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Alcoholics Anonymous attendance following 12-step treatment participation as a link between alcoholic fathers' treatment involvement and their children's externalizing problems

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Abstract

We investigated longitudinal associations between alcoholic fathers' 12-step treatment involvement and their children's internalizing and externalizing problems ($N=125$, $M_{age}=9.8\pm3.1$), testing the hypotheses that fathers' greater treatment involvement would benefit later child behavior, and that this effect would be mediated by fathers' post-treatment behaviors. The initial association was established between fathers' treatment involvement and children's externalizing problems only, while structural equation (SEM) results supported mediating hypotheses. Fathers' greater treatment involvement predicted children's lower externalizing problems 12 months later, and fathers' post-treatment behaviors mediated this association: greater treatment involvement predicted greater post-treatment Alcoholics Anonymous (AA) attendance, which in turn predicted greater abstinence. Finally, fathers' abstinence was associated with lower externalizing problems in children. Theoretical and practical implications of these findings are discussed.

Keywords

Children of alcoholics (COA); alcoholism treatment; Alcoholics Anonymous (AA); Structural Equation Modeling (SEM)

Current epidemiological studies estimate that more than 25% of all children in the US live in families marked by alcohol abuse or dependence (Grant, 2000), placing them at risk for a multitude of adverse developmental outcomes (Chassin, Pitts, DeLucia, & Todd, 1999; Sher et al., 1991; West & Prinz, 1987), and defining children of alcoholics (COA) as a high-risk group of major public health (Grant, 2000) and clinical concern (Rydellius, 1997). Indeed, there is a call for more comprehensive intervention and prevention efforts aimed at this at-risk population (Grant, 2000; SAMHSA, 2003). However, given that substance-abusing parents are generally unwilling to permit their children to participate in any form of treatment (Fals-

⁸We used a first-order delta method to compute the standard error for a sequential indirect effect involving two mediating variables and

three mediating paths (vs. traditional models with one mediator and two paths): $se_{abc} = \sqrt{b^2 c^2 se_a^2 + a^2 c^2 se_b^2 + a^2 b^2 se_c^2}$, where a , b , and c are unstandardized mediating path coefficients, and se_a , se_b , and se_c are corresponding standard errors. The asymptotic significance test was an extension of the standard Sobel test (1982) applied to the case involving 3 sequential mediating paths;

$z = \frac{abc}{se_{abc}}$, wherein values of $z > 1.96$ are statistically significant at $p < .05$. Note however that this test is rather conservative and that other, less stringent methods have recently become available for computation of standard error for mediated indirect effects (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002).

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Stewart, Fincham, & Kelley, 2004), it may be imperative to explore other less direct means when targeting COA. A promising first step in this direction may be to examine whether COA would indirectly benefit from their alcoholic parents' treatment participation, which would presumably alleviate the child's risk status as the severity of parental alcoholism decreases (O'Farrell & Feehan, 1999). This study aimed to explore these questions by examining longitudinal processes linking COA outcomes with their fathers' treatment and post-treatment characteristics. In short, we sought to find out whether treatment participation of alcoholic fathers may have a beneficial effect on their children's outcomes, and if so, to better understand the post-treatment processes through which this benefit occurs.

Parental alcoholism as a risk factor for child behavioral outcomes

Developmental psychopathology theories have postulated parental substance abuse as a risk factor for children, as COA generally have greater maladjustment rates than children from general population or children from non-COA samples (West & Prinz, 1987). This is especially true of externalizing behaviors, such as aggression and delinquency (Loukas, Zucker, Fitzgerald, & Krull, 2003; Merikangas, Dierker, Szamari, 1998; Obot & Anthony, 2004; Sher et al., 1991), which have been a somewhat of a marker for sons of alcoholic men. Even though internalizing behaviors, such as anxiety and depression have also been investigated among COA, it is less clear that parental alcoholism represents a specific risk for such types of problems. For example, even though parental drinking was found to be associated with greater internalizing problems within COA samples (Chassin, Pitts, DeLucia, & Todd, 1999; El-Sheikh & Flanagan, 2001; Kuperman, Schlosser, Lidral & Reich, 1999), several reports found no differences between COA and non-COA on such symptoms (Kelley & Fals-Stewart, 2004; Merikangas et al., 1998; Obot & Anthony, 2004; Ohannessian et al., 2004). Some researchers suggest that parental mental health problems other than or comorbid with alcoholism may be the primary risk for internalizing symptoms in COA (El-Sheikh & Flanagan, 2001; Merikangas et al., 1998; Ohannessian et al., 2004). Given that COA risk may vary for internalizing and externalizing problems, these two aspects of child outcomes were examined separately in the present study.

Children of alcoholic parents in treatment

Despite the body of empirical evidence documenting a range of difficulties in COA, relatively little is known about the COA whose parents are receiving treatment to eliminate or reduce their drinking problems, even though parental treatment involvement may be one of the most logical, albeit indirect, interventions for COA (O'Farrell & Feehan, 1999). A handful of studies investigating this question reported encouraging results of significant improvements in COA following their fathers' treatment participation and subsequent reduced drinking. Moos and Billings (1982) reported that COA whose fathers were stably remitted for two years were comparable to non-COA in terms of emotional and physical health. Similar results were obtained by Burdzovic Andreas, O'Farrell and Fals-Stewart (2006), where 12 months after fathers' alcoholism treatment COA were found to have significantly improved and to have overall and clinical-level psychosocial problems comparable to children from the general population. Like Moos and Billings (1982), Burdzovic Andreas et al. (2006) and Callan and Jackson (1986) reported better psychosocial outcomes in children of stably recovered, as opposed to children of relapsed alcoholics. Even though they did not utilize a non-COA comparison sample, Kelley and Fals-Stewart (2002) also observed significant improvements in COA following their fathers' alcoholism treatment.

In general, these studies reported positive results that COA functioning significantly improved following their fathers' treatment participation. However, the possible differences in nature and extent of fathers' treatment were not examined in relation to COA outcomes, with the

exception of Kelley and Fals-Stewart (2002) who compared outcomes in COA whose alcoholic fathers attended one of three different types of treatment: behavioral couples therapy, individual-based treatment, or couples-based psychoeducational control treatment. No study investigated possible differences in COA adjustment as a function of variations in the amount of treatment received by alcoholic fathers. In other words, it is not known whether differences in the extent of fathers' treatment are important for later child outcomes.

Further, the implied assumption in these studies of COA with parents in treatment was that the COA "risk" status would be lessened as a result of treatment-related positive changes in parents (O'Farrell & Feehan, 1999). However, these hypotheses were usually investigated in the most basic way, by examining parental treatment outcomes as a dichotomous construct and by comparing children of relapsed and remitted fathers. When fathers' post-treatment drinking problems were accounted for in a non-dichotomous way, they were found to predict child outcomes (Burdzovic Andreas & O'Farrell, 2007; Moos & Billings, 1982; Kelley & Fals-Stewart, 2002), but whether they linked fathers' initial treatment with children's adjustment was not investigated. In short, most prior studies did not examine post-treatment drinking in fathers as a complex construct of a varying degree, nor did they explicitly test for how such problems mediate the relationship between fathers' initial treatment and later child outcomes. Thus, the chain of events and behaviors linking COA functioning to their fathers' alcoholism treatment involvement remains relatively unidentified.

Rationale for the current study

Attempting to bridge these gaps, we focused on fathers' post-treatment behaviors because prior research has identified such behaviors as having an important impact on drinking outcomes in alcohol-dependent men. As recent studies reported that greater involvement in and affiliation with self-help groups such as AA strongly contributes to maintenance of positive therapy gains in alcohol-dependent men, we were interested in fathers' post-treatment Alcoholics Anonymous (AA)¹ attendance as a potential link connecting the initial professional treatment with longitudinal outcomes in both these alcoholic fathers and their children.

For example, Montgomery, Miller, and Tonigan (1995) reported lower alcohol consumption at 6-month follow-up in male patients who had greater AA involvement after participating in a 28-day treatment program. Similar 6-month follow-up results were reported by Fiorentine (1999), who reported greater abstinence rates in those patients who attended AA with greater frequency following professional treatment, even after controlling for possible confounds such as motivation to change. Identical results were obtained in a sample of alcoholic patients who received intensive 30-day alcoholism treatment, wherein patients who had greater post-treatment AA affiliation also had lower drinking rates at 1- and 6-month follow-ups, after controlling for their pre-treatment alcoholism severity, commitment to abstinence, and self-efficacy (Morgenstern et al., 1997). Further, McCrady, Epstein, and Kahler (2004) reported that AA attendance prospectively predicted abstinence rates in alcoholics who received different types of treatment across five 3-month follow-ups, but that the reverse was not the case (i.e., abstinence did not prospectively predict AA attendance). The longitudinal effects of post-treatment AA attendance held even after drinking from previous assessments was statistically accounted for. In sum, there is a compelling evidence for AA being a beneficial after-treatment resource for alcohol-dependent men, contributing to greater abstinence.

Thus, we aimed to both replicate and extend these previously established associations between treatment involvement, AA attendance, and post-treatment outcomes in alcohol-dependent

¹AA is a 12-step oriented voluntary fellowship concerned with the recovery and continued abstinence of the alcoholics who turn to it for help. AA advocates the disease model of alcoholism. (Alcoholics Anonymous, 1976)

men. While we expected to replicate prior findings of post-treatment AA attendance aiding in the recovery process and leading to greater abstinence rates, we were much more interested in extending these models to see whether they could be linked to behavioral outcomes in children of these alcoholic men. In short, the aim of this study was to examine the longitudinal association between COA outcomes and the extent of their fathers' alcoholism treatment. We first asked, is there a benefit of fathers' greater treatment involvement on their children's behavioral outcomes one year later? If so, we next asked, what would account for such a beneficial relationship? We hypothesized that this "secondary" treatment benefit for the COA would be best explained by a sequence of alcoholic fathers' post-treatment behaviors, including their AA attendance and abstinence. To our knowledge, this is the first study to extend the known models of alcoholism treatment, AA attendance, and post-treatment drinking outcomes in alcoholic men to include their children's outcomes as well.

The present study used a sample of 125 alcoholic men and their children followed for 12 months after fathers' alcoholism treatment. Our prior reports on this sample have shown greater overall and clinical-level problems among children of relapsed than remitted alcoholic fathers (Burdzovic Andreas et al., 2006) and temporal associations between alcoholic fathers' heavy drinking patterns and overall, externalizing, internalizing, and clinical-level problems in their children (Burdzovic Andreas & O'Farrell, 2007). These prior reports documented general relations between drinking problems in alcoholic fathers and behavioral outcomes in their children, but did not explore any mediating mechanisms. This is the first report using these data to address the questions of putative mechanisms and explanatory pathways, linking alcoholic fathers' treatment involvement and post-treatment outcomes with their children's outcomes over time.

Method

Sample

Participants were selected from a sample of 301 married or cohabiting men entering outpatient treatment at either of two alcoholism clinics in the northeastern US mainly serving patients from nearby small town or rural areas.² Data about parents and children were collected as part of an overall evaluation of program services. Men seeking treatment were eligible for the study if they met these criteria: (a) between 20 and 60 years of age; (b) married for at least 1 year or living with a female partner for at least 2 years; (c) current alcohol abuse or alcohol dependence diagnosis according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994); and (d) neither the male patient or the female partner met *DSM-IV* criteria for a severe mental illness (e.g., schizophrenia or other psychotic disorder).

From these 301 couples, 144 had children ages 4 to 16 residing at home, which was an additional selection requirement for the present study of child outcomes. Of these 144 families, 125 (87%) completed both the baseline and a follow-up assessment 12 months later and were included in the longitudinal analyses reported here.³ For families with multiple children in the age range, one child was randomly selected as the target child, to guard against the violation of statistical assumptions regarding independent observations. All of the children were

²Earlier reports based on this sample have contained data on partner violence (O'Farrell et al., 2003), on behavioral differences among children of relapsed and remitted alcoholics (Burdzovic Andreas et al., 2006), and on temporal associations between heavy drinking patterns in alcoholic fathers and their children's problem behavior patterns (Burdzovic Andreas & O'Farrell, 2007). These articles did not examine the nature or extent of fathers' treatment involvement or its relation to child outcomes, as they focused primarily on post-treatment outcomes. Further, our prior reports did not address the questions examined in this report regarding the associations between behavioral problems in children and treatment and post-treatment characteristics of their alcoholic fathers; specifically, the extent of fathers' treatment participation, AA attendance and abstinence rates.

biological children of the female participants, but 26 children (20.8%) were stepchildren of the alcoholic male.

Each couple was interviewed separately with the substance use modules of the Structured Clinical Interview for *DSM-IV* (SCID; First, Spitzer, Gibbon, & Williams, 1995), administered by one of two master's-level interviewers. Most male patients (i.e., fathers) from this sample had a *DSM-IV* current alcohol dependence diagnosis (94%), and 6% had alcohol abuse diagnosis. Ten fathers (8%) also had a comorbid *DSM-IV* drug abuse or dependence diagnosis, with 4 patients each receiving a SCID diagnosis for marijuana, cocaine, and heroin (note that 2 patients were positive for two substances). On average, these alcohol-dependent men reported being abstinent less than 25% of the time in the year before treatment entry ($M = 23.2\%$, $SD = 19.3$). They also reported drinking problem of many years duration ($M = 13.9$, $SD = 6.9$), and had elevated scores on measures of drinking problems such as the Michigan Alcoholism Screening Test (MAST; Selzer, 1971; $M = 31.0$, $SD = 10.2$) and the Alcohol Dependence Scale (ADS; Skinner & Allen, 1982; $M = 18.3$, $SD = 1.8$). Only 16% of the mothers were identified though the SCID as having any *DSM-IV* alcohol-related diagnosis at baseline (with 7.2% having current alcohol abuse and 8.8% alcohol dependence).

Alcoholic fathers on average had a high school education, were 40 years of age ($M = 40.4$, $SD = 7.3$) with annual family income of about \$30,000 ($M = \$31,180$, $SD = 11,476$). Most of the children were Caucasian (81%), and nearly 60% were boys. At first assessment, children were between the ages of 5 to 16, and averaged nearly 10 years old ($M = 9.8$, $SD = 3.1$). Majority of children were in middle childhood or pre-adolescence (ages 7–13; 64%), with some in early childhood (ages 5–6; 22%) and adolescence (ages 14–16; 14%). Burdzovic Andreas et al. (2006) and Burdzovic Andreas & O'Farrell (2007) provide additional demographics for this sample of alcohol-dependent men, their families and children, while O'Farrell, Fals-Stewart, Murphy, & Murphy (2003) describe the general recruiting and sampling procedures in more detail.

Procedures

Alcoholic fathers' initial treatment—The treatment program at the two alcoholism clinics consisted of 26 planned sessions including an intake assessment, a physical examination, 8 individual therapy sessions, and 16 group therapy sessions over a 12-week period. For the 125 alcoholic patients studied, the average number of treatment sessions received was 20 ($SD = 7.0$). Over two-thirds of the patients (i.e., 88/125) completed at least the minimum number of treatment sessions needed to be considered by the clinics' policy as having been meaningfully engaged in treatment (i.e., 4 individual therapy sessions and 12 group therapy sessions).

Eleven state-certified alcoholism counselors treated patients in this study. Therapists' responses on the Drug and Alcohol Program Treatment Inventory (DAPTI; Swindle, Peterson, Paradise, & Moos, 1995) showed that therapists perceived their program as being (a) characterized most strongly by an AA/12-Step orientation ($M = 24.0$, $SD = 0.0$, indicating that each therapist gave the highest possible rating to each of 8 DAPTI items reflecting an AA/12-Step orientation) and (b) not strongly characterized by 7 other orientations (e.g., marital-family systems, medical, cognitive-behavioral).⁴ The DAPTI measures staff perceptions of a

³Children and families who did not complete the study ($n = 19$) were similar to the rest of the sample at study entry. They did not differ in terms of child gender, stepchild status, number of siblings, or the baseline PSC scores (all p 's $> .20$), nor on parental race, education, marital status, family income, or the fathers' baseline alcoholism indicators (PDA at baseline, MAST or ADS scores, all p 's $> .40$). They also did not differ in terms of alcohol-related *DSM-IV* diagnosis for mothers, but not a single father from the drop-out group out had a comorbid *DSM-IV* drug diagnosis ($p < .001$). However, both children and parents from dropout families were younger than the rest of the sample ($p < .01$), leading to the related findings of dropout fathers having shorter history of alcohol-related problems at treatment entry ($p = .06$), and being in a relationship for a somewhat shorter period of time ($p = .13$). We believe that these 19 cases were lost to random attrition, but whether (and if so, how) this modest attrition would impact our findings is unknown.

program's emphasis on 8 treatment orientations; it has shown promising subscale internal consistency, and discriminant and concurrent validity (Swindle et al, 1995). In addition to the treatment program, these alcoholic patients also had the opportunity and were strongly encouraged to participate in local AA meetings. During their post-treatment interview, these alcoholic patients reported attending (in addition to their regular treatment program sessions) an average of 38.5 ($SD = 15.8$) AA meetings during the 12-week treatment period.

Baseline and follow-up data collection—The Timeline Follow-Back interview (TLFB; Sobell & Sobell, 1996), other drinking adjustment measures, and the Pediatric Symptom Checklist (PSC, Jellinek et al., 1999) measure of child adjustment problems were collected before (baseline) and after the 12-week treatment program (post-treatment assessment). TLFB retrospective daily reports of substance use show correlations $\geq .80$ for test-retest reliability over a 2-week interval and for concurrent validity (i.e., agreement between patient and collateral reports) for alcohol (Sobell & Sobell, 1996) and for illicit drugs including opioids, cocaine, and benzodiazepines (Fals-Stewart, O'Farrell, Rutigliano, Freitas, & McFarlin, 2000). In this sample, partner-patient collaterals for the primary drinking measure were all above .70 (all p 's $> .001$) as previously reported in O'Farrell et al. (2003). In the year after treatment, the TLFB and other drinking measures were collected quarterly and the PSC was collected at 6 and 12 months (M6 and M12) after the end of treatment. In this report, only baseline and M12 PSC scores were used.

Measures

COA Behavioral Outcomes—Mothers completed the 35-item Pediatric Symptom Checklist (PSC; Jellinek et al., 1999) about their child. The PSC was designed as a screening test that allows the medical or mental health practitioner to identify psychosocial problems in children ages 4 to 16 as rated by the child's parent. The sensitivity and specificity of the PSC is comparable to the Children's Global Assessment Scale and the Child Behavior Checklist (e.g., Murphy et al., 1992; Simonian & Tarnowski, 2001; Walker et al., 1989). In addition, format and conceptualization of PSC are similar to the commonly used Child Behavior Checklist (CBCL; Achenbach, 1991), as both the CBCL and PSC instruments (a) capture frequencies of various symptoms (such as "Fights with others", "Worries a lot", etc.) on a three-point scale (where 0 = never, 1 = sometimes, and 2 = often), and (b) differentiate children's externalizing and internalizing problems on separate subscales.

Externalizing and Internalizing PSC subscales have been previously empirically derived through factor analysis (Gardner et al., 1999), and are computed as a sum of, respectively, 7 and 5 relevant PSC items. Higher subscale scores indicate greater behavioral problems in a given domain. Both types of behavioral problems were considered in this report, and both utilized PSC subscales had high internal consistency (PSC Externalizing Cronbach's $\alpha_{\text{Baseline}} = .91$ and $\alpha_{\text{M12}} = .89$; PSC Internalizing, $\alpha_{\text{Baseline}} = .80$ and $\alpha_{\text{M12}} = .77$).

Fathers' initial treatment involvement—Fathers' initial treatment involvement was a sum of all treatment sessions and all AA meetings attended during the 12-week treatment period, and this variable was used in all further analyses. Because the treatment program had a strong AA/12-Step orientation, combining these two indicators (i.e., the number of treatment sessions which averaged 20.0 ($SD = 7.0$) and AA meetings attended which averaged 38.5 ($SD = 15.8$)) provided a meaningful measure of alcoholic fathers' initial treatment involvement. All fathers attended AA while in treatment (i.e., there were no alcoholic patients who received treatment sessions only). Average initial treatment involvement for these alcoholic fathers was 58.5 ($SD = 17.4$) sessions.

⁴For brevity purposes, the term 'treatment' in further text should be understood as the '12-step oriented treatment'.

Alcoholic fathers' post-treatment outcomes—In the year following treatment, two main outcomes were assessed for these alcoholic fathers: (a) continued AA attendance, (b) abstinence.

- a. As part of quarterly TLFB interviews, fathers reported the number of days on which they attended AA meetings (Breslin, Borsol, Cunningham, & Koski-Jännes, 2001). TLFB quarterly reports were summed to obtain the annual number of days on which the patients attended AA, and this measure of AA attendance was used in further analyses.⁵ In the year following treatment participation, these alcohol-dependent men reported an average of 81.1 days ($SD = 98.3$) on which they attended an AA meeting, while 45 (36%) reported no post-treatment AA attendance. Whereas these alcoholic men sought and/or attended any alternative post-treatment resources is not known.
- b. As part of quarterly TLFB interview, both fathers and mothers also reported the number of days on which the father drank alcohol, drank heavily (i.e., > 6 standard drinks), or was abstinent. TLFB quarterly reports were summed to obtain an annual indicator for each father. Because the primary goal of AA is to promote abstinence, the number of abstinent days in the year following treatment was selected as the primary outcome, and was based on fathers' self-report. This count was re-coded into a commonly used measure of percent days abstinent (PDA) within a given period. In the 12-months following treatment, the fathers from this sample had a PDA average of 79.7%.

Control variables—A set of control variables included: (a) alcoholic fathers' drinking problems at treatment entry measured by PDA for year before treatment, number of years of problematic alcohol use, and scores on two standard measures of drinking problems: the Alcohol Dependence Scale (ADS, Skinner & Allen, 1982) and Michigan Alcoholism Screening Test (MAST, Selzer, 1971); (b) presence of any *DSM-IV* alcohol-related diagnoses in mothers; and (c) presence of any *DSM-IV* drug related diagnosis in alcoholic fathers.

Data Analysis Plan

In our analyses, we drew heavily upon the causal chain analysis approach by examining the mediation sequence associated with treatment participation and post-treatment outcomes in alcohol-dependent men (Longabaugh & Wirtz, 2001). However, we modified and extended this general approach to include and examine behavioral outcomes in children of alcohol-dependent fathers as well. Our starting hypothesis was that the extent of alcoholic fathers' initial treatment involvement would be associated with the extent of their children's behavioral problems one year later, such that internalizing and externalizing behavioral problems would be lower in children of fathers with greater initial treatment involvement. We further hypothesized that this association would largely be explained by fathers' post-treatment behavior. We proposed a causal chain model, in which alcoholic fathers' treatment involvement predicts their post-treatment AA attendance, which, in turn, predicts post-treatment abstinence. Finally, we hypothesized that fathers' abstinence would predict their children's behavior at final assessment, even after controlling for the initial levels of child problems, child demographic, and measures of parental substance use problems at baseline.

These longitudinal hypotheses can appropriately be tested through the Structural Equation Modeling (SEM, Bollen, 1989) and mediation approach (Baron & Kenny, 1986; Cole & Maxwell, 2003; Longabaugh & Wirtz, 2001). The main advantage of SEM over traditional regression is its ability to model "latent" (or "true") scores, as opposed to not fully reliable

⁵Note that even though quarterly assessments of AA participation bolstered recall and validity of these self-reports, unfortunately only the total annual counts were made available in the data set, precluding examination of more complex longitudinal models.

observed measures only (Bollen, 1989; Jöreskog & Sörbom, 1996). This is possible because SEM can account for measurement error, usually by using multiple indicators of a single latent construct, or by disattenuation of measurement error from a single scale. In addition, SEM can model complex relations among these latent constructs, and it can test how well such models fit the data, none of which is possible in more standard regression models. All structural models reported in this study were estimated using LISREL 8.2 software (Jöreskog & Sörbom, 1996) and maximum likelihood estimates. Prior levels of primary dependent variable (i.e., child adjustment problems) and other conceptually important variables (i.e., extent of fathers' pre-treatment drinking problems) were accounted for in the hypothesized theoretical model (Cole & Maxwell, 2003), shown in Figure 1. One model for child internalizing, and one for externalizing adjustment problems was hypothesized.

Results

Descriptive Analyses

Over the 15-month study period, COA from our sample experienced significant reductions in their behavioral problems. PSC Externalizing scores were significantly reduced from baseline ($M = 3.7$, $SD = 4.5$) to M12 assessment ($M = 2.1$, $SD = 3.3$), paired $t(124) = 5.6$, $p < .001$, $ES_r = .198$. Similarly, COA's PSC Internalizing scores were significantly reduced from baseline ($M = 2.1$, $SD = 2.6$) to M12 assessment ($M = 1.2$, $SD = 1.9$), paired $t(124) = 4.6$, $p < .001$, $ES_r = .19$.

Preliminary analyses testing the association between the extent of fathers' treatment involvement and their children's outcomes 12 months later, showed this association for externalizing, but not internalizing problems; $r = -.26$, $p = .004$ for PSC Externalizing, and $r = -.03$, $p = .73$ (*ns*) for PSC Internalizing subscale. In other words, fathers who reported greater initial treatment involvement had children with lower externalizing problems 12 months later. Thus, only this type of problems is considered in subsequent analyses, and the hypothesized model (Figure 1) is applied only to externalizing problems. Partial correlation between fathers' treatment and PSC Externalizing at M12, accounting for PSC Externalizing at baseline was $r = -.19$, $p = .038$.

Table 1 shows means, standard deviations, and zero-order correlations among observed variables of interest, including (a) *child demographic characteristics* (race, gender, and age), (b) *child behavioral outcomes* (i.e., child externalizing problems at baseline and M12 follow-up), (c) *the extent of fathers' initial treatment involvement* (i.e., number of treatment program sessions + number of AA meetings attended during the 12-week treatment period), (d) *fathers' post-treatment outcomes*, including continued AA attendance and PDA for the year following treatment, and (e) *control variables*, including fathers' drinking problems at treatment entry (i.e., PDA for year before treatment, years of problematic use, ADS, and MAST), presence of any *DSM-IV* alcohol-related diagnoses in mothers, and presence of any *DSM-IV* drug related diagnosis in fathers.

Note that of demographic variables, only gender was significantly associated with M12 outcome. Therefore, child's age⁶ and race were omitted from the model. Similarly, all of the baseline control variables except for presence of drug-related diagnosis in fathers, were omitted from the model, as none of them was significantly related to the M12 PSC Externalizing scores (see Table 1). Even though some of the variables were skewed, the models were fitted without transformations.⁷

Structural Equation Modeling - Model Specifications

Model variables were specified as shown in Figure 1. Because fathers' initial treatment involvement, post-treatment AA attendance, and PDA were based on simple counts, these variables were entered as observed variables to the SEM model (i.e., error variances set to 0). Next, child externalizing problems at baseline and M12 were included in the SEM model as latent constructs, created by disattenuation of measurement error from the PSC Externalizing scales at each time (cf. Jöreskog & Sörbom, 1996, *p.* 37). This measure had excellent reliability at both times ($\alpha_{\text{Baseline}} = .91$ and $\alpha_{\text{M12}} = .89$). Corresponding error variances were computed for each assessment [i.e., $(1-\alpha) * \sigma^2$] and specified as such in the SEM model. Because child gender and fathers' baseline drug-related diagnosis were the only control variable correlated with the M12 child outcome, they were included in the model as observed indicators as well.

SEM Results

Figure 2 shows a graphic representation of the completely standardized solution, in which the longitudinal association between the alcoholic fathers' initial treatment involvement and their children's externalizing problems one year later was hypothesized to be mediated by the sequence of fathers' AA attendance and abstinence rates in the year following treatment. The predictors in the model accounted for 73% of the variance in our outcome of interest (i.e., in COA's Externalizing problems at M12 follow-up). This model displayed an excellent fit with the data, as shown by the non-significant χ^2 value ($\chi^2 = 10.03$, $df = 8$, $p = .26$); the RMSEA value lower than the recommended cut-off of .06 (RMSEA = .04); and the general fit indices values exceeding the recommended cut-off of .90 (GFI = .98; AGFI = .92). These results were additionally evaluated following the guidelines for probing of mediation effects in general (Holmbeck, 1997, 2002) and in SEM longitudinal models in particular (Cole & Maxwell, 2003). These guidelines and recommendations include: (1) test of direct paths; (2) test of omitted paths; and (3) Sobel test for significance of mediation path.

⁶Even though we found no significant associations of children's age with the main outcomes (see Table 1) we were concerned that the effect of the developmental stage was present but that it was possibly non-linear or limited to a smaller subgroup of children (such as adolescents, for example). To investigate this possibility, we first visually examined the scatterplot of M12 PSC Externalizing Problems as a function of children's chronological age. These graphs reiterated the lack of associations between children's age and their externalizing problems, as no trends (linear or non-linear) were observed. Next, we conducted a one-way ANOVA examining externalizing symptoms as a function of 4 developmentally relevant categories: (1) 'early childhood' (ages 5–6; $n = 28$), (2) 'middle childhood' (ages 7–10; $n = 43$), (3) 'preadolescence' (ages 11–13; $n = 37$), and (4) 'adolescence' (ages 14–16; $n = 17$). These results also revealed no significant differences in externalizing behaviors between COA from these 4 developmental stages, $F(3, 121) = .72$ (*ns*). Identical non-significant results were obtained when COA were categorized into 2 general age categories – 'younger' (ages 5–10; $n = 71$) and 'older' (ages 11–16; $n = 54$), $t(123) = .80$ (*ns*).

Finally, we replicated the correlation matrix shown in Table 1 separately for 'younger' ($n = 71$) and 'older' ($n = 54$) COA. Even though some significance levels were attenuated due to the reduction in sample size, the main associations of interest remained identical to those from the entire sample and did not appear to differ significantly across age groups. Most importantly, the initial link between fathers' treatment involvement and children's outcomes was similar across children's age groups, such that both younger ($r = -.28$, $p = .02$) and older children ($r = -.22$, $p = .10$) of fathers who had greater treatment involvement had lower externalizing problems 12 months later. Further, associations between fathers' initial treatment involvement and post-treatment AA attendance also were similar for both younger ($r = .35$, $p < .01$) and older ($r = .23$, $p = .09$) children, as were the associations between fathers' AA attendance and their M12 PDA ($r = .21$, $p = .08$ for younger, and $r = .35$, $p < .01$ for older children). Finally, greater PDA in fathers in the year following treatment was associated with lower externalizing problems in both younger ($r = -.30$, $p = .01$) and older ($r = -.49$, $p < .001$) children at M12. In short, our set of exploratory analyses revealed no significant age-related trends or differences across younger and older children.

This lack of anticipated age differences was most likely due to the majority of COA from our sample being in middle childhood or pre-adolescence (65%), which made this sample more developmentally homogenous. Because these exploratory analyses revealed no age effects and because there were no large variations in age, children's age was not considered in further analyses and structural models. How these associations and models would have differed in COA samples with majority of much older or much younger children is not known.

⁷Decision to retain model variables in their original format was based on the set of preliminary analyses revealing that both ln and square root transformations not only did not improve but actually worsened the skew of some variables. Specifically, "fathers' PDA for the year following treatment" showed worse skew with these transformations than in its original form (i.e., $-.91$ vs. -1.32 with square root, and $-.91$ vs. -1.88 with ln transformation). While the associations among remaining variables remained similar regardless of whether the original or transformed format was used, the correlations with the "post-tx PDA" variable worsened when the transformed versions of this variable were used. In order to preserve clarity and interpretation of results, we decided to keep all variables in their original format, as the transformations either did not significantly improve (or have actually worsened) associations among model variables.

First, there was a significant association between the fathers' initial treatment involvement and child outcomes, such that children of fathers who had greater treatment involvement had lower externalizing problems one year later ($r = -.26, p = .004$; see Table 1). This significant zero-order association was replicated in the measurement model (in which all structural constructs were allowed to covary freely), and was additionally strengthened as would be expected due to the disattenuation of measurement error, $r = -.28, p < .001$.

Second, this association was weakened to $\gamma = -.12, p = .065$ after a causal sequence of mediating paths was added to the model (i.e., fathers' AA attendance and PDA in the year following treatment, see Figure 2). These results suggest that the initial link between fathers' treatment and subsequent child outcomes was to a large extent accounted for by this mediating sequence. These conclusions were additionally supported by the results from a trimmed model ($\chi^2 = 13.47, df = 9, p = .14$; RMSEA = .06; GFI = .97; AGFI = .91), in which the direct path from fathers' initial treatment to M12 child outcome was omitted. Comparison between the original and trimmed model supported full mediation, as the addition of the direct path from fathers' treatment to child M12 adjustment would not significantly improve the model fit, $\Delta\chi^2 = 3.44, df = 1, p = .064$ (Holmbeck, 1997).

Finally, as shown in Figure 2, all three paths in the mediating sequence were statistically significant. As hypothesized, fathers' greater initial treatment involvement predicted their greater AA attendance in the year to follow ($\gamma = .29, p < .001$). Next, greater annual AA attendance was associated with greater PDA during the same period ($\beta = .26, p < .01$). Finally, fathers' abstinence predicted child outcomes, such that fathers with greater annual PDA had children with lower externalizing problems at M12 ($\beta = -.25, p < .01$). These paths remained significant even though our SEM model controlled for possible direct effects of child externalizing problems at baseline, fathers' drug diagnosis, and child gender ($\gamma = .76, p < .001$; $\gamma = .12, p = .05$; and $\gamma = -.02, ns$, respectively).

We estimated the magnitude of the sequential indirect effect (by multiplying the three unstandardized paths in question), and tested its significance (by dividing it by its standard error) using an extension of the traditional Sobel test (1982).⁷ The obtained value was $z = -1.966, p = .05$, indicating a statistically significant mediating effect accounting for 90% of the total effect of fathers' treatment participation on child externalizing problems.⁹

Probing Longitudinal Effects

Even though our hypothesized model showed an excellent fit with the data according to all indicators, the question remains about the directionality of effects and of the causal chain sequence, especially between concurrently measured post-treatment AA attendance and PDA. Ideally, this question would be addressed with a multi-wave panel model reflecting true longitudinal sequence (cf. Cole & Maxwell, 2003). However, given the nature of our data set, in which post-treatment AA attendance was available only as an annual count, this was not possible. Instead, the question of causal ordering was addressed by two different approaches: (a) fitting of an additional SEM model in which all mediators remained identical to our hypothesized model (Figure 2) but the ordering of fathers' AA attendance and PDA in the year following treatment was reversed, and (b) fitting of an additional SEM model in which fathers' annual PDA was replaced by the PDA for the last quarter (i.e., reflecting PDA only for the last 90 days of the study period, $M = 72.4, SD = 28.0$).

⁹Computed as $1 - \frac{abc}{d}$, where a, b , and c are mediating path coefficients and d is the total effect of fathers' treatment on M12 child adjustment (<http://davidakenny.net/cm/mediate.htm#IE>).

(a) Reversed ordering model—The results indicated that this model had an exceptionally poor fit with the data, as shown by the statistically significant χ^2 value ($\chi^2 = 35.52$, $df = 8$, $p < .001$); high RMSEA (RMSEA = .16); and low general indices (GFI = .93; AGFI = .75). Thus, a better solution was provided by the originally hypothesized model, in which the longitudinal association between fathers' initial treatment involvement and child problems 12 months later was explained by the sequence of fathers' post-treatment behaviors: their AA attendance and PDA, in this specific order.

(b) M12 PDA model—The results indicated that this model fit the data well, as indicated by a non-significant χ^2 value ($\chi^2 = 10.22$, $df = 8$, $p = .25$); the low RMSEA (RMSEA = .04); and high general fit indices (GFI = .98; AGFI = .92). In this model, the original association between the fathers' initial treatment involvement and children's outcomes was also reduced from $-.28$, $p < .01$ to $-.11$, $p = .08$ after the addition of the hypothesized mediating sequence, and this sequential path was significant at the statistical trend level ($z = -1.87$, $p = .064$). Thus, similar results were obtained even when post-treatment PDA in fathers was not measured by the annual rate concurrent with post-treatment AA attendance, but by the PDA for the 3-month period at the end of study.

In short, results from both of the above approaches converged to offer additional support for the longitudinal sequence proposed by the original model.

Discussion

Because a major way to intervene with COA may be indirectly through their alcoholic parents' treatment (Fals-Stewart et al., 2004; O'Farrell & Feehan, 1999), the aim of this study was to investigate whether the extent of treatment involvement in alcoholic fathers has a beneficial effect for their children's outcomes, and whether this benefit occurs through intermediate paternal behaviors: continued AA attendance and greater abstinence in the year following treatment. We examined outcomes in COA within the larger context of parental alcoholism risk and help-seeking behaviors, and to our knowledge, this is the first study to prospectively investigate COA in relation to their fathers' treatment involvement, post-treatment AA attendance, and post-treatment abstinence.

The hypothesized longitudinal benefit of fathers' greater treatment involvement on child outcomes was observed, but only for externalizing problems, such that fathers with greater initial treatment involvement had children with lower externalizing problems at M12. Why the internalizing problems in COA appeared not to be affected by the extent of their fathers' treatment (even though these problems also declined in the follow-up year) is not discernable from our results. It is possible that factors other than parental alcoholism per se influenced this type of symptoms (cf. Merikangas et al., 1998; Ohannessian et al., 2004), or that parental treatment was more effective in reducing family-level risks that more strongly affect externalizing, not internalizing problems. In short, our further analyses focused on COA externalizing problems at M12 follow-up, as they were significantly predicted by their fathers' treatment involvement one year earlier.

We further proposed that this beneficial effect of fathers' treatment on child behavior occurs indirectly, through fathers' post-treatment behavior. The hypothesized SEM model had an excellent fit (Figure 2), showing that alcoholic fathers' greater treatment involvement predicted their greater post-treatment AA attendance in the year following treatment, which in turn, predicted greater abstinence in the same time period. Finally, father's abstinence predicted their children's outcomes, such that fathers who spent more time sober had children who had lower externalizing problems at final assessment. These associations remained significant even though child externalizing problems and father's drug problems at baseline were accounted

for in the model. The proposed sequential mediating path accounted for 90% of the total effect and was statistically significant. In short, our findings demonstrated that better outcomes in COA whose fathers had greater initial treatment involvement were fully explained by their fathers' continued and more frequent help-seeking behaviors and subsequent abstinence in the year following treatment.

These results have twofold theoretical and clinical implications, as they are important in considering both (a) prevention and intervention efforts when targeting at-risk COA populations, and (b) broader implications of treatment and AA participation in alcohol-dependent fathers. First, developmental psychopathology theories describing parental alcoholism as a risk factor for child adjustment were supported by these results, as was the related notion that treatment participation would benefit not only the treatment participants but also their children, albeit indirectly (O'Farrell & Feehan, 1999). Indeed, our results show that greater abstinence in fathers predicted lower externalizing problems in children, replicating prior studies in our own lab (Burdzovic Andreas et al., 2006; Burdzovic Andreas & O'Farrell, 2007) and in other samples (Callan & Jackson, 1986; Moos & Billings, 1982) where outcomes in COA were reflective of their fathers' post-treatment status, thus adding to the literature identifying COA as "children at risk" (especially at risk for externalizing symptomatology, cf., Kuperman et al., 1999; Loukas et al., 2003; Obot & Anthony, 2004). More importantly, our results show that alleviation of the alcoholism risk from the COA environment resulted from greater treatment involvement and continued AA attendance by alcoholic fathers. Thus, greater help-seeking behaviors, both in terms of 12-step oriented professional treatment and self-help groups, proved valuable for both these alcohol-dependent men and their children, as they promoted abstinence and lessened the severity of COA risk status. These results suggest that indirect interventions, through alcoholic fathers' greater treatment involvement may have positive effects on COA adjustment. This an important consideration at times when many children in the US face this risk (Grant, 2000) and when this may be the only way to reach them (Fals-Stewart et al., 2004). Even though this indirect effect was small (.02), it can nevertheless have important real-life, social and policy implications for at-risk children (McCartney & Rosenthal, 2000).

Second, these results replicate and extend recent findings on the importance of AA attendance during and after professional alcoholism treatment, as we replicated previous findings on AA as a link from professional treatment to better post-treatment outcomes, but we also extended these findings to include children of alcohol-dependent patients. Our results established a prospective link between the initial treatment and subsequent AA attendance, and are consistent with the McCrady et al. (2004), where greatest post-treatment AA participation was observed in alcoholics who simultaneously participated in AA during their alcohol behavioral couples treatment. The professional treatment received by alcoholic patients in our sample had a strong 12 Step/AA orientation, and, just as in McCrady et al. (2004) these patients were encouraged to attend AA meetings in addition to their professional treatment. It is possible that the degree of their treatment involvement reflected these patients' other characteristics (such as their motivation to change and/or their commitment to abstinence) but we did not have measures of these characteristics available in our study.¹⁰ In short, the extent of alcoholic fathers' initial treatment involvement was the significant first element in the causal chain model we hypothesized, as it led to their continued AA attendance.

Next, the positive association we observed between greater post-treatment AA attendance and greater abstinence echoes recent reports from McCrady et al. (2004), Morgenstern et al. (1997), and Ritsher et al. (2002). These studies also reported both short-term and long-term

¹⁰But prior research often accounted for such factors, still reporting independent AA effects on later drinking outcomes (Fiorentine, 1999; McKellar et al., 2003; Morgenstern et al., 1997).

positive effects of AA involvement, including lower alcohol-related life problems, greater abstinence rates, and higher remission rates in alcoholic patients who attended AA following treatment. Thus, this report offers additional support for the idea that AA attendance is as a meaningful strategy for maintaining professional treatment gains (McCrary et al., 2004) as our overall results testify to the positive impact of post-treatment AA attendance on short-term drinking outcomes in alcohol-dependent fathers. Because fathers' abstinence was positively related to children's outcomes, our findings of AA benefits indirectly extend to COA as well.

Limitations

Even though this study furthered our understanding of processes associated with adjustment in children of alcoholic fathers in treatment, some limitations still remain. These were primarily methodological limitations, based on the nature of data collected, sample size, and sample characteristics. Because we were bound by the parameters of the original treatment study which examined only alcoholic men, the present study included only alcoholic fathers and not mothers. Thus, the important question of maternal alcoholism impact on child outcomes was beyond the scope of the present report. Further, relatively small sample size precluded testing separate models for boys and girls, or more detailed tests of differences among adolescent and pre-adolescent COA, all of which could have been differently impacted by fathers' drinking problems. Next, measures of fathers' AA attendance and abstinence rates were cumulative measures of annual experiences, which precluded more definite causal conclusions. Even though we addressed this issue statistically, having a truly longitudinal multi-wave design would have enabled examination of reciprocal associations, and replication of McCrary et al. (2004) who were able to appropriately test such effects. The cumulative nature of these measures also prevented more complex examination of the timing and duration of AA attendance, as variations in such patterns have been found to predict drinking outcomes in alcoholic men (Moos & Moos, 2004).

Also, the proposed models do not identify psychological mechanisms through which AA attendance impacted fathers' abstinence, or through which fathers' abstinence impacted child outcomes. It is likely that AA worked through some of the common change processes identified in past research, such as self-efficacy or abstinence commitment (Morgenstern et al., 1997). However, positive effects of AA often remained strong even when such potential mediation processes were accounted for (Fiorentine, 1999; McCrary et al., 2004; McKellar et al., 2003), supporting the view of AA's unique contribution to abstinence. Similarly, it is likely that our findings of COA of more sober fathers having lower externalizing problems were an indirect effect as well. Fathers' alcoholism possibly contributed to a host of family and parenting problems, which could feasibly mediate the relation between fathers' drinking and child behavior. For example, families of alcohol-dependent men are often marked by elevated marital violence (O'Farrell & Murphy, 1995) and by generally impaired family and/or parenting functioning (El-Sheikh & Flanagan, 2001; Fals-Stewart et al., 2004; Kelley & Falls-Stewart, 2002; Loukas et al., 2003), all of which could be the potential link between parental drinking and child maladjustment, and all of which could have been improved with fathers' abstinence. However, none of these mediating hypotheses could be directly tested, because we lacked the appropriate measures of these family and parental characteristics. Also, our primary goal was limited to testing the basic prospective associations between alcoholism treatment in alcoholic fathers and behavioral outcomes in their children. In other words, in this report we did not aim to test more complex mediating paths, many of which could certainly exist.

Further, our sample was limited to two-parent lower to middle-class families where fathers were primarily alcohol dependent, and where the great majority of mothers did not have alcoholism problem. We included additional control variables in the model (i.e., fathers' drug-related diagnosis and mothers' alcohol-related diagnosis), but they held little predictive power;

most likely because our sample of alcohol-dependent men was relatively homogenous. These fathers also did not have additional severe mental illness diagnosis (i.e., schizophrenia), while other paternal psychopathology (such as anti-social personality disorder, depression, etc.) was not assessed in this study. Hence, is not known how possible presence of these additional parental disorders may have impacted our results, as children of parents with comorbid disorders generally have more severe psychological and behavioral problems (Ohannessian et al., 2004). However, positive effects of AA/12-step programs participation have been observed in alcoholic patients with additional mental health diagnoses (McKellar, Stewart, & Humphreys, 2003) as well as in drug-abusing patients (Fiorentine, 1999). We also did not have additional behavioral and personality measures for these parents, which could have acted as possible mediators and moderators of the observed relations. In short, whether similar results would have been obtained in children from demographically different samples, or in children of alcoholic mothers or drug-abusing parents or of parents suffering from additional psychiatric disorder is difficult to say. Finally, our sample was limited to alcohol-dependent men attending an intensive 12-step oriented professional treatment program. Whether treatment programs of different orientations would achieve similar effects is not known, as no treatment comparison group was available. Patients averaged 20 treatment sessions plus 38.5 AA meetings over a 12-week period, so the results may not generalize to patients who receive less intensive treatment; and we do not know from the present results how much parental treatment was necessary for children to receive beneficial reductions in externalizing behaviors.

In sum, this study extends research on the importance of post-treatment AA involvement in alcohol-dependent men, by showing that not only these men, but their children also benefited from their more frequent AA attendance. The initial result of lower externalizing problems in COA whose fathers had greater treatment involvement was fully explained by the sequence of fathers' post-treatment behaviors, including their AA attendance and abstinence. Practical implications of such results are important, especially the notion that COA can reap indirect benefits from their fathers' professional alcoholism treatment and AA attendance. These secondary preventive effects established through treatment- and AA-associated reductions in parental alcoholism are even more important knowing, (1) that even though they are at greater risk for adverse outcomes, COA are unlikely to receive individual treatment due to their parents' unwillingness to enroll them in such programs (Fals-Stewart et al., 2004), and (2) that neither the initial treatment program nor AA directly addressed or had as a goal improving parenting behaviors specifically. While these results add additional support to the hypotheses of causal linkages between problematic parental and problematic child functioning, they also testify to the broader positive impact of both professional alcoholism treatment and AA attendance in alcohol-dependent men.

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Figure 1.

Hypothesized theoretical model for alcoholic fathers' initial treatment involvement, their post-treatment outcomes, and their children's behavioral outcomes.

Note: Shown is the hypothesized causal sequential model. Latent constructs (i.e., constructs where measurement error was accounted for) are shown in ovals; observed indicators (including measures used to construct latent constructs as well as measures in which measurement error is assumed to be non-existent) are shown in rectangles. Measures providing data about children are shown in gray tones, while the measures providing data about parents are shown in white. Two identical models are hypothesized; one for children's internalizing (using PSC Internalizing subscale) and one for externalizing (using PSC Externalizing subscale) adjustment problems.

PSC = Pediatric Symptom Checklist; PDA = percent days abstinent of father. Control variables (collected at baseline) include: fathers' PDA in the year before treatment entry; number of problematic alcohol use for fathers; fathers ADS and MAST scores; maternal DSM-IV alcohol-related diagnosis; and paternal DSM-IV drug-related diagnosis.

$\chi^2 = 10.03$, $df = 8$, $p = .26$; RMSEA = .04; GFI = .98; AGFI = .92

Figure 2.

Completely standardized solution for the fitted model for alcoholic fathers' initial treatment involvement, their post-treatment outcomes, and their children's behavioral outcomes.

$\chi^2 = 10.03$, $df = 8$, $p = .26$; RMSEA = .04; GFI = .98; AGFI = .92

Note: Shown is the completely standardized solution. Measures providing data about children are shown in gray tones, while the measures providing data about alcoholic fathers are shown in white. Non-significant paths are shown in dotted lines. PSC Ext = Pediatric Symptom Checklist, Externalizing Problems subscale; PDA = percent days abstinent of father. Drug diagnosis = presence of *DSM-IV* drug-related diagnosis in fathers at baseline. * $p < .05$; ** $p < .01$; *** $p < .001$

Effects of Fathers' Treatment Involvement on Child Externalizing Problems at M12:

Indirect (though post-treatment AA attendance and PDA): $.29 \times .26 \times (-.25) = -.02$

Table 1
Means, standard deviations and correlation coefficients among observed study variables.

Observed variables in the model	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
<i>Child demographics</i>														
1. Caucasian (vs. minority)	--													
2. Girl (vs. boy)	-.03	--												
3. Age (years)	-.01	.09	--											
<i>Child Adjustment Problems</i>														
4. SCS Externalizing (Pre-Ad)	.05	-.27*	.04	--										
5. SCS Externalizing (Mid)	-.03	-.21*	-.06	.73**	--									
<i>Fathers' tx involvement</i>														
6. x + AA meetings	-.18*	.03	.11	-.18*	-.26**	--								
<i>Fathers' post-tx outcomes^a</i>														
7. Days of AA attendance	.02	-.05	.03	-.09	-.11	.28*	--							
8. Father PDA (Mid)	.16	-.05	.19*	-.17	-.37**	.07	.26**	--						
<i>Post-tx control variables^b</i>														
9. Father PDA	-.02	.07	.08	-.19*	-.09	-.05	-.03	.05	--					
10. Father years of problem use	-.009	-.02	.44**	-.009	-.07	.06	.03	.10	-.11	--				
11. Father ADS	-.18*	-.05	.17	.08	.09	.04	-.03	.01	-.40**	.15 ^f	--			
12. Father MAST	-.22*	-.04	.13	.05	.04	.06	.05	.03	-.42**	.21*	.89**	--		
13. Mother DSM-IV (alcohol)	.05	-.02	.03	-.07	-.02	-.01	.20*	.06	-.11	-.02	.07	.07	--	
14. Father DSM-IV (drug)	.07	-.01	-.04	.04	.18*	-.03	-.11	-.17	.11	.13	.01	-.05	.03	--

^f $p < .10$;

* $p < .05$;

**
 $p < .01$;

 $p < .001$

PSC = Pediatric Symptom Checklist, ADS = Alcohol Dependence Scale (Skinner & Allen, 1982); MAST = Michigan Alcoholism Screening Test (Selzer, 1971), PDA = percent days abstinent of father.

Pre = pre-treatment (baseline) assessment; M12 = month 12 follow-up.

^a All post-treatment variables document alcoholic fathers' self-reported experiences for the entire 12-month period following treatment participation.

^b All control variables document the extent of fathers' alcohol-related problems at treatment entry, with the exception of "Mother DSM-IV (alcohol)" and "Father DSM-IV (drug)". These are dichotomized variables coding for presence of any current DSM-IV alcohol diagnosis (in mothers) or presence of any current DSM-IV drug diagnosis (in fathers) where 1 = diagnosis present and 0 = diagnosis absent. PDA at Pre codes for the percent of days fathers remained abstinent during the 12-month period immediately preceding treatment entry.