

Patterns of Self-Reported Alcohol Use, Depressive Symptoms, and Body Mass Index in a Family Sample: The Buffering Effects of Parentification

Lisa M. Hooper¹, Kirsten Doehler², Peter J. Jankowski³, and Sara E. Tomek¹

Abstract

Although the impact of parentification on children and adolescents' psychological health and outcomes has long been studied and well documented, little is known about the impact of parentification on children and adolescents' physical health and medical outcomes. Moreover, the potential buffering effects of parentification have been examined very rarely. The data in the current study were collected from an understudied, high-priority adolescent population ($N = 51$ rural adolescent-parent dyads). The authors examined the bivariate relations between parent health (alcohol use, depressive symptoms, and body mass index [BMI]), adolescent health (alcohol use, depressive symptoms, and BMI), and parentification. The effect size of the significant bivariate correlations ranged from small to large ($r = .29$ to $r = .62$). Parentification was positively associated with parent BMI and adolescent depressive symptoms. Parent alcohol use was strongly associated with adolescent alcohol use. Regression analyses were performed to determine if parentification moderates the relation between parental health and adolescent health. Parentification was found to function as a buffer of the relation between parent alcohol use and adolescent alcohol use. Parentification did not function as a moderator of the relation between parent depressive symptoms and adolescent depressive symptoms nor in the relationship between parent BMI and adolescent BMI. However, parentification did moderate the association between parent alcohol use and adolescent depressive symptoms.

Keywords

parentification, alcohol use, depressive symptoms, body mass index, parental health, adolescent health, tests of moderation, health risk factors

The transmission of psychological, emotional, and physical functioning and conditions from parent to child has long been described in the clinical and empirical literature (see Bowen, 1978; Bowlby, 1969; Minuchin, Montalvo, Guerney, Rosman, & Schumer, 1967). A family systems orientation suggests that various relationship, psychological, and physical conditions and patterns, though seen and measured at one level (e.g., parent level), can have lasting effects at other levels (child and grandchild level) and may be passed down from generation to generation—that is, in a multigenerational transmission process. This process, proposed by family systems theorists (see Bowen, 1978; Bowlby, 1969; Minuchin et al., 1967), can account for how psychological health and physical health conditions of parents can be evinced in their children and the adults those children become. The notion that parentification—the specific focus of the current study—is transmitted from one generation to another has also been empirically studied and discussed in the literature (Chase, Demming, & Wells, 1998; Hooper & Newman, 2011; Jurkovic, 1998; Macfie, McElwain, Houts, & Cox, 2005; Zeanah & Zeanah, 1989).

Because of its ubiquitous nature (Byng-Hall, 2008a; Hooper, 2007a, 2007b; Jurkovic, 1997), parentification may be an important risk factor, protective factor, and explanatory factor in understanding the transmission of psychological, emotional, and physical health functioning and symptoms from parent to adolescent. Family systems theory is an ideal theoretical framework to clarify the relation or transmission of physical and psychological health conditions from the parent generation to the adolescent generation in conjunction with parentification. For

¹ Department of Educational Psychology, The University of Alabama, Tuscaloosa, USA

² Department of Mathematics and Statistics, Elon University, Elon, NC, USA

³ Counseling Psychology Program, Bethel University, St. Paul, MN, USA

Corresponding Author:

Lisa M. Hooper, Department of Educational Psychology, Research Methodology, and Counseling, The University of Alabama, Box 870231, 315 Graves Hall, Tuscaloosa, AL 35487, USA
Email: lhooper@bamaed.ua.edu

example, Chase, Demming, and Wells (1998) emphasized the multigenerational nature of parentification. Specifically, Chase et al. defined parentification as the “dynamic in which the emotional deficits of a previous generation are inherited as emotional debts to be paid or resolved by the subsequent generation” (p. 105). Similar to Boszormenyi-Nagy and Spark (1973), Chase (1999) also asserted that parentification almost always involves wrongs enacted by previous generations that are carried forward into current generations, who must “settle the score” or “balance the ledger” (p. 10) for the aforementioned wrongs. These behaviors carried out by the younger generation traditionally require the child to take on the role of parent (a role sometimes termed adultification; see Burton, 2007) or mate (a role sometimes termed spousification; see Sroufe & Ward, 1980), or both, for his or her own parent/parents or family members. Parentification is defined in the current study as a type of role reversal, boundary distortion, and inverted hierarchy between parents and other family members in which adolescents assume developmentally inappropriate levels of responsibility in the family of origin (Boszormenyi-Nagy & Spark, 1973; Hooper, 2011; Jurkovic, 1997; Kerig, 2005; Minuchin et al., 1967).

Empirical research on the effects of parentification has accumulated over the past four decades (Boszormenyi-Nagy & Spark, 1973; Hooper, 2011; Minuchin et al., 1967). Overwhelmingly, the significant associations that have emerged over the past 40 years between parent psychological and physical health and adolescent parentification have been shown to be pervasive and deleterious. A few researchers have suggested that these significant associations may be a function of the level or “dose” of parentification experienced by children and adolescents (see Byng-Hall, 2008b; Hooper 2007b; Jurkovic, Thirkield, & Morrell, 2001; Stein, Rotheram-Borus, & Lester, 2007). In other words, not all parentification is equally deleterious; low levels of parentification may be beneficial, whereas high levels of parentification may account for the commonly reported link between parentification and psychopathology (Hooper, DeCoster, White, & Voltz, 2011). Therefore, recent researchers have been challenging the proposition that negative outcomes are the only possible outcomes that may be observed (see East, 2010; Gelman & Greer, 2011; Hooper, 2007b; Ireland & Pakenham, 2010a; Kelley et al., 2010; Pakenham, Chiu, Bursnall, & Cannon, 2007; Walker & Lee, 1998).

Although the impact of parentification on psychological functioning across generations has long been studied and well documented, little is known about the impact of parentification on children and adolescents’ physical health, health risk factors, and medical outcomes. Moreover, the protective buffering effects of parentification have been examined very rarely. Consequently, the present study may be useful in mapping the relations among parentification, parent health, and adolescent health. Using a family systems framework, the present investigation addressed these limitations in the empirical literature. The present study extends the literature base by focusing on an understudied, high-priority adolescent population, specifically by examining

parentification in a convenience sample of rural, racially diverse families.

Background

Parentification

Parentification, along with the related psychology of families, is a process, whereby parental roles and responsibilities are abdicated by parents and carried out by children and adolescents. Some scholars have asserted that the parentification process can be equated with a type of psychological control parents exert over their children (Barber, 1996, 2001). Parentification is a ubiquitous phenomenon often seen in a range of populations (Byng-Hall, 2008b; Chase, 1999; Hooper, 2007a, 2007b).

The term *parentification* was introduced by family systems theorists Minuchin and colleagues (1967), who asserted that in the process of parentification, “the parent(s) relinquishes executive functions by delegation of instrumental roles to a parental child or by total abandonment of the family psychologically and/or physically” (p. 219). The family system and structure in which parentification takes place often is by definition embedded in or composed of (a) subsystems (parental, child, and sibling) with loose or nonexistent boundaries (Kerig, 2005); (b) inverted hierarchies, where the child acts as the parent and the parent acts as the child (Minuchin et al., 1967); and (c) systems (family, community, and neighborhood) in which stressors (e.g., parents with serious medical conditions, divorce, significant crime, and low-resource communities), adversity, and trauma are often present (Burton, 2007; Garber, 2011; Hooper, 2007b; Stein, Riedel, & Rotheram-Borus, 1999).

Numerous reasons have been put forward as to why some parents may parentify their children. Three of the most commonly reported factors that are evidenced in the family psychology and adolescent health literature include (a) parents’ health and serious medical conditions, (b) parents’ psychological health and distress, and (c) parents’ use of alcohol and other substances (Stein et al., 1999). Overwhelmingly, the associations between parent psychological and physical health and adolescent parentification are pervasive and negative. The next subsections briefly describe empirical studies that have examined relations among parent health, adolescent health, and parentification.

Parent Alcohol Use and Parentification

A significant number of studies have examined the association between parent alcohol use and parentification experienced by adolescents and emerging adults. Often but not always, the research has revealed positive associations between parent alcohol use and parentification; that is, higher levels of parent alcohol use and dependence correlate with higher levels of parentification. For example, Bekir, McLellan, Childress, and Gariti (1993) found that alcohol, more often than not, serves as a precursor to the assigned spousal or parental role of the child. More specifically, they found that adults who abuse

alcohol or drugs often are unable to perform their parental duties, so the parentified child is consequently forced into a role providing self-care, sibling care, and parental care. Bekir et al. also found that the parentified child is often inclined to repeat the same behaviors with his or her own children, to use drugs and alcohol to self-medicate during stressful times, or to become overresponsible children and then later overresponsible adults. These findings support the idea that parentification-related roles, responsibilities, and processes may be transmitted across generations.

Chase et al. (1998) examined young adults who had assumed a parentified role in their family of origin and sought to determine, after grouping students according to levels of parental alcohol use, whether the degree or level of parentification would differ between the groups. Children of parents who abused alcohol (COA) were found to be more parentified than either non-COAs or children of problem drinkers. Chase et al. concluded that their findings cohered with other studies that have examined the association between parental alcohol use and parentification of children. For example, their findings were similar to the findings of Goglia, Jurkovic, Burt, and Burge-Callaway (1992). Goglia et al., using the same instruments, had also found that a higher level of participants' parentification scores (as measured by the Parentification Questionnaire) meant a greater likelihood that they were also COAs. Similarly, the results of a study by Godsall and colleagues (2004) uncovered those adolescents with parents who reported excessive use of alcohol had higher levels of parentification.

Walker and Lee (1998), on the other hand, strongly contended that researchers and clinicians must study and understand the wide-ranging types of families in which alcohol use and dependence are present. In their meta-analysis of the literature looking at the families with alcoholic members, they found that many researchers had only looked for and measured individuals from a deficit framework and had consequently failed to uncover the strengths of the population who experience both alcoholism and resilience in the family and its members, even parentified ones. Walker and Lee found in their review that some families and individual members actually become strong in the face of adversity and thus establish ways to cope effectively with family members who use alcohol and other substances. The other theme that emerged from Walker and Lee's systematic review was the automatic assumption that parentification is destructive in every family where alcohol use and dependence is present. They asserted that parentification may in fact be a positive, resourceful way for the family to cope temporarily with alcoholism in the family. Accordingly, Walker and Lee declared that the role reversal (i.e., parentification) that often coexists in an alcoholic family actually may engender individuation, adaptation, and an increase in a child's self-esteem. They concluded that an exploration of the protective side of parentification is needed. Like Walker and Lee (1998) and Jurkovic and Casey (2000) argued that moderate levels of parentification in childhood may be beneficial and may teach children how to be caring parents, responsible adults, and overall good caregivers in adulthood.

The current study is informed by these varied empirical outcomes and multiple points of view about the links between parent alcohol use, adolescent alcohol use, and parentification. The findings support the need to continue exploring the phenomenon of parent alcohol use and parentification so that correlates, antecedents, and consequences are delineated and so that, in the process, clinically and statistically significant factors are uncovered that may inform and be tested in future studies. The current study considers possible significant correlates among family systems factors and individual health factors. In addition, given the propensity for alcohol use in many families, uncovering its influence on health outcomes of children and adolescents, including the feasibility of buffering the effects of parental alcohol use, is an important area of study.

Parent Psychological Health and Parentification

Past research has found that parent psychological health (primarily that of mothers) plays a central and significant role in the phenomenon, whereby children or adolescents are forced into a parentified role in their family system (Carroll & Robinson, 2000; Champion et al., 2009; Garber, 2011; Hooper & Wallace, 2010; Stein et al., 2007). When parents experience mental illness such as depression, often they are unable to function fully or at all and are consequently unable to carry out their traditional roles and responsibilities in the family system (Burton, 2007; Earley, Cushway, & Cassidy, 2007; East, 2010; East & Weisner, 2009; Hooper, 2007a, 2007b; Shifren & Kachorek, 2003). Children and adolescents are then called upon to perform these family duties (Stein et al., 1999).

For example, Champion et al. (2009) described the impact of family caretaking behaviors on adolescents' functioning. Specifically, they examined the extent to which mothers' level of depression relates to types of adolescent caretaking behaviors, adolescents' level of depression, and adolescent competence. In their sample of 72 mother-adolescent dyads, they found positive associations between emotion-focused parentification roles and responsibilities, adolescent depression, and adolescent competence. Adolescents with mothers diagnosed with current depression or a history of depression reported higher levels of depression and anxiety symptoms. These adolescents also reported higher levels of emotional parentification. Interestingly, higher levels of parentification were also associated with higher levels of adolescent competence (as reported by mothers). This positive correlation may suggest that mothers are unaware of the potential burden on adolescents who are parentified, or alternatively, parentification may be associated with positive outcomes such as self-efficacy or competence (see Stein et al., 1999; Stein et al., 2007).

Parent Physical Health and Parentification

Numerous studies have examined how parents' (primarily mothers') physical health conditions (both chronic and unexpected) correlate with parentification-related roles and responsibilities. Past research has found that parents' physical health

conditions (Alzheimer's disease, cancer, dementia, HIV/AIDS) often thrust the child or adolescent into a parentified role in the family system (Banks et al., 2001; Duryea, 2007; Gates & Lackey, 1998; Gelman & Greer, 2011; Ireland & Pakenham, 2010b; Luecken & Lemery, 2004; Shifren, 2001; Stein et al., 1999; Svanberg, 2010).

In a recent case study report, Gelman and Greer (2011) described the impact that a diagnosis of early-onset Alzheimer's disease in one parent had on family members, including the parent without Alzheimer's. They uncovered common problems often suggested when parentification is evinced in the family system. Of significance, parentification was observed by the researchers and reported by both children (ages 12 and 16) in this case study family. For example, both children were required to supervise their father (the parent who was diagnosed with early-onset Alzheimer's disease) and to take over a significant number of chores, including laundry, cooking, and cleaning. They were also expected to provide companionship and emotional support to the mother, who was charged with being the sole breadwinner of the family. In the mother's absence, the 16-year-old daughter reported feeling overwhelmed, sad, and at times angry about being held responsible for managing the entire household. Everyone in this family reported a sense of loss and sadness as roles changed (e.g., both children became part of the parent subsystem) and were reconfigured, and as additional stressors were added because of the father's illness.

In another study, Ireland and Pakenham (2010b) investigated what factors serve as significant predictors of adjustment in youths whose parents had a serious physical illness or disability, compared to youths whose parents had a serious mental illness. Results related to poorer youth adjustment were differentiated by the type of parentification-related activities and by parents' type of illness. Significant predictors of youth adjustment included the onset of a parent's illness or disability (acute vs. delayed onset); the type of illness (physical vs. mental); gender of the child; and the parentification-related roles, responsibilities, and processes. With regard to parentification, youths who reported higher levels of isolation as a result of caring for parents, higher levels of discomfort in performing their caregiving role, lower levels of maturity, and higher levels of confidence in performing caregiving roles also reported lower levels of adjustment. Additionally, youths of parents with a self-reported mental illness experienced lower levels of adjustment than did youths of parents with a self-reported physical illness. Conversely, youths of parents with a physical illness reported greater levels of worry than did youths of parents with a mental illness. Findings from this study accorded with findings from another study conducted by Pakenham, Bursnall, Chiu, Cannon, and Okochi (2006).

Shifren (2001) also reported on the possible long-term effects when children and adolescents provide care for parents with serious health conditions. Shifren examined the current health status of participants who had cared for (i.e., provided physical assistance and emotional support for) parents who experienced cancer, Alzheimer's disease, arthritis, stroke,

Parkinson's disease, substance use, and concomitant mental health disorders. Shifren found that participants who cared for parents with mental health disorders and comorbid substance use reported higher depressive symptoms than did participants who cared for parents with only serious physical health conditions (e.g., cancer, Alzheimer's disease, arthritis, stroke, Parkinson's disease). Additionally, a vast majority of Shifren's small sample ($N = 12$) reported their caregiving experiences as more positive (as measured by well-being scores on the Center for Epidemiologic Studies Depression scale [CES-D]) than negative (as measured by depressive symptoms on the CES-D). Shifren concluded that the long-term effects of caring for parents and family members may not be "universally negative and pervasive" (p. 190). Shifren's conclusions are also supported by other researchers (Gelman & Greer, 2011; Hooper, 2011; Kam, 2011; Vernig, 2011).

In conjunction with the significant negative effects that may be engendered by caregiving for a parent with a serious medical condition, Gelman and Greer (2011) reported on possible positive impacts of the parentification process. Specifically, Gelman and Greer stated, "Children as young as 10 years old caring for parents with illnesses such as cancer, multiple sclerosis, and arthritis, have reported that caregiving strengthened their relationships, increased their insights into the illness, improved their spiritual growth, increased their coping skills, and gave them a sense of pride and identity" (p. 31).

Taken together, these studies consider the impact of the parents' physical health on the parentified role. Very few studies, if any, have examined adolescent physical health along with parent physical health and parentification in the same study. There has been an overwhelming focus on psychological sequelae when studying the effects of parentification. Notably absent are possible physical outcomes for children and adolescents (e.g., obesity, diabetes, and somatization) that may be associated with parentification (Hooper, 2011; Johnston, 1990; Luecken & Lemery, 2004; Pakenham et al., 2006; Shifren & Kachorek, 2003; Stein et al., 1999) underscoring the need for the current study.

Importantly, the current section reviewed studies that have examined the link between parentification and *other* health conditions rather than obesity and weight status. However, because obesity (as measured by body mass index [BMI]) has been linked to and reported as a physical health risk factor for many of the health conditions reviewed herein (e.g., see Field et al., 2001; Institute of Medicine, 2011; Must et al., 1999), we have included it as a proxy for physical health in the current study. Additionally, we have included BMI as an important health risk factor because depression and obesity (as measured by BMI) are two of the most common adolescent and adult public health issues and they are likely to cooccur (Reeves, Postolache, & Snitker, 2008; Stunkard, Faith, & Allison, 2003). Finally, previous empirical investigations have considered physical health conditions less frequently than psychological symptoms and conditions and specifically BMI rarely if ever in an effort to better understand the moderating effects associated with parentification. Exploring depressive

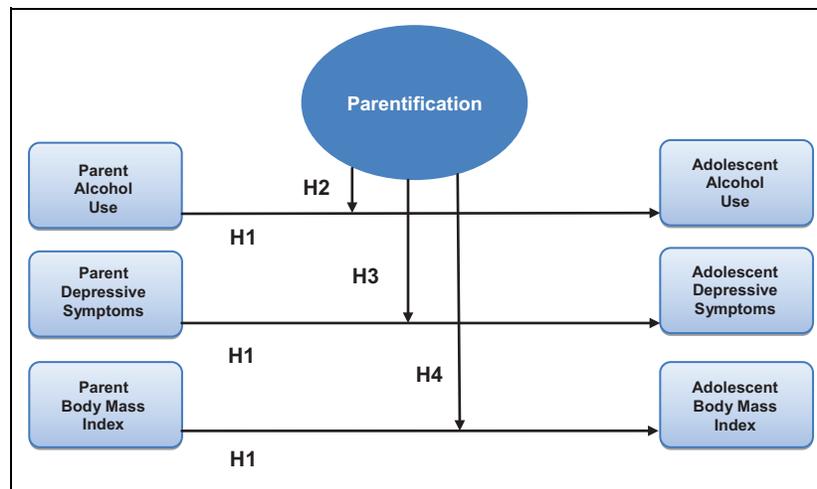


Figure 1. Hypothesized model of the bivariate relations and moderating effect of parentification. Note. H1 = Hypothesis 1; H2 = Hypothesis 2; H3 = Hypothesis 3; H4 = Hypothesis 4.

symptoms, parentification, and BMI in the same study gives the current investigation unique value and meaning and fills an important gap in the literature on family and adolescent health.

Parent Health, Parentification, and Adolescent Health: The Current Study

Undergirded by family systems theory, and in the context of the family systems literature, some researchers have questioned the extent to which negative outcomes are the only possibility when parentification occurs (Duryea, 2007; East, 2010; Gelman & Greer, 2011; Hooper, 2007b; Shifren, 2001). For example, some researchers have contended that pathology and thriving/competency may result from parentification. Specifically, the parentification process may engender independence, hardiness, and self-efficacy for some children and adolescents—even under some of the most adverse conditions and traumatic family and community contexts (Hooper, Marotta, & DePuy, 2009; Jurkovic, Morrell, & Thirkield, 1999; Sahoo & Suar, 2010; Stein et al., 1999; Walker & Lee, 1998).

Early discussions of parentification by family systems scholars (e.g., Minuchin et al., 1967) included the understanding of potential benefits of being parentified. Nonetheless, bimodal outcomes are not commonly examined. Therefore, one plausible consideration for new research is to examine how parentification may lead to positive outcomes. A second consideration is the extent to which parentification may buffer the relation between the transmission of negative parent outcomes to negative adolescent outcomes.

In the current study, we considered how three health conditions or health risk factors in parents—alcohol use, depressive symptoms, and BMI—relate to the same three health conditions or health risk factors in adolescents. Because research is mixed regarding the extent to which parentification, the experience of being parentified, and the family psychology and ecologies in which parentification takes place engenders

only negative, deleterious, and poor outcomes, we explore parentification as a potential buffer of the relation between parent health and adolescent health. It could be that children and adolescents who have been parentified may demonstrate increased skill and ability to thwart negative outcomes (such as poor psychological and physical health and substance use) as a result of parent factors and contextual factors (Walker & Lee, 1998).

Study Hypotheses

Given the foregoing literature review, the gaps and limitations in the literature, and the exploratory nature of our study, we tested a correlational hypothesis for each of the three predictors and outcome measures and three moderator hypotheses (see Figure 1). The three moderator hypotheses represent tests of the proposition that parentification buffers the direct linear effects of parent health on adolescent health. We tested the following four hypotheses:

Hypothesis 1: Parent health variables (alcohol use, depressive symptoms, and BMI) are positively correlated with adolescent health variables (alcohol use, depressive symptoms, and BMI).
Hypothesis 2: Parentification serves as a significant buffer between parent alcohol use and adolescent alcohol use (Carroll & Robinson, 2000; Chase et al., 1998; Goglia, Jurkovic, Burt, & Burge-Callaway, 1992; Stein et al., 2007).

Hypothesis 3: Parentification serves as a significant buffer between parent psychological health and adolescent psychological health (Burton, 2007; Earley et al., 2007; East, 2010; East & Weisner, 2009; Hooper, 2007a, 2007b; Shifren & Kachorek, 2003).

Hypothesis 4: Parentification serves as a significant buffer between parent physical health (as measured by BMI) and adolescent physical health (Duryea, 2007; Gates & Lackey, 1998; Gelman & Greer, 2011; Ireland & Pakenham, 2010a, 2010b; Luecken & Lemery, 2004; Shifren, 2001).

Method

Participants

Participants were recruited using a convenience sampling from a southeastern rural community. The sample consisted of 54 adolescent and parent paired responses. However, three participants (three adolescent and parent paired responses) were excluded because the adolescents did not meet the age criterion for the study (i.e., 12–18), thus are final study sample was comprised of 51 adolescent and parent paired responses. The adolescents ranged in age from 12 to 17 years, with a mean age of 13.80 years ($SD = 1.28$). The adolescent sample was 51% ($n = 26$) female and 47% ($n = 24$) male, with a gender response missing from 2% ($n = 1$). Of the adolescent sample, 43% ($n = 22$) considered themselves Black American, 2% ($n = 1$) listed Asian American, 53% ($n = 27$) listed White American, and 2% ($n = 1$) were unsure of what to list. The parents ranged in age from 30 to 65 years with a mean age of 41.74 years ($SD = 6.64$). The parent sample was approximately 92% ($n = 47$) female and 2% ($n = 1$) male, and 6% ($n = 3$) did not report a gender.

Procedure

We used multiple recruitment methods in the current study. More specifically, we used flyers, invitation letters, e-mail, and voice mail messages to recruit participants in the community. When participants were contacted by phone, the researchers used a telephone script similar to the description in the invitation letters, which explained the purpose of the study: an exploration of the link between family factors, parent health, and adolescent health. Data collection took place during evening hours at local schools. Participants were provided with detailed information about the purpose of the study and were given an informed consent form, an assent form, and a packet of self-report questionnaires. To reduce participant burden (i.e., length of time to complete surveys) and cost to the conduct the study all data were self-reported or self-rated on the paper-and-pencil questionnaires. The principal investigator and trained graduate-level research assistants administered the questionnaires. On average, participants took approximately 30 to 40 min to complete the questionnaires. All study procedures were approved by the University Institutional Review Board.

Measures

Demographic information. Youth were asked for information regarding year in school, race, ethnicity, current age, and gender. A separate form, created for the study, asked parents to report their completed years of education, age, gender, and location of primary residence.

Alcohol use in adolescents. We used a single question to assess how often adolescents consumed alcohol. The single question was derived from the Youth Risk Behavior Surveillance System (Centers for Disease Control and Prevention [CDC],

2009), which measures health risk behaviors and leading causes of morbidity and mortality among American youth. All participants responded to the following question only: “During the past 30 days, on how many days did you have at least one drink of alcohol?” Participants gave an answer on a scale from 0 (*0 days*) to 7 (*all 30 days*). This single item measure was taken from the full survey in an attempt to assess the adolescents use and exposure to alcohol.

Alcohol use in parents. We used a single question from the Alcohol Use Disorders Identification test (AUDIT; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001) to assess how often parent-participants consumed alcohol. The AUDIT is a widely used 10-item self-report instrument that measures excessive, problematic patterns of alcohol use and harmful alcohol consumption. All participants responded to the following question only: “How often do you have a drink containing alcohol?” Participants gave an answer on a scale from 1 (*never*) to 5 (*four or more times a week*). This single item comes from the factor measuring hazardous alcohol use and indicates the amount of exposure the adults have to alcohol.

Depressive symptomatology. The Beck Depression Inventory II (BDI-II; Beck, Steer, & Brown, 1996) is composed of 21 self-report questions that capture depressive symptomatology. Scores for each item range from 0 to 3. The maximum possible total score is 63, and higher scores reflect greater severity of depressive symptomatology and a greater likelihood of a diagnosis for major depression disorder.

The BDI-II has been used with a range of populations, including racially diverse adolescents (Byrne, Stewart, Kennard, & Lee, 2007; Dozois, Dobson, & Ahnberg, 1998; Osman et al., 2008) and racially and culturally diverse adults (Dozois et al., 1998; Gary & Yarandi, 2004; Steer & Clark, 1997; Ward, 2006). Some researchers have suggested that the factor structure of the BDI-II differs based on the type of sample (e.g., clinical vs. nonclinical; Carmody, 2005; Storch, Roberti, & Roth, 2004). Likewise, it is assumed that the factor structure of the BDI-II may vary based on the cultural background of the sample (Black Americans vs. White Americans).

In terms of construct validity, although the BDI-II cannot confirm a diagnosis of depression, the scores can point to probable depression (Beck et al., 1996). Findings from Osman and colleagues (1997) suggested that BDI-II scores yield sound convergent, construct, and discriminant validity. Research conducted by Dozois and colleagues (1998) and Storch and colleagues (2004) provided evidence for construct validity based on the relations between scores on the BDI-II, the State-Trait Anxiety Inventory-Depression, and the State-Trait Anxiety Inventory-Anxiety factors scores.

The BDI-II is one of the most widely used instruments to measure depressive symptoms, and scores from this instrument have been shown to have good reliability and validity (Beck et al., 1996). In the present study, the BDI-II was used to capture depressive symptomatology in accordance with the *Diagnostic and Statistical Manual of Mental Disorders* (American

Psychiatric Association, 1994). Consistent with other studies, the obtained reliability in the current study was more than adequate. The BDI-II was used to assess depressive symptoms in both adolescents and parents. The obtained reliability from the BDI-II scores for parent-participants was .94 and for adolescent participants was .92.

BMI in adolescents. We used BMI to assess physical health and weight status. To ascertain BMI, all adolescent-participants' height (in feet and inches) and weight (in pounds) were self-reported, and these were converted to meters and kilograms, respectively. In youth participants, the age-adjusted z score for BMI was used for the regression analyses. Among youth participants, "overweight" and "obese" were categorized as having BMIs greater than the 85th percentile and 95th percentile, respectively, which is consistent with the CDC (n.d.) guidelines for children and adolescents. We calculated BMI continuous and categorical scores for study analyses so that our findings would be comparable to previous studies that used both approaches.

BMI in parents. Physical health was again assessed using BMI. To ascertain BMI, all parent-participants' height (in feet and inches) and weight (in pounds) were self-reported, and these were converted to meters and kilograms, respectively. BMI was then calculated using the following standardized formula: weight (in kilograms) divided by height squared (in meters). Parents' BMIs were then categorized as recommended by the CDC (2006a, 2006b) in the following way: "normal weight" is characterized as 18.5–24.9 kg/m²; "overweight" is characterized as 25.0–29.9 kg/m²; and "obese" is characterized as 30.0 kg/m² or greater. We calculated BMI continuous and categorical scores for study analyses so that the findings would be comparable to previous studies that used both approaches.

Parentification questionnaire—youth (PQ-Y). The PQ-Y (Godsall & Jurkovic, 1995) is a 20-item instrument that measures caregiving behaviors in youths' families of origin. Respondents are asked to rate if they have engaged in caregiving roles and responsibilities, and they can respond by selecting "yes" or "no." Sample items include "I'm told that I act older than my age," "I'm often asked to do more than my share of the work in my family," and "I often feel more like an adult than a child in my family." In previous samples, reliability coefficients have been reported in the range of .75 to .83 (Godsall & Jurkovic, 1995; Godsall, Jurkovic, Emshoff, Anderson, & Stanwyck, 2004). The obtained reliability from the PQ-Y score was .80 in the current study. Parentification was dichotomized using a median split (median = 5) when incorporated into regression analyses in order to test for differences between individuals with "high" versus "low" levels.

Data Analysis Plan

We employed the following data analytic procedures to examine the data. First, descriptive data (means and standard

deviations) for all parent and adolescent study variables were examined. Second, Pearson product-moment correlation coefficients were used to test our correlational hypothesis. As recommended for moderation analyses (Aiken & West, 1991), all independent variables were standardized ($M = 0$, $SD = 1$) to reduce multicollinearity before we included them in the tested model. Multicollinearity was analyzed by use of the variance inflation factor. Levels below 10 indicate that multicollinearity is not a problem (Kutner, Nachtsheim, & Neter, 2004).

Regression models to predict adolescent alcohol use, BDI-II (Beck et al., 1996) score, and BMI were tested based on parent alcohol use, parent BDI-II score, and parent BMI, respectively. We extended these models to the multiple regression setting by also basing our predictions on parentification and a possible interaction term of the parent health variable with parentification in order to determine if a moderation effect exists. As part of an exploratory analysis, we generated models to predict adolescent BDI-II score and BMI based on parent alcohol use, parentification, and an interaction term to determine if parentification moderates additional variable relations other than those proposed in our research questions. All of our regression models have a power of .90 to detect a moderate effect size of .30.

An a priori significance level of .05 was used in the current study. All analyses were conducted with Statistical Package for the Social Sciences software (version 15.0) and Statistical Analysis System (version 9.1).

Results

Intercorrelations Between Study Variables

Means, standard deviations, and correlations among BDI-II scores, BMIs, alcohol use scores, and parentification levels are shown in Table 1. Possible ranges are also given for some variables. Correlation coefficients significant at the .05 or .0001 α levels are noted in the table. There were low to moderate positive correlations of parent BMI with both adolescent BMI, $r = .29$, $p < .05$, and adolescent parentification, $r = .31$, $p < .05$. Adolescent parentification was found to have a moderate positive correlation with adolescent BDI-II, $r = .40$, $p < .05$. Not surprisingly, a large positive correlation was found between parent alcohol use and adolescent alcohol use, $r = .62$, $p < .0001$.

Adolescent Alcohol Use

Parentification and parent alcohol use were found to significantly predict adolescent alcohol use, $R^2 = .35$, $F(2, 44) = 13.25$, $p < .0001$. Parameter estimates and model fit summaries are shown in Table 2. Adolescent alcohol use was found to increase significantly as the parent alcohol use increased, $\beta = 0.61$, $t = 5.14$, $p < .0001$. However, parentification was found to have no affect on adolescent alcohol use, $\beta = 0.06$, $t = .30$, $p = .7634$. Additionally, the interaction between these two variables was found to be significant, R^2 Change = .06, F change = 4.61, $p = .0374$, indicating a significant moderation effect of parentification.

Table 1. Possible Ranges, Means, Standard Deviations, and Correlations Among Study Variables

Variable	Possible Range	M	SD	1	2	3	4	5	6
1. Parent BMI	—	30.97	7.06	—					
2. Parent BDI-II	0–63	8.00	9.79	.00	—				
3. Parent alcohol use	0–4	.50	.86	-.11	.08	—			
4. Adolescent BMI	—	23.45	7.24	.29*	-.12	-.28	—		
5. Adolescent BDI-II	0–63	6.68	9.25	.04	.03	.26	-.06	—	
6. Adolescent alcohol use	0–6	.24	.84	.06	.05	.62**	-.04	.17	—
7. Adolescent parentification	0–20	6.51	3.97	.31*	.05	.05	.06	.40*	.14

Note. BDI-II = Beck Depression Inventory-II; BMI = body mass index.

* $p < .05$. ** $p < .0001$.

Table 2. Summary of Hierarchical Analysis Predicting Adolescent Alcohol Use From Parent Alcohol Use

Step	Variables	β	R^2	R^2 Change	F Change	p Value
1	Main Effects		.38	.38	29.84**	
	Parent Alcohol Use	0.60**				<.0001
2	Model 2		.38	.00	.09	
	Parent Alcohol Use	0.61**				<.0001
	Adolescent Parentification	0.06				.7634
3	Interaction Effects		.44	.06	4.61*	
	Parent Alcohol Use	0.80**				<.0001
	Adolescent Parentification	0.33				.1662
	Parent Alcohol Use \times Adolescent Parentification	-0.50*				.0374

* $p < .05$. ** $p < .0001$.

When parent alcohol use is low, there is no difference between the two groups, high versus low parentification. However, adolescents with high parentification show a significantly greater increase of alcohol use when parental alcohol use increases as compared to the group with low parentification. A graphical display of this interaction can be found in Figure 2.

Subsamples of Black American adolescents ($n = 22$) and White American adolescents ($n = 27$) were also analyzed. Neither main effects nor the interaction effect was significant in the subsample of Black American adolescents. However, in the sample of White Americans, parent alcohol use was found to be a significant predictor of child alcohol use, $\beta = 0.875$, $t = 4.51$, $p = .0002$, which is not the case in the sample of Black Americans. Similar to the Black American sample, neither parentification nor the interaction term was significant.

Adolescent Depressive Symptoms

Adolescent BDI-II scores were shown to be unrelated to parent depressive symptoms and parentification, as well as their interaction, $R^2 = .05$, $F(3, 42) = .79$, $p = .5046$. Model fit summary is shown in Table 3. A similar lack of significant relation was found for the subsamples of Black American adolescents and White American adolescents.

Additional exploratory analyses, however, found adolescent depression to be significantly related to parent alcohol use and parentification, $R^2 = .30$, $F(3, 43) = 6.17$, $p = .0014$. Parameter estimates are found in Table 4. Although parent alcohol

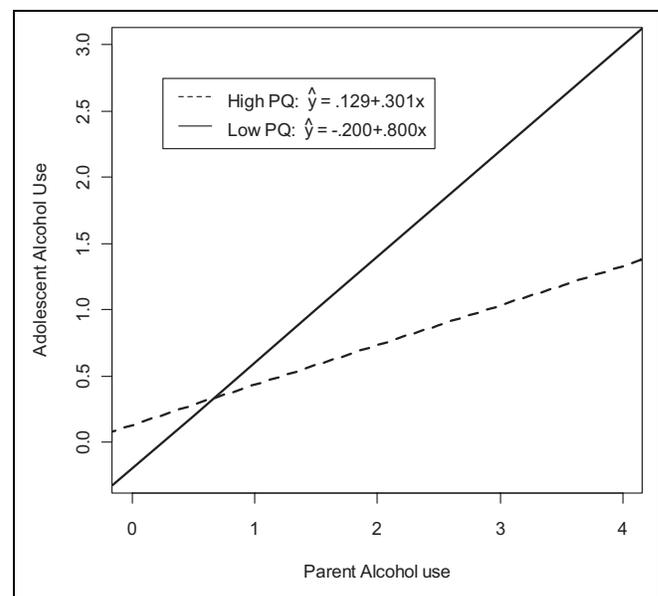


Figure 2. Interaction effect of parentification and parent alcohol use on adolescent alcohol use.

use, $\beta = -1.218$, $t = -.70$, $p = .4885$, and parentification, $\beta = -1.022$, $t = -.36$, $p = .7169$, were not found to be significant predictors of adolescent depression, the interaction term was significant, $\beta = 9.62$, $t = 3.45$, $p = .001$, indicating a significant moderation effect of parentification. For adolescents

Table 3. Summary of Hierarchical Analysis Predicting Adolescent Depressive Symptoms From Parent Depressive Symptoms

Step	Variables	β	R^2	R^2 Change	F Change	p Value
1	Main Effects		.00	.00	.05	
	Parent Depressive Symptoms	0.03				.8162
2	Model 2		.05	.05	2.43	
	Parent Depressive Symptoms	0.01				.9624
	Adolescent Parentification	4.35				.1264
3	Interaction Effects		.05	.00	.00	
	Parent Depressive Symptoms	0.01				.9706
	Adolescent Parentification	4.39				.2766
	Parent Depressive Symptoms \times Adolescent Parentification	-0.01				.9862

Table 4. Summary of Hierarchical Analysis Predicting Adolescent Depressive Symptoms From Parent Alcohol Use

Step	Variables	β	R^2	R^2 Change	F Change	p Value
1	Main Effects		.07	.07	3.50	
	Parent Alcohol Use	2.79				.0674
2	Model 2		.11	.05	2.41	
	Parent Alcohol Use	2.54				.1010
	Adolescent Parentification	4.11				.1274
3	Interaction Effects		.30	.19	11.93*	
	Parent Alcohol Use	-1.21				.4485
	Adolescent Parentification	-1.02				.7169
	Parent Alcohol Use \times Adolescent Parentification	9.62*				.001

* $p < .05$.**Table 5.** Summary of Hierarchical Analysis Predicting Adolescent Body Mass Index From Parent Body Mass Index

Step	Variables	β	R^2	R^2 Change	F Change	p Value
1	Main Effects		.09	.09	4.05	
	Parent BMI	0.30*				.0504
2	Model 2		.14	.05	0.02	
	Parent BMI	0.32*				.0234
	Adolescent Parentification	0.29				.8840
3	Interaction Effects		.14	.00	.24	
	Parent BMI	0.22				.3721
	Adolescent Parentification	-4.04				.6605
	Parent Body Mass Index \times Adolescent Parentification	0.14				.6296

* $p < .05$.

experiencing low parentification, there is not a significant change in adolescent depression as parent alcohol use increases. The parameter is slightly negative, but not significant. With high parentification, there is a significant increase in adolescent depression as parent alcohol use increases. Thus, parentification demonstrated an exacerbating effect as a moderator of the association between parent alcohol use and adolescent depression. When looking at the two race subsamples, no significant results were found.

Adolescent BMI

Parent BMI was found to be a significant predictor of adolescent BMI in the main effects model, $\beta = 0.32$, $t = 2.36$, $p = .0234$. However, neither parentification nor the interaction was

found to add to the relationship; see Table 5. Plotting the regression lines for high and low parentification categories revealed nearly horizontal lines. The near-parallelism of the lines supports an insignificant interaction term between parentification category and parent BMI. Thus, no figure is included for this nonsignificant finding. Similar results were found for the subsamples of Black American adolescents and White American adolescents.

Discussion

One purpose of this cross-sectional, correlational study was to explore whether parent health—defined in the current study as alcohol use, depressive symptoms, and BMI—positively relates to adolescent alcohol use, depressive symptoms, and

BMI. A second purpose was to determine the extent to which parentification buffers the relation between parent health and adolescent health, including BMI, which is shown to be a direct link to health outcomes. Because of the exploratory nature of the study, we tested three separate models for each adolescent health outcome variable (i.e., alcohol use, depressive symptoms, and BMI). In this section, we discuss the findings related to our analyses among our study variables, followed by a discussion of the hypothesized and exploratory moderating effects of parentification.

Bivariate Relations Among Study Variables

The results with our rural family sample revealed several significant positive correlations and one negative association among the study variables. The effect sizes as defined by Cohen (1992) and Cohen and Cohen (1983) for these significant associations ranged from small ($r = .10$) to large ($r = .60$). Parent self-reported BMI is associated with adolescent parentification; higher levels of parentification are associated with higher levels of parent BMI. Parent alcohol use and adolescent alcohol use are strongly, positively correlated. Contrary to our study hypothesis, parent BDI-II score is not correlated with adolescent BDI-II score. However, adolescent BDI-II score is positively related to adolescent parentification. The foremost finding from our bivariate analyses is the significant, positive correlations between parent BMI and adolescent BMI. These significant positive relations add to the literature base and have important meaning for future research. First, although we narrowly defined and operationalized physical health in the current study as BMI, ample empirical research supports the association between BMI and a plethora of other serious physical health conditions, such as diabetes and asthma (e.g., see Field et al., 2001; Institute of Medicine, 2011; Must et al., 1999). Nonetheless, going forward, researchers should examine the link between parentification and other physical health or serious medical conditions (e.g., asthma, diabetes, etc.) in addition to BMI. Second, although we did not find a significant association between parentification and adolescent BMI, as hypothesized, we did find a significant positive relation between parentification and parent BMI. Moreover, a significantly understudied area has been the link between childhood parentification and adolescent BMI. Therefore, this nonsignificant finding should not detract from the need to examine physical health—in both the parents and the parentified child—in future studies. Finally, the strongest bivariate relationship uncovered in our study was that of parent alcohol use and adolescent alcohol use. This finding, too, is important and had the most robust contribution to the results in our moderation models described below. In sum, we found partial support for our correlational hypothesis.

Moderating Effects of Parentification

The foremost finding from our tested regression models reveals partial support indicating that parentification buffers the effect

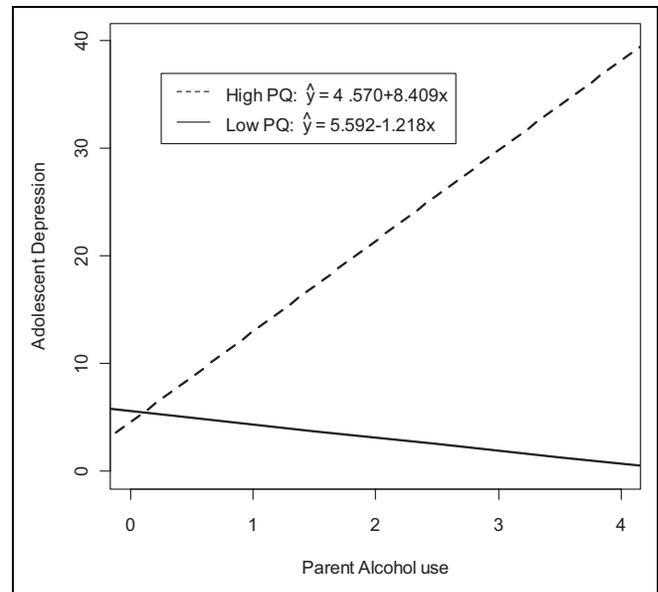


Figure 3. Interaction effect of parentification and parent alcohol use on adolescent depressive symptoms.

of parent alcohol use on adolescent alcohol use. More specifically, the analyses revealed a significant main effect and an interaction effect for parentification moderating the effect of parent self-reported alcohol use on adolescent self-reported alcohol use. The ability of parentification to moderate the relation between a parent health measure and an adolescent health measure is consistent with nascent empirical research in the last decade examining bimodal outcomes of and associations with parentification (Hooper, Marotta, & Lanthier, 2008; Jurkovic, Morrell, & Casey, 2001; Stein et al., 2007). More specifically, the current study showed that higher levels of parentification had a buffering effect on adolescent alcohol use (a negative health measure) in adolescent participants. This effect may be considered counterintuitive or paradoxical by some researchers and practitioner–scientists (Byng-Hall, 2008b). However, it is consistent with the findings of other scholars who believe that parentification and the related family psychology, ecology, and processes may engender resilience, competence, and hardiness in adolescents that they may not otherwise experience (Byng-Hall, 2008b; East, 2010; Hooper, 2007b, 2011; Hooper et al., 2008; Stein et al., 1999; Walker & Lee, 1998; Walsh, 1998).

In addition to the buffering effect uncovered in our results described above, parentification had an exacerbating effect on the relation between parent alcohol use and adolescent depression. More specifically, the moderation analysis of the association between parent alcohol use and adolescent depression revealed that parentification had an exacerbating effect on the relationship such that higher levels of parentification were associated with an increased association between parent alcohol use and adolescent depression (see Figure 3). In contrast, lower levels of parentification resulted in a negative association between parent alcohol use and adolescent depression. Lower

levels of parentification appear to lessen the adolescents' experience of depression even as parent alcohol use increases, while increased parentification seems to correspond to increased depressive symptoms as parent alcohol use increases.

Taken together, the buffering and exacerbating effects noted in this study depict a complexity of interpersonal processes and individual functioning within family systems that is consistent with existing research predicting adolescent health measures. For example, Fischer, Forthun, Pidcock, and Dowd (2007) demonstrated the mediating effects of parenting processes and affect regulation in a multiply mediated association between parent alcohol use and college student alcohol use. In fact, research has consistently found associations between emotional dysregulation and increased alcohol consumption (Fischer, Forthun, Pidcock, & Dowd, 2007; Simons, Carey, & Gaher, 2004). It would seem that the adolescents in this study may not have been using alcohol to cope with their depressive symptoms, although drinking motives were not directly assessed. The lack of a significant bivariate correlation between adolescent depressive symptoms and adolescent alcohol use would also seem to support the suggestion that the adolescents were not using alcohol to regulate their depressive symptoms.

It may also be that while parent alcohol use was depressing to the adolescents as a function of the increased parentification, the increased parentification simultaneously buffered the adolescents from alcohol use. This would seem to suggest that parentification perhaps served a paradoxical self-regulating function for the adolescents. The interpretation seems consistent with theorizing and research findings which suggest that filial responsibility promotes a sense of identity through experiences of role fulfillment and belonging (Telzer & Fuligni, 2009). A strong sense of family identity may facilitate behavioral self-regulation, thereby buffering alcohol consumption, even while simultaneously having negative emotional consequences for the individual. Consistent with family systems theories (Boszormenyi-Nagy & Spark, 1973; Bowen, 1978; Minuchin et al., 1967), individuals in families often experience the dialectic tension of togetherness and separateness forces or experience the pull of family loyalty and the push for autonomy. The same family loyalty that may deter alcohol use may also foster an ethic of sacrificing personal well-being for the sake of the system. The consequence may therefore be increased depressive symptoms in the parentified individual.

Finally, the findings may also suggest that some of the families in this study held cultural beliefs that both supported parentification and protected against adolescent alcohol use, as family belief systems have been found to promote resilience processes in families (Walsh, 1998). Cultural factors that support filial responsibility seem to lessen the deleterious consequences of parentification (Hooper & Wallace, 2010; Jurkovic et al., 2001; Kam, 2011; Telzer & Fuligni, 2009). Similarly, cultural factors, such as religious beliefs and practices, appear to function as protective factors reducing alcohol consumption among youth (Hill, Burdette, Weiss, & Chitwood, 2009; Walker, Ainette, Wills, & Mendoza, 2007). Whether cultural factors such as the families' belief systems accounted for

the buffering effect observed in this study cannot be directly ascertained. Nevertheless, there does appear to be some phenomenon at play, whereby increased parentification in the context of parent alcohol use seems to buffer adolescent alcohol consumption. For example, Stein, Rotheram-Borus, and Lester (2007) found that increased parentification scores at baseline when participants were young adolescents predicted lower alcohol use at a 6-year follow-up. Parental substance use significantly predicted parentification scores at baseline (Stein et al., 1999). In contrast, there may be less of a framework in these same families for making sense of and managing an individual's negative emotional experiences such as depression, particularly given the loyalty and togetherness dynamics at work in parentified systems. Clearly though, future research could benefit from including measures that directly assess affect regulation, cultural factors such as religiosity, and additional indicators of family functioning. Future research might also explore mediation models in addition to moderation models, or perhaps explore complex models of conditional indirect effects or moderated mediation (Preacher, Rucker, & Hayes, 2007).

Study Limitations and Directions for Future Research

This study contributes to the family systems and adolescent health literature by documenting (1) a buffering effect for parentification, (2) a bivariate association between parentification and parent health, and (3) bivariate associations between parent and adolescent health, namely, the correlations for alcohol use and BMI. Concurrent with our results, limitations of the current cross-sectional, correlational study must also be considered.

First, our study was limited by the self-selected convenience sample, which may have resulted in an unrepresentative sample and thus affected the generalizability of the study's findings. Additionally, we used multiple strategies to recruit our convenience sample. Thus, it is unclear if these strategies impacted our results or engendered some of our participants to be more motivated to participate than others. Moreover, because we did not use random sampling methods or comparison group in the current study our results must be considered preliminary and interpreted with caution.

Second, and also related to our study sample, is the small number of fathers in the parent sample. There has long been a call for researchers to include fathers in family systems research, to examine the implications of the father-adolescent relationship, and to explore how the father-adolescent relationship may impinge upon a range of types functioning in adolescence (e.g., psychological, emotional, physical, and academic; see Garber, 2011; Nielsen, 2011) and adulthood. Future research should systematically aim to recruit and include fathers in the study of parentification and adolescent health outcomes.

A third limitation is that the data were all derived from self-rated information and it is unclear if social desirability influenced how our study participants responded to our survey questions. For example, height and weight used to calculate BMI was self-reported. Self-reporting of current weight status

among study participants—as compared with actual measured weight—could have significantly influenced the results of the study. Underreporting or overreporting of weight status can exist (Davis & Gergen, 1994; Strauss, 1999; Tienboon, Wahlqvist, & Rutishauser, 1992). Of significance, findings might change if the constructs were examined based on data from multiple informants or on observation data. Future research should address this measurement limitation. Fourth, one of our key study constructs, namely, alcohol use, was measured by a single item. This single item is limited in its ability to capture alcohol use rather than looking at both alcohol use and abuse, as those are not always the same. Moreover, there are complete measures available to assess for alcohol use and abuse. Because we were concerned with participant burden we elected to use a single item, which may have attenuated the results of the study. Future studies could include measures that assess not only physical health risk factors but also physical health conditions (e.g., Alzheimer's disease, cancer, dementia, HIV/AIDS) discussed previously.

A fifth limitation in our discussion also related to our artificial creation of a dichotomized parentification score from a continuous scale. This was due to the interest in examining differences in high versus low reports of parentification. As described in the literature (see DeCoster, Galluci, & Iselin, 2011), there are advantages and disadvantages to this approach with data. Alternatively, the moderator analysis could have been conducted using parentification as a continuous variable.

Parentification itself may be related to other outside factors that are driving these moderation analyses. Our data as a whole has the limitation of being a relatively low-risk sample, with low mean values of both parent alcohol use and depression. This may be a factor of sample size, measurements used, or of the area from which the sample was collected. Finally, given the cross-sectional, nonexperimental nature of the current study, no conclusions related to cause and direction of the effects and significant relations can be drawn.

Conclusion

Our study is unique in that we examined youths' physical health, psychological health, and alcohol use in the same study with current adolescent parentification in an understudied, high-priority population, that is, Black American and White American rural families. Our study tentatively and preliminarily confirms the need to broaden the long-held clinical and theoretical axiomatic position and myopic view that parentification engenders negative outcomes only. The buffering effect of parentification that emerged in our study may help inform future research, prompting researchers to include measures of wellness and psychopathology in their empirical studies. For example, future studies should consider adding measures such as the Satisfaction with Life scale (Diener, Emmons, Larsen, & Griffin, 1985) and the Posttraumatic Growth Inventory (Tedeschi & Calhoun, 1996) to further clarify how, when, and for whom parentification may be linked to wellness, growth, thriving, and high functioning (Garber, 2011; Hooper, 2007b;

Hooper et al., 2009; Kam, 2011). As previously mentioned, a careful consideration of the measures selected to assess for alcohol use and physical health is also warranted.

The current study adds to the literature base by demonstrating that parentification is not associated only with negative sequelae. Certainly additional studies that replicate the results found in the current sample are needed, and larger, more diverse, longitudinal studies are warranted. Moreover, the current findings do not negate the overwhelming and convergence of findings evinced in numerous studies—including the current study—indicating that childhood parentification is associated with negative outcomes in childhood and adulthood as well. As evidenced in the current study, bivariate correlations revealed that higher levels of parentification were associated with higher levels of depressive symptomatology in adolescents and parentification demonstrated an exacerbating effect as a moderator of the association between parent alcohol use and adolescent depression.

Nonetheless, and of importance, research to uncover the protective nature of parentification on differential outcomes has been absent from the empirical literature until recently (Stein et al., 2007). Research has overwhelmingly focused on the pernicious and risky nature of parentification (see Burton, 2007; Byng-Hall, 2008b; East, 2010). Understanding the possible buffering effects of parentification is equally as important and informative as clarifying the exacerbating effects of parentification. Well-balanced studies that explore bimodal outcomes, as well as protective factors, risk factors, and explanatory factors will likely generate new, innovative, and unexpected findings that may inform prevention and intervention efforts going forward (East, 2010; Garber, 2011; Hooper, 2011; Kam, 2011).

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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