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The Role of Parental ADHD in Sustaining the Effects of a Family-School Intervention for ADHD

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Abstract

Objective—This study investigated the extent to which parental Attention-Deficit/Hyperactivity Disorder (ADHD) symptoms impact child and parent outcomes following a multimodal family-school intervention, the Family School Success (FSS) program, when compared to an active-control condition (CARE).

Method—Participants were 139 children with ADHD (67% male; 91% non-Hispanic; 77% Caucasian; grades 2–6) and their primary caretaker (91% female; aged 26–59) who participated in a randomized clinical trial evaluating the efficacy of FSS. Associations were examined between parent-reported ADHD symptoms at baseline and intervention outcomes reported by parents and teachers after treatment and at a 3-month follow-up, including child homework and classroom impairments, child ADHD and oppositional defiant disorder symptoms, parenting behaviors, and parent-teacher relationship quality.

Results—Across both treatment conditions, parental ADHD was not associated with parent or child outcomes at post-assessment. However, differences emerged between the two treatment groups at follow-up for parents with ADHD, particularly when an empirically-supported symptom cutoff was used to identify parents at-risk for having ADHD. In FSS, but not in CARE, parental ADHD was associated with declines in treatment gains in the quality of the parent-teacher relationship and the child's homework performance.

Conclusions—Parents at-risk for ADHD had difficulty maintaining treatment effects for themselves and their child in the FSS intervention, but not in CARE. The supportive and

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educational components central to the CARE intervention may be helpful in promoting the sustainability of psychosocial interventions for children with ADHD who have parents with elevated ADHD symptoms.

Keywords

ADHD; family-school intervention; adult ADHD; treatment sustainability

Attention-Deficit/Hyperactivity Disorder (ADHD) affects 5–7% of school-aged children (APA, 2013; Willcutt, 2012) and, on average, at least one child in every classroom in the United States (Froehlich et al., 2007). Children with ADHD experience significant impairment across home, school, and peer domains of functioning (Barkley, 2006). Specifically, children with ADHD achieve significantly less academically and are more likely to incur disciplinary action and dropout of school than children without ADHD (Kent et al., 2011; Loe & Feldman, 2007). Consequently, the estimated cost of ADHD to society is substantial (Robb et al., 2011).

In order to manage this disorder and improve the functioning of children with ADHD, a plethora of treatments have been empirically examined. Behavioral interventions implemented by parents at home (i.e., parent training) and by teachers at school (i.e., classroom management) are well-established treatments for children with ADHD (Evans, Owens, & Bunford, 2013). However, only a limited body of research has examined the impact of combining the efforts of both parents and teachers in the care of children with ADHD (e.g., Piffner et al., 2007; Owens, Murphy, Richerson, Girio, & Himawan, 2008). Multimodal treatments may be beneficial because they enable providers to address impairments directly in multiple domains and because they allow for synergistic effects through parent-teacher collaboration. Recent findings suggesting that a cooperative effort between parents and teachers can have positive effects in diverse child populations, specifically children with disruptive behaviors (e.g., Sheridan, Ryoo, Garbacz, Kunz, & Chumney, 2013), points to the need to investigate the benefit of enhancing parent training for children with ADHD with strategies for increasing family involvement in their child's academic life. Family School Success (FSS; Power et al., 2012) is a novel, integrative psychosocial intervention for children with ADHD combining components of efficacious interventions to improve children's behavioral and academic functioning at home and at school (e.g., behavioral parent training, daily report card, and conjoint behavioral consultation; Evans et al., 2013; Owens et al., 2008; Sheridan & Kratochwill, 2008). By combining these treatment approaches across home and school settings, FSS aims to improve parenting, parent involvement in education, and parent-teacher collaboration in order to ultimately improve children's behavior and academic performance. Power and colleagues (2012) conducted a large randomized clinical trial and demonstrated the efficacy of FSS compared to an active control condition. The active control condition, titled *Coping with ADHD through Relationships and Education* (CARE; see Power et al., 2012), took place over the same amount of weeks as FSS and was meant to provide psychoeducation about ADHD and a supportive context for parents to discuss challenges in coping with their child's difficulties and progress with home behaviors. Although the clinician was prohibited from discussing standard features of behavioral parent training (BPT) and engaging in

problem-solving with parents, parents were allowed to discuss challenging situations and share potential solutions. Parents and children met in separate group settings; children's behaviors in the child group were managed through a token economy system. One family-school meeting was held in which the clinician, parent, and teacher met to discuss school progress. CARE was implemented to control for the non-specific effects of the FSS intervention.

Overall in the original outcome paper (Power et al., 2012), FSS showed modest efficacy across several of the examined child and parent outcomes, which included child homework problems, parent-teacher relationship quality, parent use of ineffective discipline, and parent self-efficacy as an educator. Outcomes were assessed immediately following the intervention (post-assessment) as well as three months following the cessation of the intervention (follow-up assessment). Originally, FSS displayed superior post-assessment treatment outcomes in parent-report of their self-efficacy as an educator of their child, their children's homework performance (i.e., less inattention and avoidance of work), and their parenting practices (i.e., decrease in negative or ineffective discipline) than CARE. At the follow-up assessment, FSS displayed superior outcomes in the quality of the family-school relationship in addition to parenting practices (Power et al., 2012).

However, given the integral role of parents in structured treatments such as FSS, or other behavioral treatments for children with ADHD, it is important to examine factors that may influence how well parents act as agents of change, particularly when they are held responsible for implementing regimented components of an intervention for their children. Considering that ADHD is highly familial and heritable (Faraone et al., 2005), with recent estimates indicating that between 20–40% of children with ADHD have at least one biological parent with ADHD (Takeda et al., 2010), parental ADHD may be one such parental factor that influences intervention outcomes for children. There is reason to be concerned about parental ADHD in the context of treatment for children, as evidence indicates that parents with elevated ADHD symptoms inconsistently monitor and manage their child's behavior and do not follow through with procuring services for themselves and their child when needed (Johnston & Lee-Flynn, 2011; Johnston et al., 2012). It is easy to see how inattention, forgetfulness, disorganization, and the tendency to choose non-effortful activities may make it difficult for parents with ADHD to make changes to their parenting practices suggested during BPT or more comprehensive interventions (e.g., FSS). In addition, even if these parents are able to make changes initially, symptoms of ADHD may limit their ability to sustain the use of new parenting practices after terminating treatment, when the support of clinicians and parenting groups are withdrawn. As parents are the key agents of change in most evidence-based psychosocial interventions for child ADHD, it stands to reason that children with ADHD may not respond as well to these treatments if their parents have elevated ADHD symptoms.

These concerns about parental ADHD have led to several studies examining the impact of elevated parent ADHD symptoms on treatment outcomes for children. Sonuga-Barke, Daley, and Thompson (2002) reported that preschool children whose mothers reported relatively high levels of ADHD symptoms did not demonstrate improvements in ADHD symptoms immediately after BPT or at follow-up 15 weeks later. In contrast, children of mothers with

relatively low levels of self-rated ADHD symptoms made significant gains on a composite outcome measure combining ADHD symptoms and/or oppositional behaviors at both time points. It should be noted that Sonuga-Barke and colleagues (2002) assigned the maternal level of ADHD (i.e., “low,” “mid,” “high”) by creating groups based on sample-specific cutoffs for ADHD symptoms, as opposed to applying an evidence-based criterion for adult ADHD classification (e.g., reporting 4 or more symptoms; Barkley, Murphy, & Fischer, 2008). Additionally, the intervention implemented in this study did not include treatment targets in addition to parent-reported child ADHD symptoms (e.g., academic impairments), thus limiting its implications for understanding the effects of parental ADHD on the range of measurable outcomes that are relevant to childhood ADHD.

Similarly, Chronis-Tuscano and colleagues (2011) found that 6–10 year old children of mothers who reported more ADHD symptoms displayed less improvement in their disruptive behavior symptoms immediately following BPT than did the children of parents reporting fewer ADHD symptoms. Like Sonuga-Barke and colleagues (2002), this study examined BPT only. Further, this study examined parent-rated and observer-coded outcomes related to ADHD symptoms, impairment, and parenting behavior. This study also lacked a follow-up assessment, precluding an examination of the effects of parental ADHD on child or parent outcomes when treatment supports are removed.

In a randomized trial of a friendship coaching intervention, Griggs and Mikami (2011) examined the effects of parental ADHD symptoms on the social functioning of 6–10 year old children with ADHD. These authors found that higher self-rated parental inattention symptoms predicted lower child peer acceptance and higher peer rejection at post-treatment. Griggs and Mikami (2011) also found that parent ADHD was associated with poorer parental facilitation during child playgroups (i.e., poorer provision of positive support to their child during peer interactions). Therefore, parental ADHD was shown to negatively impact parents’ ability to perform important roles in treatment (e.g., friendship coach) and to negatively affect vital areas of the children’s functioning (e.g., peer acceptance). Although these researchers extended the literature base by testing outcomes beyond child symptoms, they only focused on social functioning outcomes. They also did not include a follow-up assessment in their study, limiting the ability to assess effects beyond active involvement in the intervention.

Overall, these few studies demonstrate that parental ADHD can affect both parent and child response to treatment. However, there are gaps in this small body of literature. Specifically, no studies have examined interventions targeting academic outcomes, and two of the three studies only focused on ADHD symptoms and global impairment, and did not examine specific areas of impairment. This is a notable limitation as academic concerns, such as homework problems, are common among children with ADHD (Sheridan, 2009), and research highlights that impairment, as opposed to symptom severity, is more predictive of long-term outcomes (Mannuzza & Klein, 1999). Demonstrating that parental ADHD has an effect on children’s real-world functioning would heighten concerns about parent ADHD relevant to treatment implementation. Two of the three studies reviewed above also did not investigate the persistence of effects after treatment termination. Sustainability is increasingly becoming a focus of intervention development (Evans, Owens, Mautone,

DuPaul, & Power, 2013), which is particularly important for ADHD interventions given that this disorder is chronic in nature (Wolraich et al., 2011). Therefore, the transition to a chronic care or life course model that emphasizes the sustainment of treatment effects appears essential for this impaired population (see Evans et al., 2013). In this vein, it is important to explore factors that may contribute to poor sustainability. Given the high heritability of ADHD and the parenting difficulties associated with adult ADHD, it is likely that parental ADHD contributes to discouraging trends in the maintenance of treatment effects. Finally, none of these studies investigated the role of parental ADHD in the context of a regimented behavioral intervention versus a less regimented control condition; as such, the mechanisms for which parental ADHD might impede intervention outcomes are less clear.

The present study extends the literature by examining the effects of parental ADHD on the immediate and 3-month follow-up response to FSS, a multi-modal, integrative psychosocial treatment for children with ADHD. The effects of parental ADHD on treatment response will also be compared to effects of parental ADHD on a less regimented active-control condition. Not only will be the first study to investigate the effects of parent ADHD on academic outcomes, but it will be the first study to place these findings within the context of an active-control group. Given the specific parenting difficulties that are common among adults with ADHD (Johnston et al., 2012), we predicted that, within the context of FSS, parents with ADHD would manifest problems implementing effective parenting practices in the home. We also predicted that they would have difficulty maintaining a strong working relationship with their child's teacher, which may have an effect on their ability to co-implement (with teachers) contingency management techniques such as the daily report card, potentially weakening the overall beneficial effects of the treatment; as such, a variety of child and parent outcomes were examined. However, we predicted that parents with ADHD might benefit more from, or be less impaired in responding to, interventions meant to support them, that require less of the skills that adults with ADHD may lack (e.g., organization, attentiveness, etc.), such as the strategies included in the active control condition of this study.

The primary aim of the present study was to examine how parental ADHD impacted FSS outcomes for children with ADHD. Consistent with previous research, we hypothesized that elevated parental ADHD symptoms would be associated adversely with (a) child outcomes after treatment and at follow-up, and (b) parent outcomes at post-treatment and follow-up for both FSS and CARE conditions. We also hypothesized that the pattern of results, in terms of how parental ADHD affects outcomes, would vary across FSS and the active control condition, CARE; however, without prior research the direction of this difference was not hypothesized.

Method

Participants

Data used in this study were collected as part of a randomized clinical trial (see *blinded for review*) examining the effectiveness of the Family School Success (FSS) intervention over an active-control intervention with respect to improvement in home and school outcomes of

children with ADHD. Details regarding recruitment and selection procedures, as well as inclusion and exclusion procedures, are presented elsewhere (Power et al., 2012).

The primary focus of the current investigation was on families assigned to the FSS, though families who were assigned to the CARE condition were included to provide a point of reference. Only families where the primary caregivers completed the *Conner's Adult ADHD Rating Scale* (CAARS; FSS: $n=65$; CARE: $n=74$) were included in the analyses. Because the CAARS was added to the assessment battery later in the trial (i.e., during the 4th out of 13 FSS/CARE cohorts), 35 families in FSS and 25 families in CARE included in the original study (Power et al., 2012) were not included in the present sample. Within the FSS sample, no differences were detected on any of the primary outcome measures (evaluated at baseline, post-treatment, and follow-up) or covariates between families who were or were not included; however, parents who completed the CAARS (65%) were more likely to have a child with internalizing problems at baseline than parents who did not complete the CAARS (35%), $X^2(1, n=100) = 3.87, p=.049$. Within the CARE sample, parents who completed the CAARS endorsed less general psychopathology on the SCL-90 ($T=-2.33, p=.022$) and reported higher inattention/avoidance on homework (HPC, Factor 1) than parents who did not complete the CAARS ($T=2.55, p=.012$). No significant differences were detected across groups (FSS/CARE) on any demographic variable.

Of the primary caregivers (hereafter also interchangeably referred to as parents), 126 (90.6%) were women (122 mothers, 2 grandmother, 2 step-mother) and 13 (9.4%) were men (12 fathers, 1 step-father). Notably, 23 families (11 in FSS and 12 in CARE) included a primary caregiver reporting 4 or more clinically-significant DSM-IV symptoms of ADHD on the CAARS Self-report short form (Conners et al., 1999). Additional background and diagnostic information for the sample are presented in Table 1.

Procedure

Parents participating in FSS had 12 weekly sessions: two were conjoint family-school consultation, six were clinic-based parent group meetings with concurrent child group sessions, and four were individualized family therapy sessions. Parents participating in CARE also had 12 weekly sessions: 11 group sessions for parents and children separately, and one family-school meeting to obtain information about school performance. In the present analyses we included data collected at baseline (pre-treatment), post-treatment (immediately after the 12th FSS/CARE session), and at a follow-up assessment (3 school months after treatment ended). All outcomes assessed in the original RCT were included for examination in order to provide a comparison when the potential effects of parental ADHD is considered for each intervention outcomes.

Measures

Parent ADHD—The CAARS-Short form (Conners et al., 1999) was used to assess ADHD symptoms in the primary caregiver. Items assessing DSM-IV criteria for ADHD were rated on a 4-point scale (0= *not at all, never*; 1= *just a little, once in a while*; 2= *pretty much, often*; 3= *very much, very frequently*). From the self-reported CAARS DSM-IV items, we created a dimensional variable and a dichotomous variable. To create the dimensional variable, we

calculated a mean item score based on the 18 DSM-IV items self-reported by each participant ($\alpha = .91$). To create the dichotomous variable, individuals who endorsed four or more clinically-significant symptoms (occurring “often” or “very much”) of hyperactivity/impulsivity, inattention, or both were considered at increased risk of having ADHD. There is much empirical support for a 4-symptom cutoff as an indicator of ADHD risk in adults (Barkley et al., 2008; Solanto, Wasserstein, Marks, & Mitchell, 2012). These studies largely indicated that adults having at least four symptoms of ADHD have significant impairments when compared to adults without ADHD, and therefore, warrant treatment (see Barkley et al., 2008).

Potential Covariates—Child gender, family’s socioeconomic status, child’s ADHD medication status, child’s baseline ADHD severity and parent’s overall psychological difficulties were examined as potential covariates in the analyses. Child gender was examined as minor gender differences have been indicated for children with ADHD for symptoms, impairment, and parental distress levels (Gershon & Gershon, 2002; Podolski & Nigg, 2001). Family socioeconomic status (as determined by the Hollingshead [1975] index) was examined because it has been found to affect treatment outcomes and treatment adherence for children with ADHD or other externalizing behaviors, particularly for parent-involved treatments (Firestone & Witt, 1982; Reyno & McGrath, 2006). Medication status (as reported at baseline) was evaluated given its potential to confound effects of treatment. Because pre-treatment severity of child ADHD is often found to predict treatment outcomes for both the parent and the child (e.g., Owens et al., 2003), we formed an aggregate child ADHD severity index by averaging parent and teacher item ratings on the 18-item *Swanson, Nolan, and Pelham Questionnaire* (SNAP-IV; Swanson et al., 2001; $\alpha = .91$). Finally, to rule out parental psychopathology beyond ADHD symptoms as a risk factor for treatment outcomes, we evaluated the global severity index T-scores from the *Symptom Checklist-90-Revised* (SCL-90-R; Derogatis & Savitz, 2000) as a potential covariate.

Child Behavioral Outcomes—The *Homework Problem Checklist* (HPC; Anesko, Schoiock, Ramirez, & Levine, 1987) was used to assess parent perceptions of their child’s homework performance. The HPC measures two factors, Inattention/Avoidance (HPC-IA) and Poor Productivity and Nonadherence with Rules (HPC-PP). This measure has demonstrated acceptable validity (see Power, Werba, Watkins, Angelucci, & Eiraldi, 2006). In the current sample, coefficient alphas were .89 for the HPC-IA factor and .78 for the HPC-PP factor.

The Student Responsibility factor from the *Homework Performance Questionnaire Teacher Version* (HPQ; Power, Dombrowski, Watkins, Mautone, & Eagle, 2007) was used to assess the teacher-ratings of the student’s responsibility on homework. This 7-item measure has demonstrated both concurrent and discriminant validity for children with ADHD (Mautone, Marshall, Costigan, Clarke, & Power, 2011). Internal consistency was $\alpha = .90$.

A 12-item subscale from the *Academic Performance Rating Scale* (APRS; DuPaul, Rapport, & Perriello, 1991), assessing the teacher-report of the students’ academic productivity, was used. The psychometric properties of the APRS are acceptable (DuPaul et al., 1991) as was the coefficient alpha in this sample, $\alpha = .86$.

To assess child ADHD and Oppositional-Defiant Disorder (ODD) symptoms, parents and teachers completed the 26-item *SNAP-IV* (Swanson et al., 2001). A unitary index of symptomology was used by creating a mean item composite score for the parent and teacher report, respectively (see Swanson et al., 2001). The coefficient alpha for both the parent and teacher rating in the present sample was excellent, $\alpha = .93$.

Parenting Outcomes—A 10-item version of the *Parent as Educator Scale* (PES; Hoover-Dempsey, Bassler, & Brissie, 1992) was used to assess whether caregivers believe that they can influence their children's school success. Each item is rated on a 5-point scale (1 = *strongly disagree* to 5 = *strongly agree*). The reliability of this scale was high in prior studies ($\alpha = .89$; Hoover-Dempsey et al., 1992; $\alpha = .83$, Power et al., 2012) and in the present study, $\alpha = .82$.

One 11-item factor from the *Parent-Teacher Involvement Questionnaire* (PTIQ; Kohl, Lengua, McMahon, & Conduct Problems Prevention Research Group, 2000) was used to assess the quality of the parent-teacher relationship as perceived by both the parent and the teacher. The reliability of this scale was high in the present sample $\alpha = .88$. However, items comprising the Quality of the Parent-Teacher factor may not load as well onto a unitary factor as they do onto a two factor model separated by informant (i.e., parent factor and teacher factor; Mautone, Marcelle, Tresco, & Power, in press). Accordingly, as a secondary examination, we examined this variable as a parent-factor and a teacher-factor. The separate parent and teacher factors had strong internal consistency ($\alpha = .90$ and $\alpha = .81$, respectively).

The *Parent-Child Relationship Questionnaire* (PCRQ) was used to assess parent perceptions of the parent-child relationship. This scale was divided into two factors, a 22-item Positive Involvement (PCRQ-PI) factor and a 12-item Negative/Ineffective Discipline (PCRQ-NI) factor (factor structure supported by Furman & Giberson, 1995; and Hinshaw et al., 2000). In the current sample, coefficient alphas were .89 and .84, respectively.

Analytical Plan

As an initial step, we explored the suitability of the potential covariates for inclusion in the final regression models. Covariates were retained in the analyses if they demonstrated at least one significant association with an outcome variable at post treatment or follow-up. Next, Mplus 7.11 (Muthén & Muthén, 2012) maximum likelihood estimation, robust to data nonnormality, was used to test the path models. Treatment group (FSS vs. CARE) was tested as a moderator of associations between parental ADHD symptoms and outcomes at post and at follow-up respectively, by assessing for model invariance as part of multiple group analyses. Initially, all regression paths in the models were allowed to vary across participants in FSS and CARE. Then, the association between parent ADHD and the outcome variable at post-treatment was constrained to be equal across participants in FSS and CARE. Next, the association between parental ADHD and the outcome variable at follow-up was constrained to be equal across groups. If constraining either regression path to be equivalent did not cause model fit to worsen significantly over the base model (i.e., chi-square difference < 3.84 ; Muthén & Muthén, 2012), then the strength of the association between parent ADHD and the specific outcome variable was considered to be the same across CARE and FSS.

Conversely, if constraining the paths of interest caused the model fit to worsen significantly (i.e., chi-square difference > 3.84), then the strength of the association between parent ADHD and the specific outcome variable was considered different between CARE and FSS and interpreted separately across groups.

Every model controlled for correlations among all baseline variables (parent ADHD symptoms, baseline level of outcome variable, covariates). Non-significant ($p > .20$) correlations between baseline variables were set to zero in each path model. Each outcome measure at post-intervention and at follow-up was regressed upon corresponding baseline scores on the measure, all covariates, and parent ADHD symptoms. Models were first tested with the dimensional measure of parent ADHD symptoms and then with the dichotomous measure of parent ADHD symptoms. Model fit indices used in this study were χ^2 , root mean square error of approximation (RMSEA) and comparative fit index (CFI). A model is generally considered a good fit for the data when χ^2 is non-significant, RMSEA $\leq .06$, and CFI $\geq .95$ (Hu & Bentler, 1999). In models with good fit when using the dichotomous method of determining parent ADHD, effect sizes ($M^1 - M^2 / \sigma^2$ pooled; Cohen's d ; Cohen, 1988) reflecting differences in outcome from post to follow-up assessment were calculated as a function of parental ADHD status. However, because the sample of individuals with four or more symptoms of ADHD was relatively small, the confidence intervals produced around the effect sizes are likely wide (i.e., including zero), reflecting an unreliable estimate. As such, 95% confidence intervals for effect sizes that straddle zero should be interpreted with caution.

Results

Preliminary Analyses

Intercorrelations between parent ADHD, other potential covariates and outcome variables at post and follow-up are presented in Table 2. Because all covariates of interest were significantly associated with parent ADHD or at least one outcome variable, and because multicollinearity among covariates was not a significant concern (all correlations < .31), we retained all five covariates in the main analyses. Table 3 provides the means and standard deviations of each outcome measure at each time point across both treatment groups divided by parental ADHD status.

Path Model Analyses

Six of the ten path models including parent ADHD symptoms as a risk factor for post-treatment and 3-month follow-up outcomes fit the data very well, within the confines outlined by Hu and Bentler, 1999 (see Table 4)¹. Accordingly, only these outcomes are discussed.

Moderation Analyses—When conducting multiple group analyses to test for model invariance across CARE and FSS, the pattern of results appeared to generally be similar

¹The models assessing the outcomes obtained from the Parent-Child Relationships Questionnaire (positive and negative involvement) and the outcomes obtained from the parent and teacher report on the SNAP (child ODD/ADHD) symptoms did not meet the fit statistic criteria when analyzed in multiple group analyses.

across the FSS and CARE samples when parental ADHD was assessed dichotomously at post-assessment (see Table 4), indicating that families with a primary caretaker at risk for ADHD (i.e., four or more symptoms of self-reported ADHD) generally performed with similar patterns across both groups immediately following treatment. However, when assessed three months later, there appeared a clear difference between the pattern of results between FSS and CARE. Within these analyses, three of the six outcomes significantly differed across the CARE and FSS sample (see Table 4). In analyses including the homework inattention/avoidance variable (HPC-IA), a trend was detected within the FSS condition indicating that children with a parent at risk for ADHD declined at follow-up (Cohen's $d = .32$; $CI = -.54, 1.14$). In contrast, for the CARE condition there was trend indicating improvement during this period (Cohen's $d = -.63$; $CI = -1.43, .21$). Moreover, children whose parents reported 4 or more clinically significant ADHD symptoms tended to have more difficulty with work productivity and adherence to homework rules (HPC-PP) at 3-month follow-up in the FSS condition (Cohen's $d = .53$; $CI = -.36, 1.39$; see Figure 1) whereas there was a trend indicating an improvement in performance in the CARE condition during this time (Cohen's $d = -.35$; $CI = -1.15, .47$). Additionally, in families in which a parent reported risk for ADHD, those in the FSS condition displayed a significant decline in the quality of the parent-teacher relationship (PTIQ; Cohen's $d = -.86$; $CI = -1.74, .09$) that was not displayed by families in CARE (Cohen's $d = -.12$; $CI = -.69, .91$). Because the PTIQ variable is collapsed across parent and teacher report, secondary analyses were conducted to examine the association between parent ADHD and parent- and teacher-reported parent-teacher relationship quality separately within the FSS sample. The model fit the data well for both parent- ($\chi^2(16) = 9.54, p = .89, RMSEA = .00, CFI = 1.00$) and teacher-reported quality of the parent-teacher relationship ($\chi^2(13) = 11.06, p = .61, RMSEA = .00, CFI = 1.00$). However, the results indicated that parental ADHD risk was associated with worse parent-teacher relations at follow-up when assessed by parent ratings ($\beta = -.33, p = .001$; see Figure 1), but not teacher ratings ($\beta = -.12, p = .373$).

We observed trends for differential associations between parental ADHD risk and two other outcomes for families in CARE vs. FSS at the follow-up assessment. The association between parent ADHD and APRS and PES were non-significant for both CARE and FSS, indicating that the risk of parental ADHD was not strongly associated with follow-up treatment gains/declines in these domains for either treatment group. However, a trend was detected within the CARE group indicating that parental risk of ADHD predicted greater parent-reported self-efficacy as an educator to their child (Cohen's $d = .57$; $CI = -.27, 1.36$). That is, parents at risk for ADHD whose children were in the CARE condition reported increases in self-efficacy at the follow-up assessment that were not detected in the FSS condition (Cohen's $d = -.33$; $CI = -1.18, .55$).

Similarly, when multiple group analyses were used to test model invariance across CARE and FSS for parental ADHD assessed dimensionally, few differences emerged between CARE and FSS at post assessment (Table 4). One exception is that the association between parental ADHD symptoms assessed dimensionally and their child's homework productivity (HPC-PP) when assessed immediately after treatment varied across CARE and FSS treatment groups. Specifically, a high level of parent ADHD symptoms was associated with a non-significant decline in homework productivity for families in the FSS condition, but

greater parental ADHD symptomatology was associated with a marginally significant improvement in parent-report of child homework productivity for CARE.

At follow-up assessment, there were two significant differences detected when assessing parental ADHD dimensionally: parent-report of their children's homework performance productivity (HPC-PP), and teacher-report of children's homework responsibility (HPQ; Table 4). The association between parental ADHD and HPC-PP was negative and non-significant in the CARE group, but positive and marginally significant in the FSS group, indicating that parent-report of their child's homework productivity declined in FSS, but not in CARE. A significant, and therefore noteworthy effect, was detected for HPQ. Parental ADHD symptoms significantly predicted declines in teacher-reported student homework responsibility at follow-up assessment in FSS, but not CARE. As such, these results indicate that children of parents with more symptoms of ADHD declined more at follow-up, according to the teacher's report of their homework responsibility, in FSS than in CARE.

Main Effects—Outcomes that did not present significant differences across the FSS and CARE conditions were then assessed collapsing across groups in order to detect the main effects of parental ADHD symptomatology on each outcome respectively. Parental ADHD symptomatology significantly predicted only one outcome at post-assessment that demonstrated a similar pattern of results across the FSS and CARE conditions. Specifically, when parental ADHD was assessed dichotomously, but not dimensionally, it appeared that families with a parent at risk for ADHD reported significant improvements in the child's homework productively (HPC-PP; $\beta = -.13$, $p = .031$, Cohen's $d = -.16$; $CI = -.42, .75$) immediately following the cessation of treatment across both intervention groups. No other main effects were detected for associations between parental ADHD and outcomes at post- or follow-up time points.

Discussion

This study provided evidence in accordance with previous research findings (Chronis-Tuscano et al., 2011, Griggs & Mikami, 2011; Sonuga-Barke et al., 2002) that parental ADHD adversely affects the outcomes of behavioral psychosocial interventions, like FSS, for children with ADHD. However, contrary to previous research, parent ADHD in the present study was predominantly unrelated to immediate treatment outcomes; rather, the unique risks associated with elevated parental ADHD symptoms were seen at the 3-month follow-up assessment in the FSS intervention. Notably, the failure to maintain gains was associated with those parents whose self-reported ADHD symptoms met a symptom threshold of 4-symptoms (c.f., Barkley et al., 2008) and largely not when parental ADHD was measured dimensionally (i.e., ADHD symptom severity). When parents self-reported the presence of four or more ADHD symptoms, both the child and the parent in the FSS group generally failed to maintain gains made during the intervention stage; however, this was not the case in the CARE group. Of the six outcomes investigated that proved a sufficient fit to the data, the effect of parental ADHD status was moderated significantly by treatment condition for three outcome measures, and there was a marginally significant effect for two additional outcomes, indicating that parents with elevated ADHD symptoms in FSS generally failed to maintain gains, but those in CARE did maintain gains. The

difference in findings when using the dimensional versus dichotomous method for assessing parent risk for ADHD suggests that, in general, self-reported parent ADHD may need to cross a threshold to have a detrimental effect on the maintenance of treatment gains made by parents and children. The pattern of findings detected, based on a consideration of significant and marginal effects (Cohen, 1994), clearly suggests an effect of parent risk for ADHD on outcomes at follow-up for the structured, multimodal, FSS intervention.

Our findings deviate from previous research (Chronis-Tuscano et al., 2011, Griggs & Mikami, 2011; Sonuga-Barke et al., 2002) and from our first hypothesis in that parents with and without elevated, self-reported ADHD symptoms and their children with ADHD displayed similar gains at the completion of both the FSS and CARE interventions. One potential explanation for the deviation from previous findings is that the multimodal FSS program and the supportive CARE intervention may offer some advantages over standard BPT for parents at risk for ADHD. For example, FSS places an emphasis on collaborative connections between family and school and offers numerous opportunities for teachers to support parents, aided by a trained clinician. CARE provides parents with repeated opportunities to provide and receive support from other parents and to receive extensive psychoeducation about ADHD and its causes, impact, and developmental course, which may have an especially beneficial effect for a parent with elevated ADHD symptoms. Additional research is needed to identify specific elements of treatment that are uniquely beneficial for parents with elevated ADHD symptoms.

Although parent risk for ADHD had little effect on outcomes immediately following FSS, such risk was associated with a pattern of poorer outcomes 3-months following treatment termination for the FSS condition. The one other study that did track follow-up outcomes found results comparable to the current findings (Sonuga-Barke et al., 2002). That is, higher levels of parental ADHD predicted poorer child outcomes at 4-month follow-up. Again, an important distinction is that in the Sonuga-Barke et al. (2002) study using a BPT program, parental ADHD symptoms were linked with poorer treatment response at both post-treatment and follow-up. In contrast, in this study using the FSS intervention, the effect of parent risk of ADHD largely was isolated to the follow-up period. One explanation that may have contributed to the difference in findings between studies is that children in the Sonuga-Barke et al. (2002) study were preschoolers, whereas those in this study were in elementary school (grades 2 to 6). Regardless, these findings highlight the need to monitor outcomes at follow-up when implementing family interventions for youth with ADHD. Our results indicate that during the follow-up period, when the level of teacher involvement and clinician support likely was reduced, parents at risk for ADHD in the FSS condition struggled to maintain treatment gains. On the contrary, in the psychoeducation and support condition, CARE, the adverse effect of parental ADHD at follow-up was largely undetected.

Understanding the pattern of results in the FSS condition in comparison to the CARE condition raises important questions about potential mechanisms for change during the follow-up period. FSS was an intensive and collaborative intervention involving the family, school, and clinician. FSS provided structured parent training and guided family-school collaboration, requiring implementation of behavioral strategies and a high level of organization from parents. In contrast, CARE was a non-directive intervention that focused

on educating parents and establishing a mechanism for providing support to one another. Although CARE did not specifically offer parents guided problem solving, parents in this condition sometimes engaged spontaneously in problem solving on their own. Comparing the two conditions, of the parents at risk for ADHD, only those in the CARE condition were able to maintain treatment gains at follow-up. It may then be, counter to prior presumptions, that parents with ADHD, when left to their own devices after therapy has ended, struggle to maintain the benefits of highly structured, behavioral intervention programs like FSS; in contrast, they may derive benefits, at least in the short-term, from group programming aimed at providing a context of support for families of children coping with ADHD. Our findings suggest that the supportive and educational components of the control intervention may help to promote the maintenance of treatment effects. These findings suggest that incorporating elements of CARE into the FSS intervention (i.e., providing parents more opportunities to learn about ADHD, its causes, the impact of parental ADHD on parenting; and giving them frequent opportunities to support one another) may be a useful strategy for sustaining the effects of FSS.

In addition to exploring outcomes at multiple time points and in multiple settings, the current study extends previous research by examining treatment effects on academic impairment. In the current study, children of parents with four or more symptoms of ADHD in the FSS condition were less likely to maintain improvements in academic functioning at follow-up assessment, specifically homework productivity as reported by their parent, despite making significant improvements in this variable at the post-assessment period across both treatment conditions. Children of parents with increased ADHD symptomatology in the FSS condition were also less likely to maintain improvements in their responsibility with homework as reported by their teachers. Thus, children of parents at risk for ADHD or with increased ADHD symptomatology, displayed greater impairment in homework performance at follow-up assessment as reported by two different reporters (teachers and parents) within FSS. This result is worrisome as homework impairments have been shown to be associated with meaningful long-term academic outcomes, such as lower grades and standardized test scores (see Cooper, Robinson, & Patall, 2006). This pattern of findings suggests that parental risk for ADHD may be associated with declines in child homework performance after the cessation of a structured family-school psychosocial intervention.

Primary caretakers with four or more symptoms of ADHD in the FSS condition also failed to maintain gains in the quality of the parent-teacher relationship, which was an important target of FSS. It is unclear why this drop in parent-teacher relations occurred after treatment termination. One possibility is that the FSS clinician played a critical role in supporting the relationship between the parent and the teacher. After clinician support was removed, the quality of the parent-teacher relationship may have declined. Interestingly, secondary analyses revealed that the drop in the parent-teacher relationship for parents with ADHD occurred solely from the parent's perspective, and not the teacher's perspective. It is possible that during treatment, clinicians aided parents in reinterpreting teacher behaviors and understanding the school's perspective in a way that the parents at-risk for ADHD could not sustain after treatment ended.

Several limitations of the current study must be considered. First, the number of parents in the ADHD group using the dichotomous method of identification was relatively few, which limited the statistical power to detect significant findings. We decided to dichotomize the parent ADHD variable to examine the effect of clinically meaningful elevations in ADHD symptomatology, in contrast to previous studies that examined parent ADHD using only a dimensional method. By so doing, we identified a relatively low prevalence of parents with clinically-elevated ADHD symptoms in this sample of children with ADHD (16.5%). This rate is comparable to other child ADHD treatment studies that sampled only one parent. For example, the prevalence obtained by Chronis-Tuscano et al (2011) in their sample of children in treatment for ADHD who had a mother meet the 4-symptom cutoff for adult ADHD was 14% ($n = 10$). Nonetheless, it is possible that the relatively low prevalence of adult ADHD in this study reflects underreporting among the parents of their own ADHD symptoms (e.g., Zucker, Morris, Ingram, Morris & Bakeman, 2002), or perhaps, is indicative of an unrepresentative sample. On that note, it must be acknowledged that deriving symptom counts from the CAARS to dichotomize parental ADHD varied from the method recommended by Conners et al (1999; i.e., use of elevated T-Scores). Although our method deviates from the established scoring method, our approach aligns with DSM-IV diagnostic criteria and established scoring methods from parallel rating scales inclusive of the DSM-IV criteria (e.g., Vanderbilt ADHD Rating Scale; Wolraich, Hannah, Baumgaertel, & Feurer, 1998), and allowed us to assess the empirically supported 4-symptom cutoff (Barkley et al., 2008; Solanto et al, 2012).

Second, parental ADHD status was identified through self-report only and did not include a clinical interview and ancillary reporters. As such, identification fell short of best practice for diagnosis of adult ADHD (Sibley et al., 2012). Third, parents participating in this study were generally highly motivated, as they were self-referred and followed through with multi-stage screening, evaluation, and outcome assessment procedures prior to starting treatment. Thus, families less likely to engage in treatment likely were underrepresented in this sample. Further, this sample included families who were predominantly of middle to upper middle social status. Similar research should be extended to families of lower socioeconomic status in order to better generalize findings to the population.

Fourth, given that parents rated their own ADHD symptoms as well as several of their and their child's outcomes, some of the significant findings may be attributed to shared method variance. Furthermore, shared method variance may be an additional confound given that we collapsed across parent- and teacher- report of child baseline ADHD symptoms to measure the covariate of ADHD severity to maintain consistency, rather than assessing these reporters separately for each respective outcome. However, the similarity in findings when homework performance was assessed using both parent and teacher reports for analyses based on dimensional parental ADHD mitigates, to some extent, the concern about shared method variance.

Given the present findings indicating that parental ADHD is detrimental to the maintenance of behavioral treatment gains for parents and children, future research should, in line with recommendations from Chronis-Tuscano et al. (2012), explore the best approach to maintain treatment effects for parents and children with ADHD. Research is needed to explore the

mechanisms underlying the interaction of child and parental ADHD, with particular attention paid to the role of adult ADHD on parenting practices (Johnston & Lee-Flynn, 2011; Johnston et al., 2012). To this end, future studies should examine the factors that underlie poor maintenance of treatment gains in families that have a primary caretaker with ADHD in structured psychosocial interventions while investigating extended follow-up periods (i.e., follow-up assessments longer than 3-months). For example, given that parents with ADHD have more difficulty following through with schedules and managing details (Johnston et al., 2012; Weiss, Hechtman, & Weiss, 2000), it may be that parent inattention symptoms play a central role in the loss of treatment gains. Conversely, parents with ADHD may struggle to adhere to treatment strategies when therapist scaffolding is removed. Moreover, parents struggling with ADHD symptoms may benefit from added supportive and validating components in structured treatments, mirroring components of CARE.

The drop off in treatment effects for FSS during follow-up highlights the importance of creating interventions that equip families with the skills necessary to maintain treatment gains. One option for treating parents with ADHD may be pharmacological treatments. Preliminary reports have indicated that treating parents with ADHD with stimulant medication reduces both symptoms of ADHD and negative parenting practices (Evans, Vallano, & Pelham, 1994). However, Chronis-Tuscano et al. (2012) demonstrated that pharmacological treatment of parents with ADHD may be necessary but not sufficient, and it may be most prudent to combine pharmacological and psychosocial treatments to promote sustainability.

Another option for treating families with multi-generational ADHD is to transition to a step-down approach. That is, once treatment dosage reaches effective levels, the frequency of treatment sessions could systematically be reduced. For example, treatment could transition from weekly to quarterly sessions. Also, as suggested by this study, offering parents more psychoeducation about ADHD (e.g., discussing the causes of ADHD and the potential impact of parental ADHD) and providing them frequent opportunities to receive and offer support to one another may help to maintain gains. Finally, the use of technologies, such as text messaging, smartphone applications, and web-based portals may prove useful in sustaining treatment effects (see Mautone, Carson & Power, in press). Investigating the effectiveness of these sustainability practices in isolation and combination is an important direction for future research.

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Figure 1.

Depiction of the pattern of change across FSS treatment and into follow-up comparing children with ADHD who had or did not have a parent with self-reported risk of ADHD in parent-reported homework productivity (HPC-PP) and in the parent-report of the quality of the parent-teacher relationship (PTIQ). Lower scores on the HPC-PP factor reflect better performance, and higher scores on the PTIQ factor reflect a stronger parent-teacher relationship.

Table 1

Descriptive information of about participants in the FSS and CARE sample who completed the CAARS.

Variable	CAARS <i>n</i> = 139	Parents < 4 ADHD Symptoms <i>n</i> = 116	Parents ≥ 4 ADHD Symptoms <i>n</i> = 23	Difference
Child Age, <i>M</i> (<i>SD</i>) ^a	9.33(1.25)	9.34(1.28)	9.26 (1.12)	<i>p</i> = .794
Child Sex, % female	33.1%	36.2%	17.4%	<i>p</i> = .080
Grade level, <i>M</i> (<i>SD</i>) ^a	3.42(1.18)	3.46 (1.22)	3.26 (.96)	<i>p</i> = .469
Single parent status, %	20.9%	22.4%	13.0%	<i>p</i> = .312
Parent SCL-90 Global Severity ^a	51.00(9.76)	49.23(9.25)	59.83(7.22)	<i>p</i> = .000
Ethnicity, % ^b				<i>p</i> = .506
Hispanic	9.4%	8.6%	13.0%	
Non-Hispanic	90.6%	91.4%	87.0%	
Race % ^b				<i>p</i> = .017
African American (AA)	18.7%	21.6%	4.3%	
White	76.3%	72.4%	95.7%	
Asian	1.4%	1.7%	-	
Multiracial	2.8%	4.3%	-	
SES, %, Levels III, IV, V on Hollingshead	98.6%	98.3%	100%	<i>p</i> = .526
ADHD, combined type, %	48.9%	47.4%	56.5%	<i>p</i> = .425
ADHD, inattentive type, %	51.1%	52.6%	43.5%	<i>p</i> = .425
Baseline Child ADHD Severity ^a	1.53(.46)	1.52(.47)	1.58(.41)	<i>p</i> = .559
Learning disability status, % with disability	26.6%	26.7%	26.1%	<i>p</i> = .701
Externalizing disorder, % with disorder	25.2%	25.0%	26.0%	<i>p</i> = .913
Internalizing disorder, % with disorder	22.3%	23.3%	17.4%	<i>p</i> = .536
Medication status at baseline, % on medication	39.6%	38.8%	43.5%	<i>p</i> = .675

Note. SES refers to socioeconomic status, as assessed by the Hollingshead (1975) index of social status. Levels III, IV, and V reflect the middle to high levels of the scale.

^aIndependent samples t tests were used to compute these comparisons. For all other comparisons, chi-square tests were used.

^bChi-square test for ethnicity compared Hispanic to non-Hispanic; Chi-square test for race compared white to non-white.

Table 2

Correlations between predictors and outcome variables at post and follow-up.

Predictor	Post Outcomes ^a									
	PES	PCRQ-PI	PCRQ-NI	PTIQ	HPC-1A	HPC-PP	HPQ	APRS	SNAP-P	SNAP-T
Medication Status	.059	-.035	.027	.197*	.003	.111	.050	.253**	-.054	-.220*
Child Gender	-.038	.041	.149 ^T	-.039	.126	.177*	-.173*	-.021	.143 ^T	.206*
SES	.133	.115	.060	.040	.079	-.108	.202*	.106	.014	-.182*
Parent Global Severity	-.141	-.244**	.112	.063	.049	.027	.099	.068	.144	-.046
Child Baseline ADHD Symptoms	-.165 ^T	-.089	.151 ^T	-.108	.254**	.277**	-.294**	-.413**	--	--
Dimensional Parent ADHD	-.026	-.132	.072	-.060	.066	-.083	.133	.059	.129	-.099
Dichotomous Parent ADHD	.031	-.057	.031	.101	.047	-.111	.051	.025	.083	-.052

Predictor	Follow-up Outcomes ^a									
	PES	PCRQ-PI	PCRQ-NI	PTIQ	HPC-1A	HPC-PP	HPQ	APRS	SNAP-P	SNAP-T
Medication Status	.114	.014	.031	.086	-.028	.027	.127	.131	-.032	-.085
Child Gender	.019	-.052	.061	.008	.049	.078	-.173*	-.132	.065	.249*
SES	.147 ^T	.118	.031	.016	.006	-.097	.288**	.171 ^T	-.005	-.210*
Parent Global Severity	-.170	-.091	.212*	.092	.082	.026	.073	.042	.161	-.132
Child Baseline ADHD Symptoms	-.133	-.078	.188*	-.047	.084	.072	-.301**	-.373**	--	--
Dimensional Parent ADHD	-.124	-.040	.145	-.028	.060	-.079	.075	.057	.091	-.099
Dichotomous Parent ADHD	.014	-.002	.054	-.029	.038	-.045	-.011	.025	.035	-.040

Note.

p<.01,

*<.05,

^T<.10;

Medication status was coded 0 = not on ADHD medication and 1 = on ADHD medication. Child sex was coded 0=female and 1=male. SES = socioeconomic status, as determined by the Hollingshead (1975) index (categories range from 1 to 5). PES = Parent as Educator Scale; PCRQ—PI = Parent–Child Relationship Questionnaire—Positive Involvement factor; PCRQ—NI = Parent–Child Relationship Questionnaire—Negative/Ineffective Discipline factor; PTIQ = Parent–Teacher Involvement Questionnaire; HPC—1A = Homework Problem Checklist, Inattention/Avoidance factor; HPC—PP = Homework Problem Checklist, Poor Productivity Factor; HPQ = Homework Performance Questionnaire–Student Responsibility Factor—Teacher Version; APRS = Academic Performance Rating Scale; SNAP = Swanson Nelson and Pelham ADHD Questionnaire, combining ADHD and ODD symptoms, (P=parent report, T=teacher report).

Table 3

Means and standard deviations for primary outcomes across three data collections periods across groups and parental ADHD status.

Measure	CARE Low Risk of Parental ADHD n = 62			FSS Low Risk of Parental ADHD n = 54			CARE High Risk of Parental ADHD n = 12			FSS High Risk of Parental ADHD n = 11		
	Baseline		Post	Baseline		Post	Baseline		Post	Baseline		Post
	M (SD)	Follow-up		M (SD)	Follow-up		M (SD)	Follow-up		M (SD)	Follow-up	
PES	3.39(.58)	3.77(.45)	3.81(.60)	3.28(.61)	3.97(.46)	3.94(.45)	3.13(.64)	3.85(.38)	4.08(.44)	3.32(.33)	3.93(.62)	3.68(.88)
PCRQ-PI	4.02(.37)	4.06(.32)	4.06(.39)	3.98(.43)	4.08(.43)	4.07(.41)	3.94(.51)	4.06(.38)	4.16(.38)	3.91(.31)	3.96(.35)	3.97(.30)
PCRQ-NI	2.92(.52)	2.74(.51)	2.57(.56)	2.89(.57)	2.36(.60)	2.35(.55)	3.19(.62)	2.69(.55)	2.41(.65)	3.01(.45)	2.53(.58)	2.72(.54)
PTIQ	3.00(.58)	3.03(.59)	3.01(.61)	2.94(.60)	3.23(.44)	3.05(.61)	3.15(.61)	3.11(.82)	3.20(.76)	3.24(.36)	3.31(.33)	2.90(.58)
HPC-IA	2.14(.52)	1.55(.57)	1.46(.60)	2.05(.55)	1.17(.56)	1.21(.52)	2.38(.34)	1.67(.38)	1.37(.56)	1.92(.59)	1.24(.45)	1.41(.61)
HPC-PP	.96(.56)	.64(.39)	.69(.55)	.91(.54)	.48(.35)	.54(.48)	1.05(.35)	.45(.26)	.36(.28)	.92(.40)	.49(.35)	.76(.61)
HPQ	2.70(1.17)	2.74(1.05)	2.84(1.19)	2.87(1.01)	3.09(.81)	3.11(.92)	3.31(.48)	3.21(.46)	3.32(.57)	2.75(1.02)	2.84(.71)	2.49(.91)
APRS	3.12(.74)	3.24(.68)	3.32(.81)	3.03(.66)	3.51(.66)	3.23(.70)	3.05(.44)	3.19(.43)	3.58(.42)	3.24(.61)	3.37(.65)	3.32(.79)
SNAP-P	1.49(.47)	1.24(.45)	1.15(.53)	1.45(.56)	.92(.45)	1.10(.56)	1.62(.47)	1.33(.46)	.96(.44)	1.44(.48)	1.23(.53)	1.24(.60)
SNAP-T	1.15(.68)	1.09(.68)	1.11(.81)	1.20(.54)	.93(.57)	1.09(.57)	1.06(.47)	.92(.42)	.88(.54)	1.15(.48)	1.10(.58)	1.04(.72)

Note.

PES = Parent as Educator Scale; PCRQ—PI = Parent–Child Relationship Questionnaire—Positive Involvement factor; PCRQ—NI = Parent–Child Relationship Questionnaire—Negative/Ineffective Discipline factor; PTIQ = Parent–Teacher Involvement Questionnaire; HPC—IA = Homework Problem Checklist, Inattention/Avoidance factor; HPC—PP = Homework Problem Checklist, Poor Productivity Factor; HPQ = Homework Performance Questionnaire—Teacher Version; APRS = Academic Performance Rating Scale; SNAP = Swanson Nelson and Pelham ADHD Questionnaire, (P-parent report, T-teacher report).

^aAdditional information for Means and SD (e.g., collapsed across groups) can be provided upon request

Table 4

Path coefficients for models with parental ADHD in multiple group analyses.

Dichotomous P-ADHD	Post Change				Follow-up Change			
	FSS	CARE	Model Fit χ^2 /diff test		FSS	CARE	Model Fit χ^2 /diff test	
Outcome	P-ADHD β	r^2	P-ADHD β	r^2	P-ADHD β	r^2	P-ADHD β	r^2
PES	-.01	.17*	.14 ^T	.30**	-.14	.31**	.16 ^T	.31**
PTIQ	-.06	.61**	-.06	.47**	-.34**	.45**	-.001	.46**
HPC-IA	.02	.18*	-.04	.42**	.20 ^T	.28*	-.15	.27**
HPC-PP	-.05	.39**	-.22*	.50**	.29**	.36**	-.11	.37**
HPQ	-.08	.70**	-.05	.68**	-.16 ^T	.65**	-.01	.62**
APRS	-.05	.76**	-.03	.65**	-.09	.54**	.11	.54**
								.287 ^T
								3.94*
								4.52*
								5.91*
								1.12
								2.87 ^T
Dimensional P-ADHD	Post Change				Follow-up Change			
	FSS	CARE	Model Fit χ^2 /diff test		FSS	CARE	Model Fit χ^2 /diff test	
Outcome	P-ADHD β	r^2	P-ADHD β	r^2	P-ADHD β	r^2	P-ADHD β	r^2
PES	-.11	.18*	.22*	.30**	-.16	.31**	-.07	.28**
PTIQ	-.15*	.64**	-.04	.46**	-.16	.41**	.02	.46**
HPC-IA	.17	.20*	-.09	.43**	.07	.25*	-.03	.24*
HPC-PP	.16	.41**	-.19 ^T	.49**	.24 ^T	.33**	-.14	.38**
HPQ	.01	.70**	-.06	.69**	-.24*	.65**	.04	.62**
APRS	-.06	.76**	-.04	.65**	-.11	.54**	.06	.53**
								1.67

Note.

** $p < .01$,* $p < .05$,^T $p < .10$.

χ^2 = total variance in outcomes assessed by full model.

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