



Children's ADHD Interventions and Parenting Stress: A Meta-Analysis

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Abstract

In order to increase our understanding of parenting stress, this study examined underutilized data on the effect of interventions for child ADHD on parenting stress. This project employed meta-analytic techniques to evaluate whether interventions for a child's attention-deficit hyperactivity disorder (ADHD) reduce parenting stress in families of children with ADHD, as reported on the Parenting Stress Index (PSI) or its variants. This study also assessed whether this efficacy varies based on whether the intervention is pharmacological or psychosocial, and within the psychosocial domain whether parents are directly involved or targeted in some way by the intervention. A number of parent, child, and study characteristics were also evaluated as potential moderators. A comprehensive search identified 43 (29 published, 14 unpublished) manuscripts relevant to the meta-analysis. Thirty-three studies compared levels of total parenting stress before and after treatment (within-subjects), producing a moderate effect size, $d = 0.50$, 95% CI [0.42, 0.59], $p < .001$. Nine studies compared total parenting stress between treatment and control groups (between-subjects), $d = 0.53$, 95% CI [0.33, 0.72]. Parent gender was the only statistically significant moderator, with studies with a larger proportion of mothers showing a larger effect, $Q = 5.44$, $p = .02$, $k = 17$. This study advances our understanding of parenting stress and identifies areas for future research, including research on fathers and longitudinal studies of families of children with ADHD.

Keywords Attention-deficit hyperactivity disorder · Parenting stress · Meta-analysis · Motherhood · Families

Introduction

Parenting stress (PS) is a distinct type of stress that arises when a parent's perceptions of the demands of the role of parenting outstrip his or her resources for dealing with them (Deater-Deckard 2004a). Abidin's theory of PS (Abidin 1976, as cited in Abidin 1995), although dated, continues to dominate the literature (Theule 2010). Abidin holds that PS is determined by parent factors (e.g., sense of competence), child factors (e.g., adaptability), and situational factors (e.g., role restriction). Life stressors are seen as factors that can exacerbate PS without having any direct effects on it. Extensive prior research has concluded that parents of children with attention-deficit/hyperactivity disorder (ADHD) experience greater levels of PS than parents of children without ADHD (for a meta-analysis see Theule

et al. 2013). Elevated levels of PS have been found to negatively affect the parent-child relationship, sense of parental efficacy, and parenting practices (Abidin 1992; Belsky 1984; Crnic and Ross 2017; Morgan et al. 2002; Rodgers 1998; Webster-Stratton 1990).

ADHD has prevalence rates of ~5% (Polanczyk et al. 2014), making it one of the most commonly diagnosed childhood disorders (American Psychiatric Association [APA] 2013). ADHD is a chronic, pervasive condition characterized by inattention, impulsivity, and hyperactivity that typically arises during childhood (APA 2013). In childhood especially, boys are more likely to be diagnosed with ADHD than girls (APA 2013; Kessler et al., 2006). Until recently, ADHD was considered a childhood disorder; however, recent research indicates that in most cases, ADHD is a lifelong disorder, existing in at least a partial remission form into adulthood (Barkley et al. 2002; Faraone et al. 2006). Current research points to strong genetic influences on the development of ADHD (Levy et al. 2006) and family studies have consistently found elevated rates of ADHD in the relatives of children with ADHD (e.g., Faraone et al. 2000). A recent meta-analysis indicated that 20%

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of parents of children with ADHD have ADHD themselves (Cheung and Theule 2016).

Parents of children with ADHD also have elevated rates of depressive disorders, anxiety disorders, and substance-related disorders compared with parents of children without ADHD (Cheung and Theule 2016; Lahey et al. 1988). Furthermore, some researchers have found a relationship between parental psychopathology and PS in families of children with ADHD (e.g., Anastopoulos et al. 1992; Breen and Barkley 1988; van der Oord et al. 2006) and early studies even equated parental psychopathology and PS (Gillberg et al. 1983; Sandberg et al. 1980). Notably, Abidin's seminal model of PS posits that parental depression is a key factor in PS (1995) and parental ADHD has also been strongly linked to PS in more recent work (Theule et al. 2011). This suggests that PS is not arising solely in response to child factors, including child ADHD, but is also impacted by parental factors, consistent with Abidin's theory (1995).

The dominant measure of PS (Crnic and Ross 2017; Johnston and Mash 2001; Theule et al. 2013) is the Parenting Stress Index (PSI; Abidin 1983, 1990, 1995, 2012). The full form of the PSI provides scores in two domains: child and parent domain stress, as well as a total stress score and a life stress score (Abidin 2012). The total stress score is the sum of the parent and child domain scores. Child domain stress refers to stress arising directly from factors within the child, whereas parent domain stress refers to stress related more to factors arising from within the parent.

Although experiencing some PS is considered normal (Crnic and Greenberg 1990), when it rises to clinical levels, it negatively impacts child development, in addition to affecting parenting (Deater-Deckard 2004b). Parents who experience extreme levels of PS may also be less able to implement interventions to help their children (Kazdin 1995). In contrast, reductions in PS are linked to increases in the efficacy of parent management training (PMT; Kazdin and Whitley 2003), a very commonly used intervention with children with ADHD (Kazdin 1997). Other commonly used interventions for children with ADHD include medication, child-focused interventions (e.g., cognitive-behavioural therapy or social skills training), and combined protocols where various aspects of the above three interventions are combined (Van der Oord et al. 2008). A number of reviews and meta-analyses on interventions for child ADHD have been recently completed (e.g., Corcoran and Dattalo 2006; Fabiano et al. 2009; Hodgson et al. 2014; Storebø et al. 2015; Van der Oord et al. 2008), but none have examined PS as an outcome variable.

Targeted and effective interventions for PS have been developed (e.g., Havighurst and Kehoe 2017; Kazdin and Whitley 2003; Treacy et al. 2005), but there is also a large body of research where interventions aimed at addressing a child's ADHD have tangentially assessed their impact on

PS. Logically, treatment for child ADHD may reduce demands on parents as a result of reduced child symptomatology, improved parenting practices, and/or reductions in co-occurring behaviour problems, thus decreasing PS. Given the variety of negative outcomes associated with elevated PS, it is important to comprehensively consider routes to its reduction. Specifically, we are interested in whether PS and child ADHD symptoms are bi-directionally related, such that efforts to reduce one, lead to a reduction in the other, without directly addressing other associated factors, such as parental psychopathology. Meta-analysis provides a well-suited opportunity to address this question given that there is a large body of research that has already peripherally addressed this question through the inclusion of the PSI, but without significant consideration of this particular element of the results. Through collating, integrating, and statistically analyzing the existing research on this topic, a meta-analysis will allow us to address this issue with much greater power than could be achieved in a primary study. Accordingly, our study assessed the overall efficacy of interventions for child ADHD on PS, as well as considered whether this efficacy varied based on whether the intervention was pharmacological or psychosocial, and within the psychosocial domain whether parents were directly involved or targeted in some way by the intervention. Considering variations in efficacy based on the format of the intervention (i.e., pharmacological vs. psychological, and parental involvement or not) is important in furthering our understanding of ADHD and PS, especially given the familial aspects of the disorder (Cheung and Theule 2016; Johnston and Mash 2001).

We also considered other factors relevant to ADHD and PS as moderators. These included parent and child gender, given differential rates of ADHD in boys and girls (APA 2013; Williamson and Johnston 2015), and the differential effects of parent gender with regards to ADHD (Johnston et al. 2013). Child age was also examined, given developmental changes in ADHD over time (e.g., Hart et al. 1995; Willoughby 2003). To address study quality, we considered other intervention characteristics including how long the study was conducted for (frequency, duration, and total contact hours), who it was provided by (e.g., psychologist, nurse, student), the measure of child ADHD, and publication status. Specifically, we examined the following research questions: (a) Is PS reduced by treatment of child ADHD? (b) Do pharmacological interventions differ in their reduction of PS in comparison to psychosocial interventions? (c) Does parental involvement in psychosocial interventions affect the reduction of PS? (d) What moderators affect the relationship between PS and children's ADHD treatment? The sociodemographic variables of gender and age of child and gender of parent were examined, as well as the publication status of the study, and

methodological moderators such as child ADHD measure, version of PSI, treatment duration, treatment frequency, and treatment provider.

Method

Search Strategy and Identification of Studies

The Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines were followed in terms of the identification, screening, and eligibility of the reports included in the study (Moher et al. 2009). We searched the following six databases systematically for both published and unpublished reports prepared up to July 2016: PsycINFO, Medline, Educational Resources Information Center (ERIC), Dissertations & Theses (ProQuest), EMBASE, and Google Scholar. Both published and unpublished reports were included in this study to reduce possible publication bias, where significant results are more likely to be published resulting in an overestimation of effect size (Card, 2012; Dwan et al. 2008; Moher et al. 2009). The following keywords were used in Boolean searches in all databases to obtain relevant articles: attention-deficit/hyperactivity disorder, attention deficit disorder, attention deficit disorder with hyperactivity, parent, parenting, parental, stress, distress, caregiver burden, and burnout. Attempts were also made to obtain additional studies by checking relevant review articles, books, and conference proceedings.

The articles produced by the searches were screened for eligibility in two stages. First, based on the title and abstract, studies that were not relevant and/or did not adhere to the criteria listed below were excluded. Studies that passed title and abstract screening were reviewed based on their full-text. The number of articles that were included and excluded at each stage was recorded and is presented in the PRISMA flow diagram (Fig. 1). Furthermore, the reference list of each eligible manuscript was reviewed to ensure no eligible studies were missed, and any articles citing an eligible article were also reviewed for eligibility (Card, 2012).

Criteria for Study Selection

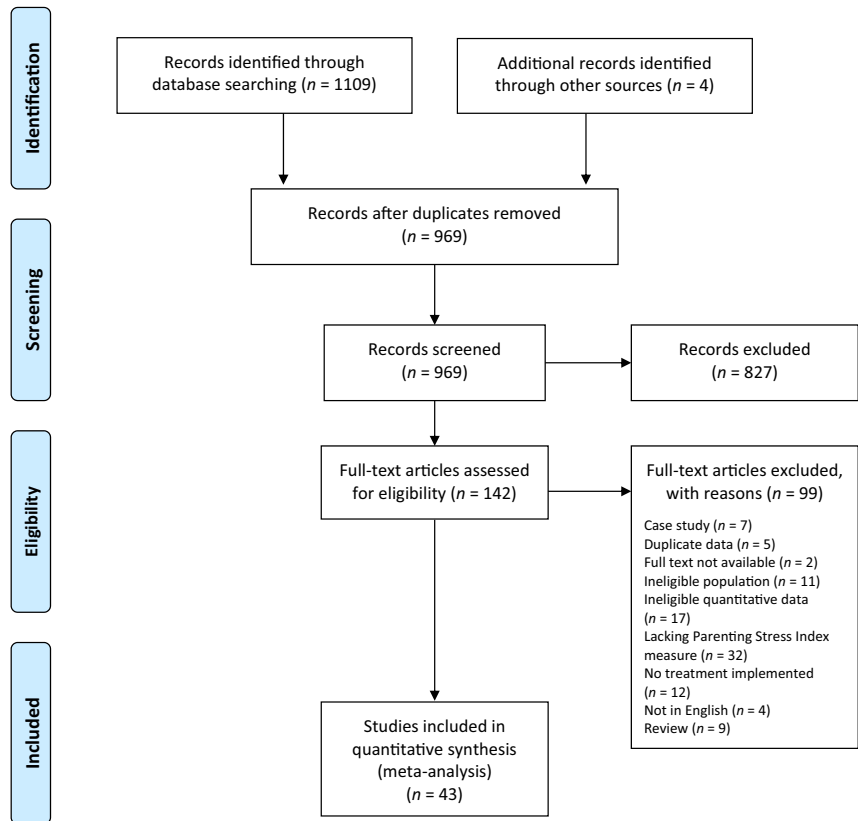
Studies were eligible for inclusion in the analysis if they were published or prepared in English up to July 2016, implemented an intervention for child/adolescent ADHD, and quantitatively reported on the outcome with respect to PS. To ensure that the studies included in our meta-analysis could be fairly compared with one another we restricted ourselves to studies using the PSI, its variants, and derivatives (e.g., the PSI-Short Form, the Stress Index for Parents of Adolescents [SIPA; Sheras et al. 1998]). Of note, Theule

et al.'s 2013 meta-analysis on ADHD and PS found that the PSI and its variants were used in 37 of their 44 included studies. As a result, we anticipated that this small trade-off in sample size would be justified by this greater consistency in outcomes. Restricting ourselves to the PSI and its variants also acted as a basic marker of primary study quality, given this measure's strong psychometric properties (Clare 2014; Young 2014). Furthermore, included studies must have been restricted to children diagnosed with ADHD, attention deficit disorder, or hyperactivity by a qualified health professional, or have restricted its participants to those falling in the clinical range on a standardized assessment measure of ADHD. They must have used a pre/post or intervention/control design. Using a PICO (Population, Intervention, Comparison, Outcome) framework (Melnik and Fineout-Overholt, 2005), the population was parents of children (under 18 years) with ADHD; the intervention was psychosocial and/or pharmacological treatments targeting child ADHD; the comparison was either within-subjects (i.e., pre-post designs), or between-subjects (i.e., treatment-control); and the outcome was PS as measured by the PS Index or its variants. We elected to include both within-subjects (uncontrolled) and between-subjects (controlled) studies to maximize our sample size, but separated them in the analysis for consistency in results. Moreover, this separation enables readers who would prefer to focus on the more externally valid between-subjects findings to do so, while enabling us to maximize our sample size and power to detect an effect by the inclusion of the within-subjects studies (which formed the majority of studies included).

Data Extraction and Missing Data

The data from each of the eligible studies was recorded and entered into Comprehensive Meta-Analysis (CMA) software 3.0 (Borenstein et al. 2013) for analysis. When multiple reports of the same study were available, the most comprehensive report was used to ensure independence between data points. If a potential study met all other eligibility criteria, but did not provide sufficient data to calculate an effect size, an effort was made to contact the principal investigator or proxy to request the missing data (Card, 2012). Missing outcome data or clarification regarding sample sizes were requested from four authors via email. Two of the four authors provided the necessary data to be included in this meta-analysis (Chacko 2006; Youcha et al. 2010). Data was coded using a formal coding manual and form (available upon request from the first author). Two coders worked on this project. The first was a doctoral graduate student with extensive previous experience in conducting meta-analyses. The second was an undergraduate student in psychology thoroughly trained in meta-analytic procedure by an experienced faculty member.

Fig. 1 PRISMA Flow Diagram



Reliability

Reliability was assessed at two stages of the meta-analysis. The searches were verified for extraction reliability by having a second searcher review all eligible studies in an effort to find others that cited studies already deemed eligible or were listed as references for other studies deemed eligible. Only four additional records were identified in this manner, supporting the thoroughness of the searches. To assess for intercoder reliability, a secondary coder coded ~25% ($n = 12$) of the eligible studies. There were no disagreements between the primary coder and the secondary coder (100% agreement on all variables).

Statistical Analysis

Given that heterogeneity across the studies was assumed, a random effects model was used throughout the analyses (Borenstein et al. 2010; Lipsey and Wilson, 2001). Individual meta-analyses were performed to address each of the research objectives. First, a standardized mean difference was used to analyze whether PS was reduced by treatment of child ADHD. Univariate meta-regression using a mixed effects model (Method of Moments) was used to address the remaining research questions, all of which investigated sources of variability in PS reduction after child treatment.

Results

Study Characteristics

Search results from six databases produced 969 unique articles. Secondary search strategies identified four additional eligible studies (Ghorbanshirodi 2015; Gonring 2016; Haydicky 2014; van den Hoofdakker et al. 2007). One-hundred-forty-two full-text articles were reviewed and screened for eligibility. Forty-three (29 published, 14 unpublished) manuscripts met the criteria to be included in the meta-analysis (Fig. 1). These studies represented a total sample of 1677 participants. Ninety-nine studies were excluded. Specific reasons for each exclusion are available in Fig. 1 (further details are available from the first author upon request). Included studies were published from 1992 through 2016. Seventy-nine percent of the studies were conducted in North America ($n = 34$), with the majority of the studies conducted in the U.S. ($n = 29$) and five studies conducted in Canada. The remaining studies were conducted in the Netherlands ($n = 3$), Iran ($n = 3$), South Korea ($n = 2$), and New Zealand ($n = 1$).

The percentage of mothers in the samples in each study ranged from 50 to 100%. The percentage of girls in the samples in each study ranged from 0 to 50%. The age range in the samples of children was 4–18 years. The majority of

Table 1 Participant characteristics of included studies

| Study | <i>N</i> ^a | Child age in years <i>M</i> (<i>SD</i>) ^b | % Child female ^b | % Mothers ^b | Type of intervention | Parental involvement |
|----------------------------|-----------------------|--|-----------------------------|------------------------|----------------------|----------------------|
| Abikoff et al. (2007) | 78 | 4.39 (0.72) | 19.67 | 50.43 | Pharmacological | No |
| Alaniz (2011) | 21 | 7.88 (1.84) | 28.57 | 71.43 | Psychosocial | Yes |
| Aman (2001) | 61 | 7–12 | – | – | Psychosocial | Yes |
| Anastopoulos et al. (1993) | 34 | 8.14 (1.06) | 26.47 | 100.00 | Psychosocial | Yes |
| Atamanoff Gambert (2008) | 14 | – | – | – | Psychosocial | Yes |
| Barkley et al. (2000) | 158 | 4.8 (0.4); 4.8 (0.5); 4.9 (0.5) | 29.31 | – | Psychosocial | Yes |
| Bakhshayesh et al. (2015) | 36 | 6–12 | 0.00 | 100.00 | Psychosocial | Yes ^c |
| Chacko (2006) | 120 | – | 28.75 | 100.00 | Psychosocial | Yes |
| Chacko et al. (2008) | 12 | 9.0 (2.5) | 16.67 | 100.00 | Psychosocial | Yes |
| Corkum et al. (2005) | 28 | – | – | – | Psychosocial | Yes |
| Corrin (2004) | 55 | 6.62 (N/A) | 29.09 | – | Psychosocial | Yes ^d |
| Danforth (1998) | 8 | 5.7 (N/A) | 37.50 | 100.00 | Psychosocial | Yes |
| Danforth et al. (2006) | 42 | – | – | 100.00 | Psychosocial | Yes |
| Driskill (1999) | 89 | 7–12 | 30.00 | – | Psychosocial | Yes |
| Gerdes et al. (2012) | 20 | 7.85 (1.53) | 25.00 | 100.00 | Psychosocial | Yes |
| Ghorbansirodi (2015) | 60 | – | – | 50.00 | Psychosocial | Yes |
| Gonring (2016) | 25 | 12.5 (0.8) | 28.00 | 76.00 | Psychosocial | Yes |
| Hall (2003) | 21 | 6.38 (1.31) | 28.00 | 50.00 | Psychosocial | Yes |
| Hauch (2005) | 19 | 4.08–7.92 | 21.05 | 50.00 | Psychosocial | Yes ^c |
| Haydicky (2014) | 17 | 15.50 (1.58) | 27.78 | 94.12 | Psychosocial | Yes |
| Heath et al. (2015) | 43 | 7–12 | 20.93 | – | Psychosocial | Yes |
| Hwang et al. (2013) | 495 | 10.4 (2.8) | 21.0 | – | Pharmacological | No |
| Isler (2003) | 9 | 6.37(1.3); 6.78(1.2) | 47.06 | – | Psychosocial | Yes ^d |
| Jones (2000) | 39 | 8.84 (2.25) | 7.69 | 100.00 | Pharmacological | No |
| Kim et al. (2013) | 132 | 8.82 (1.47) | 18.18 | – | Pharmacological | No |
| Lamb (2006) | 36 | 8.7 (4.79) | 31.6 | 68.2 | Psychosocial | Yes |
| Lehner-Dua (2002) | 48 | 6–10 | 31.25 | 70.83 | Pharmacological | No |
| McGoey et al. (2005) | 57 | 3.97 (0.71) | 12.03 | – | Psychosocial | Yes |
| Musten (1996) | 24 | 4–6 | 12.50 | – | Pharmacological | No |
| Newman (2000) | 17 | 5–13 | 9.52 | – | Psychosocial | Yes |
| Pisterman et al. (1992) | 46 | – | – | – | Psychosocial | Yes |
| Pouretmad et al. (2009) | 8 | 4–10 | – | 100.00 | Psychosocial | Yes |
| Reddy et al. (2002) | 50 | 6.25 (1.17) | 18.00 | – | Psychosocial | Yes |
| Springer (2004) | 51 | 6.22 (1.08) | 17.65 | – | Psychosocial | Yes ^c |
| Steele et al. (2006) | 145 | 6–12 | 16.55 | – | Pharmacological | No |
| Treacy et al. (2005) | 42 | 6–15 | – | 100.00 | Psychosocial | Yes |
| Tse et al. (2015) | 37 | 9.15 (2.45) | 33.3 | – | Psychosocial | Yes |
| | 10 | 11–15 | – | 100.00 | Psychosocial | Yes |

Table 1 (continued)

| Study | <i>N</i> ^a | Child age in years <i>M</i> (<i>SD</i>) ^b | % Child female ^b | % Mothers ^b | Type of intervention | Parental involvement |
|-------------------------------------|-----------------------|--|-----------------------------|------------------------|----------------------------------|----------------------|
| van de Weijer-Bergsma et al. (2012) | | | | | | |
| van Den Hoofdakker et al. (2007) | 94 | 7.4 (1.9) | 19.1 | 52.81 | Psychosocial | Yes |
| van der Oord et al. (2007) | 45 | 9.76 (1.13) | 11.11 | – | Pharmacological and psychosocial | Yes ^d |
| Vitiello et al. (2007) | 140 | 4.4 (0.7) | 25.71 | – | Pharmacological | No |
| Wells et al. (2000) | 579 | 8.5 | – | 86.4 | Pharmacological and psychosocial | Yes ^c |
| Youcha et al. (2010) | 172 | – | 35.51 | 79.61 | Pharmacological | No |

^aThis sample size represents the total of the treatment group and the control group participants that were included in the analysis

^bThis demographic information is based on participants in the treatment group who completed the study; if this information was unavailable, demographic information representing the overall sample – including those in other treatment groups and/or those who dropped out of the study early – was used. This strategy was implemented to provide sample demographics that most accurately represented the participants included in the analysis. Age ranges are included for studies in which the mean age of the children in the sample is not available. If a study had multiple treatment groups and did not provide a total mean, means and standard deviations for each group are provided

^cOnly two of the three treatment conditions included parental involvement

^dOnly one of the two treatment conditions included parental involvement

the studies examined psychosocial interventions alone ($n = 31$; 72%). Nine studies examined pharmacological interventions alone (21%). Three studies examined combined psychosocial/pharmacological interventions (7%). Parents were involved in 32 of the studies (74%). See Table 1 for participant characteristics of each included study.

The majority of studies included a previous diagnosis of ADHD as part of the eligibility criteria for the child participants ($n = 39$; 91%). Many additionally used various questionnaires (e.g., Child Behavior Checklist, Behavior Assessment Scale for Children, Conners) or interviews (i.e., the Diagnostic Interview Schedule for Children) to confirm clinically significant symptoms of ADHD. To assess PS, 22 studies used various editions of the full form of the PSI, while 19 used the PSI-Short Form. Two studies used the Stress Index for Parents of Adolescents (SIPA), which is the upward extension of the PSI.

PS Reduction After Child Treatment

Thirty-three studies compared levels of total PS before and after treatment (within-subjects). They produced a moderate effect size, $d = 0.50$, 95% CI [0.42, 0.59], $p < .001$. See Table 2 for further information, including individual study weightings, and a listing of each study included in this analysis. Twenty-five studies compared child domain stress (on the PSI-SF called “difficult child,” on the SIPA called “adolescent domain”) before and after treatment, producing

a moderate effect size, $d = 0.71$, 95% CI [0.54, 0.88], $p < .001$, see Table 3. Twenty-four studies looked at parent domain stress (on the PSI-SF called “parental distress”) before and after treatment, again producing a moderate effect size, $d = 0.49$, 95% CI [0.31, 0.66], $p < .001$, see Table 4. Nine studies compared total PS between treatment and control groups (between-subjects). They also produced a moderate effect size, $d = 0.53$, 95% CI [0.33, 0.72], $p < .001$. See Table 5.

Moderators of PS Outcomes

A total of 13 moderator analyses were conducted using meta-regression (see Table 6). To maximize power, all meta-regression was conducted using the pre-post (or between-subjects) data as it had a larger number of primary studies. All p -values reported are two-sided. These analyses sought to answer questions about variables associated with greater or lesser reductions in PS after treatment of child ADHD. Gender composition of the parent sample was the only moderator that was statistically significant ($Q = 5.44$, $p = .02$, $k = 17$), with samples with higher percentages of mothers having greater reductions in PS. Pharmacological interventions did not differ from psychosocial interventions in their efficacy, nor did parental involvement in the therapy in the case of psychosocial interventions. Neither of the child sample moderators (age, gender) examined were significant predictors. In terms of study quality/characteristics

Table 2 Meta-analysis using pre- and post-treatment data: total stress score

| Study | Mean effect size <i>d</i> | <i>p</i> -value | 95% Confidence interval | | Effect size weightings |
|-------------------------------------|---------------------------|-----------------|-------------------------|-------------|------------------------|
| | | | Lower limit | Upper limit | |
| Abikoff et al. (2007) | 0.26 | .212 | −0.15 | 0.68 | 3.22 |
| Alaniz (2011) | 0.84 | .009 | 0.21 | 1.47 | 1.59 |
| Aman (2001) | 0.20 | .271 | −0.16 | 0.56 | 4.06 |
| Anastopoulos et al. (1993) | 1.29 | .000 | 0.59 | 1.99 | 1.32 |
| Atamanoff Gambert (2008) | 0.48 | .169 | −0.20 | 1.16 | 1.40 |
| Chacko (2006) | 0.45 | .005 | 0.13 | 0.76 | 4.83 |
| Chacko et al. (2008) | 0.29 | .526 | −0.60 | 1.17 | 0.86 |
| Corkum et al. (2005) | 0.93 | .001 | 0.38 | 1.48 | 2.02 |
| Corrin (2004) | 0.41 | .043 | 0.01 | 0.81 | 3.44 |
| Danforth et al. (2006) | 0.71 | .002 | 0.27 | 1.15 | 2.93 |
| Driskill (1999) | 0.32 | .085 | −0.04 | 0.68 | 3.98 |
| Gerdes et al. (2012) | 0.44 | .168 | −0.19 | 1.07 | 1.61 |
| Ghorbanshirodi (2015) | 1.48 | <.001 | 0.91 | 2.05 | 1.90 |
| Gonring (2016) | 0.33 | .246 | −0.23 | 0.89 | 1.97 |
| Hall (2003) | 0.51 | .059 | −0.02 | 1.03 | 2.20 |
| Hauch (2005) | 0.56 | .090 | −0.09 | 1.21 | 1.51 |
| Haydicky (2014) | 0.05 | .892 | −0.62 | 0.71 | 1.46 |
| Heath et al. (2015) | 0.54 | .014 | 0.11 | 0.97 | 3.04 |
| Hwang et al. (2013) | 0.50 | .000 | 0.36 | 0.64 | 10.35 |
| Isler (2003) | 0.35 | .483 | −0.62 | 1.31 | 0.73 |
| Jones (2000) | 0.50 | .030 | 0.05 | 0.95 | 2.83 |
| Kim et al. (2013) | 0.74 | <.001 | 0.49 | 1.00 | 6.32 |
| Lamb (2006) | 0.11 | .706 | −0.48 | 0.71 | 1.78 |
| Lehner-Dua (2002) | 0.57 | .054 | −0.01 | 1.15 | 1.86 |
| Newman (2000) | 0.29 | .407 | −0.39 | 0.96 | 1.41 |
| Reddy et al. (2002) | 0.55 | .009 | 0.14 | 0.97 | 3.23 |
| Springer (2004) | 0.42 | .037 | 0.02 | 0.81 | 3.51 |
| Steele et al. (2006) | 0.51 | <.001 | 0.28 | 0.74 | 6.89 |
| Tse et al. (2015) | 0.38 | .104 | −0.08 | 0.84 | 2.74 |
| van de Weijer-Bergsma et al. (2012) | 0.47 | .298 | −0.42 | 1.36 | 0.85 |
| van der Oord et al. (2007) | 0.48 | .024 | 0.06 | 0.90 | 3.17 |
| Vitiello et al. (2007) | 0.13 | .416 | −0.18 | 0.45 | 4.82 |
| Youcha et al. (2010) | 0.69 | <.001 | 0.43 | 0.95 | 6.16 |
| Weighted mean effect size | 0.50 | <.001 | 0.42 | 0.59 | |

variables, none of the publication status of the study, study year, measure of ADHD, PSI version, treatment duration (in days), treatment hours, treatment frequency, or treatment provider were statistically significant.

Discussion

The results of our meta-analysis indicate that child ADHD interventions reduce PS (as measured by the PSI) even

when it is not targeted specifically. Furthermore, it is reduced similarly in child and parent domains, as well as overall (total stress), suggesting the results are not restricted to child effects. That said, there are concerns in the literature that the Child Domain score on the PSI conflates child behaviour problems (including ADHD symptoms) and parenting stress (Theule et al. 2011). Pharmacological treatments were not more efficacious in reducing PS than psychosocial treatments. Furthermore, the inclusion of parents in treatment interventions did not promote better

Table 3 Meta-analysis using pre- and post-treatment data: child domain stress score

| Study | Mean effect size <i>d</i> | <i>p</i> -value | 95% Confidence interval | | Effect size weightings |
|----------------------------------|---------------------------|-----------------|-------------------------|-------------|------------------------|
| | | | Lower limit | Upper limit | |
| Aman (2001) | 0.15 | .403 | −0.20 | 0.51 | 5.20 |
| Anastopoulos et al. (1993) | 1.26 | <.001 | 0.57 | 1.96 | 3.17 |
| Atamanoff Gambert (2008) | 0.66 | .041 | 0.03 | 1.30 | 3.48 |
| Bakhshayesh et al. (2015) | 2.18 | <.001 | 1.55 | 2.82 | 3.47 |
| Corrin (2004) | 0.52 | .011 | 0.12 | 0.92 | 4.90 |
| Danforth (1998) | 1.16 | .032 | 0.10 | 2.22 | 1.87 |
| Gerdes et al. (2012) | 0.67 | .040 | 0.03 | 1.30 | 3.47 |
| Hall (2003) | 0.65 | .016 | 0.12 | 1.19 | 4.06 |
| Haydicky (2014) | 0.37 | .276 | −0.30 | 1.04 | 3.30 |
| Isler (2003) | 0.68 | .182 | −0.32 | 1.67 | 2.04 |
| Jones (2000) | 1.50 | <.001 | 1.00 | 2.00 | 4.24 |
| Kim et al. (2013) | 0.83 | <.001 | 0.58 | 1.09 | 5.83 |
| Lamb (2006) | 0.27 | .375 | −0.33 | 0.86 | 3.70 |
| Lehner-Dua (2002) | 0.62 | .036 | 0.04 | 1.20 | 3.78 |
| McGoey et al. (2005) | 0.59 | .074 | −0.06 | 1.24 | 3.40 |
| Musten (1996) | 0.74 | <.001 | 0.33 | 1.16 | 4.81 |
| Newman (2000) | 0.30 | .381 | −0.37 | 0.98 | 3.27 |
| Pisterman et al. (1992) | 0.98 | <.001 | 0.54 | 1.41 | 4.69 |
| Pouretamad et al. (2009) | 0.81 | .118 | −0.21 | 1.83 | 1.98 |
| Reddy et al. (2002) | 1.01 | <.001 | 0.57 | 1.44 | 4.67 |
| Springer (2004) | 0.64 | .002 | 0.25 | 1.04 | 4.91 |
| Treacy et al. (2005) | 1.01 | .007 | 0.28 | 1.73 | 3.03 |
| van Den Hoofdakker et al. (2007) | 0.52 | .014 | 0.11 | 0.93 | 4.83 |
| Vitiello et al. (2007) | 0.21 | .196 | −0.11 | 0.52 | 5.46 |
| Wells et al. (2000) | 0.32 | <.001 | 0.19 | 0.46 | 6.43 |
| Weighted mean effect size | 0.71 | <.001 | 0.54 | 0.88 | |

outcomes with respect to reducing PS. The one investigated moderator that was significant was parental gender. Specifically, the relationship between child ADHD treatment and PS reductions was strongest in samples with greater numbers of mothers. No other sociodemographic variables (child age, gender) were significant. None of the study quality/methodological moderators we investigated were significant. That is, publication status, publication year, ADHD measure, PSI version, study duration, study frequency, and treatment provider did not significantly affect the relationship between child ADHD treatment and PS outcomes.

Our findings suggest that child ADHD likely has a relationship to PS, beyond the effects of parental depression or parental ADHD (although of course impacts of these variables may also be present), given that there were no significant differences between studies that involved parents (which could result in PS changes through general positive therapeutic encounters) and those that did not. This finding

is consistent with the results of Williamson and Johnston (2017) who also found that parental ADHD symptoms are not sufficient to understand PS. That said, the very idea that help is being provided may reduce PS somewhat. These findings also confirm that PS is not synonymous with parental psychopathology as early studies held (Gillberg et al. 1983; Sandberg et al. 1980). Moreover, the reductions in PS seen here indicate that this construct is open to change, indicating that it can reasonably be a focus of intervention, consistent with the work of Treacy et al. (2005), Kazdin and Whitley (2003), and Havighurst and Kehoe (2017). It should be kept in mind, however, that we did not directly assess child response to treatment in our consideration of treatment efforts and PS reductions.

Similar to Treacy et al. (2005), we found greater reductions in PS in samples with larger percentages of mothers. This may be due to lower levels of pretreatment PS amongst fathers (see Theule et al. 2013) which results in a floor effect as suggested by Treacy et al. Indeed, one of our

Table 4 Meta-analysis using pre- and post-treatment data: parent domain stress score

| Study | Mean effect size <i>d</i> | <i>p</i> -value | 95% Confidence interval | | Effect size weightings |
|----------------------------------|---------------------------|-----------------|-------------------------|-------------|------------------------|
| | | | Lower limit | Upper limit | |
| Aman (2001) | 0.13 | .471 | −0.22 | 0.49 | 5.49 |
| Anastopoulos et al. (1993) | 0.82 | .015 | 0.16 | 1.48 | 3.56 |
| Atamanoff Gambert (2008) | 0.42 | .179 | −0.19 | 1.04 | 3.83 |
| Bakhshayesh et al. (2015) | 1.98 | <.001 | 1.32 | 2.64 | 3.56 |
| Corrin (2004) | 0.24 | .240 | −0.16 | 0.63 | 5.23 |
| Danforth (1998) | 2.04 | .001 | 0.83 | 3.25 | 1.66 |
| Gerdes et al. (2012) | 0.19 | .539 | −0.43 | 0.82 | 3.79 |
| Hall (2003) | 0.47 | .080 | −0.06 | 0.99 | 4.36 |
| Haydicky (2014) | 0.31 | .360 | −0.36 | 0.98 | 3.54 |
| Isler (2003) | 0.38 | .435 | −0.58 | 1.35 | 2.30 |
| Jones (2000) | 0.63 | .006 | 0.18 | 1.09 | 4.82 |
| Lamb (2006) | 0.04 | .885 | −0.55 | 0.63 | 3.96 |
| Lehner-Dua (2002) | 0.48 | .103 | −0.10 | 1.05 | 4.06 |
| McGoey et al. (2005) | 0.15 | .647 | −0.49 | 0.79 | 3.70 |
| Musten (1996) | 0.45 | .031 | 0.04 | 0.85 | 5.16 |
| Newman (2000) | 0.17 | .625 | −0.51 | 0.84 | 3.50 |
| Pisterman et al. (1992) | 0.59 | .006 | 0.17 | 1.01 | 5.07 |
| Pouretamad et al. (2009) | 0.66 | .198 | −0.35 | 1.67 | 2.17 |
| Reddy et al. (2002) | 0.65 | .002 | 0.23 | 1.07 | 5.06 |
| Springer (2004) | 0.21 | .283 | −0.18 | 0.60 | 5.26 |
| Treacy et al. (2005) | 2.90 | <.001 | 1.92 | 3.88 | 2.25 |
| van Den Hoofdakker et al. (2007) | 0.25 | .221 | −0.15 | 0.66 | 5.15 |
| Vitiello et al. (2007) | 0.07 | .671 | −0.25 | 0.38 | 5.77 |
| Wells et al. (2000) | 0.19 | .006 | 0.06 | 0.33 | 6.75 |
| Weighted mean effect size | 0.49 | <.001 | 0.31 | 0.66 | |

included studies (Gerdes et al. 2012) does present data separately by parent gender. Their findings show lower pretreatment stress levels among fathers than mothers, and a statistically significant drop in PS only in mothers, and not in fathers. Greater reductions in PS in samples with larger percentages of mothers may also be related to issues related to the match between child and parent ADHD symptoms, which vary by parent gender (see Williamson et al. 2017). That is, fathers typically show higher levels of ADHD symptoms than mothers, and so mothers with ADHD may be more impaired than fathers with ADHD.

The effects found in the current study were in the moderate range, which is striking given that reducing PS was not the target of intervention in most of the included studies. However, the effect sizes found here were generally smaller than those of Treacy et al. (2005; notably an included study) and Kazdin and Whitley (2003; which targeted a wider variety of child behaviours), who directly targeted PS, suggesting that although PS is reduced incidentally through child treatment, greater reductions can be

observed with direct intervention. That said, taken together, this may suggest that in cases of mild to moderate elevations in PS, it may be more efficient to address child ADHD symptoms alone than to also address PS specifically. We should also note here that we were unable to directly test the association between a reduction in child ADHD symptomology and decreases in parenting stress due to the unavailability of this data, and this marks a significant limitation of the current study.

Importantly, none of our study quality moderators were statistically significant, suggesting that we can have greater confidence in our results as none of these factors were significantly systematically impacting outcomes. Furthermore, our reliability was very strong, adding further confidence to our results. Another strength of the current meta-analysis is the decision to include unpublished research in our analyses. Of the 43 studies included in our meta-analysis, 14 were unpublished. We opted to include these works to reduce the effects of publication bias, whereby published studies are more likely to have statistically

Table 5 Meta-analysis using treatment and control group data: total stress score

| Study | Mean effect size d | p -value | 95% Confidence interval | | Effect size weightings |
|----------------------------|----------------------|------------|-------------------------|-------------|------------------------|
| | | | Lower limit | Upper limit | |
| Abikoff et al. (2007) | 0.32 | .170 | −0.14 | 0.77 | 10.82 |
| Anastopoulos et al. (1993) | 0.85 | .018 | 0.15 | 1.56 | 5.89 |
| Youcha et al. (2010) | 0.31 | .055 | −0.01 | 0.63 | 15.35 |
| Barkley (2000) | 0.36 | .006 | 0.10 | 0.61 | 17.90 |
| Chacko (2006) | 0.30 | .060 | −0.01 | 0.61 | 15.51 |
| Driskill (1999) | 0.77 | <.001 | 0.37 | 1.17 | 12.32 |
| Ghorbanshirodi (2015) | 1.26 | <.001 | 0.70 | 1.81 | 8.38 |
| Lamb (2006) | 0.46 | .185 | −0.22 | 1.14 | 6.27 |
| Lehner-Dua (2002) | 0.73 | .017 | 0.13 | 1.32 | 7.56 |
| Weighted Mean Effect Size | 0.53 | <.001 | 0.33 | 0.72 | |

Table 6 Moderator analyses

| Moderator | No. studies | Q | p -value | R^2 |
|--|-------------|-------|------------|-------|
| Type of intervention: pharmacological or psychological | 33 | 0.21 | .901 | −0.29 |
| Parental involvement | 27 | 0.02 | .880 | −0.16 |
| Gender composition of child sample (% girls) | 18 | 2.86 | .091 | 0.13 |
| Mean age of the child sample | 13 | 0.00 | .979 | −1.15 |
| Gender composition of parent sample (% mothers) | 17 | 5.44 | .020 | 1.00 |
| Publication Status | 33 | 1.99 | .159 | 0.02 |
| Publication Year | 33 | 0.88 | .350 | −0.01 |
| Measure of child ADHD | 25 | 3.75 | .289 | −0.08 |
| Parenting stress index version | 33 | 6.81 | .147 | 0.30 |
| Treatment duration (in days) | 22 | 3.14 | .925 | −1.67 |
| Total contact hours | 15 | 0.00 | .950 | −0.40 |
| Treatment frequency | 25 | 0.55 | .456 | −0.15 |
| Treatment provider | 19 | 13.60 | .137 | 1.00 |

significant effects. Unpublished studies, however, have not undergone peer review, thus raising potential concerns about study quality. In an attempt to address some of these concerns, we did conduct a number of moderator analyses to assess for systematic bias in matters of quality or publication – none were significant.

Overall, this study has added to our understanding of PS in families of children with ADHD by collating an under-considered and underexamined literature on the effects of child ADHD treatment on PS. Given the far-reaching effects of PS on both children and parents (see Deater-Deckard 2004a), it is important that we move forward with research in this area by further investigating the nature of PS amongst fathers. We need more research on levels of PS amongst fathers, the interaction between paternal role (i.e.,

as the primary caregiver or not) and paternal PS, and the role of family structure (e.g., single parenthood, blended families) in these relationships. Investigations into the role of ethnicity and socioeconomic status are also needed. Longitudinal research investigating the development and course of child ADHD symptoms, parental psychopathology (most notably parental depression and ADHD), and PS are critical to further understanding of this important construct. Furthermore, we need more research directly examining the role of reduced child ADHD symptoms in reduced PS.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval This article does not contain any studies with human participants performed by any of the authors.

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