Examining Mechanisms Underlying the Effect of Family Disruption in Childhood on Parenting Provided to Offspring in Adulthood

by

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A Thesis Presented in Partial Fulfillment
of the Requirement for the Degree of
Master of Arts

Approved November 2019 by the
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December 2019

ABSTRACT

Family disruption, or the separation of children from caregivers, has been well-established in prior literature as a risk factor for child maladjustment; however, little is known about how family disruption impacts youth into adulthood, particularly how it influences children's later parenting of their own offspring. The present study examined whether cumulative family disruption (i.e., parental hospitalization, death, incarceration, divorce) in childhood exerts effects on children's parenting of their own offspring in adulthood, beyond other demographic characteristics and risk factors. Further, several potential mechanisms were hypothesized to underlie the association between family disruption in the first and second generation (G1-G2) family and later parenting provided from second-generation (G2) adults to third-generation (G3) children. Mediators included conflict and disorganization in the G1-G2 family and dysregulation in the G2 child.

Participants (N = 236 in models that included multiple G2 siblings; N = 110 in models without siblings) were drawn from a larger sample of at-risk (i.e., alcoholic) and comparison families followed longitudinally for over 30 years and across three generations. Four mediation models were estimated to examine effects of two separate G1-G2 family disruption components (deviance-related and health-related disruption) on parenting of G3, mediated by family conflict, family disorganization, and G2 dysregulation. Results indicated that health-related disruption impairs consistency of parenting provided to G3 offspring through conflict in the G1-G2 family. A direct effect of health-related disruption was also seen on parental monitoring. There were no direct or mediated effects of deviance-related disruption on parenting. Implications and future directions will be discussed.

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Introduction

Family disruption is the separation of children from caregivers through events such as parental divorce/separation, death, hospitalization, incarceration, or a change in caregiver. The detrimental effects of family disruption on child development have been well-established across a large body of literature (Rutter, 1971; Peterson & Zill, 1986; Fergusson et al., 1994; Paksarian & Eaton, 2015). Extended separation of parents from offspring is common in the United States; approximately 40 to 50 percent of married couples in the United States divorce, and many of these couples have children (APA, 2019). Furthermore, nearly 1 in 28 children is separated from a parent due to parental incarceration (The Pew Charitable Trusts, 2010), and up to two million children experience the deployment of one or both parents for military service (Department of Defense, 2012). Other events leading to family disruption (e.g., informal parental departures) may go unrecorded and are difficult to track, but likely affect a sizeable number of children. Thus, family disruption represents a significant public health concern for children and parents alike.

Given the heterogeneity in constructs in prior literature examining family-related adversity, it is imperative to distinguish family disruption, the construct examined in the present study, from similar but distinct constructs. When considered cumulatively across time, the accumulation of family disruption events resembles a similar but distinct construct: family instability. Research has previously defined family instability broadly as the number of caregiving transitions, residential changes, and stressful family events (Ackerman et al., 1999). More recent literature has further specified the construct, eliminating stressful family events and focusing more narrowly on instability in

caregiving, residence, and parental employment (Forman & Davies, 2003; Milan et al., 2006; Marcynyszyn et al., 2008). Other research has employed even further specificity, solely examining the impact of number and type of family structure transitions, such as caregiver entries and exits from household via parental separation, divorce, and remarriage (Martinez Jr. and Forgatch, 2002; Fomby & Bosick, 2013; Pasqualini et al., 2015). No research to date, however, has measured the impact of cumulative family disruption.

Research on family disruption is imperative to understanding the development of the child within the family context because it represents a distinct set of adverse family events that challenge the parent-child attachment relationship. It captures a broader subset of events involving caregiver-child separation than family structure transitions but limits the scope of events to those occurring within the family context, thereby eliminating stressors such as changes in residency and parental employment. Investigating the cumulative impact of family disruption is important for a number of reasons. First, events such as residential moves and parental job loss, while stressful, do not inherently challenge the attachment relationship between parent and child, and thus capture a type of familial stress distinct from family disruption. Second, expanding research on family structure transitions beyond measuring the effects of changes in family structure based on parental romantic relationship status allows us to further incorporate the impact of events such as parental death, incarceration, and hospitalization, which have all demonstrated negative impacts on children (Worden & Silverman, 1996; Miller, 2006; Grasso & Ford, 2012). Thus, the present study will measure the consequences of cumulative family disruption for family-level and individual child outcomes. Nevertheless, because this

study is the first to operationalize family disruption in this manner, evidence for the proposed hypotheses will emerge from prior literature on the impact of family instability, changes in family structure, family unpredictability, and parental divorce. In the subsequent literature review, these constructs will heretofore be referred to more generally as family disruption.

Family disruption exerts a negative, dose-dependent effect on children's outcomes (Cavanagh and Huston, 2008; Fomby and Cherlin, 2007; Lee and McLanahan, 2015). Higher levels of family disruption are associated with poorer academic achievement (Martinez Jr. & Forgatch, 2002; Marcynyszyn et al., 2008), increases in internalizing and externalizing problems (Milan et al., 2006), lower social skills (Marcynyszyn et al., 2008), and greater substance use (Pasqualini et al., 2015; Marcynyszyn et al., 2008). Prior research has also identified distinct moderators of effects of family disruption. Family disruption typically exerts a stronger effect if it occurs earlier in childhood (Pasqualini et al., 2015) and has a worse impact on boys than girls (Cavanagh & Huston, 2008), although this may differ based on the outcome examined. The effects of childhood family disruption may last into adulthood, with negative consequences observed in several domains. Individuals exposed to family disruption demonstrate lower rates of college completion and earlier childbearing, marriage, and entry into the labor force (Fomby & Bosick, 2013). Despite the growing body of research revealing detrimental effects of family disruption on children into adulthood, few studies have examined its impact on one specific domain: parenting provided to one's own offspring in adulthood. Only one prior study has demonstrated that cumulative family disruption experienced in childhood predicts poorer-quality parenting of offspring in adulthood, above and beyond family

socioeconomic and psychosocial factors, and characteristics of the third-generation child (Friesen et al., 2017). The authors found that children's exposure to a greater number of parental separations/divorces between birth and age 15 predicted greater parental overreactivity and physical punishment, and lower parental sensitivity and warmth, in parenting provided to offspring 30 years later. However, this study did not identify any mechanisms by which this effect occurs, limiting the ability to address potential targets of intervention for children who experience family disruption.

Mechanisms underlying effects of family disruption on parenting

There are several plausible mechanisms through which family disruption in childhood impacts children's later parenting of their own offspring, and these fall into two primary categories: individual-level mediators and family-level mediators. One possibility is that family disruption exerts effects on the individual child, which then influence how the child then parents his or her own offspring in adulthood. There is very little research specifically investigating individual-level mechanisms underlying effects of family disruption on later parenting behavior, but there is more evidence identifying individual-level mediators of effects of early adversity on later parenting, which can inform predictions in the current study (Whitbeck et al., 1997; Choi et al., 2019; Yehuda & Lehrner, 2018). A second possibility is that early family disruption influences later parenting of offspring in adulthood through deterioration of the childhood family environment. Although this indirect effect has not been specifically examined, one prior study has found that cumulative family disruption is associated with parenting difficulties, which impair child outcomes by negatively shifting their perceptions of the family environment (Forman & Davies, 2003). A larger body of research underscores the

importance of parenting and family environment in determining child outcomes following single-event disruptions, such as parental divorce (Lengua et al., 2000; Sandler et al., 2003; Sandler et al., 2012) and death (Haine et al., 2006; Kwok et al., 2005). More broadly, prior research has found that adverse parenting begets adverse parenting in the subsequent generation (Lomanowska et al., 2017). In particular, intergenerational transmission of parenting behavior may occur through learning mechanisms (Quinton, 1988) in which the child emulates parenting behavior through social learning (Simonton, 1983). Of course, family-level and individual-level mediators are not mutually exclusive and may have interrelated effects.

Individual-level mechanisms

Behavioral dysregulation. One possible individual-level mechanism underlying the effect of cumulative family disruption in childhood on later parenting of offspring is behavioral dysregulation. Family disruption may increase behavioral dysregulation by creating an "unpredictability schema" in which instability acts as a cue that outcomes are uncertain (Ross & Hill, 2002). This schema may result in delay discounting (Hill et al., 2008), a preference for small, immediate rewards over larger, delayed rewards (Kirby & Marakavic, 1995).

Although difficult to measure empirically, life history theory provides an evolutionary basis for the plausibility of this mechanism. This theory proposes that one's early environment directs one towards "fast" or "slow" life history strategies based on efforts underlying resource allocation strategies (Ellis et al., 2009). Stable, safe, resource-laden early environments predict longer lifespans, encouraging the development of slow life history strategies that reflect long-term planning, investment in offspring, and careful

mate selection (Figuerdo et al., 2006). Meanwhile, more uncertain or dangerous environments call for the adoption of fast life history strategies. These strategies are based on the assumption that one's lifespan will be shorter and early reproduction is imperative. Such strategies may include early childbearing, greater sexual activity (to ensure a greater number of offspring), and elevated risk-taking.

Two proposed domains of environmental risk that predict the adoption of fast life history strategies are environmental harshness and unpredictability (Ellis et al., 2009). Harshness represents environmental exposures that place physical strain on an organism, threatening mortality through scarcity in resources or violence. Unpredictability captures the degree of consistency in one's environment. Although both domains have demonstrated associations with fast life history strategies, environmental unpredictability appears to be a stronger predictor than environmental harshness (Belsky et al., 2002; Simpson et al., 2012).

In the context of the family environment, cumulative family disruption, repeated separations from a caregiver, represents a dimension of risk that confers instability in the family unit. Indeed, a wealth of literature has demonstrated that behavioral dysregulation (e.g., impulsivity, antisocial behavior, risk-taking) is predicted by family unpredictability and disruption. Perceptions of parental unpredictability put adolescents at risk for antisocial behavior, including substance use (Ross & Hill, 2002; Vicary & Lerner, 1986). Family instability has also been shown to predict increases in children's impulsivity (McCoy & Raver, 2014). Work by Hartman and colleagues (2018) revealed that caregiver separations, more so than residential changes and parental job loss, predict

adolescents' sexual risk-taking and externalizing behavior, suggesting family disruption as an especially salient marker of environmental unpredictability.

Prior research has not only demonstrated that behavioral dysregulation in childhood is predicted by family disruption, but has also revealed that various markers of behavioral dysregulation are related to difficulties parenting one's own offspring in adulthood. Antisocial behavior is associated with poorer parenting, both when measured concurrently to parenting (Smith & Farrington, 2004; Simons et al., 1993) and preceding parenting in adolescence (Thornberry et al., 2009). Additionally, low parental self-control has a negative impact on domains of family environment such as cohesion, conflict, and efficacy (Meldrum et al., 2016). Deficits in inhibitory control, furthermore, demonstrate negative associations with parental sensitivity and responsiveness (Shaffer & Obradovic, 2017). Given that unpredictable family environments increase behavioral dysregulation in childhood, and behavioral dysregulation is a salient predictor of poorer parenting in adulthood, it follows that behavioral dysregulation may mediate the association between cumulative family disruption in childhood and parenting provided to offspring in adulthood.

Emotion dysregulation. A second individual-level domain that may mediate the impact of childhood family disruption on parenting provided to offspring in adulthood is emotion dysregulation. Attachment theory informs much of the present literature on the relationship between facets of family disruption and emotion regulation. Secure attachment to a caregiver is vital to the development of effective emotion regulation strategies (Bowlby, 1969; 1973; 1980). Early in development, children lack independent self-regulation strategies and instead adopt strategies – namely, seeking proximity to

caregivers – that encourage their caregivers to aid them in regulating their emotions (Bowlby, 1973). However, when attachment figures are unavailable or major disruptions in attachment occur, proximity-seeking strategies fail to relieve distress, resulting in the development of emotion dysregulation. Insecure attachment to caregivers is associated with poorer emotion regulation, not only in childhood (Spanger & Zimmerman, 1999; Waters et al., 2010; Calkins & Leerkes, 2004), but also in adolescence (Allen & Miga, 2010) and adulthood (Karreman & Vingerhoets, 2012). Because family disruption events represent interruptions of the attachment relationship, cumulative family disruption may be especially predictive of children's emotion regulation strategies.

A burgeoning literature suggests that in addition to early parenting serving as a predictor of emotion regulation in children and adolescents, emotion regulation may serve as a predictor of later parenting provided to offspring. At present, most research examining associations between parental emotion regulation and parenting behavior is cross-sectional, with an absence of literature employing longitudinal methods to examine the link between childhood emotion regulation and parenting provided to offspring in adulthood. Nevertheless, prior research has shown associations between parental emotion dysregulation and insensitive parenting (Rutherford et al., 2015), invalidation of children's emotions (Buckholdt et al., 2014; Jones et al., 2014), hostile parenting (Saritas et al., 2013), unsupportive parenting (Morelen et al., 2014), reduction in positive parenting (Shaffer & Obradovic, 2017), and worse family functioning (Crandall et al., 2016). Further research is needed to determine whether emotion dysregulation in childhood is stable into adulthood and confers risk for poorer parenting of offspring. However, it seems theoretically likely that this would be the case. Moreover, it is

plausible that family disruption in childhood demonstrates long-lasting effects on emotion dysregulation, thereby impairing parenting provided to offspring in adulthood.

Cognitive dysregulation. Lastly, regulatory functions related to cognition may be impacted by cumulative family disruption. Cognitive self-regulation represents top-down domains of self-regulation involving attention, planning, and inhibitory control (e.g., executive control; Espy et al., 2011; McCoy et al., 2011). Prior research has found that family instability is associated with deficits in cognitive control (Lewis et al., 2007). Specific types of family disruption have also been implicated in the development of cognitive dysregulation. For example, parental divorce and incarceration are associated with elevations in children's attention problems (Harland et al., 2002; Liu et al., 2000; Geller et al., 2010). Furthermore, parental exits from the home predict greater HPA axis dysregulation (Blair et al., 2011), which is related to impairments in cognitive control (Arnsten, 2000). Finally, residential mobility, which is likely related to family disruption, predicts poorer cognitive self-regulation in children (Ziol-Guest & McKenna, 2014; Roy et al., 2014).

A number of studies suggest that cognitive dysregulation may not only result from experiences of family disruption in childhood, but also impairs parenting of children's own offspring in adulthood. The majority of such research examines effects of maternal ADHD or executive functioning on parenting. In a review of the effects of maternal ADHD on parenting behavior, Johnston and colleagues (2006) proposed a model in which difficulties with executive functioning, such as working memory, inhibitory control, and planning may impact a variety of parenting behaviors (e.g., parental monitoring, planning, guidance provision), parenting cognitions (e.g., sense of parenting

efficacy, parenting stress), and emotional responsiveness. Other research has tested cognitive dysregulation as a mediating mechanism of childhood adversity on later parenting of offspring. For example, maternal executive functioning has been shown to mediate the effect of socioeconomic risk on maternal sensitivity (Sturge-Apple et al., 2017). Only one study has tested cognitive dysregulation as a mediator of family disruption on parenting, and found evidence for an indirect effect; however, because the predictor was an index of both family disruption and maltreatment experiences, the specific impact of family disruption alone cannot be concluded from this study (Gonzalez et al., 2012). Nevertheless, given prior evidence that family disruption increases risk for cognitive dysregulation, and that cognitive dysregulation predicts poorer parenting, it is possible that family disruption in childhood exerts effects on parenting of offspring in adulthood through its impact on cognitive self-regulation.

Global dysregulation. Because prior research has demonstrated that behavioral, emotional, and cognitive dysregulation are all separately related to both family disruption and parenting behavior, it is also plausible that these domains of dysregulation work in tandem as a mediating mechanism. A single "dysregulation profile," made up of each of these three domains, has been validated in a number of studies with the CBCL Dysregulation Profile (CBCL-DP), which utilizes responses on the Child Behavior Checklist (Achenbach & Edelbrock, 1978) to capture significant elevations in the aggressive behavior, attention problems, and anxious/depressed subscales (Holtmann et al., 2010; De Caluwe et al., 2012; Althoff et al., 2011). Some studies measure the CBCL-DP dichotomously by determining whether clinical cutoffs are simultaneously met for all three subscales (e.g., Ayer et al., 2009; Mbekou et al., 2014), whereas others sum T-

scores of the three subscales to create a continuous measure of dysregulation (e.g., Kim et al., 2012; Althoff et al., 2011). The former method reflects comorbidity among the three scales (Holtmann et al., 2007), whereas the latter captures an underlying dysregulation "syndrome" (Kim et al., 2012), similar to the general psychopathology factor (Haltigan et al., 2018). Behavioral genetic studies have evidenced CBCL-DP to be separate from its subcomponents, thereby supporting the use of the "syndrome" method (Boomsma et al., 2006; Althoff et al., 2006). Research using this method has found that this general dysregulation syndrome is not linked to a specific disorder but is related to general dysfunction in adulthood (Ayer et al., 2009), which could presumably include parenting. Further, the CBCL-DP has been linked to adversity in childhood (Jucksch et al., 2011) and has been shown to be fairly stable across time (McQuillan et al., 2018). Thus, it follows that cumulative family disruption experienced in childhood may produce a syndrome-like pattern of behavioral, cognitive, and emotional dysregulation that is stable into adulthood and impairs parenting provided to offspring.

Family-level mechanisms

Family conflict. One potential domain of family environment that may underlie effects of early family disruption on later parenting is family conflict. A wealth of literature suggests that parental conflict mediates the effect of divorce on child outcomes (Amato & Sobolewski, 2001; Fabricius & Luecken, 2007; Grych, 2005) but less is known about whether it similarly mediates the effect of cumulative family disruption. One study found that family conflict predicted child externalizing behavior over and above family disruption, but it did not examine family conflict as a mediator of the impact of disruption. Given prior evidence for intergenerational continuity in family conflict

(Rothenberg et al., 2016), it is possible that cumulative family disruption heightens conflict within the childhood family environment, hindering the quality of parenting provided to the next generation (e.g., by increasing conflict within the environment of the family that is created in adulthood).

Family disorganization. Family disorganization represents another potential mechanism through which family disruption in childhood displays effects on parenting of offspring in adulthood. Family disorganization captures the extent to which families engage in set routines and predictable activities (Bloom, 1985). There is evidence that divorced families have less stable and predictable family routines (Ross & Miller, 2009; Holdnack, 1993). Therefore, it makes theoretical sense that, relative to divorce, repeated disruptions in family structure may even more strongly undermine the predictability and organization of the family unit. Relatedly, despite a lack of research on the impact of childhood family disorganization on later parenting of offspring in adulthood, there is evidence for intergenerational transmission of family disorganization (Fiese et al., 1992; Denham et al., 2003). Moreover, it has been found that disorganized families display lower parenting competence (Fiese et al., 2002; Coldwell et al., 2006). Parents who model family environments from the disorganized environments they experienced in childhood may, then, exhibit related difficulties in parenting their own offspring. Thus, there is a strong conceptual basis for the premise that family disorganization may mediate the effects of cumulative family disruption in childhood on later parenting in adulthood.

Method

The original study

Participants. Participants for the present study were drawn from a 3-generation, longitudinal study of at-risk (i.e., children of alcoholics, or COAs) and comparison families (Chassin et al., 1991; 1993; 1999; 2004). A total of 454 adolescents (Generation 2, or "G2") and their parents (Generation 1, or "G1") were recruited at Wave 1. Fifty-four percent of G2s in the sample had at least one biological and custodial parent with alcohol use disorder (AUD), whereas the remaining 46% were demographically-matched comparison participants. Once per year for three years (Waves 1-3), adolescents and parents were interviewed. Beginning at Wave 4, participants were interviewed as part of a long-term follow-up; 327 of G2s' biological siblings were also added to the study and interviewed at this time. Follow-up interviews continued every five years through Wave 6. At Wave 6, 745 children of G2s ("G3s") were added to the study. Interviews for G3s occurred at three waves: the baseline assessment at Wave 6, a subsequent assessment approximately 18 months later, and a third assessment approximately 18 months later.

There was minimal attrition for G1s and G2s, and moderate attrition for G3s. At the Wave 4 follow-up, 90% of the original G2s were interviewed; furthermore, 91% of the original G2s and 92% of siblings were retained at Wave 5. At the 18-month Wave 6 follow-up, 580 G3s were retained, and at the 36-month follow-up, 68% returned.

Recruitment. Recruitment of COA families was based on several sources for identifying G1s with potential AUD, including court records, telephone surveys, and health maintenance organization (HMO) wellness questionnaires. Families were eligible to participate if they had a child between 11-15 years old, were of Hispanic or non-

Hispanic Caucasian ethnicity, were born between 1927 and 1960, had Arizona residency, and included at least one biological parent who met DSM-III criteria for AUD.

Comparison families who lived in the same neighborhood as COA families were identified and recruited through reverse directories. Families were matched based upon (1) demographic traits including child age, ethnicity, and socioeconomic status, and (2) family composition (single versus married/cohabitating parents). Furthermore, neither biological or custodial parent could meet lifetime criteria (via the DSM-III or FH-RDC) for AUD or alcohol abuse. Seventeen comparison families who reported sub-clinical levels of alcohol problems were removed.

Recruitment biases. Two potential sources of recruitment bias were examined: bias between those who were contacted versus not contacted, and bias between those who agreed versus did not agree to participate.

To evaluate contact biases in COA families, archival records (e.g., HMO surveys, court records) of contacted and noncontacted individuals were compared. Contacted individuals from the court sample were more likely to have higher SES, be male, married, and non-Hispanic Caucasian (p <.05 for all). However, no significant differences were found in BAC at time of arrest, number of convictions, or Michigan Alcoholism Screening Test (MAST) scores. Similarly, contacted individuals from the HMO sample were more likely to be female. There were no differences between contacted and noncontacted individuals in the HMO sample on alcohol indicators.

Although data on refusal biases was not currently available for the larger study, this information has been reported elsewhere (Chassin et al., 1992). Individuals identified from court records were more likely to participate upon screening if they were unmarried

and non-Hispanic Caucasian. No differences in alcohol indicators were found. Those who were identified from HMO questionnaires demonstrated no differences based on refusal status. Comparison of participants and non-participants in the comparison group revealed that those who agreed to participate were more likely to be Hispanic, but no other differences emerged.

Procedure. At each wave of the study, trained interviewers began by administering informed consent procedures to parents and children. Interviewers informed families that the aim of the study was to understand why some people develop issues with drugs and alcohol while others do not. Interviews were conducted at Arizona State University or at families' homes, depending on which was more convenient for the family. Interviews were conducted by phone if the family had relocated. Family members were interviewed individually in order to ensure confidentiality of responses. Interviewers entered participants' verbal response onto the laptop for them, except when privacy was compromised, in which case participants entered their responses on a number pad. Interviews lasted 1-2 hours. Families were financially compensated for their participation at each wave, with the amount varying by wave.

The current study

Participants. To be included in the current analysis, G2s needed to (1) have at least one child by Wave 6, and (2) have complete data on the Wave 1 family disruption index. G2 full biological siblings were also included in analyses if they were age 18 or younger at Wave 1 (to ensure that they would have experienced the same family disruption events as the "target" G2s). These criteria yielded a sample size of 236 G2s.

This subsample was 64.4% (N = 152) female and 63.8% (N = 150) non-Hispanic Caucasian. G2s were a mean of 14 years old (range: 8-18) at baseline (Wave 1).

Differences between included versus excluded G2 participants were examined (see Table 1). Participants included in the subsample were significantly older than excluded participants, presumably because those who had children were more likely to be older. Included G2s were also less likely than excluded participants to be college graduates, and more likely to be Hispanic/Latino and female.

Measures. The measures used for the current study, to be further detailed, were part of a larger battery of measures. Descriptive statistics for all variables can be seen in Table 2.

Demographics. At Wave 1, G2 participants self-reported gender (1 = male, 0 = female) and ethnicity (0 = non-Hispanic Caucasian, 1 = Hispanic/Latino or other). G2s reported their age at each wave. G2 siblings, who were added to the study at Wave 4, first reported their age, gender, and ethnicity at this timepoint. Thus, G2 sibling ages at Waves 1, 2, and 3 were calculated by taking the difference between target G2s' age at Wave 4 and ages at Wave 1, 2, and 3, and subtracting these difference scores from the corresponding siblings' ages at Wave 4. At Wave 6, G3 children self-reported their gender, (0 = female, 1 = male), ethnicity (0 = non-Hispanic Caucasian, 1 = Hispanic/Latino or other), and age.

G1-G2 Family Disruption. At Wave 1, both G1 parents (if applicable) separately answered questions about history of separation from the G2 child while they were living with the child. Parents reported on number of times they had been divorced/separated from their partner, with responses ranging from (0) "Never" to (4) "More than three

times." If parental responses differed, suggesting divorces/separations from multiple partners, the higher of the two responses was used. Parents also reported the number of times they had been hospitalized for a physical illness or mental health problem and the number of times they had been to jail; coding reflected the same system used for parental divorce. Finally, parents reported if the child had ever experienced the death of a parent (0 = no, 1 = yes). For G2 siblings, who were missing data during Wave 1, family disruption data was obtained by using the corresponding target child's data. Final wave 1 family disruption events were represented as follows: parental divorce/separation (higher of two reporters), parental hospitalization (sum of two reporters), parental incarceration (sum of two reporters), and parental death (counted as "yes" if either parent endorsed). Single parent responses did not require choosing or summing across responses. Frequency of each family disruption event seemed reasonable, with parental hospitalization being the most frequent, followed by divorce, arrest, and death.

The wave 1 family disruption index was derived from a Principal Component Analysis (PCA) of the four disruption variables. PCA is a method to reduce the dimensionality of many observed variables into a smaller number of "principal components," which each represent a linear weighted combination of the original variables (Jolliffe, 2011). A polychroric correlation matrix of the variables was created to be used as input for the PCA. Proc PRINQUAL, a SAS procedure for PCA, was used to extract principal components (Kuhfeld et al., 1985). Finally, a web-based parallel analysis application was utilized to determine the number of factors to be extracted (Patil et al., 2017). In parallel analysis, the eigenvalues from the PCA are compared to eigenvalues created from randomly generated correlation matrices. When the eigenvalues

from the observed data exceed the eigenvalues from the random data for corresponding components, components are retained (Horn, 1965). This is similar to Kaiser's (1991) rule of retaining components with eigenvalue exceeding 1, except that in a parallel analysis, the benchmark value to exceed varies by component.

Results indicated that two components should be extracted from the PCA; results are presented in Table 3. For the first component, parental divorce and arrest showed high positive loadings, while parental death showed moderate negative loadings; loadings for hospitalization were low. For the second component, parental hospitalization showed high positive loadings and parental death showed moderate positive loadings, whereas loadings for arrest and divorce were low. The first component may be interpreted as a measure of family disruption related to parental "problem behavior" or "deviance" (i.e., marital problems, criminality), whereas the second may capture disruption due to parental health problems (i.e., hospitalization, death). Both family disruption scores will be entered as parallel predictors in each mediation model. Levels of both heath-related and deviance-related disruption at wave 1 were fairly low (Table 2).

At Waves 2 and 3, family disruption information was gathered from a combination of G1 and G2 report. Children were asked whether one or more parents went to jail (no = 0, yes = 1) or experienced a divorce/separation (no = 0, yes = 1) in the past year. Parents separately reported whether they had been hospitalized in the past year. Given the low rates of parental death during this timespan (N = 2), death was not included in the Wave 2-3 family disruption index. Binary variables were created for each type of disruption across waves 2 and 3, such that experiencing parental hospitalization, divorce, or arrest at either wave 2 or 3 was coded as (1) "yes," and the absence of the

event at both timepoints was coded as (0) "no." Again, family disruption information for siblings was gathered from "target child" data. PCA was utilized to create the Wave 2-3 family disruption index, and results suggested that one component should be extracted. The one-component PCA score had high positive loadings on arrest and divorce, and moderate positive loadings on hospitalization. The Wave 2-3 family disruption score will be included as a covariate in all analyses. There were again low levels of family disruption at waves 2-3 (Table 2).

G1-G2 Family Stressors. In order to parse out the effects of family disruption from other types of family stressors, a G1-G2 "other family stress" score was created. At Wave 1, life stressors were measured with items adapted from the Children of Alcoholics Life Events Schedule (Roosa et al., 1988) and the General Life Events Schedule for Children (GLESC; Sandler et al., 1986). 29 items in total were administered in the original study (see Appendix A), which were chosen from the Children of Alcoholics Life Events Schedule and the GLESC to tap events that were negative and uncontrollable. In the current study, items were further omitted if they were used in the family disruption index, related to covariates (e.g., parental AUD) or mediators (e.g., family conflict), or were endorsed too infrequently (i.e., residential changes). G2 children reported whether they had experienced a variety of family-related stressors in the past three months: a sibling having serious trouble (e.g., with the law, school, drugs), a sibling experiencing a serious illness or injury, parents having serious financial problems, parent losing a job, or the death of a close family member other than a parent.

The "other stress" index was created with PCA, which revealed two components (see Table 2). Component 1 displayed high positive loadings on parental financial

problems and parental job loss, and moderate negative loadings of sibling illness or injury; loadings for death of a family member and sibling problem behavior were low. Component 2 displayed high positive loadings for sibling problem behavior and death of a family member, with low loadings for the other three variables. To reduce the complexity of the final mediation models, and given that Component 2 displayed no significant correlation with other study variables, it will not be retained in analyses. Component 1, which captures G1-G2 family financial problems, will be included as a correlated predictor in mediation analyses. Low levels of family stressors were seen in the present sample (Table 2).

G1-G2 Family Environment. G1 mothers reported on their perceptions of the G1-G2 family environment during the past three months at Wave 1. Parent report of family environment was utilized because of the absence of self-report data from G2 age-eligible siblings at Wave 1. Maternal report, specifically, was chosen due to evidence that mothers are typically more involved in family interactions than fathers (Phares et al., 2008; McBride & Mills, 1993). Family environment was measured with two scales, family conflict and family disorganization, from Bloom's Family Processes Scale (BFPS; Bloom, 1985). All items were scored on a scale from (1) "strongly agree" to (5) "strongly disagree." Family conflict items captured the extent to which family members argued, hit each other, got angry at each other, threw things, lost their tempers, and criticized each other. Family disorganization reflected the extent to which family members had irregular schedules, had difficulties making plans due to unexpected events, and had trouble counting on each other's promises. Prior research on the psychometric properties of the BFPS has revealed good internal consistency and reliability for both family conflict (α =

.84; r = .88) and disorganization (α = .73; r = .87; Bloom & Naar, 1994) and the measure has been widely used. In the current sample, maternal report of family conflict (α = .69) and family disorganization (α = .75) had fair internal consistency. Family conflict and disorganization were fairly normally distributed; see Table 2.

G2 Dysregulation. G2 dysregulation was measured at Wave 1 using G1 maternal report of the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1978). Prior studies have validated the use of the CBCL Dysregulation Profile (CBCL-DP), which captures severe behavioral, cognitive, and emotional dysregulation by summing T-scores for the aggressive behavior, attention problems, and anxious/depressed subscales of the CBCL (Holtmann et al., 2010; De Caluwe et al., 2012; Althoff et al., 2011). Estimates of internal consistency in prior studies range from .81 to .92 and estimates of reliability are approximately .82 (De Caluwe et al., 2012; Masi et al., 2015). In the current study, only items of the CBCL that loaded onto both boys and girls age 12-15 were provided; thus, T-scores could not be calculated. Instead, raw scores for all available items in the three subscales (see Appendix B) were summed. Responses for individual items ranged from (1) "almost never" to (5) "almost always." High scores on the CBCL-DP reflect higher levels of dysregulation. Overall, levels of dysregulation in the sample were relatively low (see Table 2). The CBCL-DP scale in the current sample had excellent internal consistency (α = .93). Because dysregulation was measured at Wave 1, when only "original" G2s (not siblings) participated, the sample size will be smaller for models in which dysregulation is used as a mediator. There were moderately low levels of dysregulation in the sample (Table 2).

G2 Parenting Provided to G3. At Wave 6, G2 adults and G3 children reported on the parenting provided to G3s. Children and parents completed measures of parental monitoring, parental support, and parental consistency of discipline. Although some G2 parents had multiple children, only the oldest child's report of parenting was chosen, as their experience of G2 parenting was temporally closest to the time of G1-G2 family disruption. Levels of parent- and child-reported monitoring, support, and consistency tended to be high in the sample (see Table 2).

Parental support was measured with seven items from the Network of Relations Inventory (Furman & Burmeister, 1985). Example items include "How much can you count on your parent to be there when you need them?" and "How much does your parent treat you like you're admired and respected?" Participants responded on a five-point scale, ranging from (1) "little to none" to (5) "the most possible." Higher scores reflect higher levels of support. Internal consistency in the original measure was excellent (α = .90; Furman & Burmeister, 1985). In the current sample, internal consistency for child-report of support was .88 for mothers and .89 for fathers; α = .77 and .83 for mothers' and fathers' reports.

Five items assessing parental monitoring were taken from a measure assessing adolescents' perspectives on what their parents know about their interests and activities; prior research has demonstrated good internal consistency (α = .72; Lamborn et al., 1991). Items asked about how much they thought their parents (or in the case of self-report, how much they, themselves) asked about things such as "who [the child's] friends were" and "where [the child] was at night." Higher scores for monitoring indicate higher levels of parental monitoring. Internal consistency was .79 for maternal self-report and

.75 for paternal self-report; for child-report, internal consistency was .88 for maternal monitoring and .93 for paternal monitoring.

Parental consistency of discipline was indexed using 10 items taken from the Children's Report of Parental Behavior Inventory (CRPBI; Schaefer, 1965). Items measured consistency of parental discipline with statements such as "My parent didn't pay much attention to my behavior" and "My parent soon forgot the rules s/he had made." Items were answered on a scale ranging from (1) "strongly disagree" to (5) "strongly agree" and were reverse-scored so that higher scores represented greater consistency in discipline. Reliability of the original measure was .78 (Schaefer, 1965). In the present sample, internal consistency was good (α = .87 and .89 for children's report of maternal and paternal consistency, and α = .83 and .80 for maternal and paternal self-report).

Two latent factors were created by aggregating the three indicators, described above, for each reporter separately. A Tobit model was used in confirmatory factor analyses to account for ceiling effects in parenting scores in M*plus* Version 8.3. Tobit regression uses a latent underlying score instead of the observed score; this latent score is not constrained by the highest possible observed score and is thereby free to take on all values (McBee, 2010). Loadings for the parent-report latent factor were moderately strong (range .36-.57) and significant. For the child-report latent factor, the loading for parental support was strong (.72); loadings for parental consistency and monitoring were low (.19 and .21, respectively). Therefore, mediation models predicting child-report of G2 parenting will include all three scores as separate outcomes, whereas models predicting parent-report of parenting will use the latent factor score.

G1 Alcohol Use Disorder. Because the original study oversampled families with alcohol use disorder (AUD), parental (G1) AUD will be tested as a covariate. Information on lifetime diagnoses of DSM-III AUD was gathered using the computerized Diagnostic Interview Schedule (DIS, Version 3; Robins et al., 1981). Lifetime AUD diagnoses were obtained for noninterviewed parents with the Family History-Research Diagnostic Criteria (FH-RDC, Version 3; Endicott et al., 1975). A dichotomous variable was created to reflect whether at least one parent had a lifetime diagnosis of AUD. Less than half of the sample (39.4%) had at least one parent with AUD; see Table 2.

G1 Psychopathology. Analyses in the present study also tested G1 psychopathology as a covariate. G1 lifetime diagnoses of affective disorder (major depression and dysthymia) and antisocial personality disorder were assessed with the DIS-III (Robins et al., 1981) at Wave 1. Information on lifetime diagnoses of anxiety disorder was gathered at Wave 4, and age of onset data were used to determine whether the anxiety disorder had occurred prior to Wave 1. A dichotomous variable was created to capture whether at least one parent had a lifetime diagnosis of an affective disorder, anxiety disorder, or antisocial personality disorder. Approximately one-third (30.9%) of G2s had at least one parent with psychopathology (Table 2).

G1 Level of Education. G1 parents reported on the highest level of education they had reached. The higher level of education of the two parents (if applicable) was taken, and the variable was dichotomized to reflect college graduates and non-graduates. Less than a third (27.9%) of G1 parents had a college degree.

G3 Behavior Problems. In order to parse out child-driven effects on parenting outcomes, G3 conduct problems were tested as a covariate. G3 self-reported conduct

problems were measured at Wave 6 using the rule-breaking and aggression subscales of the Youth Self Report externalizing scale (Achenbach & Rescorla, 2001). These subscales have demonstrated excellent internal consistency (α = .81 and .86 for rule-breaking and aggressive behavior, respectively) and test-retest reliability (r = .83 and .88 for rule-breaking and aggressive behavior, respectively; Achenbach & Rescorla, 2001). Item responses ranged from (0) "not true" to (2) "very true." In the current sample, internal consistency was .89. Levels of G3 conduct problems were generally low; see Table 2.

Data Analysis

Covariates. Several variables were tested as potential covariates due to their theoretically plausible relationship to parenting (Belsky, 1984). First, G2 gender and ethnicity were tested as covariates. G2 gender was associated with levels of parental monitoring and G2 ethnicity was related to the G2 self-report parenting factor score; therefore, they will be included as covariates. Furthermore, G1-G2 family disruption occurring after Wave 1 will be entered as a covariate; given prior literature demonstrating that family disruption occurring earlier in development exerts a greater effect on children (Pasqualini et al., 2015; Milan et al., 2006; Sigle-Rushton and McLanahan, 2004), we are primarily interested the effect of earlier levels of disruption. G3 characteristics, including age and behavior problems, were also tested as potential covariates. Both were associated with self-report and child-report of parenting variables and will be included in all models. Lastly, variables related to family socioeconomic and psychosocial risk (e.g., G1 alcohol use disorder, psychopathology, and level of education) were included as covariates in order to parse out the effect of family disruption from that of other risk variables.

Multicollinearity. Multicollinearity between predictors was examined through the Variance Inflation Factors (VIFs) of all independent variables and mediators. First, initial correlations between variables were examined. Wave family disruption (deviance) was significantly associated with Wave 2-3 family disruption, G1 AUD diagnosis, psychiatric diagnosis, family disorganization, and family conflict. Wave 1 family disruption (health) was related to G1 psychiatric diagnosis and family conflict. Family disorganization and conflict were also correlated with each other. "Other stress" was related to Wave 2-3 family disruption and G3 behavior problems. G2 dysregulation was correlated with G2 ethnicity and G3 behavior problems. When VIFs were calculated, none were problematic. All values were between 1 and 2; values above 5 typically indicate multicollinearity (Sheather, 2009).

Intraclass correlations and design effects. In order to measure the degree of clustering in the present sample (i.e., between G2 siblings) and determine whether multilevel mediation analyses would be necessary for producing accurate standard errors, DEFT scores (the square root of the unconditional design effect; McNeish & Stapleton, 2016) were calculated for variables with potential clustering: G2- and G3-report parenting scores. A DEFT score was not calculated for G2 dysregulation, as only target G2s completed the CBCL at wave 1; their siblings did not. Further, DEFT scores were not computed for family disorganization and conflict because maternal report was used, and G2 siblings had equivalent data for these variables.

The DEFT score captures inflation of the standard error of the mean due to clustering, compared to inflation in data from a simple random sample (McNeish & Stapleton, 2016). The following formula is used to compute DEFT:

$$\text{DEFT} = \sqrt{1 + (m - 1) \times \text{ICC}}$$

The ICC is the intraclass correlation, and *m* represents the average cluster size. To compute DEFT scores for each variable, the intraclass correlation (ICC) is first calculated by dividing the between-cluster variability (τ_{00}) by the total variability ($\tau_{00} + \sigma^2$; McCoach & Adelson, 2010). A larger ICC suggests that there is greater homogeneity between clusters. An ICC of 0 indicates that DEFT = 1, and the Level 1 residual variance is equivalent to the total residual variance. If this is the case, multilevel modeling is not necessary for obtaining proper standard errors in analyses.

Mediation analyses. The present study utilized a series of four multiple mediation analyses to test the proposed hypotheses. M*plus* (Muthén & Muthén, 1998-2007) were used to test bootstrapping-based mediation. This is a method of mediation that utilizes nonparametric re-sampling and produces more statistical power than traditional procedures, such as the Sobel test (MacKinnon, Krull, & Lockwood, 2000; Preacher & Hayes, 2008). Parameter estimates and 95% bias-corrected and accelerated (BCa) confidence intervals were estimated for total, direct, and indirect effects using 5000 bootstrapped samples; statistical significance is met when zero is not included within the interval range (Preacher & Hayes, 2008).

Four separate multiple mediation models were tested. Model 1 (N = 236) tested (1) G1-G2 family disorganization and conflict as mediators of the effect of (1) deviance-related family disruption, (2) health-related family disruption, and (3) "other stress" on the G2 self-report factor score of parenting provided to G3 offspring, controlling for G1 AUD, G1 psychiatric diagnoses, G1 education, G2 ethnicity, G3 age, G3 behavior problems, and Wave 2-3 family disruption (see Figure 1). Model 2 (N = 236) examined

G1-G2 family disorganization and conflict as mediators of the effect (1) deviance-related family disruption, (2) health-related family disruption, and (3) "other stress" on child (G3) report of G2 parental consistency, monitoring, and support, controlling for G1 AUD, G1 psychiatric diagnoses, G2 gender, G3 age, G3 behavior problems, and Wave 2-3 family disruption (see Figure 2). Model 3 (N = 110; G2 siblings not included) analyzed G2 dysregulation as a mediator of the effect of (1) deviance-related family disruption, (2) health-related family disruption, and (3) "other stress" on G2 self-report factor score of parenting, controlling for G1 AUD, G1 psychiatric diagnoses, G1 education, G2 ethnicity, G3 age, G3 behavior problems, and Wave 2-3 family disruption (see Figure 3). Finally, model 4 (N = 110) examined G2 dysregulation as a mediator of the effect of (1) deviance-related family disruption, (2) health-related family disruption, and (3) "other stress" on child (G3) report of G2 parental consistency, monitoring, and support, controlling for G1 AUD, G1 psychiatric diagnoses, G2 gender, G3 age, G3 behavior problems, and Wave 2-3 family disruption (see Figure 4). Path models were estimated to simultaneously test (1) the effect of G1-G2 family disruption and other stress variables on mediators, (2) the effect of the mediators on G2 parenting of offspring, (3) the effect of family disruption and other stress variables on G2 parenting of G3 through the mediators (i.e., the mediated effect), (4) the direct effect of family disruption and other stress variables on G2 parenting of G3 (i.e., the unique effect of family disruption and other stress independent of the mediators), and (5) the total effect of family disruption and other stress on parenting (i.e., the indirect and direct effect combined). Clustering within families was taken into account in all analyses.

Results

Intraclass correlations and design effects. DEFT scores were calculated for the G2-report parenting factor score and the three G3-report parenting variables in order to determine the strength of clustering in the data. Intraclass correlations were calculated by specifying an unconditional multilevel model in *Mplus*. All ICCs were low (range: .011 - .107). The average cluster size (number of G2 siblings per family) was 1.35. DEFT scores were then computed, and all were close to 1 (range: 1 – 1.02), indicating that levels of clustering were low and multilevel modeling was not necessary for producing accurate standard errors in subsequent analyses.

Mediation analyses.

Model 1. Wave 1 family disruption variables, other family stress, and all covariates accounted for 4.7% of family disorganization (R^2 = .047) and 10.2% of family conflict (R^2 = .102). All predictors and covariates accounted for 18.7% of the variance in G2 self-report of parenting (R^2 = .187). The model demonstrated moderately good fit; X^2 = 122.27(44), p < .001; SRMR = .080; RMSEA = .087. Controlling for G1 AUD, G1 psychiatric diagnoses, G1 education, G2 ethnicity, G3 age, G3 behavior problems, and Wave 2-3 family disruption, the total effects of health-related family disruption, deviance-related family disruption, and other stress on parenting were not significant. Health-related family disruption predicted greater family conflict, but not family disorganization. Deviance-related disruption was related to greater family disorganization, but not conflict. Other stress did not show significant direct effects on family conflict or disorganization. Family conflict and disorganization did not have significant direct effects on parenting, controlling for all covariates. Neither deviance-

related disruption, health-related disruption, nor other stress had direct effects on parenting. All direct effects are shown in Table 8; total and specific indirect effects appear in Table 9. None of the total or specific indirect effects of health-related disruption, deviance-related disruption, or other stress on parenting, through family disorganization and conflict, were significant.

Model 2. Wave 1 family disruption and stress and all covariates accounted for 4.8% of the variance in family disorganization and 10.1% of the variance in family conflict. All predictors and mediators accounted for 16.7% of the variance in G3 report of G2 parental consistency, 14.9% of the variance in parental monitoring, and 11.4% of the variance in parental support. The model fit moderately well; $X_2 = 83.13(29)$, $p < .001$; SRMR = .069; RMSEA = .089. Controlling for G1 AUD, G1 psychiatric diagnoses, G2 gender, G3 age, G3 behavior problems, and Wave 2-3 family disruption, the total effects of deviance-related disruption, health-related disruption, and other stress on parental consistency were nonsignificant. The total effects of other stress and deviance-related family disruption on parental monitoring were not significant, but there was a significant total effect of health-related family disruption on parental monitoring; greater disruption was associated with lower levels of monitoring. The total effect of other stress on parental support was significant – greater stress predicted higher support – but the total effect of health- and deviance-related family disruption on support was not. Higher levels of health-related family disruption were related to greater family conflict, but not disorganization. Deviance-related family disruption predicted greater family disorganization, but not conflict. Other stress was not related to either family conflict or disorganization. Controlling for all covariates, family conflict predicted lower parental

consistency, but not support or monitoring. Family disorganization was not associated with parental consistency, support, or monitoring. There were no direct effects of disruption or other stress on parental consistency when controlling for family disorganization and conflict. Furthermore, greater health-related family disruption, but not deviance-related disruption or other stress, was associated with lower parental monitoring. Finally, higher levels of "other stress," but not family disruption, were related to greater parental support. All direct effects of predictors on family environment mediators and parenting outcomes are presented in Table 10.

Total and specific indirect effects of family disruption scores and other stress on parental monitoring and support were nonsignificant, as were total and specific indirect effects of deviance-related disruption and other stress on parental consistency. There was a specific indirect effect of family conflict, but not disorganization. The total indirect effect of health-related family disruption on parental consistency, through family disorganization and conflict, was significant. Total and specific indirect effects are presented in Table 11.

Model 3. Family disruption, other stress, and covariates accounted for 2.6% of the variance in G2 dysregulation. All predictors, including G2 dysregulation, accounted for 20.1% of the variance in G2 report of parenting provided to G3 children (R_2 = .201). The model fit moderately well; X_2 = 36.06(19), p = .010; SRMR = .055; RMSEA = .085. Direct effects of all predictors on G2 dysregulation and G2 report of parenting are available in Table 12. The total effects of deviance-related disruption, health-related disruption, and other stress on parenting were not significant, controlling for G1 AUD, G1 psychiatric diagnoses, G1 education, G2 ethnicity, G3 age, G3 behavior problems,

and Wave 2-3 family disruption. There were no significant direct effects of either family disruption score or other stress on dysregulation. Controlling for all covariates, dysregulation did not predict parenting. Neither family disruption variables, nor other stress, was associated with parenting. There were no indirect effects of health-related family disruption, deviance-related family disruption, and other stress on parenting through dysregulation; see Table 13.

Model 4. Deviance-related family disruption, health-related family disruption, other stress, and all covariates accounted for 2.6% of the variance in G2 dysregulation. All predictors, including G2 dysregulation, accounted for 25.0% of parental consistency, 18.5% of parental monitoring, and 19.0% of parental support. The model demonstrated moderately good fit; $X_2 = 15.22(8)$ $p = .055$; SRMR = .039; RMSEA = .086. Controlling for G1 AUD, G1 psychiatric diagnoses, G2 gender, G3 age, G3 behavior problems, and Wave 2-3 family disruption, there were no significant total effects of either disruption variable on parental consistency, monitoring, or support. There was a significant total effect of other stress on parental support, but not on parental consistency or monitoring; other stress predicted greater support. Direct effects are presented in Table 14. Neither deviance-related disruption, health-related disruption, nor other stress had a significant direct effect on dysregulation. Dysregulation was not related to parental monitoring or support, but greater dysregulation predicted lower consistency of parenting. Neither family disruption variable predicted parenting. Other stress was directly related to greater parental support, but not monitoring or consistency. None of the indirect effects of disruption or other stress to parental consistency, monitoring or support were significant (see Table 15).

Discussion

The present study examined, in a longitudinal sample of three generations, whether early family disruption in the G1-G2 family prospectively predicted poorer parenting provided to G3 offspring once G2 children reach adulthood. Further, it examined several individual-level and family-level mechanisms that may underlie this association: G1-G2 family environment (i.e., conflict, disorganization) and G2 dysregulation. Analyses yielded a number of interesting findings that inform current literature on family disruption.

Mechanisms underlying effect of family disruption on parenting

G2 dysregulation. Despite prior evidence for effects of family disruption on dysregulation (e.g., Hartman et al., 2018; Karreman & Vingerhoets, 2012; Lewis et al., 2007) and dysregulation on parenting (e.g., Thornberry et al., 2009; Rutherford et al., 2015; Johnston et al., 2006), there were no effects of family disruption on G2 dysregulation, and G2 dysregulation did not mediate the effect of G1-G2 family disruption on G2 parenting provided to G3 offspring.

Importantly, prior evidence for these pathways has related family disruption and parenting to specific domains of dysregulation, including dysregulation in behavior, cognition, and affect. Given common findings among these domains, the present study tested *global* dysregulation (i.e., co-occurring dysregulation in all three domains) as a mediating mechanisms underlying the relation between early family disruption and later parenting provided to offspring. Perhaps regulatory mechanisms in this pathway are more domain-specific than domain-general. For example, there is abundant evidence for effects of family disruption on behavioral and cognitive dysregulation (Ross & Hill, 2002;

Vicary & Lerner, 1986; Hartman et al., 2018; McCoy & Raver, 2014; Lewis et al., 2007; Harland et al., 2002; Liu et al., 2000; Geller et al., 2010). However, hypotheses about effects of family disruption on emotion dysregulation relied upon the assumption that this disruption impairs emotion regulation by undermining parent-child attachment. It may be the case that parent-child attachment is impervious to even major disruptions (e.g., parental divorce) in some cases, depending on the quality the parent-child relationship (Altenhofen et al., 2010; Solomon & George, 1999). Given this, future research should test more specific domains of dysregulation (i.e., behavioral, cognitive) as mediators of the effect of family disruption on later parenting behavior.

Another possible reason for these null findings is the heterogeneity in symptom presentation captured by the CBCL-DP. Prior research has yielded two methods of calculating the CBCL-DP: categorial measurement (child fits the profile if clinical cutoffs for aggressive behavior, attention problems, and anxiety/depression are all met concurrently), and continuous measurement (summing scores on all three subscales). Although prior research has validated the use of both methods (Kim et al, 2012), it is likely that continuous measurement represents a more heterogeneous profile; two children could receive the same score, for example, if one is elevated on both aggression and anxiety/depression while the other is elevated on attention and aggression. As such, the sum-score CBCL-DP may not truly represent a consistent profile of global dysregulation.

Although direct effects of family disruption on dysregulation and indirect effects of dysregulation were nonsignificant, there was a significant direct effect of dysregulation on later consistency of parenting provided to G3 offspring. This suggests that

dysregulation in adolescence is stable enough to impair the consistency of parenting provided to children in adulthood, and confidence in this finding is bolstered by the use of multiple reporters: G2 dysregulation was reported by G1 parents, whereas parental consistency was reported by G3 offspring. However, it remains unclear why parental dysregulation would undermine consistency of discipline, but not parental monitoring or support. Perhaps dysregulation exerts a particularly deleterious effect on parents' ability to harness self-regulatory skills in service of employing consistent rules and limits. whereas it is less consequential for their ability to provide support and compassion to their children. Moreover, parental consistency may differ from parental monitoring in that it requires greater effort from parents. Parents' knowledge of their children's friends and whereabouts may be driven more by child disclosure and thus less susceptible to effects of parental factors.

G1-G2 family environment. Mediation models testing the indirect effects of G1-G2 family disruption on parenting of G3s through G1-G2 family environment yielded mixed findings. Health-related family disruption predicted poorer parental consistency provided to G3 offspring, in part due to elevations in conflict in the G1-G2 family. In other words, it appears as though stress in the family due to parental death or hospitalization increases family conflict, which impairs children's ability to provide consistent discipline to their children once they reach adulthood. Again, parental consistency seemed more sensitive to predictors than parental monitoring and support, although the reason for this is unclear. Despite this significant indirect effect, the effect is small (*B* = -.023, 95% CI: -.054 – -.001) and was found in the context of numerous mediational pathways tested in four models; thus, interpretations must be made with

caution. This finding may also be the result of third variables (e.g., parental AUD and psychopathology) that presumably predict both parental mortality rates/health problems and family conflict. In fact, the measure of parental hospitalization included psychiatric hospitalization, further suggesting that the health-related disruption component may partially represent parental psychopathology.

In contrast to evidence for family conflict as a mediator of the effect of health-related family disruption on G3 report of parenting, there were no indirect effects of health-related disruption on parenting quality through family disorganization, and no indirect effects of deviance-related disruption or other stress on parenting through either family environment variable. It was surprising that deviance-related disruption and other stress did not predict family conflict, as prior research has demonstrated effects of parental divorce and incarceration on conflict within the family (Amato & Sobolewski, 2001; Fabricius & Luecken, 2007; Aaron & Dallaire, 2010). Although it did not predict family conflict, deviance-related disruption was associated with greater family disorganization, suggesting that events such as parental incarceration and divorce hinder the family's ability to adhere to predictable routines and schedules when one parent is absent.

Although there was one specific indirect effect of family disruption on G3 report of parenting, neither family disorganization nor family conflict mediated the effect of family disruption on G2 self-report of parenting. Capturing G2 report of parenting with a latent factor score may have obscured effects of family disruption and environment on specific domains of parenting (namely parental consistency, which appears to be the domain most sensitive to predictors in the present study). Alternatively, it is possible that

parental consistency, monitoring, and support are too conceptually distinct to be captured in a latent factor; factor loadings were significant but only moderate in strength (range .36-.57). Lastly, G2 parents may be worse reporters of the parenting that they provide since do not directly experience it and may be more susceptible to social desirability effects (Morsbach & Prinz, 2006).

Direct effects of family disruption on parenting

Several direct effects of family disruption on parenting of offspring emerged, over and above the effects of mediators and covariates. Greater health-related disruption in the G1-G2 family, but not deviance-related disruption, predicted poorer parental monitoring of G3s. Although neither family environment nor G2 dysregulation mediated this effect, perhaps other mediating mechanisms are at play, such as G2 depressive symptoms. Parental death and health problems are associated with greater depressive symptoms in children (Kendler et al., 2002; Siegel et al., 2002), which may then hinder their ability to later monitor their own children (Jones et al., 2003). Though not a predictor of interest, "other stress" in childhood was related to greater parental support provided to G3s in adulthood. However, these two variables were not associated in the zero-order correlations (see Table 5), which indicates that this may have been a spurious effect. In addition, despite the indirect effect of health-related family disruption on parenting consistency through family conflict, there was no direct effect, suggesting that this process occurs primarily through a mediated mechanism. Lastly, there were no direct effects of either family disruption variable on G2 parent-report of parenting, which again may reflect measurement error or biased reporting in this variable.

These findings stand in contrast to Friesen and colleagues' (2017) results, which demonstrated that the number of parental separations experienced in childhood for 6 months or more predicted physically abusive parenting and warmth, sensitivity, and overreactivity of parenting provided to offspring in adulthood, controlling for other sociodemographic characteristics. The authors did not find effects on consistency of parenting, as we did in the present study. This may be because family disruption only affects parenting consistency through indirect effects on the family environment, which were not tested in their study. They did not examine effects on parental monitoring, limiting the extent to which comparisons can be made in this domain. A number of methodological advantages in Friesen and colleagues' study may have contributed to their findings. First, their sample was much larger than ours (N = 1,265) and only tested direct effects, allowing for greater statistical power. Second, although survey measures relied on parental self-report, observational measures of parenting were also utilized, which lent greater objectivity to measurement. Finally, the authors utilized more stringent criteria for coding family disruption: only separations of 6 months or more were counted towards the sum score. We did not have information on the timespan of each disruption event in the present sample; however, parents were asked at wave 1 if they had ever been separated from their child for 6 months or more. More stringent testing of the current models could be accomplished by only including family disruption events that co-occurred with parental endorsement of this separation variable (i.e., by running a crosstabs analysis of each disruption type and the separation variable).

Direct effects of covariates on parenting

Although they were not effects of primary interest, there were effects of several covariates on parenting. First, G3 age and behavior problems were significantly or marginally related to all domains of parenting; older children and children with greater behavior problems evoked poorer parenting. These effects lend credence to our overall findings by demonstrating child-driven effects on parenting in the expected direction (Belsky, 1984). Furthermore, G2 gender was associated with G3 report of parental monitoring, such that female G2s monitored their children more. This result is consistent with prior findings demonstrating that mothers generally know more than fathers about their children's activities and whereabouts (Crouter et al., 1993). Finally, G1 psychiatric diagnosis predicted greater consistency of parenting provided from G2s to G3 children. However, this effect may be spurious, given that the two variables were not related in bivariate correlations (Table 5).

Strengths and limitations

The present study possesses a variety of strengths that add to the current literature on family disruption. First, a prospective longitudinal design was used to examine effects of early family disruption on later parenting provided to offspring in a sample that spans three generations. In addition, multiple reporters were used for study variables. G1 parents reported on G1-G2 family disruption, G1-G2 family environment, and G2 dysregulation; G2 children endorsed various family disruption events contributing to the family disruption indices and self-reported the quality of parenting they provided to their G3 children in adulthood; and G3 offspring reported on parenting provided to them by the G2s. Furthermore, effects of family disruption on parenting were tested above and

beyond a variety of other sociodemographic and risk factors, such as G1 parental psychopathology and substance use disorder, G1-G2 socioeconomic status, other stressors within the G1-G2 family, and G3 age and behavior problems. This allows for greater specificity in interpretation of effects. Lastly, unlike previous research that has examined effects of specific family disruption events (e.g., parental divorce or incarceration) or broad constructs like family instability that also capture instability outside of the parent-child relationship, the current study specifically focused on the cumulative impact of disruptions in the parent-child relationship. Using PCA, two components of family disruption, health-related and deviance-related disruption, were identified. These components appeared to have distinct implications for family environment and later parenting behavior: health-related disruption increases family conflict and impairs parenting of the subsequent generation, whereas deviance-related disruption heightens family disorganization and does not appear to have effects on parenting provided to next-generation offspring. This finding calls into question the methodology of previous literature on family disruption, which has almost exclusively measured cumulative disruption as a unitary construct. Future research should seek to validate the family disruption components identified in the present study and aim to elucidate further components.

The strengths of this study, however, must be taken in the context of several limitations. The sample size was relatively small, especially in the models tested without G2 siblings, which limited statistical power for analyses. In analyses that did include siblings (i.e., models that tested family conflict and disorganization as mediators), sibling report of family environment at wave 1 was not available; therefore, maternal report was

used across participants. Although this was likelier a better method than substituting target G2 child reports for their siblings' reports, it is not a perfect solution. Mothers' perceptions of the family environment may not match children's perceptions, which are what theoretically should predict later parenting that those children provide to their offspring. Another limitation is the concurrent measurement of predictors and mediators; retrospective reports of family disruption and reports of current levels of family conflict, family disorganization, and child dysregulation were all measured at wave 1. This was done in order to prevent a time lag between disruption events and mediators but nonetheless may have created bias. Furthermore, because mediators were measured at wave 1, prior levels of the mediators (preceding the disruption events) could not be covaried; thus, it is possible that the negative family environment or child dysregulation may have preceded or even contributed to family disruption. In addition, although parental death and hospitalization formed a single component, which was conceptualized to represent health-related disruption, the cause of parental death was not asked. As such, there is no way to confirm whether these deaths were truly the result of health-related issues or were instead caused by other factors. A final limitation is that effects of family disruption were analyzed *above and beyond* the effects of other distal risk factors. However, it appears that these risk factors, such as parental AUD and psychopathology, are important pieces of the puzzle, as they are correlated with family disruption. These distal risk factors may, in fact, initiate the chain of family disruption that exerts effects on parenting of the subsequent generation. If this is the case, parsing out the effects of these variables likely limited predictive power in the current models.

Implications and future directions

Results of the present study suggest that there is some evidence for the effects of early family disruption on quality of parenting provided to offspring in adulthood. Namely, the accumulation of health-related family disruption events, such as parental hospitalization and death, appears to undermine the quality of parental monitoring and consistency provided to the subsequent generation. For parental consistency in particular, this effect is mediated by conflict in the first- and second-generation family. This indicates that effects of family disruption on later parenting behaviors may occur through children's modeling of the dysfunctional family environment once they reach adulthood and create families of their own.

Despite these implications, given certain limitations of the present study, future research should aim to build upon these findings by incorporating several methodological advancements. First, pathways of early family disruption and later parenting outcomes should be modeled as part of the larger developmental sequalae of G1 parental psychopathology and AUD. It is likely that these distal predictors initiate patterns of family disruption and thus should be modeled as predictors themselves, not simply as covariates. Second, future research should aim to characterize the impact of cumulative family disruption occurring throughout childhood (birth to age 18), rather than early disruption. Although prior research indicates that family disruption occurring earlier in childhood exerts more deleterious effects, the measurement of "early" disruption in the present study was still relatively late (in adolescence) and only tested effects of disruption up to wave 1, above and beyond the effects of disruption events occurring one and two years later at waves 2 and 3. Capturing disruption throughout childhood would create

more variability in the family disruption index and reflect the impact of all disruption events experienced during this developmental period. Third, future research would benefit from genetically-informed designs, such as twin designs, to test these models more robustly. It is possible that associations between family disruption, family environment, child dysregulation, and children's later parenting of their offspring are simply reflective of shared genetic risk among parents and children, and analyses in the current study were unable to control for this. Fourth, utilizing Friesen and colleagues' (2017) more stringent criteria for family disruption (i.e., separation of parent and child for 6 months or more) would likely improve analyses by removing more minor events, such as hospital visits, that did not truly result in an extended separation. Finally, research in this area can be extended by examining alternative mediators of the effect of family disruption on parenting, effects on different domains of parenting, and consequences for other outcomes in adulthood *besides* parenting.

Table 1. *Comparison of included to excluded participants*

	Included		**Excluded**			
	N	***Mean (SD)***	***N***	***Mean (SD)***	***t***	***p-value***
G2 Age (W1)	236	14.00(2.28)	543	13.29(2.52)	-4.24	**<.001***
G2 Dysregulation (W1)	111	16.19(10.85)	300	16.92(11.46)	.575	.566
G1-G2 Family Conflict (W2)	222	2.69(.78)	518	2.63(.78)	-.936	.350
G1-G2 Family Disorganization (W2)	222	2.61(.71)	518	2.53(.72)	-1.39	.166
G1 Arrest (W1)	234	.30(.76)	506	.29(.73)	-.148	.883
G1 Divorce (W1)	234	.56(.98)	506	.51(.92)	-.756	.450
G1 Hospitalization (W1)	234	.61(.49)	506	.67(.47)	1.56	.119
	N	***%***	***N***	***%***	***Chi-Square***	***p-value***
G2 Gender	236		543		38.23	**<.001***
1 = Male		35.6% Male		59.7% Male		
0 = Female		64.4% Female		40.3% Female		
G2 Ethnicity					13.07	**<.001***
0 = Caucasian	235	63.8% Cauc.	535	76.4% Cauc.		
1 = Hispanic		36.2% Hisp.		23.6% Hisp.		
G1 Psychopathology	236		542		.035	.851
0 = No		69.2% No		69.7% No		
1 = Yes		30.8% Yes		30.3% Yes		
G1 AUD	236		543		1.43	.232
0 = No		39.4% Yes		44.0% Yes		
1 = Yes		60.6% No		56.0% No		
G1 Level of Education	215		519		4.49	**.034***
0 = Not college grad		27.9% Yes		34.0% Yes		
1 = College grad		72.1% No		64.0% No		
G1 Death	234		507		1.34	.247
0 = No		5.1% Yes		3.4% Yes		
1 = Yes		94.9% No		96.6% No		

Table 2. *Descriptive statistics for subsample of G1, G2, and G3 participants*

	N	Min.	Max.	Mean (SD)	Skewness (SE)	Kurtosis (SE)
G2 Age (W1)	236	8.00	18.00	14.00(2.28)	.02(.16)	-.71(.32)
G3 Age (W6)	236	7.36	17.44	12.86(1.95)	.13(.16)	-.35(.32)
Family Disruption: Deviance (W1)	236	-1.36	6.06	0.00(1.19)	2.89(.16)	9.76(.32)
Family Disruption: Health (W1)	236	-1.43	4.59	0.00(1.06)	1.82(.16)	4.76(.32)
Family Disruption (W 2-3)	216	-.55	5.59	0.00(1.22)	2.93(.17)	8.99(.33)
Other Family Stress (W1)	236	-1.19	2.80	0.00(1.12)	1.24(.16)	.63(.32)
Family Conflict (W1)	222	1.20	4.80	2.69(.78)	.69(.16)	.31(.32)
Family Disorganization (W1)	222	1.17	4.50	2.61(.71)	.32(.16)	-.27(.33)
G2 Dysregulation (W1)	111	0.00	57.00	16.19(10.85)	.97(.23)	1.15(.46)
G2 Parenting Factor Score, Self-Report (W6)	226	-3.05	1.85	0.00(.86)	-.51(.16)	.18(.32)
G2 Parenting Consistency, G3 Report (W6)	227	2.40	5.00	4.03(.60)	-.34(.16)	7.58(.32)
G2 Parental Monitoring, G3 Report (W6)	227	1.00	5.00	4.42(.72)	-2.50(.16)	7.58(.32)
G2 Parental Support, G3 Report (W6)	227	2.60	5.00	4.18(.56)	-.50(.16)	-.36(.32)
G3 Conduct Problems (W6)	226	0.00	21.00	2.51(3.42)	2.56(.16)	8.23(.32)

	N	%
G2 Gender	236	64.4% (N = 152) Female
G2 Ethnicity	235	63.6% (N=150) Non-Hispanic Caucasian
G1 Psychopathology	236	30.9% (N = 73) Yes
G1 Alcohol Use Disorder	236	39.4% (N = 93) Yes
G1 Level of Education	215	27.9% (N = 60) College Grads
G3 Gender	225	63.6% (N = 143) Female
G3 Ethnicity	160	43.6% (N = 103) Non-Hispanic Caucasian
		28.7% (N = 46) Hispanic
		1.3% (N = 3) African American
		3.4% (N = 8) Other

Note. Higher values of each continuous variable indicate higher levels of that variable.

Table 3. *Component Loadings for Principal Component Analysis Scores*

	Wave 1 Family Disruption		**Waves 2-3 Family Disruption**		**Wave 1 Other Stress**	
Variable	**Comp. 1**	**Comp. 2**	**Comp. 1**	**Variable**	**Comp. 1**	**Comp. 2**
Parent Divorce	.824	.156	.897	Sibling Deviance	.176	.807
Parent Death	-.626	.499	N/A	Sibling Illness	-.589	.140
Parent Arrest	.773	.025	.852	Financial Problems	.861	.084
Parent Hospitalization	.181	.906	.436	Parent Job Loss	.753	.007
				Family Death	-.169	.812

Table 4. *Correlations between variables in model 1*

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Disrupt: deviance W1	--												
2. Disrupt: health W1	0	--											
3. Disrupt W2-3	.214**	-.102	--										
4. Other stress W1	.106	-.048	.216**	--									
5. G1 AUD	.327**	.125	.035	.090	--								
6. G1 Psych Dx	.267**	.186**	139*	.089	.136**	--							
7. G1 Education	-.074	.159*	.004	.047	-.073	.177**	--						
8. G2 Ethnicity	.028	-.068	-.128	.028	.086	-.257*	-.240**	--					
9. G1-G2 fam disorg. W1	.170*	.124	.240**	.064	.094	.063	-.017	.035	--				
10. G1-G2 fam con. W1	.170*	.228**	.126	.173*	.252**	165**	.018	-.062	.363**	--			
11. G3 age W6	.101	-.120	-.075	.074	.124	.013	-.138*	.188**	.004	.046	--		
12. G3 bx problems W6	.107	.-.032	.152*	.180**	.021	.064	-.123	-.085	.051	.099	.116	--	
13. G2 parenting (self-report)	-.095	.026	-.037	.004	-.118	.096	.147*	-.147**	-.055	-.042	-.271**	-.302**	--

Note. *$p < .05$, **$p < .01$

Table 5. *Correlations between variables in model 2*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Disrupt: deviance W1	--														
2. Disrupt: health W1	0	--													
3. Disrupt W2-3	.214**	-.102	--												
4. Other stress W1	.106	-.048	.216**	--											
5. G1 AUD	.327**	.125	.035	.090	--										
6. G1 Psych Dx	.267**	.186**	139*	.089	.136**	--									
7. G1 Education	-.074	.159*	.004	.047	-.073	.177**	--								
8. G2 Gender	-.116	.038	.054	-.084	-.056	-.038	.005	--							
9. G1-G2 fam disorg. W1	.170*	.124	.240**	.064	.094	.063	-.017	.-.054	--						
10. G1-G2 fam con. W1	.170*	.228**	.126	.173*	.252**	165**	.018	-.050	.363**	--					
11. G3 age W6	.101	-.120	-.075	.074	.124	.013	-.138*	-.130	.004	.046	--				
12. G3 bx problems W6	.107	.-.032	.152*	.180**	.021	.064	-.123	-170*	.051	.099	.116	--			
13. G2 parental consist. (G3 rep)	-.102	.032	-.125	-.155*	-.102	.124	.048	.078	-.122	-.159	-.189**	-.266**	--		
14. G2 parental mon. (G3 rep)	.022	-.096	.031	.041	-.099	-.011	.011	-.236**	-.062	-.039	-.183**	-.124	.119	--	
15. G2 parental supp. (G3 rep)	-.058	-.001	-.066	.095	-.078	-.061	.057	-.047	.013	-.050	-.134**	-.255**	.364**	.312**	--

Note. *$p < .05$, **$p < .01$

Table 6. *Correlations between variables in model 3*

	1	2	3	4	5	6	7	8	9	10	11	12
1. Disrupt: deviance W1	--											
2. Disrupt: health W1	.052	--										
3. Disrupt W2-3	.255**	-.081	--									
4. Other stress W1	.201*	.029	.231*	--								
5. G1 AUD	.345**	.116	-.028	.238**	--							
6. G1 psych Dx	.305**	.122	.212*	.066	.173	--						
7. G1 education	-.083	.113	-.004	.088	-.069	.068	--					
8. G2 ethnicity	.070	.095	-.106	.080	.183*	-.229*	-.229**	--				
9. G2 dysregulation W1	-.033	.033	.155	.145	.118	.093	-.062	-.191*	--			
10. G3 age W6	.157	.003	-.007	.123	.207*	.026	.089	.207*	.085	--		
11. G3 bx problems W6	.093	-.007	.051	.225*	.060	.116	-.139	-.034	.262**	.152	--	
12. G2 parenting (self-report)	-.134	.027	-.031	.037	-.078	.074	-.074	-.183*	-.066	-.244**	-.297**	--

Note. *$p < .05$, **$p < .01$

Table 7. *Correlations between variables in model 4*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Disrupt: deviance W1	--													
2. Disrupt: health W1	.052	--												
3. Disrupt W2-3	.255**	-.081	--											
4. Other stress W1	.201*	.029	.231*	--										
5. G1 AUD	.345**	.116	-.028	.238**	--									
6. G1 psych Dx	.305**	.122	.212*	.066	.173	--								
7. G1 education	-.083	.113	-.004	.088	-.069	.068	--							
8. G2 gender	-.089	.009	.021	-.145	-.134	-.109	.028	--						
9. G2 dysregulation W1	-.033	.033	.155	.145	.118	.093	-.062	-.017	--					
10. G3 age W6	.157	.003	-.007	.123	.207*	.026	.089	-.131	.085	--				
11. G3 bx problems W6	.093	-.007	.051	.225*	.060	.116	-.139	-.246**	.262**	.152	--			
12. G2 parental consist. (G3 report)	-.018	-.071	-.099	-.235*	-.138	.097	.030	.104	-.262**	-.346**	-.260**	--		
13. G2 parental mon. (G3 report)	.016	-.058	.055	.095	-.092	.095	.070	-.222*	-.009	-.268**	-.108	.225*	--	
14. G2 parental supp. (G3 report)	-.011	-.064	-.098	.138	-.058	-.028	.096	-.103	.030	-.134	-.252**	.249**	.431**	--

Note. $*p < .05$, $**p < .01$

Table 8

Model 1: direct effects of predictors on family environment and G2 report parenting

	β	*p*
Parenting		
Family Disorganization	-.027	.679
Family Conflict	-.002	.966
W1 Disruption (Deviance)	-.038	.555
W1 Disruption (Health)	-.043	.377
W1 Other Stress	.093	.153
G1 AUD	-.075	.187
G1 Psych Dx	.111	.069
G1 Level of Education	.038	.570
G2 Ethnicity	-.098	.144
G3 Age	**-.223**	**.001****
G3 Behavior Problems	**-.288**	**.001****
W2-3 Family Disruption	.055	.376
Family Disorganization		
W1 Disruption (Deviance)	**.171**	**.022***
W1 Disruption (Health)	.121	.223
W1 Other Stress	.039	.667
Family Conflict		
W1 Disruption (Deviance)	.158	.093
W1 Disruption (Health)	**.220**	**.001****
W1 Other Stress	.143	.089

Table 9

Model 1: mediation by family disorganization/conflict on family disruption/stress and G2 report parenting

		95% BCa bootstrap CI	
	β	Lower	Upper
Family Disruption (Deviance)			
Parenting			
Family Conflict	.001	-.017	.017
Family Disorganization	-.005	-.027	.018
Total Indirect	-.005	-.031	.021
TOTAL	-.043	-.166	.080
Family Disruption (Health)			
Parenting			
Family Conflict	.001	-.024	.023
Family Disorganization	-.001	-.020	.014
Total Indirect	-.001	-.031	.024
TOTAL	.092	-.146	.054
Other Stress			
Parenting			
Family Conflict	-.001	-.016	.015
Family Disorganization	-.003	-.009	.007
Total Indirect	-.004	-.019	.016
TOTAL	-.046	-.040	.223

Note. Point est. = point estimate of the indirect effect; BCa bootstrap CI = Bias corrected and accelerated confidence intervals.

Table 10

Model 2: direct effects of predictors on family environment and G3 report parenting

	β	*p*
Parental Consistency		
Family Disorganization	-.054	.403
Family Conflict	**-.106**	**.040***
W1 Disruption (Deviance)	-.045	.527
W1 Disruption (Health)	.005	.927
W1 Other Stress	-.078	.290
G1 AUD	-.051	.433
G1 Psych Dx	**.194**	**.001****
G3 Age	**-.145**	**.036***
G3 Behavior Problems	**-.218**	**.001****
W2-3 Family Disruption	-.064	.352
Parental Monitoring		
Family Disorganization	-.075	.208
Family Conflict	.014	.729
W1 Disruption (Deviance)	.056	.180
W1 Disruption (Health)	**-.101**	**.030***
W1 Other Stress	.066	.217
G1 AUD	-.098	.092
G1 Psych Dx	-.007	.904
G2 Gender	**-.268**	**.001****
G3 Age	**-.206**	**.004****
G3 Behavior Problems	**-.172**	**.008****
W2-3 Family Disruption	.034	.440
Parental Support		
Family Disorganization	.057	.420
Family Conflict	-.036	.669
W1 Disruption (Deviance)	.002	.978
W1 Disruption (Health)	-.026	.635
W1 Other Stress	**.179**	**.006****
G1 AUD	-.062	.385
G1 Psych Dx	-.037	.541
G3 Age	-.112	.064
G3 Behavior Problems	**-.261**	**.001****
W2-3 Family Disruption	-.082	.250
Family Disorganization		
W1 Disruption (Deviance)	**.175**	**.020***
W1 Disruption (Health)	.120	.228
W1 Other Stress	.037	.682
Family Conflict		
W1 Disruption (Deviance)	.158	.293
W1 Disruption (Health)	**.218**	**.001****
W1 Other Stress	.144	.085

Table 11

Model 2: mediation by family environment on family disruption/stress and G3 report parenting

		95% BCa bootstrap CI	
	β	Lower	Upper
Family Disruption (Deviance)			
Parental Consistency			
Family Conflict	-.017	-.042	.009
Family Disorganization	-.009	-.034	.015
Total Indirect	-.026	-.065	.013
TOTAL	-.071	-.205	.062
Parental Monitoring			
Family Conflict	.002	-.011	.015
Family Disorganization	-.013	-.036	.009
Total Indirect	-.011	-.035	.013
TOTAL	.045	-.036	.126
Parental Support			
Family Conflict	-.006	-.032	.020
Family Disorganization	.010	-.016	.036
Total Indirect	.004	-.021	.029
TOTAL	.006	-.116	.128
Family Disruption (Health)			
Parental Consistency			
Family Conflict	**-.023**	**-.054**	**-.001**
Family Disorganization	-.006	-.024	.009
Total Indirect	**-.029**	**-.060**	**-.004**
TOTAL	-.024	-.138	.071
Parental Monitoring			
Family Conflict	.003	-.014	.020
Family Disorganization	-.009	-.030	.012
Total Indirect	-.006	-.031	.019
TOTAL	**-.107**	**-.208**	**-.006**
Parental Support			
Family Conflict	-.008	-.045	.029
Family Disorganization	.007	-.013	.026
Total Indirect	-.001	-.035	.033
TOTAL	-.027	-.127	.073
Other Stress			
Parental Consistency			
Family Conflict	-.015	-.037	.006
Family Disorganization	-.002	-.012	.008
Total Indirect	-.017	-.044	.009
TOTAL	-.096	-.239	.048
Parental Monitoring			
Family Conflict	.002	-.009	.013
Family Disorganization	-.003	-.017	.012
Total Indirect	-.001	-.019	.018
TOTAL	.065	-.041	.170
Parental Support			
Family Conflict	-.005	-.029	.018
Family Disorganization	.002	-.009	.013
Total Indirect	-.003	-.024	.018
TOTAL	**.176**	**.051**	**.300**

Note. Point est. = point estimate of the indirect effect; BCa bootstrap CI = Bias corrected and accelerated confidence intervals.

Table 12

Model 3: direct effects of predictors on G2 dysregulation and G2 report parenting

	β	*p*
Parenting		
G2 Dysregulation	-.068	.479
W1 Disruption (Deviance)	-.126	.296
W1 Disruption (Health)	.030	.698
W1 Other Stress	.192	.057
G1 AUD	-.009	.929
G1 Psych Dx	.088	.331
G1 Level of Education	-.057	.573
G2 Ethnicity	-.187	.058
G3 Age	-.171	.050
G3 Behavior Problems	**-.302**	**.001****
W2-3 Family Disruption	-.075	.461
G2 Dysregulation		
W1 Disruption (Deviance)	-.067	.422
W1 Disruption (Health)	.033	.682
W1 Other Stress	.156	.055

Table 13

Model 3: mediation by G2 dysregulation on family disruption/stress and G2 report parenting

		95% BCa bootstrap CI	
	β	Lower	Upper
Family Disruption (Deviance)			
Parenting			
Dysregulation	.005	-.013	.022
TOTAL	-.121	-.358	.115
Family Disruption (Health)			
Parenting			
Dysregulation	-.002	-.015	.010
TOTAL	.028	-.124	.180
Other Stress			
Parenting			
Dysregulation	-.011	-.044	.023
TOTAL	.182	-.023	.387

Note. Point est. = point estimate of the indirect effect; BCa bootstrap CI = Bias corrected and accelerated confidence intervals.

Table 14

Model 4: direct effects of predictors on G2 dysregulation and G3 report parenting

	β	*p*
Parental Consistency		
G2 Dysregulation	**-.189**	**.040***
W1 Disruption (Deviance)	.055	.538
W1 Disruption (Health)	.-.089	.274
W1 Other Stress	-.137	.124
G1 AUD	-.058	.524
G1 Psych Dx	**.180**	**.030***
G3 Age	**-.294**	**.001****
G3 Behavior Problems	**-.163**	**.011***
W2-3 Family Disruption	-.106	.157
Parental Monitoring		
G2 Dysregulation	.064	.458
W1 Disruption (Deviance)	.054	.375
W1 Disruption (Health)	-.064	.197
W1 Other Stress	.130	.112
G1 AUD	-.131	.171
G1 Psych Dx	.079	.184
G2 Gender	**-.241**	**.001****
G3 Age	**-.275**	**.003****
G3 Behavior Problems	**-.187**	**.001****
W2-3 Family Disruption	-.001	.990
Parental Support		
G2 Dysregulation	.122	.207
W1 Disruption (Deviance)	.070	.468
W1 Disruption (Health)	-.085	.316
W1 Other Stress	**.260**	**.002****
G1 AUD	-.110	.253
G1 Psych Dx	-.006	.943
G3 Age	-.111	.158
G3 Behavior Problems	**-.313**	**.001****
W2-3 Family Disruption	**-.193**	**.019***
G2 Dysregulation		
W1 Disruption (Deviance)	-.065	.449
W1 Disruption (Health)	.026	.749
W1 Other Stress	.158	.050

Table 15

Model 4: mediation by G2 dysregulation on family disruption/stress and G3 report parenting

		95% BCa bootstrap CI	
	β	Lower	Upper
Family Disruption (Deviance)			
Parental Consistency			
Dysregulation	.012	-.022	.047
TOTAL	.068	-.113	.249
Parental Monitoring			
Dysregulation	-.004	-.019	.011
TOTAL	.050	-.068	.167
Parental Support			
Dysregulation	-.008	-.029	.013
TOTAL	.062	-.129	.254
Family Disruption (Health)			
Parental Consistency			
Dysregulation	-.005	-.035	.025
TOTAL	-.094	-.247	.059
Parental Monitoring			
Dysregulation	.002	-.010	.013
TOTAL	-.062	-.159	.034
Parental Support			
Dysregulation	.003	-.017	.024
TOTAL	-.082	-.254	.089
Other Stress			
Parental Consistency			
Dysregulation	-.030	-.067	.008
TOTAL	-.167	-.342	.008
Parental Monitoring			
Dysregulation	.010	-.018	.038
TOTAL	.140	-.023	.303
Parental Support			
Dysregulation	.019	-.014	.053
TOTAL	**.279**	**.118**	**.441**

Note. Point est. = point estimate of the indirect effect; BCa bootstrap CI = Bias corrected and accelerated confidence intervals.

Figure 1. *Family disorganization and conflict mediating the effect of family disruption on later parenting of own offspring (self-report).*

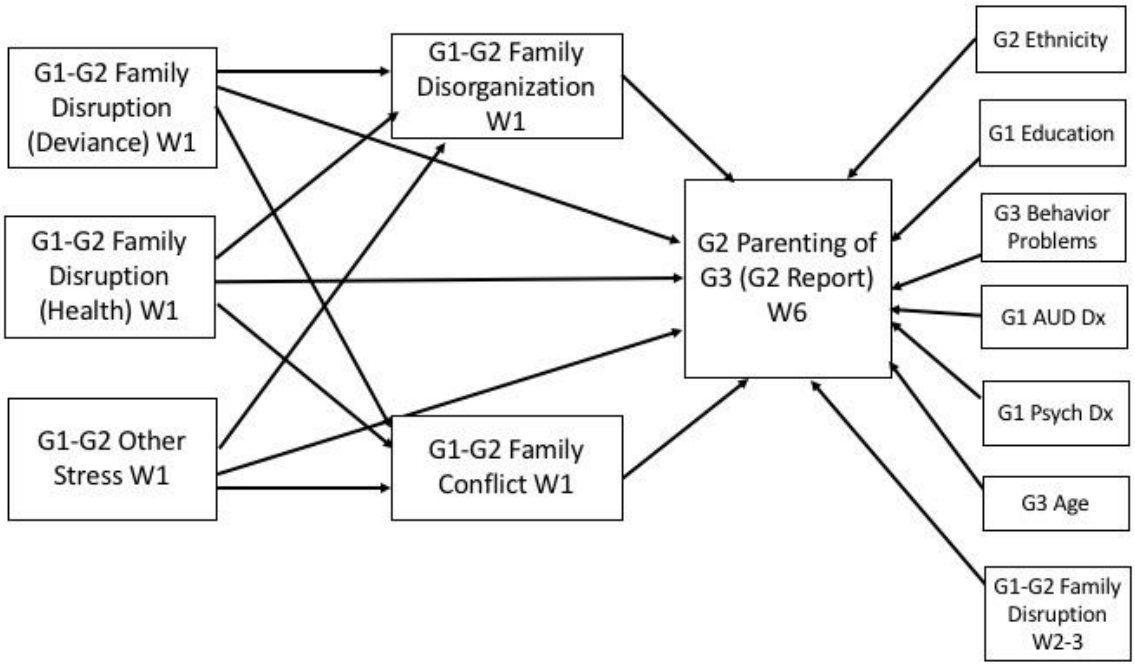


Figure 2. *Family disorganization and conflict mediating the effect of family disruption on later parenting of own offspring (child-report).*

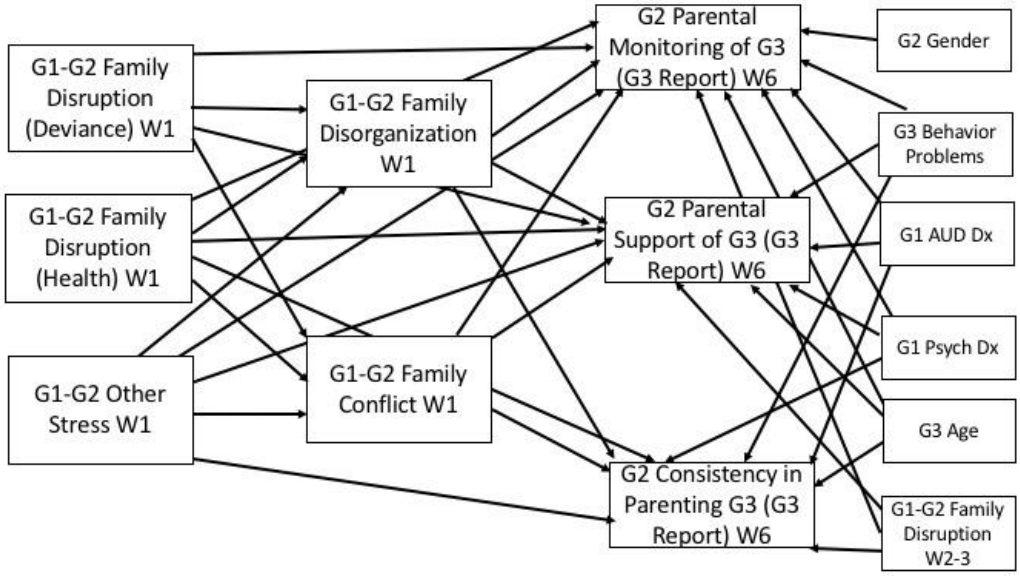


Figure 3. *Dysregulation mediating the effect of family disruption on later parenting of own offspring (parent-report).*

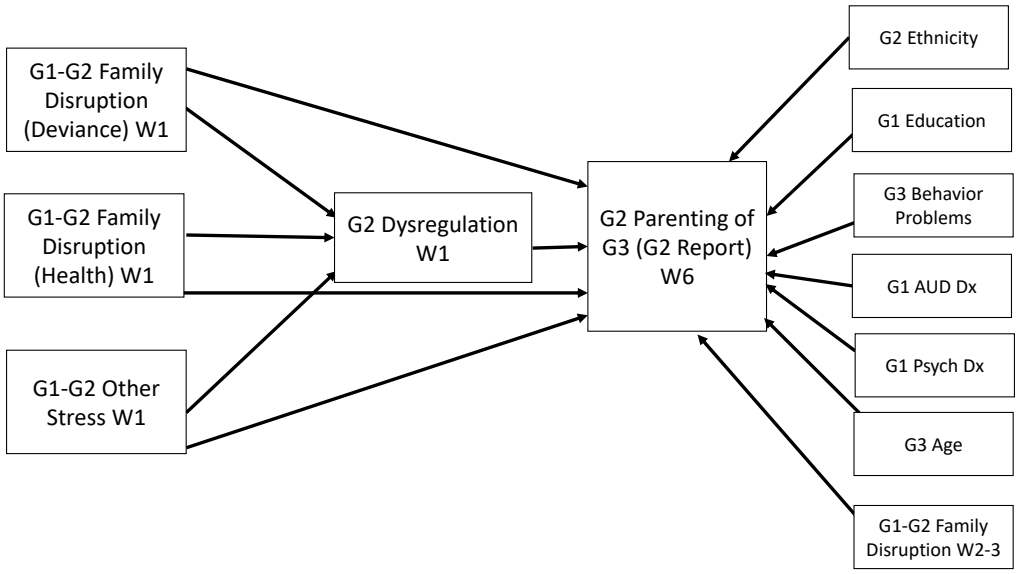
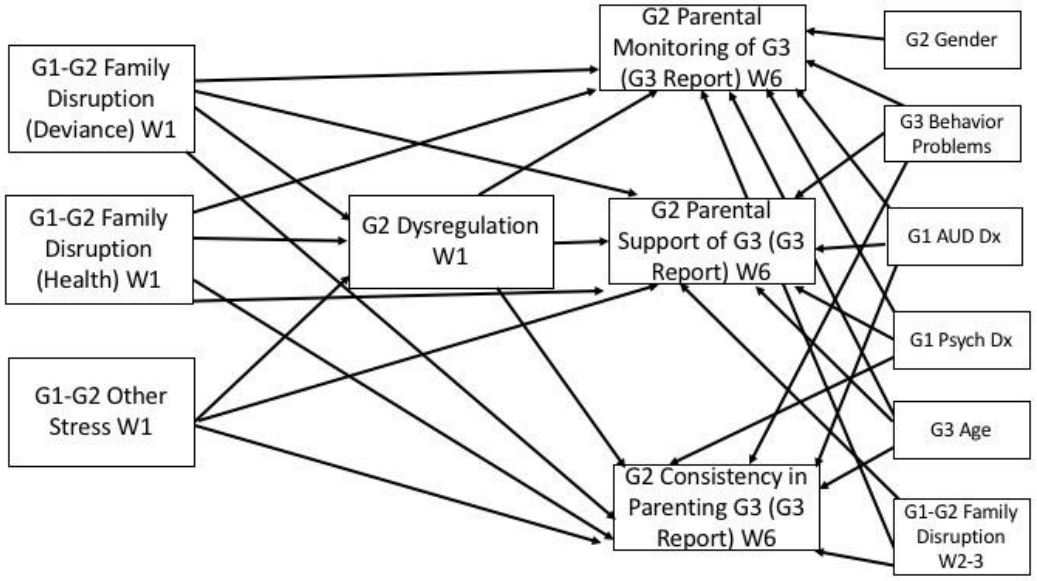


Figure 4. *Dysregulation mediating the effect of family disruption on later parenting of own offspring (child-report).*



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APPENDIX A

ITEMS IN THE LIFE STRESS SCALE

Appendix A: Items in the Life Stress Scale

Rating scale: 1 “Yes,” 2 “No,” 3 “Not applicable”

Here is a list of things that happen to people. Which happened to you in the past 3 months?

1. Your brother or sister had serious trouble (with the law, school, drugs, etc.).*
2. Your brother or sister suffered a serious physical illness or injury.*
3. You suffered a serious physical illness or injury.
4. Your close friend had serious troubles, problems, illness, or injury.
5. Your mom or dad suffered a serious illness or injury.
6. Your mom or dad talked about having serious money troubles.*
7. Your relatives said bad things about your mom or dad.
8. Your mom or dad fought or argued with your relatives.
9. People in your neighborhood said bad things about your mom or dad.
10. Your mom or dad acted badly in front of your friends.
11. You saw your mom or dad drunk.
12. Your mom or dad forgot to do important things for you that they promised they would do (such as take you someplace or go to school or athletic activities).
13. Your mom or dad was arrested or sent to jail.
14. Your mom or dad lost their job.*
15. A close family member died.*
16. You changed schools because of a family move.
17. A close friend of yours died.
18. A close friend of yours moved away.
19. Your mom and dad got divorced or separated.
20. You were the victim of a crime.
21. Your mom and dad argued in front of you.
22. You saw your mom or dad drunk in public.
23. Your mom or dad spent one or more nights away from home when they should have been home.
24. You took care of your mom or dad when they were drunk.
25. Your mom or dad criticized things you’ve done well.
26. Your mom said bad things about your dad.
27. Your dad said bad things about your mom.
28. Your mom or dad screamed, shouted, or broke things.
29. Your boyfriend or girlfriend broke up with you.

*Items tested in PCA for the family stress scale. Only items 6 and 14 were retained in the final component.

APPENDIX B

ITEMS IN THE CBCL DYSREGULAITON PROFILE (CBCL-DP)

Appendix B: Items in CBCL Dysregulation Profile (CBCL-DP)

Rating scale: 0 "Not true," 1 "Somewhat or sometimes true," 2 "Very true or often true"

1. Argues a lot
2. Brags
3. Complains of loneliness
4. Cries a lot
5. Daydreams or gets lost in his/her thoughts
6. Easily distractible
7. Fears he/she might think or do something bad
8. Feels he/she has to be perfect
9. Fidgety, has difficulty sitting still
10. Fears or complains that no one loves him/her
11. Feels worthless or inferior
12. Immature
13. Nervous, high-strung, or tense
14. Explosive
15. Too fearful or anxious
16. Feels too guilty
17. Self-conscious or easily embarrassed
18. Stares blankly
19. Mean or cruel to others
20. Unhappy, sad, or depressed
21. Worrying
22. Jealous of others
23. Destroys his/her own things
24. Feels others are out to get him/her
25. Destroys things belonging to others
26. Disobeys at home
27. Disobeys at school
28. Starts fights
29. Acts without stopping to think
30. Physically attacks people
31. Screams a lot
32. Shows off or clowns
33. Moods/feelings change suddenly
34. Talks too much
35. Teases a lot
36. Quick-tempered
37. Threatens people
38. Unusually loud

APPENDIX C

IRB AND FUNDING ACKNOWLEDGEMENTS

Appendix C: IRB and Funding Acknowledgements

This work was supported by the National Institute of Alcohol Abuse and Alcoholism: R01 AA016213 and R21 AA022097-02 to Laurie Chassin. Approval was obtained by the Institutional Review Boards at Arizona State University (IRB# 0506000017).