

Characterizing the Magnitude of the Relation Between Self-Reported Childhood Parentification and Adult Psychopathology: A Meta-Analysis

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Several decades of research have shown that people who experience parentification in childhood are at an increased risk of experiencing psychopathology in adulthood. A meta-analysis was conducted to examine the magnitude of the relation between self-reported parentification experienced in childhood and psychopathology evidenced in adulthood. Results from 12 nonoverlapping studies ($N = 2,472$), which were conducted between 1984 and 2010, revealed a small significant but reliable effect ($r = .14$; 95% confidence interval = .10 to .18). Moderator analyses were performed to explore possible explanations for the variance evidenced between parentification and psychopathology. Moderators that were examined include population factors, methodological factors, and type of psychopathology. The present findings indicate that four factors—types of psychopathology, type of sample, race, and parentification measure used—moderated the relation between parentification and psychopathology. The meta-analytic findings that emerged highlight the need for additional empirical research. Possible explanations and clinical implications of the findings and directions for future research are considered. © 2011 Wiley Periodicals, Inc. *J Clin Psychol* 67:1–16, 2011.

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Several decades of research have shown that people who experience parentification in childhood are at an increased risk of experiencing psychopathology in adulthood. Parentification, or the experiences and processes where children take on instrumental and emotional roles and responsibilities usually reserved for adults, has long been linked with negative sequelae (Boszormenyi-Nagy & Spark, 1973). Over the past four decades, the parentification discourse has focused largely on associations with pathology, such as the following conditions and disorders: trauma, distress, and adversity (Alexander, 1992; Boszormenyi-Nagy & Spark, 1973; Hooper, Marotta, & Lanthier, 2008; Karpel, 1976; Lackie, 1999; Minuchin, Montalvo, Guerny, Rosman, & Schumer, 1967); eating disorders (Rowa, Kerig, & Geller, 2001); mood disorders (Shifren & Kachorek, 2003); substance use disorders (Carroll & Robinson, 2000; Chase, Demming, & Wells, 1998; Godsall, Jurkovic, Emshoff, Anderson, & Stanwyck, 2004); dissociative disorders (Jones & Wells, 1996; Wells & Jones, 1998); and personality disorders (Jones & Wells, 1996).

The above negative outcomes have long been discussed and almost accepted as axiomatic (Byng-Hall, 2008; Chase, 1999; Cree, 2003; Hooper, 2007b; Jurkovic, 1997). Moreover, 40 years after the phenomenon of parentification was first described, the clinical narrative reviews and empirical research on parentification remains primarily focused on pathology (Fitzgerald et al., 2008; Hooper, in press, 2007a,b; Telzer & Fuligni, 2009; Thirkield, 2002).

Thus, although psychopathology has been the overriding phenomenon discussed routinely in the parentification clinical literature, and examined empirically in the parentification research literature, the magnitude of these relations and the rigor of the studies on which these

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links have been advanced remain unclear. A meta-analysis study was needed to clarify the scope of the relation between parentification and psychopathology.

The current meta-analytic study examines the magnitude of the relation between self-reported childhood parentification and adulthood psychopathology using a narrow and specific operationalization of psychopathology (i.e., Axis I and Axis II disorders and symptoms from the *Diagnostic Statistic Manual of Mental Disorders Fourth Edition* (DSM-IV; American Psychiatric Association, 1994)). We contend that this study—the first meta-analytic study of parentification—will add significantly to the understanding of a long-held assumption regarding the deleterious effects and negative sequelae of childhood parentification. Moreover, we believe that the current study will allow clinical psychologists, mental health care providers, and researchers to synthesize and examine concurrently the extent to which the research and clinical community has overemphasized or underemphasized results from a single study.

In this article, we first briefly describe the literature base of the aforementioned associations between parentification and psychopathology. We then provide a detailed description and summary of the research design and results of the current study. Finally, we discuss the clinical implications of the findings and directions for future research.

Relation Between Parentification and Psychopathology

As previously mentioned, psychopathology is a common correlate of parentification (Chase, 1999). A considerable body of evidence suggests that the consequences of parentification are often destructive, crippling, traumatic, and negative (Alexander, Teti, & Anderson, 2000; Boszormenyi-Nagy & Spark, 1973; Byng-Hall, 2008; Chase; Hooper, Marotta, & Depuy, 2009; Jurkovic, 1997, 1998; L'Abate, 1998; Lackie, 1999). Explorations of this link have been undergirded by attachment theory and family systems theory and have generated a substantial amount of research. The most commonly explored disorders—both in empirical studies and in narrative summary reviews—have been mood, personality, and substance use disorders.

For example, in a recent study exploring the link between depression and parentification (caregiving), Shifren and Kachorek (2003) found that of the 24 participants, 42% ($n = 10$) of the participants received a score on the Center for Epidemiological Studies Depression Scale (CES-D) that suggested the presence of depression. The authors found that the amount of time since removal from the caregiving role did not affect the participants' current mental health, but the length of time the participants had served in a caregiving role was positively correlated with current mental health (Shifren & Kachorek, 2003). The authors also found that the participants' perceptions of warmth and empathy from their parents during their caregiving years were related to their current levels of mental health. The authors caution against drawing conclusions about all caregivers based on their findings because of the small sample size, self-selected participants, cross-sectional design, and data collection method of retrospective recall questions (Shifren & Kachorek, 2003). This link between childhood parentification and adult depression has been evinced in other studies, as well (Martin, 1995; Winter, 2000; Wolkin, 1984).

In their study of 360 undergraduate college students, Jones and Wells (1996) found support for their hypothesis that parentification would be positively correlated with "self-defeating characteristics" and "narcissistic personality characteristics" (p. 147). Similar significant positive relations were found in a follow-up study (Wells & Jones, 1998), comprised of 124 undergraduate students, that explored how splitting and dissociation might be related to childhood parentification. The authors' analyses suggested a positive relationship between parentification in childhood and "defensive splitting" in adulthood, but no relationship between parentification and dissociation (p. 336). Wells and Jones asserted that parentified children may not be able to fully separate and individuate themselves from their parents, possibly contributing to the development of self-defeating and narcissistic characteristics, which may, in turn, make those individuals more likely to use defensive splitting (i.e., "an attempt to separate our good and bad experiences" or competing psychological representations of self and others; Wells & Jones, 1998, p. 334) in adulthood as a way of sheltering themselves from anxiety and emotional injury. Byng-Hall (2008) suggested that these

characterological and personality traits and behaviors (i.e., dissociation and splitting) may be a way to deal with the competing demands pulling individuals in different directions.

Parental substance abuse is positively correlated with offspring substance abuse (Chassin, Pitts, DeLucia, & Todd, 1999). Parental substance abuse is also positively correlated with parentification (Carroll & Robinson, 2000; Chase et al., 1998; Godsall et al., 2004). Subsequently, and extending this link, researchers have hypothesized that parentification may make it more likely for some children to develop substance abuse disorders in their adolescent and adult years (Locke & Newcomb, 2004). The self-medication hypothesis posits that individuals who suffer maltreatment or neglect (such as parentification) in childhood leading to psychological distress in adolescence and adulthood may have a higher likelihood of using substances to self-medicate (Locke & Newcomb, 2004).

Rationale for Study Moderator Variables

A careful review of the literature reveals that demographic factors appear to influence the course and outcomes of parentification. Historically, gender, age, and race have been advanced as factors that help researchers better understand the parentification process and outcomes, although the results are equivocal (Burton, 2007; Chase, 1999; East, 2010; McMahon & Luthar, 2007), and the implications of race and ethnicity have been the least explored demographic factor (Anderson, 1999; East, 2010).

With regard to gender, females have been considered to be at a greater risk for deleterious outcomes as compared with their male counterparts (Burt, 1992; Chase, 1999; Jurkovic, 1997). In their study sample of 123 participants, Burnett, Jones, Bliwise, and Ross (2006) found that females were more likely to report parentification than males. In agreement, Aldridge (2006) reported that a child is more likely to face parentification if that child is female and her mother is ill, single, or out of work. Additionally, Byng-Hall (2008) asserted that girls are more likely to be parentified as children than are boys, and the results of a national survey of 6,178 young caregivers conducted in the United Kingdom found that girls reported higher involvement in all types of caregiving (Dearden & Becker, 2004).

However, the intuitive hypothesis that females may be more likely to be selected for, and more willing to engage in, some of the instrumental roles often evinced in parentification does not always hold up in the empirical literature base (Champion et al., 2009). For example, Kuperminc, Jurkovic, and Casey (2009) found no association between gender and outcomes evidenced in parentified adults. Additionally, Diaz and colleagues (Diaz, Siskowski, & Connors, 2007) found that parentified males had worse outcomes than parentified females.

In her study of 133 children from divorced families, Johnston (1990) also found that neither gender nor age was correlated with parentification. In contrast to the suggestion that girls tend to do more housework in stressed families, McMahon and Luthar (2007) asserted that there is no consistent data showing that girls do more housework than boys and that tasks tend to be divided among the genders based on the type of task. McMahon and Luthar also found from their study of 356 children living in inner-city poverty that eldest children were most often responsible for household chores and that only children were most often expected to provide emotional support for their mothers.

The findings relative to age and race are also mixed (Burton, 2007; Jurkovic, Morrell, & Thirkield, 1999). Some studies reveal that when the parentification experience and family process happens at an earlier age, the long-term outcomes are more severe and enduring. However, the premise that older children engaged in the role of parentification experience greater and more severe outcomes is seen in literature, as well. East (2010) suggests that there may be an interaction between age and gender: females provide more care for parents, grandparents, and other family members, and as age increases, rates of parentification increases for females but decreases for males. However, East concurs with other researchers (Champion et al., 2009; Chase, 1999; Gilligan, 1982) that the “so-called gender effect” could be a function of gender socialization, sex-role stereotypes, propensity to nurture, and societal

norms as well as possible underreporting by males; therefore, the true prevalence rates based on gender remains unclear.

Finally, cultural-specific and race-specific values, rituals, and norms may inform gender-focused behaviors, whereby both males and females may be equally likely to assume parentification roles in the family. For example, in some Asian American families, the parentification of males may be a part of the family psychology in preparation for, and expectation of, future leadership roles (Jurkovic, 1997).

Godsall et al. (2004) posited that because family boundaries and psychology in racial minority groups may not follow the same patterns as those of the majority population in the United States, parentification in racial minorities may not have a negative and adverse connotation and, thus, may not have pathological outcomes. Additionally, the differences in views of childhood across different cultures could shape the perceived consequences and benefits of parentification. Becker (2007) suggested that the Western view of childhood may be very different from that of other cultures, in that the Western culture views childhood as a sacred time that should be reserved for children to develop and that children should be protected by adults until they become adults and are able to make adequate decisions for themselves.

Jurkovic, Thirkield, and Morrell (2001) pointed out that the Black American participants in their study reported providing higher levels of instrumental caregiving within their family of origin during childhood than did White American participants, but that the Black American participants did not report higher levels of perceived unfairness than did their peers, who reported providing less instrumental caregiving during childhood. Similar to work conducted by McMahon and Luthar (2007), Jurkovic and colleagues (2001) concluded that the results in their study show differences in expected roles and responsibilities within various races.

In their study of 356 children living in inner-city poverty with their birth mothers, McMahon and Luthar (2007) concluded that a child's level of participation in caretaking too far below or above a moderate level may indicate disorder within a family living in such conditions. The authors suggest that children in these home environments may be expected to provide some level of emotional support to their mothers in addition to instrumental support, which may serve to bring the family together but may isolate children from those outside of the family (McMahon & Luthar, 2007).

Determining the impact of age, gender, and race on parentification may be difficult because of the multifactorial and multilayered nature of these factors, which are difficult to disentangle and do not allow for the study of discreet nonoverlapping factors. Given the variable nature of the link between demographic factors and parentification, we included these factors as possible moderators in the current meta-analysis.

We also considered the plethora of research related to the *type* of parentification. Two main types of parentification—emotional and instrumental—have been delineated in the literature (Hooper & Wallace, 2010; Jurkovic, 1997, 1998; Minuchin et al., 1967). For example, Minuchin and colleagues (1967) asserted that children can experience parentification in duties such as preparing meals, doing household chores, and handling financial matters (i.e., instrumental parentification) or in duties such as responding to emotional needs of the parent or siblings or acting as the peacemaker for the family (i.e., emotional parentification). Type of parentification has long been considered an important link to the severity and acuity of outcomes in adulthood, with emotional parentification being advanced as the more severe and pathological type of parentification (Byng-Hall, 2002, 2008). Therefore, we included type of parentification as a possible moderator in the present study.

Parentification Measures

A careful review of the literature (described above) on parentification reveals the two most commonly used instruments include the Parentification Questionnaire (PQ; Jurkovic & Thirkield, 1998) and the Parentification Scale (PS; Mika, Bergner, & Baum, 1987). These instruments are self-report measures of retrospective parentification PQ (Jurkovic &

Thirkield, 1998) and PS (Mika et al., 1987); moreover, both appear to have sound psychometric properties.

Specifically, the PQ (Jurkovic & Thirkield, 1998) is a 30-item, widely used self-report instrument that measures retrospectively three dimensions of perceived parentification: instrumental parentification, emotional parentification, and perceived fairness of the parentification process. Of the 30 items, 10 pertain to instrumental parentification, 10 pertain to emotional parentification, and 10 pertain to perceived fairness. Participants rate how true the statements are on a 5-point Likert scale, ranging from 1 (strongly disagree) to 5 (strongly agree). Higher scores reflect greater parentification and/or perceived fairness. Cronbach's alphas for the PQ scores have been reported to be in a range of .82 to .92 (Burnett, Jones, Bliwise, & Ross, 2006; Hooper & Wallace, 2010; Jurkovic, Thirkield, & Morrell, 2001; Kelley et al., 2007).

The PS (Mika et al., 1987) is the second most widely used instrument that captures parentification. Designed to assess parentification, the self-report assessment includes 30 items asking adults to indicate how often they fulfilled a particular adult responsibility (Mika et al., 1987). Cronbach's alphas for the PS scores have been reported to be in a range of .76 to .86 (Fitzgerald et al., 2008).

Significance of the Present Study

Notwithstanding the small number of studies uncovered in our systematic search (described later in this article) of published and unpublished data on the association between parentification and psychopathology, the present meta-analysis study adds to the literature in the following ways. First, it summarizes quantitatively the empirical data linking parentification with psychopathology. Second, it examines published and unpublished studies. Third, with an increased power, it examines moderators in the selected combined studies, which may reveal factors that buffer the effects of parentification on psychopathology not otherwise uncovered in single studies. Finally, given the ubiquitous nature of parentification in the family psychology literature, the absence of a meta-analysis on parentification is noteworthy. The present study fills this gap.

Aims of the Present Study

Using a meta-analysis methodological framework (Glass, 1977; Lipsey & Wilson, 2001), the primary aims of the present study were twofold: (a) to clarify the strength of the relation between parentification and psychopathology and (b) to identify factors that may moderate the relation between parentification and psychopathology. More specifically, informed by the empirical literature base, our meta-analysis addressed the following three questions:

- What is the magnitude of the relation between childhood parentification and adult psychopathology?
- What study population factors moderate the relation between childhood parentification and adult psychopathology?
- What study methodological factors moderate the relation between childhood parentification and adult psychopathology?

Method

Location of Studies

The purpose of our literature search was to locate the population of studies that have investigated the relation between childhood/adolescent experiences of parentification and adult psychopathology. We searched the following computerized databases from their origin up to May 2009: PubMed, PsychInfo, Social Service Abstracts, ERIC, and Dissertation Abstracts. Our search procedure identified articles whose titles or abstracts paired terms

related to parentification (parentification, parentified, adultification, spousification, role reversal, little parent, young caregiver, young carer, burdened children, invisible carers, boundary dissolution, or boundary distortion) with terms related to psychopathology (psychopathology, depression, alcohol use, substance use, substance use disorders [SUDs], dissociation, anxiety, posttraumatic stress disorder [PTSD], trauma, personality disorder, eating disorder, anorexia nervosa, or bulimia nervosa) as defined by the DSM-IV (American Psychiatric Association [APA], 1994). The parentification and psychopathology measures could be assessed either continuously or categorically.

We used the Social Science Citation Index to locate articles that had cited Boszormenyi-Nagy and Spark (1973), Minuchin et al. (1967), or Karpel (1976), which were seminal studies or seminal theoretical reviews of parentification. We examined the references of Byng-Hall (2008) and Earley and Cushway (2002), two comprehensive recent reviews of the literature on parentification. We performed hand-searches of the *American Journal of Family Therapy* (1989 to present) and the *Journal of Family Psychology* (1995 to present), two journals that are popular outlets for research on parentification. We also contacted authors who are engaged currently in the study of parentification to uncover any in press or unpublished manuscripts that might be included in the current study. Finally, we wrote the corresponding authors of the articles identified by these methods to obtain additional work in the area of parentification and psychopathology that were unpublished or that may have been missed by our search. These procedures identified a total of 1,946 potential studies for our meta-analysis.

Inclusion Criteria

Our next step was to determine whether the 1,946 studies fit the boundaries of our meta-analysis. The inclusion criteria were as follows:

- The study must contain a quantitative estimate of the relation between parentification and psychopathology.
- Psychopathology must be assessed on the individual who was parentified, rather than the parent doing the parentification.
- Psychopathology must be assessed on the individual when they are an adult. Studies that examined concurrent measurements of parentification and psychopathology were excluded because of potential response bias.
- The measurement of psychopathology had to be consistent with the DSM-IV (APA, 1994) definition and/or symptomatology of this construct. This included all of the disorders mentioned in the search criteria above.
- The article had to be written in English.

We next examined the abstracts of the identified articles to create a “reduced candidate list” of 104 articles, removing those that clearly did not contain data relevant to our meta-analysis. We then examined the full text of these 104 articles, finally locating 12 that fit our inclusion criteria and which reported enough relevant, complete, and extractable data to compute an effect size for the relation between child/adolescent parentification and adult psychopathology. Eligible studies included published articles as well as unpublished articles or dissertations.

Coding of Moderators

Two of the authors (NW and MLV) coded independently the moderator variables to establish inter-rater reliability. The authors used and were trained extensively on the double-coding recommendations and procedures put forward by Lipsey and Wilson (2001). A copy of the moderator codebook is provided in Appendix I. Estimates of reliability were calculated using the intraclass correlation for continuous variables and Cohen’s kappa (Cohen, 1960) for categorical variables. The study moderators required few inferences on the part of the coders, and so all had perfect reliability except for type of parentification, which had a reliability of .771. Inconsistencies on this variable were resolved through discussion with the first author (LMH).

Calculation of Effect Sizes

We used the Pearson correlation r as our effect size measure, representing the correlation between child/adolescent parentification and adult psychopathology. Positive effect sizes indicated that children who were self-reported higher rates of parentification had more psychopathological symptoms as adults. Each r was transformed to Zr using Fisher's r -to- Z transformation function prior to all analyses. Effect sizes were calculated with the assistance of DSTAT version 1.10 (Johnson, 1993) and Comprehensive Meta-Analysis version 2.2 (Borenstein, Hedges, Higgins, & Rothstein, 2006). During analysis, all effect sizes were weighted by the inverse of their variances.

Use of Fixed-Effects Analyses

When performing a meta-analysis, researchers have the choice of treating the effect of study as either a fixed or a random effect in their analyses. This choice influences the results in several ways (Lipsey & Wilson, 2001). Fixed-effects analyses are more powerful, but random-effects analyses allow the researcher to generalize more broadly from the results. The inferences based on fixed-effects analyses can only be applied to the specific studies that are included in the meta-analysis, whereas the inferences based on random-effects analyses can be applied to the broader population of studies, from which the meta-analytic sample is drawn (Lipsey & Wilson, 2001). Although modern meta-analysts have typically preferred random-effects analyses, we decided to use fixed-effects analyses because our small sample size would allow for only very inaccurate estimates of the random effects of study. In addition, the reduced power that accompanies random-effects analyses would make it difficult to draw conclusions from our results. Specifically, there are no significant moderators (all p 's > .05) when a random-effects model is used.

Results

Descriptive Analyses

The studies included in our analyses reported a mean gender composition of 82.7% women. The mean racial composition was 72.4% White, 17.5% Black, and 8.0% Hispanic. The mean age of participants was 29.1 years. Table 1 reports the sample size and overall effect size from each included study. The total sample size comprised 2,472 participants from 12 studies. Table 2 presents the summary effect size characteristics. All meta-analytic computations were performed using Comprehensive Meta-Analysis version 2.2 (Borenstein et al., 2006). Each r was transformed to Zr using Fisher's r -to- Z transformation function before aggregation, but was afterward transformed back to r for ease of presentation. Each effect size was weighted by

Table 1
Studies Included in the Meta-Analysis and Overall Effect Sizes From Included Studies

Study: first author	Year	N	Published or unpublished	Effect size
Dwyer	1991	56	Published	0.22
Fitzgerald	2008	499	Published	0.01
Hooper	2010	143	Published	0.20
Jones	1996	360	Published	0.22
Katz	2009	163	Published	0.27
Martin	1995	150	Unpublished	0.09
Mayseless	2004	128	Published	0.00
Rowa	2001	95	Published	0.42
Walker	2001	302	Unpublished	0.16
Wells	1998	124	Published	0.30
Winter	2000	93	Unpublished	0.12
Wolkin	1984	359	Unpublished	0.14

Table 2
 Summary Effect Size Characteristics

Number of studies	12
Number of participants	2,472
Median r	.17
Mean weighted r	.14
Test comparing r to 0	$Z = 7.10, p < .001$
95% confidence interval around mean weighted r	(.10, .18)
Range of r	(0, .42)
Heterogeneity	$Q_w = 35.49, p < .001, I^2 = 69\%$

the inverse of its variance when calculating the weighted mean effect size. The weighted mean effect size was .14, which would be classified as a small effect by Cohen's (1992) and Lipsey and Wilson's (2001) guidelines. This effect is significantly greater than zero, indicating that participants who self-reported higher levels of parentification as children or adolescents showed more psychopathological symptoms as adults. There was a significant amount of heterogeneity among the effect sizes, indicating that there was more variability in the effect sizes than we would expect due to random chance alone. The large amount of heterogeneity (based on the guidelines of Higgins, Thompson, & Deeks, 2003) suggests the need to explain the variability using either random effects analyses (for which we lack the need number of studies) or moderator analyses.

We created a funnel plot of the effect sizes to explore the possible influence of publication bias (Light & Pillemer, 1984). This plot is provided as Figure 1, and illustrates both the observed effect sizes as well as the unpublished effect sizes that were imputed using the trim-and-fill method (Duval & Tweedie, 2000). Including these estimated studies would the weighted mean effect size from .14 to .12. Although there is some evidence of publication bias (with the plot suggesting that there are two unpublished studies not included in our analyses), it does not appear to have altered our estimated mean effect size in a substantial fashion. This may be because of the relatively large number of unpublished studies in our sample.

Moderator Analyses

As mentioned above, the heterogeneity among the effect sizes suggests the need to perform moderator analyses to determine if variability in the effect sizes can be explained by study characteristics. There were seven moderators (see Tables 3 and 4) examined in the study: type of psychopathology, type of parentification, parentification measure, type of sample, and mean age, gender, and race.

We considered the categorical moderators type of psychopathology, type of parentification, parentification measure, and type of sample. We considered the continuous moderators mean age, percent female, percent White, and percent Black. The categorical moderators were examined by testing whether the between-group heterogeneity Q_b was significantly different from zero. This statistic follows a chi-square distribution and represents the variability in the effect sizes that can be explained by group differences. The continuous moderators were tested using meta-regression, where we determined whether the slope between the moderator and effect sizes was significantly different from zero. For more information on these analytic procedures, see DeCoster (2009) or Lipsey and Wilson (2001).

Tests of the categorical moderators are presented in Table 3. The type of psychopathology being assessed did appear to impact effect sizes. Parentification had the smallest relation with on mood disorders and alcohol use and had stronger relations with anxiety disorders, personality disorders, and eating disorders. Post-hoc analyses (based on Rosenthal & Rubin, 1986) show that the effect on eating disorders is significantly greater than the relations with mood, anxiety, or alcohol use disorders. The type of parentification being assessed did not appear to affect the strength of the relations, although this was likely because only a single study differentiated parentification type. The parentification measure significantly moderated

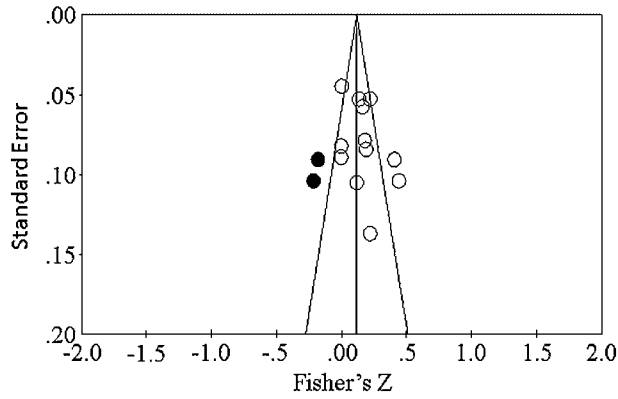


Figure 1. Funnel plot of standard error by Fisher's Z. Note: White circles represent observed data and black circles represent imputed data.

Table 3
Tests of Categorical Moderators

Type of psychopathology		
Mood disorder/depression	Number of studies	4
	<i>r</i> (95% CI)	.13 (.06, .20)
Anxiety disorder	Number of studies	1
	<i>r</i> (95% CI)	.22 (-.05, .46)
Alcohol use disorder	Number of studies	1
	<i>r</i> (95% CI)	.16 (.05, .27)
Personality disorder	Number of studies	3
	<i>r</i> (95% CI)	.24 (.16, .32)
Eating disorder	Number of studies	1
	<i>r</i> (95% CI)	.42 (.24, .57)
Test	<i>Qb</i> [4] = 10.85, <i>p</i> = .03	
Type of parentification		
Emotional	Number of studies	1
	<i>r</i> (95% CI)	.18 (.03, .33)
Undifferentiated	Number of studies	11
	<i>r</i> (95% CI)	.14 (.10, .18)
Test	<i>Qb</i> [1] = .25, <i>p</i> = .62	
Parentification measure		
PQ	Number of studies	5
	<i>r</i> (95% CI)	.21 (.15, .27)
PS	Number of studies	2
	<i>r</i> (95% CI)	.07 (.00, .13)
Other self-administered scale	Number of studies	2
	<i>r</i> (95% CI)	.27 (.15, .38)
Other interview measure	Number of studies	2
	<i>r</i> (95% CI)	.05 (-.08, .18)
Test	<i>Qb</i> [3] = 15.83, <i>p</i> = .001	
Type of sample		
Community	Number of studies	1
	<i>r</i> (95% CI)	.00 (-.17, .17)
Clinical	Number of studies	3
	<i>r</i> (95% CI)	.26 (.14, .38)
College student	Number of studies	8
	<i>r</i> (95% CI)	.14 (.10, .18)
Test	<i>Qb</i> [2] = 6.43, <i>p</i> = .04	

Note: CI = confidence interval; PQ = Parentification Questionnaire; PS = Parentification Scale.

Table 4
Tests of Continuous Moderators

Mean age	
Number of studies	10
Unstandardized slope (<i>SE</i>)	.0043 (.0027)
95% CI around slope	(-.0008, .0097)
Test of slope \neq 0	$Z = 1.65, p = .10$
% Female	
Number of studies	12
Unstandardized slope (<i>SE</i>)	-.0019 (.0011)
95% CI around slope	(-.0041, .0004)
Test of slope \neq 0	$Z = -1.65, p = .10$
% White Americans	
Number of studies	9
Unstandardized slope	-.0029 (.0013)
95% CI around slope	(-.0055, -.0002)
Test of slope \neq 0	$Z = -2.14, p = .03$
% Black Americans	
Number of studies	7
Unstandardized slope	.010 (.0027)
95% CI around slope	(.0049, .0154)
Test of slope \neq 0	$Z = 3.74, p < .001$

Note: SE = standard error; CI = confidence interval.

effect sizes. Post-hoc analyses showed that studies using the PQ (Jurkovic & Thirkield, 1998) had significantly stronger relations than those using the PS (Mika, Bergner, & Baum, 1987), and those studies using other self-administered scales had significantly stronger relations than those using other interview measures. Finally, there was a significant impact of sample on effect sizes, such that the relations of parentification were significantly stronger in clinical samples than in college student or community samples.

Tests of the continuous moderators are presented in Table 4. The mean age of the participants and the gender makeup of the sample did not significantly moderate effect sizes. The racial composition of the sample, however, did appear to relate to effect sizes. The relation between parentification and psychopathology was stronger in samples that had fewer White Americans or more Black Americans.

Relations Between Moderators

We conducted a series of analyses to determine the relations between the significant moderators (type of psychopathology, parentification measure, sample, percent White participants, and percent Black participants) to determine whether the observed results might have been caused by collinearity between the moderators. Unsurprisingly, we found a significant negative correlation ($r = -.84, p = .003$) between percent White participants and percent Black participants. We also observed significant relations of sample with percent White participants, $F(1, 10) = 8.75, p = .01$, and percent Black participants, $F(1, 8) = 6.21, p = .04$. On average, clinical samples had more Black participants and fewer White participants than college samples. None of the other relations between the moderators were significant.

Discussion

The purposes of the study were (a) to determine the magnitude and direction of the relation between parentification and psychopathology and (b) to establish the extent to which type of parentification and select population and methodological factors moderated the relation between parentification and psychopathology. Our primary finding—derived from aggregated data from 11 studies—was a significant positive small effect ($r = .14$), according to Cohen

(1992), between parentification in childhood and psychopathology in adulthood. This finding is consistent with a plethora of research. The strength and direction of the relation are also consistent with other studies.

Our second finding was the presence of significant moderator correlates of parentification and psychopathology. The results from our moderator analyses revealed four factors that explained the relation between parentification and psychopathology: one population factor (race) moderated this relation, and three methodological factors (parentification measure, type of sample, and type of psychopathology) moderated the relation.

These significant moderator factors were both expected and unexpected. For example, in accord with previous family systems literature (Alexander, 1992; Byng-Hall, 2008; Carroll & Robinson, 2000; Chase et al., 1998; Hooper et al., 2008; Jones & Wells, 1996; Rowa et al., 2001; Shifren & Kachorek, 2003; Wells & Jones, 1998), the results revealed a significant positive link between childhood parentification and adult eating, anxiety, and personality disorders. Greater levels of parentification portended greater levels of these disorders and symptomatology. Inconsistent with previous literature (Carroll & Robinson, 2000; Chase et al., 1998; Godsall et al., 2004), we found a weaker link between self-reported parentification and substance use and depression symptoms. The robustness of the link between these two disorders and parentification is discussed and empirically studied commonly in the family systems literature.

We also were surprised by the lack of significant moderating correlates related to the type of parentification. For the most part, empirical studies have shown a clear association between type of parentification and severity of outcomes, with emotional parentification being more deleterious than instrumental parentification (Hooper et al., 2008; Jurkovic, 1997). We conjecture that the absence of a significant finding is a function of the small number of studies. Thus, one possible explanation for these divergent outcomes—as compared with prior single studies—is that the present study is limited, as described in the next section.

In the present study, the results support long-held assumptions about correlates and outcomes of parentification. However, at the same time, the results fail to uncover relations often, but not always, reported in the literature (e.g., significant gender correlates related to parentification, differential findings based on type of parentification). The absence of a significant relation associated with gender may be explained by the number of male participants as compared to the number of female participants in the present meta-analysis. While there is some support for the differential findings of parentification based on gender, the research continues to be mixed, as described earlier in this paper. Future research with study samples consisting of larger percentages of male participants is needed. Possible mediators and moderators not included in the current investigation may disentangle the long-held assumption that females are at an increased risk for parentification and, thus, an increased risk for psychopathology following parentification in childhood.

Given the preliminary nature of these results, failure to continue to investigate outcomes associated with parentification would be significantly premature. The meta-analytic findings that emerged were both expected and unexpected and thereby highlight the need for additional methodologically sound empirical studies. Researchers are encouraged to continue investigating the relation between parentification and psychopathology with larger, culturally diverse samples (e.g., gender, race, geographical region).

Finally, although the current study considered the link between parentification and negative sequelae such as psychopathology, the empirical study of positive sequelae to parentification may be equally useful (East, 2010). Although relatively absent in the family systems literature, explorations related to positive sequelae of parentification are encouraged (Hooper, 2007b; Kuperminc et al., 2009; McMahon & Luthar, 2007; Shifren & Kachorek, 2003; Telzer & Fuligni, 2009). We believe a meta-analysis exploring the relation between parentification and positive outcomes (e.g., posttraumatic growth) and competency would be as useful for clinicians and practitioners as the current study, although such research may, too, be limited by a small number of studies. Importantly, more single studies and/or a meta-analysis may uncover important significant moderators that could serve as a focus of clinical interventions and treatment for individuals and families.

Limitations of the Study

We note several limitations of the present study that future researchers may address, including the issue of recall bias. Certainly, memory or motivational factors may affect accurate reporting. The stability of reporting of adverse events (as described by Briere & Conte, 1993), particularly when based on lifetime experiences, may be confounded by memory loss, underreporting, overreporting, or minimizing. Alternately, individuals may be unwilling to report events such as childhood abuse, trauma, or adversity (Baker, 2009; Brewin, Andrews, & Gotlib, 1993). First, our results are limited by the fact that the included studies were derived from cross-sectional data. Thus, causal directions of the relations evinced in the present study cannot be determined.

Second, because of the self-report nature of the data on which this study is based, caution is warranted when interpreting the findings. It is possible that other factors that were not measured or self-reported in the included studies can be implicated in the results.

Third, our findings are limited by the select population and methodological factors that were examined. Other factors that were not measured or examined (e.g., birth order, number of siblings where the parentification role and responsibilities may be shared, age of onset of the parentification role and responsibilities, duration and severity of parentification role and responsibilities) may account for the relation between parentification and psychopathology. Therefore, researchers ought to consider measuring additional variables in their research designs.

Fourth, the current meta-analysis was limited by the small number of studies, although combining studies in a meta-analysis increases the power and thus allows for the uncovering of significant differences when differences actually exist (i.e., Type II error). Nevertheless, the small number of studies remains a limitation of the study. Certain levels of our moderator variables (such as the type of psychopathology) have limited representation and so conclusions about them must be interpreted cautiously. Moreover, there are unmeasured variables they may account for the results of our study.

Fifth, we included studies that used different measures to capture parentification (e.g., PQ, PS, and so forth). It is possible that scores on the included instruments do not correspond explicitly with the parentification roles and responsibilities. Also, it is possible that the scores evidenced on these measures capture the construct of parentification differentially. Future studies may establish an inclusion criterion for studies that used one specific measure only. Additionally, studies are needed to examine the validity and reliability of the scores of instruments that measure parentification.

Finally, we used a fixed-effects approach to the data. Thus, we recognize that we cannot generalize the findings beyond the studies included in the meta-analysis. As more research accumulates on the relation between parentification and psychopathology, researchers should consider a random-effects approach, allowing for a broader application of the findings.

Implications for Practice

How might the current results inform clinical psychologists and mental health care providers working with adults who have been parentified? First, clinical psychologists ought to recognize that adults who have experienced parentification are at an increased risk for psychopathology. While childhood parentification does not foretell adult psychopathology in all situations, "parentification is a factor inherent in many forms of individual pathology" (Boszormenyi-Nagy & Spark, 1973, p. 165); thus, parentification must be included in the assessment of the family relational process. It is important for clinical psychologists and other mental health care providers to assess for level and duration of the parentification roles and responsibilities carried out in the family of origin (East & Weisner, 2009). A comprehensive assessment could inform and best match the appropriate scope and level of treatment and intervention services for effective patient care. This careful approach or strategy would also be helpful to clinical psychologists and clinicians in avoiding the overpathologization of the parentification process and related systems and outcomes. This overpathologization is a concern lamented by several

researchers and psychologists and family systems scholars (Earley & Cushway, 2002; Hooper, 2007b; Kuperminc et al., 2009).

Although we do not recommend that clinical psychologists and clinicians have a hyperfocus (i.e., over-zealousness) on parentification among Black American patients, it is noteworthy that in the present study, parentification had greater negative associations among Black participants than did White participants. This finding may point to the need for psychologists to consider treatments and intervention methods that may be culturally relevant and responsive to Black patients who have been parentified. Moreover, there may be differences in the therapeutic conversations about parentification based on racial and cultural factors. Thus, clinicians must consider how to culturally tailor their interventions to be equally relevant and sensitive to racially diverse patient populations.

Interestingly, the relation between parentification and psychopathology varied based on the specific disorder; these differential findings evidenced among the disorders was surprising, but may be explained partially by the small number of studies that examined these disorders. Other than encouraging clinical psychologists and clinicians to assess for parentification in conjunction with psychopathology, we are cautious in interpreting and translating this finding in the context of clinical practice. We believe it would be useful to continue to consider childhood parentification as a risk factor for adult psychopathology—irrespective of our specific disorder findings (e.g., anxiety, eating, mood, substance, and personality disorders).

As expected, the relation between parentification and psychopathology was stronger in clinical samples than in community or college samples. This finding is consistent with other studies. However, it is noteworthy that the relation was more pronounced for college student samples than for community samples.

Consonant with previous treatment recommendations, we believe there is a need to assess for parentification among adult patients who present with emotional distress and psychopathology symptoms. The PQ (Jurkovic & Thirkield, 1998) and the PS (Mika et al., 1987) affords clinical psychologists and mental health care providers two tools for assessing parentification. We also surmise that if adult patients report a history of parentification, there is merit in infusing family-of-origin-focused interventions and methods into treatment plans. Parentification is an important experience in the psychology of families. Therefore, parentification and its aftereffects are relevant to all family members. Thus, effective interventions and treatments are likely to include and be directed toward both individuals and family systems and subsystems.

Conclusion

The current meta-analysis of 2,329 adults assembled the first quantitative summary of the relation between parentification and psychopathology. Taken together, the results of the study are noteworthy given the small number of included studies. On the other hand, given the small number of included studies we want to underscore the preliminary nature and thus the clinical utility (significance) that may be derived from our study. Nonetheless, the results of the study buttress some of the long-held assumptions about the relation between parentification in childhood and psychopathology in adulthood. Although much is still unknown on this ubiquitous, often pernicious, family systems construct, the results of the current meta-analysis provide evidence to consider in clinical and research settings. This meta-analysis serves as a jumping off point in clarifying the scope of the relation between parentification and psychopathology, as well as indentifying factors that may buffer this relation.

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(References marked with an asterisk (*) indicate studies included in the meta-analysis.)

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