Reliability and validity of the Brief Symptom Inventory. *Psychological Assessment, 3*, 433–437.

Center for Behavioral Health Statistics and Quality. (2015). Behavioral health trends in the United States: Results from the 2014 National Survey on Drug Use and Health (HHS Publication No. SMA 15-4927, NSDUH Series H-50). Rockville: Center for Behavioral Health Statistics and Quality. Retrieved from http://www.samhsa.gov/data/.

Clark, D. B., Cornelius, J., Wood, D. S., & Vanyukov, M. (2004). Psychopathology risk transmission in children of parents with substance use disorders. *American Journal of Psychiatry, 161*, 685–691.

Cordova, D., Heinze, J., Mistry, R., Hsieh, H., Stoddard, S., Salas-Wright, C. P., & Zimmerman, M. A. (2014). Family functioning and parent support trajectories and substance use and misuse among minority urban adolescents: A latent class growth analysis. *Substance Use and Misuse, 49*, 1908–1919.

Crowe, A. H., & United States, Office of Juvenile Justice and Delinquency Prevention. (1998). *Drug identification and testing in the juvenile justice system: Summary*. Washington, DC: U.S. Dept. of Justice, Office of Justice Programs, Office of Juvenile Justice and Delinquency Prevention.

Dennis, M., Godley, S. H., Diamond, G., Tims, F. M., Babor, T., Donaldson, J., & Hamilton, N. (2004). The Cannabis Youth Treatment (CYT) Study: Main findings from two randomized trials. *Journal of Substance Abuse Treatment, 27*, 197–213.

Derogatis, L. R., & Melisaratos, N. (1983). The brief symptom inventory: An introductory report. *Psychological Medicine, 13*, 596–605.

Derogatis, L.R. (1993). Brief symptom inventory: Administration, scoring and procedures manual (4th ed.). Minneapolis: NCS, Pearson, Inc.

Fergusson, D. M., & Horwood, L. (1997). Early onset cannabis use and psychosocial adjustment in young adults. *Addiction, 92*, 279–296.

Hallfors, D., Vevea, J. L., Iritani, B., Cho, H., Khatapoush, S. & Saxe, L. (2002). Truancy, grade point average, and sexual activity: A meta-analysis of risk indicators for youth substance use. *Journal of School Health, 72*, 205–211.

Henry, K. L., & Thornberry, T. P. (2010). Truancy and escalation of substance use during adolescence. *Journal of Studies on Alcohol and Drugs, 71*, 115–124.

Hogue, A., Henderson, C. E., Ozechowski, T. J., & Robbins, M. S. (2014). Evidence base on outpatient behavioral treatments for adolescent substance use: Updates and recommendations 2007–2013. *Journal of Clinical Child and Adolescent Psychology, 43*, 695–720.

Holmbeck, G. N. (2002). Post-hoc probing of significant moderational and mediational effects in studies of pediatric populations. *Journal of Pediatric Psychology, 27*, 87–96.

Hussong, A. M., Huang, W., Curran, P. J., Chassin, L., & Zucker, R. A. (2010). Parent alcoholism impacts the severity and timing of children's externalizing symptoms. *Journal of Abnormal Child Psychology, 38*, 367–380.

Hussong, A. M., Huang, W., Serrano, D., Curran, P. J., & Chassin, L. (2012). Testing whether and when parent alcoholism uniquely affects various forms of adolescent substance use. *Journal of Abnormal Child Psychology, 40*, 1265–1276.

Jessor R., Donovan J. E., & Costa F. M. (1989). *Health behavior questionnaire*. Boulder, CO: University of Colorado.

Kaminer, Y., & Bukstein, O. G. (2007). *Adolescent substance abuse: Psychiatric comorbidity and high risk behaviors*. New York: Haworth Press.

Kamon, J. L., Stanger, C., Budney, A. J., & Dumenci, L. (2006). Relations between parent and adolescent problems among adolescents presenting for family-based marijuana abuse treatment. *Drug and Alcohol Dependence, 85*, 244–254.

King, S. M., Iacono, W. G., & McGue, M. (2004). Childhood externalizing and internalizing psychopathology in the prediction of early substance use. *Addiction, 99*, 1548–1559.

Kirillova, G. P., Vanyukov, M. M., Gavaler, J. S., Pajer, K., Dunn, M., & Tarter, R. E. (2001). Substance abuse in parents and their adolescent offspring: The role of sexual maturation and

sensation seeking. *Journal of Child and Adolescent Substance Abuse, 10*, 77–89.

Kuepper, R., Ceccarini, J., Lataster, J., van Os, J., van Kroonenburgh, M., van Gerven, J. M., & Henquet, C. (2013). Delta-9-tetrahydrocannabinol-induced dopamine release as a function of psychosis risk: 18F-fallypride positron emission tomography study. *PloS ONE, 8*, e70378.

Li, C., Pentz, M. A., & Chou, C. (2002). Parental substance use as a modifier of adolescent substance use risk. *Addiction, 97*, 1537–1550.

Marceau, K., Abar, C. C., & Jackson, K. M. (2015). Parental knowledge is a contextual amplifier of associations of pubertal maturation and substance use. *Journal of Youth and Adolescence, 44*, 1720–1734.

Marmorstein, N. R., Iacono, W. G., & McGue, M. (2012). Associations between substance use disorders and major depression in parents and late adolescent–emerging adult offspring: An adoption study. *Addiction, 107*, 1965–1973.

Mason, C. H., & Perreault, W. D. (1991). Collinearity, power, and interpretation of multiple regression analysis. *Journal of Marketing Research, 28*, 268–280.

Merikangas, K. R., Stolar, M., Stevens, D. E., Goulet, J., Preisig, M. A., Fenton, B., & Rounsaville, B. J. (1998). Familial transmission of substance use disorders. *Archives of General Psychiatry, 55*, 973–979.

Mertens, J. R., Flisher, A. J., Fleming, M. F., & Weisner, C. M. (2007). Medical conditions of adolescents in alcohol and drug treatment: comparison with matched controls. *Journal of Adolescent Health, 40*, 173–179.

Miller, S. M., Siegel, J. T., Hohman, Z., & Crano, W. D. (2013). Factors mediating the association of the recency of parent's marijuana use and their adolescent children's subsequent initiation. *Psychology of Addictive Behaviors, 27*, 848–853.

Mowbray, C. T., & Oyserman, D. (2003). Substance abuse in children of parents with mental illness: Risks, resiliency, and best prevention practices. *The Journal of Primary Prevention, 23*, 451–482.

Nakamura, B. J., Ebesutani, C., Bernstein, A., & Chorpita, B. F. (2009). A psychometric analysis of the child behavior checklist DSM-oriented scales. *Journal of Psychopathology and Behavioral Assessment, 31*, 178–189.

Palamar, J. J., Fenstermaker, M., Kamboukos, D., Ompad, D. C., Cleland, C. M., & Weitzman, M. (2014). Adverse psychosocial outcomes associated with drug use among US high school seniors: A comparison of alcohol and marijuana. *The American Journal of Drug and Alcohol Abuse, 40*, 438–446.

Power, R. A., Verweij, K. J., Zuhair, M., Montgomery, G. W., Henders, A. K., Heath, A. C., & Martin, N. G., et al. (2014). Genetic predisposition to schizophrenia associated with increased use of cannabis. *Molecular Psychiatry, 19*, 1201–1204.

Shane, P. A., Jasiukaitis, P., & Green, R. S. (2003). Treatment outcomes among adolescents with substance abuse problems: The relationship between comorbidities and post-treatment substance involvement. *Evaluation and Program Planning, 26*, 393–402.

Spirito, A. (1999). Reliability data for the drug use questionnaire. (unpublished raw data available from Anthony_Spirito@brown.edu).

Substance Abuse and Mental Health Services Administration, Center for Behavioral Health Statistics and Quality (2015). *Treatment Episode Data Set (TEDS): 2003–2013. National Admissions to Substance Abuse Treatment Services*. BHSIS Series S-75, HHS Publication No. (SMA) 15-4934. Rockville: Substance Abuse and Mental Health Services Administration, Center for Behavioral Health Statistics and Quality.

Swendsen, J., Burstein, M., Case, B., Conway, K. P., Dierker, L., He, J., et al. (2012). Use and abuse of alcohol and illicit drugs in US adolescents: Results of the National Comorbidity Survey-Adolescent Supplement. *Archives of General Psychiatry, 69*, 390–398.

Szalavitz, M. (2011). Why pot smokers are paranoid. *Time*. Retrieved from http://healthland.time.com/2011/04/06/why-pot-smokers-are-paranoid/.

Tabachnick, B. G., & Fidell, L. S. (2013). *Using Multivariate Statistics* (6th edn.). Boston: Pearson.

Tanner-Smith, E. E., Jo Wilson, S., & Lipsey, M. W. (2013). The comparative effectiveness of outpatient treatment for adolescent substance abuse: A meta-analysis. *Journal of Substance Abuse Treatment, 44*, 145–158.

Tanner-Smith, E. E., & Lipsey, M. W. (2015). Brief alcohol interventions for adolescents and young adults: A systematic review and meta-analysis. *Journal of Substance Abuse Treatment*, 511–518.

TruthOnPot.com (2013). Why does weed make you paranoid? Retrieved from http://www.truthonpot.com/2013/04/23/why-does-weed-make-you-paranoid/.

Vakalahi, H. F. (2001). Adolescent substance use and family-based risk and protective factors: A literature review. *Journal of Drug Education, 31*, 29–46.

Van Meter, A., Youngstrom, E., Youngstrom, J. K., Ollendick, T., Demeter, C., & Findling, R. L. (2014). Clinical decision making about child and adolescent anxiety disorders using the Achenbach system of empirically based assessment. *Journal of Clinical Child and Adolescent Psychology, 43*, 552–565.

Waldron, H. B., & Turner, C. W. (2008). Evidence-based psychosocial treatments for adolescent substance abuse. *Journal of Clinical Child and Adolescent Psychology, 37*, 238–261.

Weaver, T., Madden, P., Charles, V., Stimson, G., Renton, A., Tyrer, P., & Paterson, S. (2003). Comorbidity of substance misuse and mental illness in community mental health and substance misuse services. *The British Journal of Psychiatry, 183*, 304–313.

Weissman, M. M., Warner, V., Wickramaratne, P., Moreau, D., & Olfson, M. (1997). Offspring of depressed parents: 10 years later. *Archives of General Psychiatry, 54*, 932–940.