

INTERGENERATIONAL CONTINUITY IN HIGH CONFLICT FAMILY ENVIRONMENT:
INVESTIGATING A MEDIATING DEPRESSIVE PATHWAY

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ABSTRACT

William Andrew Rothenberg: Intergenerational continuity in high conflict family environments:
Investigating a mediating depressive pathway
(Under the direction of Andrea Hussong)

Experiencing family conflict is associated with numerous deleterious outcomes across ontogeny, including increases in externalizing and internalizing behavior and impairments in social functioning. Emerging evidence suggests that family conflict shows continuity across generations and that intergenerational family conflict can be more intense and deleterious than conflict experienced in a single generation. However, few investigations have identified etiological mechanisms by which family conflict is perpetuated across generations. Nor have many investigations examined for whom, or under what circumstances, family conflict persists over generations. Addressing these limitations, the current study examined whether G2 depressive symptoms measured at multiple time points across development explained continuity in family conflict from one generation to the next. Results revealed that depressive symptoms served as mediators of intergenerational family conflict in both men and women, but in different ways. Specifically, G2 women's adolescence and young adulthood each represented periods of vulnerability in which G2 depressive symptoms were especially likely to mediate intergenerational continuity in family conflict. Additionally, in both men and women, higher G1-G2 family conflict was associated with higher depressive symptoms that persisted from adolescence into young adulthood and then subsequently predicted the development of G2-G3 family conflict. Results did not support the hypothesis that G2 partner depressive symptoms

moderated the relation between G2 depressive symptoms and G2-G3 family conflict.

Implications of findings regarding the roles that G2 gender and G2 depressive symptoms play in the transmission of family conflict from one generation to the next are discussed

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INTRODUCTION

Myriad developmental scientists and clinicians have identified high family conflict as a threat to child and adult well-being (Cummings & Schatz, 2012). Family conflict is associated with the development and persistence of numerous deleterious behaviors (Dishion & Patterson, 2006; Pelton & Forehand, 2001) and is a prospective predictor of internalizing symptoms (Cummings & Schatz, 2012; Jaycox & Repetti, 1993; Herrenkohl, Lee, Kosterman & Hawkins, 2012; Kouros & Garber, 2014), externalizing symptoms (Kimonis, Frick, & McMahon, 2014; Moffitt et al., 2006), substance misuse (Pillow, Barrera & Chassin, 1998; Herrenkohl et al., 2012) and impairments in social role functioning (Cummings & Schatz, 2012; Horowitz et al., 2011; Rothenberg, Solis, Hussong, & Chassin, under review) across the life-course.

Emerging evidence suggests that family conflict can persist across generations within families, such that high family conflict in homes comprised of G1 (or first generation at baseline) parents and their G2 (or second generation) children prospectively predicts high conflict in homes comprised of that G2 parent, his or her spouse and their G3 (or third generation) children (Rothenberg, Hussong, & Chassin, 2016). Conflict that persists from G1-G2 homes to G2-G3 homes is more intense and maladaptive than conflict in a single (G2-G3) family environment (Rothenberg et al., under review). Moreover, G2s and G3s from families that show continuity in conflict across multiple generations report significantly greater psychopathology across symptom domains ($d = 0.54$ to 0.91) than those from other family types (including families that demonstrate high conflict only in one generation; Rothenberg et al., under review). These large and deleterious effects suggest that family conflict that persists across generations poses a

significant threat to healthy development and thriving across the lifespan. However, few investigations have explored the etiological mechanisms that may underlie intergenerational continuity in family conflict (Rothenberg et al., 2016). Identification of such mechanisms would inform our understanding of when and where to intervene to break these pernicious intergenerational cycles of conflict.

Therefore, the present investigation explores one mediating mechanism that could help explain intergenerational continuity in conflict; namely, a depressive pathway that persists across the life-course. I posit that this depressive pathway emerges as children experience emotional insecurity, negative affect and behavioral withdrawal due to family conflict in childhood (Cummings & Schatz, 2012) and consequently develop depressive symptoms that persist across ontogeny and influence the development of conflict in their adult families. Numerous longitudinal single-generation studies link family conflict with the subsequent emergence of depressive symptoms in adolescence (Auerbach & Ho, 2012; Habib et al., 2013; Sheeber, Hops, Alpert, Davis, & Andrews, 1997), demonstrate that depressive symptoms show relative stability from adolescence into adulthood (Hammen, Rudolph, & Abaied, 2014; Hammen & Rudolph, 2003) and show that depressive symptoms in adulthood predict conflict in the families that these adults form (Sarigiani, Heath, & Camarena, 2003).

However, there are several limitations in the existing literature that are addressed by the current study. First, though existing studies of single generation family processes have examined each of the links in the proposed depressive pathway, no investigation has followed a single cohort of individuals across ontogeny to determine whether depression as a result of conflict in one's family of origin influences conflict in one's family of destination (i.e. the family one forms in adulthood). Second, few investigations of family conflict examine G2 psychopathology across

multiple time points (e.g., in adolescence and young adulthood, though see Rothenberg et al., 2016). Therefore, it is unclear whether there is developmental specificity in the prediction of G2-G3 conflict from G2 depressive symptoms such that G2 depressive symptoms experienced at specific points in development (i.e., depression experienced in adolescence versus young adulthood) present heightened risk for the intergenerational transmission of family conflict.

Third, many existing studies find significant but only modest associations in family processes across generations (e.g., Conger et al., 2009; Rothenberg et al., 2016). Therefore, it is important to identify for whom and in what circumstances such intergenerational links may be especially strong. In particular, G2 gender (Rothenberg et al., 2016) and G2 parenting partner behavior (e.g., Conger, Schofield, Neppl, & Merrick, 2013) are two potential moderators of intergenerational conflict that hold promise for identifying when intergenerational continuity may be strongest. Specifically, emerging evidence suggests that pathways to intergenerational continuity in family conflict may be stronger for women than men (Rothenberg et al., 2016). Additionally, researchers have called for greater consideration of the role of partner behavior in perpetuating intergenerational phenomena (e.g., Conger et al., 2013), though little empirical research exists examining the effect of parenting partner behavior on intergenerational transmission of family conflict. Investigating the depressive pathway and the moderating factors that alter such a pathway, would facilitate identification of intervention targets at multiple points across the lifespan to break cycles of intergenerational family conflict and of personal and family characteristics that place individuals at greatest risk for recreating high conflict family environments.

The current study addresses these limitations by using a multigenerational sample of high-risk families and matched controls to evaluate intergenerational continuities in family

conflict as reported by multiple family members across generations. In addition, the present study tests whether a depressive pathway mediates cross-generational consistencies in family conflict, whether there is developmental specificity in the effects of the depressive pathway and whether the depressive pathway is more apparent for women and those whose partners have greater depressive symptoms. These aims are captured in the conceptual model depicted in Figure 1.

Defining Family Conflict

Existing work examines various indicators of family conflict including such parenting behaviors as harsh discipline, ignoring and aggression, poor cohesion in the family environment (Seiffge-Krenke, 1999) and conflict in both dyadic (e.g., parent-child and marital) and triadic family relationships (Emery, 1993). These multiple definitions of family conflict reflect the state of the current literature. Common to many definitions of family conflict is a focus on physical and verbal aggression, frequent criticism, and displays of anger and recurring arguments that occur across multiple relationships in the family (Choe, Stoddard & Zimmerman, 2014; Cummings & Schatz, 2012; Fosco, Caruthers, & Dishion, 2012; Habib et al., 2013; Van Ryzin & Dishion, 2012). Family theorists suggest that conflicts between parents, between parents and children, and between siblings each contribute to the overall climate of the family environment (Cummings & Davies, 2010; Cummings & Schatz, 2012). Accordingly, family conflict cannot be inferred from assessments of individual dyads only but must also be assessed as a broader, family-level construct (Cummings & Schatz, 2012; Emery, 1993). The present investigation defines family conflict as the experience of physical or verbal aggression, criticism, anger, or arguments within the family and utilizes measures of family conflict that align with this definition.

Additionally, developmental scientists suggest that intergenerational continuities in family environment are best captured when children in successive generations within these families are studied at similar points in development (Conger, Belsky & Capaldi, 2009; Kovan, Chung & Sroufe, 2009; Van IJzendoorn, 1992) because patterns of family interaction are most similar at these points. However, few studies are actually able to capture families in this way (Conger et al., 2009). Contributing to the literature, the present investigation examined conflict in families in successive generations with adolescent children as opposed to conflict in families with dissimilar structures (e.g. studying families with adolescents in one generation and families with no children in the next generation).

Theoretical Models of Intergenerational Family Conflict

Recent work demonstrated that family conflict exhibits significant continuity across generations (Rothenberg et al., 2016; Rothenberg et al., under review) and that intergenerational continuity in conflict can be partially accounted for by mediating G2 externalizing problems that develop in high-conflict families of origin, persist across time, and predict the development of family conflict in G2-G3 families (Rothenberg et al., 2016). Social Interactional Theory (Dishion & Patterson, 2006; Scaramella, Conger, Spoth, & Simons, 2002) may best explain the mechanisms underlying this externalizing pathway for intergenerational family conflict. According to this perspective, in high conflict G1-G2 family environments G2s learn to increase their own conflict behavior (e.g., shouting, yelling, noncompliance) to obtain social goals (e.g., receiving parental attention, avoiding chores, etc.) when such interaction patterns are normative and reinforced in the family. These G2 reactions to conflict are subsequently internalized and applied as effective social strategies to obtain goals in a variety of settings across the lifespan,

including in their own G2-G3 families. This pattern of behavior ultimately gives rise to similar conflict in the G2-G3 family to that seen in the G1-G2 family (Rothenberg et al., 2016).

Building on this conceptualization, I posit that the repeated aversive interactions experienced and observed by G2s in high-conflict G1-G2 family environments are likely to cause a variety of G2 reactions in addition to, or as an alternative to, those associated with externalizing behaviors. These alternative reactions could also perpetuate continuity in family conflict across generations. Specifically, I posit that G2s also experience depressive symptoms as a result of high-conflict G1-G2 family environments and that these depressive symptoms inform the development of G2-G3 family conflict through a series of three linked processes.

Most pertinent to the current study, I hypothesize that G2s may respond to high-conflict interactions within the G1-G2 family environment by withdrawing from family interactions to avoid the aversive consequences of family conflict (e.g., getting in trouble, avoiding unreasonable punishment from family members). This withdrawing behavior may be rewarding and serve as a negative reinforcer because it allows G2s to avoid aversive stimuli within the family environment (Auerbach & Ho, 2012; Cummings, Koss, & Davies, 2015). However, such withdrawal is also a functional impairment that is associated with depression more broadly (Hammen & Rudolph, 2003). Moreover, the same interparental and parent-child conflict interactions that make a child's withdrawing behavior adaptive (Auerbach & Ho, 2012) also endanger a child's sense of safety and security, causing emotion dysregulation, uncertainty, negative affect and a host of other depressive symptoms (as demonstrated by the robust body of literature supporting Cummings & Davies' Emotional Security Hypothesis; Cummings & Davies, 2010; Cummings & Schatz, 2012; Habib et al., 2013). Therefore, I hypothesize that the first link in the depressive pathway falls into place when G1-G2 family conflict contributes to the

emergence of G2 depressive symptoms through initially adaptive withdrawing behavior, negative affect and other mood dysregulation in response to that conflict.

Second, I hypothesize that due to the negatively reinforcing nature of behavioral withdrawal within the G1-G2 family environment, G2 withdrawal and associated depressive symptoms are likely to be internalized and utilized in social interactions throughout ontogeny. Consequently, G2 depressive symptoms that emerge as a result of G1-G2 family conflict will persist across time into adulthood . Though few studies have explicitly tested this specific mechanism, one investigation based from this conceptual framework found that family conflict did prospectively predict adolescent depression and that this link was sustained by interpersonal stress emerging from maladaptive interactions with peers (Auerbach & Ho, 2012). Many more investigations have found that G2 depressive symptoms often emerge in adolescence and remain relatively stable into adulthood (e.g., Angold & Egger, 2007; Barnett et al., 2010, Hammen & Rudolph, 2003; Hammen et al., 2014; Mills et al., 2011). Indeed, 5 international prospective longitudinal studies, including the Queensland High Risk Study (Hammen, Brennan, Keenan-Miller & Herr, 2008), Dunedin Multidisciplinary Health and Development Study (Bardone, Moffitt, Caspi, Dickson, & Silva, 1996), Ontario Child Health Study (Fleming, Boyle, & Offord, 1993), Oregon Adolescent Depression Project (Lewinsohn, Rhode, Klein, & Seeley, 1999) and Upstate New York study (Pine, Cohen, Gurley, Brook, & Ma, 1998) all reported high rates of recurrence of depressive symptoms throughout the life span (Hammen & Rudolph, 2003; Hammen et al., 2014). Thus, I posit that the second link in the depressive pathway falls into place when G2 depressive symptoms that emerge from high conflict G1-G2 families are internalized and remain stable across time.

Third, I expect that G2 depressive symptoms that persist across time contribute to the emergence of high conflict when G2s form their own G2-G3 families due to the effects such symptoms have on multiple family interactions. For example, a large body of research demonstrates that depression and withdrawal in one romantic partner can lead to frustration, confusion and increased demandingness within the other romantic partner. These emotional reactions are often the result of partners searching for affirmation that the depressed partner still cares for them (i.e., the demand-withdrawal pattern of marital interaction; Baucom, McFarland, & Christensen, 2010; Holley, Strum & Levenson, 2010). When this pattern happens within the family context, such interparental conflict often spills over into the family environment (Cummings & Schatz, 2012) and can be linked to similar patterns of frustrated, defiant and attention-seeking behavior found in children of depressed parents (e.g., Dishion & Patterson, 2006; McMahon & Forehand, 2003; Emery, 1993). Therefore, as a result of G2 depressive symptoms and consequent interactions with G2 romantic partners and eventually their own G3 children, the high conflict that pervaded G1-G2 families may be perpetuated in G2-G3 families.

In sum, the primary study hypotheses are guided by principles from Social Interactional Theory (Dishion & Patterson, 2006; Scaramella et al., 2002) and the Emotional Security Hypothesis (Cummings & Davies, 2010). Specifically, I posit that high conflict in one's G1-G2 family will contribute to the development and reinforcement of depressive symptoms in G2s. I further expect such G2 depressive symptoms to persist into adulthood when G2s start their own families and that such depressive symptoms will subsequently contribute to high conflict in the G2-G3 home. Though no literature has investigated this depressive pathway as a mediator of family conflict across multiple generations, support for this pathway emerges from other prospective empirical work that has found evidence for each of the pathway's component parts.

Empirical evidence for the first link in the depressive pathway (i.e., linking G1-G2 family conflict to the emergence of G2 depression in adolescence) and the third link in the depressive pathway (i.e. linking G2 depression in adulthood to the emergence of G2-G3 family conflict) will be examined further below. Empirical evidence for the second link in the depressive pathway (i.e., that G2 depressive behavior shows stability over time into adulthood) will not be further explored because this finding is robust and well-replicated in existing literature (Hammen & Rudolph, 2003; Hammen et al., 2014).

Additionally, in conceptualizing the depressive pathway that links intergenerational continuities in conflict, I have thus far considered mechanisms that are primarily psychosocial in nature. However, it is important to acknowledge that depression demonstrates significant genetic heritability (Hammen & Rudolph, 2003; Hammen et al., 2014). Moreover, longitudinal work following twin children ages 5 to 16 found an interaction between child genetic risk for depression (as measured by an equation weighting both twins' depression scores by a coefficient of genetic relatedness that was 1.0 for monozygotic twins and 0.5 for dizygotic twins) and family conflict (Rice, Harold, Shelton, & Thapar, 2006). Specifically, study results indicated that the association between family conflict and subsequent depressive symptoms 3 years later was found to be greater in children and adolescents at genetic risk for depression (Rice, Harold, Shelton, & Thapar, 2006). Therefore, because depression is heritable and the neurobiological mechanisms by which genetic risk for depression interacts with family conflict to influence subsequent depression are under continuing investigation, the present study will attempt to control for the effects of genetic risk for depression by controlling for G1 depression in all study analyses. Controlling at the phenotypic level in this way is suggested by researchers investigating genetic

effects on family conflict (Rice et al., 2006). However, these genetic effects will not be considered further in the present study.

Linking Family Conflict to the Emergence of Depressive Symptoms

The first link in a depressive pathway from G1-G2 family conflict to G2-G3 family conflict is the association between G1-G2 family conflict and the emergence of G2 depressive symptoms. Core to this conceptualization is the idea that G1-G2 family conflict may cause a constellation of depressive symptoms, including G2 withdrawal to avoid aversive interactions with family members, G2 negative affect, and G2 depressed mood as a result of interparental conflict. Here, I review literature examining these associations in adolescence. I focus on this developmental period because severe depressive symptoms most commonly emerge in adolescence (Hammen & Rudolph, 2003; Hammen et al., 2014); as such I examined studies investigating family conflict most proximal to this high-risk period.

Multiple prospective investigations have found links between family conflict and adolescent withdrawal from family interactions. For instance, Roubinov and Luecken (2013) found that adolescent utilization of disengaged coping methods mediated the relationship between family conflict and adolescent depressive symptoms. Additionally, several investigators evaluating the Emotional Security Hypothesis identified adolescent disengagement from family interactions as a common pattern of coping in high conflict families (Cummings et al., 2015; Forman & Davies, 2005) and found that high family conflict prospectively predicted a greater likelihood of adolescent disengagement (Cummings et al., 2015). Furthermore, researchers using the well-known Family Check-Up Intervention (designed to increase family member's engagement with one another as a treatment target) also found that adolescents in families who received this intervention showed significantly less escalation in family conflict over grades 6 to

9 and that, as a result, adolescents experienced significantly less growth in depressive symptoms during that time period (Fosco, Van Ryzin, Connell, & Stormshak, 2016). Taken together, these studies suggest that adolescents in high conflict families are more likely to withdraw from high conflict family interactions.

Multiple cross-sectional and longitudinal investigations also found associations between high family conflict and the development of adolescent depressive symptoms more generally. From the cross-sectional literature, one study of nearly 9,000 adolescents identified family conflict as the risk factor most strongly associated with adolescent depressive symptoms from a range of numerous risk and protective factors (Bond, Toumbourou, Thomas, Catalano, & Patton, 2005). Similarly, other cross-sectional studies of adolescents ages 10-16 found evidence for the association between family conflict and adolescent depressive symptoms in Caucasian (Herman, Ostrander, & Tucker, 2007), African American (Constantine, 2006) and ethnically diverse (Davis & Epkins, 2009; Formoso, Gonzales, & Aiken, 2000) samples. This association held even after accounting for other parenting (e.g., attachment, monitoring; Constantine, 2006; Formoso et al., 2000), peer (i.e., peer attachment; Formoso et al., 2000) and community (i.e., community violence; Holtzman & Roberts, 2012) variables. Additionally, seminal work by Sheeber and colleagues found that greater family conflict predicted higher adolescent depressive symptoms one year later (Sheeber et al., 1997). Subsequent longitudinal investigations have replicated this prospective link in a sample of Australian children (Habib et al., 2013) and across a 4.5 month time frame (Auerbach & Ho, 2012).

Taken together, these cross-sectional and longitudinal investigations demonstrate that high family conflict is a robust prospective predictor of adolescent withdrawal and other depressive symptoms. Further, this literature suggests that the link between family conflict and

adolescent depression is significant across diverse samples and remains so even after controlling for other influential risk and protective factors including parent, peer and community characteristics.

Linking Adult Depressive Symptoms to the Emergence of Family Conflict

The third link in the depressive pathway from G1-G2 family conflict to G2-G3 family conflict is the link between adult depressive symptoms and the emergence of conflict in the G2-G3 family environment. Although prospective longitudinal evidence linking parental depression and family-level conflict is still relatively rare, one longitudinal study that measured conflict at the family-, as opposed to dyadic-, level found that the presence of one parent who reported recurrent depressed mood prospectively predicted higher adolescent reports of family conflict one year later (Sarigiani et al., 2003). Additionally, ample evidence from longitudinal work has examined the link between adult depression and dyadic conflict throughout the family environment. These investigations index dyadic family conflict in the marital and parent-child relationships and support the assertion that adult depression can be linked to high family conflict. Indeed, several systematic reviews implicate parental depression as one of the primary predictors of both marital conflict and the spill-over of such conflict into the larger family environment (Cummings & Davies, 2010; Cummings & Davies, 2002; Cummings & Schatz, 2012). Other investigators have found that higher depressive symptoms in one parent or romantic partner predict higher levels of interparental conflict (Fear et al., 2009). Similarly, several meta-analyses and systematic reviews have found that parental depression prospectively predicts many components of family conflict, including maladaptive parenting behaviors (Dix & Meunier, 2009; Kane & Garber, 2004; Lovejoy et al., 2000). Specifically, one meta-analysis of 46 studies found that depressed parents displayed more negative and disengaged behaviors and fewer

positive behaviors (Lovejoy et al., 2000). Another systematic review of 152 studies similarly concluded that parent depressive symptoms reduced parents' attention given to children, lowered parental sensitivity, responsiveness and involvement, and increased negative appraisals of children and parental competence (Dix & Meunier, 2009). In sum, existing evidence from the dyadic and parenting conflict literatures and smaller but extant family conflict literature suggests that parental depression does predict high family conflict.

Taking each of these bodies of evidence into account, I hypothesize that a depressive pathway that emerges due to family conflict experienced in adolescence persists across time into adulthood, where it predicts the development of subsequent family conflict in one's family of destination (see Figure 1).

Developmental Specificity in the Depressive Pathway

Existing literature indicates the potential for periods of vulnerability in the depressive pathway, such that psychopathology at certain points in development may be an especially strong mediator of continuity in family conflict from G1-G2 to G2-G3 environments (e.g., Rothenberg et al., 2016; Conger et al., 2009). Specifically, in my own work I have found that psychopathology that emerges in G2s' adolescence mediates the relationship between G1-G2 and G2-G3 family conflict above and beyond psychopathology at other time points in development (Rothenberg et al., 2016). Moreover, other investigators reviewing the intergenerational parenting literature have noted that psychopathology experienced in G2s' young adulthood may also have an especially high impact on subsequent adult parenting behaviors, compared to that experienced at earlier time points (Conger et al., 2009). However, this literature operationalized G2 psychopathology as externalizing behavior and no one has examined developmental sensitivity in mood symptoms with regards to intergenerational patterns of family conflict.

Generalizing these findings to the depressive pathway, I posit that G2 depressive symptoms experienced in adolescence and young adulthood may be especially salient mediators of intergenerational continuity in family conflict from G1-G2 to G2-G3 families (as depicted by all of the paths labeled "2" in Figure 1).

Depressive symptoms experienced during adolescence may put G2s at elevated risk for perpetuating intergenerational family conflict for several reasons. Adolescence is the most common period of onset for adolescent depressive symptoms (Hammen & Rudolph, 2003; Hammen et al., 2014) and a time of relative tumult in family environments (Arnett, 2000). Adolescents often experience emotional distress and behavioral disengagement as they negotiate their autonomy and emerging identities and begin separating from their families of origin (Friedman, Holmbeck, DeLucia, Jandased & Zebracki, 2009; Hock, Eberly, Bartel-Haring, Ellwanger & Widaman, 2001; Wray-Lake, Crouter, & McHale, 2010). However, these hallmark parent-adolescent negotiations and associated distress can emerge as depressive symptoms in adolescents when they take place in high conflict families (Auerbach & Ho, 2012; Constantine, 2006). Within such families, coercive intrafamilial interactions around these issues can lead to adolescent withdrawal, insecurity and subsequent depression (Constantine, 2006; Vivona, 2000). Importantly, adolescence is a time period in which patterns of social interaction that influence adult functioning are likely to be established and internalized (Jaffee, Belsky, Harrington, Caspi & Moffitt, 2006; Thornberry et al., 2003). Therefore, G1-G2 family conflict may make it more likely that adolescents establish and internalize withdrawal and associated depressive symptoms that eventually lead to conflict in their G2-G3 families. Consequently, adolescence may be an especially salient risk period for the development of G2 depressive symptoms that mediate the

relationship between G1-G2 and G2-G3 family conflict because the conflict-withdrawal pattern of interaction may become less malleable after adolescence in G2s.

G2 depressive symptoms experienced in young adulthood (i.e., between the ages of 18-25) may also represent an especially salient risk for intergenerational continuity in family conflict. Individuals face enormous transitions and important decisions within their educational, occupational and romantic lives as they enter young adulthood and experience normative stress and distress while navigating these issues (Arnett, 2000; Howard, Galambos, & Krahn, 2010). I posit that such typical stress can evolve into, or exacerbate, existing symptoms of depression in G2s from high-conflict family environments who lack the requisite familial communication strategies or social support (due to growing up in a high-conflict environment) to combat such feelings of distress. Consequently, behaviors and emotions related to G2 depressive symptoms may begin to characterize G2 social interactions during the exact time period when G2s are most likely to be securing romantic partners and starting families. As a result, such depressive symptoms may catalyze the development of high conflict interactions in the nascent G2-G3 family environment. Those patterns of family interaction may then stabilize over time, thus once again perpetuating conflict across generations.

Evidence from several longitudinal investigations of young adulthood lend support to these claims. For instance, investigators have noted that as young adults reach major transitions in young adulthood like marriage, childbirth and early child rearing, they tend to contact and interact with their families of origin more than at other times (Cowan & Cowan, 2012; Cowan & Cowan, 2000). Such increases in interaction with one's family of origin during these transitions can result in increased conflict between family generations (Cowan & Cowan, 2012). Importantly, longitudinal work suggests that the extent to which such conflict occurs depends

largely on the relationship one has with one's family of origin before the role transition; if such interactions with one's family of origin were negatively valenced before the marriage or pregnancy, they are likely to be so afterward (Cowan & Cowan, 2012). Therefore, young adult G2s are likely to experience increased contact with their G1 parents during young adult role transitions and if G2s are from high conflict family environments, such interactions are likely to continue to be high in conflict.

Furthermore, such high conflict interactions have been linked to increases in G2 depressive symptoms, stress and distress longitudinally (Cowan & Cowan, 2012). Such depressive symptoms in young adulthood are associated with increases in marital and family conflict in one's family of destination in the first years of marriage (Hammen & Rudolph, 2003; Cowan & Cowan, 2012) and parenthood (Hammen et al., 2014; Cowan & Cowan, 2012). Perhaps surprisingly, longitudinal investigations demonstrate that these increases in conflict in one's family of destination tend to show stability for many years after a transition occurs (e.g., Cowan & Cowan, 2012; Cummings, DeArth-Pendley, Du Rocher Schudlich, & Smith, 2001). Indeed, group interventions for couples designed to decrease family conflict and increase adaptive family interactions showed that when such interventions were delivered to young adult couples with preschool-aged children, intervention effects persisted for periods of up to 10 years, as those originally preschool-age children were entering high school (Cowan, Cowan, & Barry, 2011).

Collectively, these investigations suggest that lack of support from high conflict G1-G2 families may exacerbate G2 young adult depressive symptoms. This may be due to the increased contact between G1-G2 families and young adult G2s as young adult G2s are forming their own families by marriage, pregnancy and early child-rearing. These elevated G2 young adult

depressive symptoms may then subsequently affect the emergence of high conflict interaction patterns in emerging G2-G3 families. Such high conflict in G2-G3 families persists across time. Thus, G2 depressive symptoms experienced in young adulthood may be especially likely to perpetuate intergenerational family conflict from G1-G2 to G2-G3 families.

Gender Specificity in the Depressive Pathway

Researchers have repeatedly noted that though continuity in family conflict is demonstrated across generations, these associations are often modest in magnitude (Conger et al., 2009; Rothenberg et al., 2016). Therefore, the current study aims to identify particular individual characteristics that may make someone more likely to traverse a depressive pathway to intergenerational continuity in family conflict. In my prior work, I found that one such individual characteristic that moderated intergenerational continuity in family conflict was G2 gender (Rothenberg et al., 2016). Specifically, I found that G2 gender moderated the relationship between G1-G2 and G2-G3 family conflict such that the indirect effects (via G2 externalizing behavior) of G1-G2 family conflict on G2-G3 family conflict were significant in women but not men and accounted for over 40% of the variance in G2-G3 family conflict scores in women. Notably, these effects were found even after other covariates known to influence family conflict, such as antisocial personality disorder, alcoholism and socio-economic status, were controlled.

I posit that G2 gender may also moderate the mediating depressive pathway between G1-G2 and G2-G3 family conflict, such that the pathway is significant in women but not in men. Existing theory and evidence indicates that women may be at elevated risk for experiencing the depressive pathway for two key reasons. First, conflict in one's family of origin may be more likely to affect the emergence of depressive symptoms in adolescent girls compared to boys (e.g., Constantine, 2006). G2 adolescent girls are more likely to experience depression as a result of

family conflict in their G1-G2 family (Hops, 1995; Sheeber et al., 1997) perhaps because G2 girls are often socialized to invest in familial and interpersonal relationships in their family of origin more so than are boys (e.g., Constantine, 2006; Jones & Costin, 1995; Monetmayor, 1982; Noller, 1994; Sheeber et al., 1997). As a result, conflict in such relationships may be more deleterious for G2 girls' depressive symptoms, especially in adolescence as instances of family conflict and experiences of depressive symptoms normatively increase (Constantine, 2006; Noller, 1994). Indeed, theorists exploring sexual selection theories of gender vulnerability (e.g., Triosi, 2001) posit that compared to boys, girls' greater emphasis on developing social support networks and enduring relationships leaves them more vulnerable to depressive symptoms as a result of interpersonal conflict throughout adolescence (Triosi, 2001). This prediction is supported by empirical evidence. Less supportive and cohesive relationships are more strongly linked to higher depression in adolescent girls than boys (Avison & McAlpine, 1992; Constantine, 2006; Windle, 1992) and some research finds family conflict predicts higher depressive symptoms over a 7 year time frame in adolescent girls but not in boys (Mazza et al., 2009). Therefore, it appears that the prospective association between G1-G2 family conflict and the G2 depressive pathway may be stronger for G2 adolescent girls than boys. As a result, I expect the association between G1-G2 family conflict and G2 depressive symptoms in adolescence to be moderated by gender, such that, among adolescent girls and boys with the same level of conflict, adolescent girls experience greater depressive symptoms.

Second, G2 women may be at greater risk for traversing the depressive pathway to intergenerational family conflict than men because in adulthood, women are more likely to adopt the central roles of caregiving and parenting within their family environment (Craig & Mullan, 2011; Powell & Greenhaus, 2010) and consequently define many family interaction patterns

(Cowan & Cowan, 2012; Rothenberg et al., 2016). Moreover, when shaping interactions in their family of destination, women are likely to emulate patterns of interaction they learned from their own families of origin (Thronberry, Freeman-Gallant, Lizotte, Krohn, & Smith, 2003).

Additionally, other researchers have suggested that women experiencing psychopathology may typically be more likely to exhibit such psychopathology in the family context because that is where, on average, women dedicate more time than men, whereas men with similar psychopathology may most often exhibit it in a work context, (e.g., Craig & Mullan, 2011; Elder, Caspi, & Downey, 1986; Thornberry et al., 2003; Hammen et al., 2014). Therefore, the depressive pathway may be more likely to lead to continuity in family conflict across generations in G2 women, as opposed to men, because G2 women are likely to have more opportunities for interactions by which their depression may cultivate family conflict. Thus, I expect G2 gender to moderate the association between G2 depressive symptoms in adulthood and the development of G2-G3 family conflict, as I predict this association may be stronger in G2 women, compared to men.

In summary, G2 gender may moderate the likelihood that G2 depressive symptoms mediate the relationship between G1-G2 and G2-G3 family conflict for two reasons. First, G2 women are more likely to experience depressive symptoms as a result of family conflict in the G1-G2 home. Second, G2 women with depressive symptoms are more likely to manifest such symptoms in the G2-G3 family environment because of the more central role women play in interacting in and shaping that environment.

The Role of Parenting Partners

Few studies have investigated how the behavior of G2 parenting partners may also underlie continuities in high conflict family environments, though several researchers have called

for future investigations to integrate parenting partner behavior into intergenerational investigations (Conger et al., 2012; Rothenberg et al., 2016). If G2s who are high in depressive symptoms select G2 partners who are also high in depressive symptoms, then both partners are more likely to engage in the withdrawing, uninvolved interaction styles associated with such behaviors, resulting in even greater G2-G3 family conflict. As a result, the same patterns of family interaction that depressed partners experienced across childhood are more likely to be perpetuated in their G2-G3 family. Supporting this hypothesis, multiple studies indicate that individuals with depressive symptoms are more likely to partner with other individuals who are also experiencing internalizing problems (e.g., Desai, Schimmack, Jidkova, & Bracke, 2012; Mathews & Resus, 2001). One meta-analysis of 17 studies found that if an individual has an affective disorder, they are two times more likely to partner with another individual who also has an affective disorder (Mathews & Reus, 2001). Another investigation that sampled more than 900 married couples annually over a 7-year period found moderate to high spousal similarity in levels of depression at the beginning of the marriage (Desai et al., 2012). Interestingly, these researchers also found that increases in one spouses' depression over time predicted increases in the other spouse's depression over time (Desai et al., 2012).

Additionally, though not specific to depressive symptoms, researchers have found that if an individual with high levels of psychopathology finds a partner with similarly high levels of psychopathology, then the maladaptive interactions between the two will likely be volatile and damaging, with negative effects for the family environment (Humbad, Donellan, Iacono, & Burt, 2010). My own work examining G2 and G2 partner similarity in externalizing behavior found evidence that families where two parents have high externalizing behavior have the highest levels of family conflict (Rothenberg et al., 2016), presenting further evidence that when two

partners both experience psychopathology, higher family conflict could result. Therefore, to the extent that G2 partners show elevated depressive symptoms, I posit that G2s' own depressive symptoms are expected to predict greater conflict in the family environment. Due to assortative mating for depressive symptoms, this may be a common occurrence for G2s with high depressive symptoms.

The Current Study

The current study prospectively examines intergenerational continuities in family conflict using multiple reporters of the family environment, incorporating repeated assessments of G2 depressive behaviors spanning adolescence to adulthood and taking into account potential moderators of this association. Specifically, I use a structural equation modeling approach to test four hypotheses using a multigenerational longitudinal study assessing families across a twenty year period (see Figure 1). My first hypothesis is that a G2 depressive pathway measured across ontogeny will mediate intergenerational continuities in family conflict (i.e., paths labeled 1 in Figure 1). My second hypothesis is that G2 depressive symptoms in adolescence and young adulthood will be especially salient mediators of intergenerational continuity in family conflict, as operationalized by finding specific indirect effects of G1-G2 family conflict on G2-G3 family conflict through these time periods (i.e., paths labeled 2 in Figure 1). My third hypothesis is that G2 gender will moderate the entire depressive pathway, such that the pathway will be significant for G2 women, but not for G2 men (i.e., path labeled 3 in Figure 1). My fourth and final hypothesis is that a stronger association between G2 depressive symptoms and high conflict G2-G3 family environments will occur for families where G2 partner's have greater depressive symptoms (i.e., path labeled 4 in Figure 1).

In investigating all study hypotheses, I control for other variables that could also be related to family conflict and /or G2 depression, including G2 age, G2 ethnicity, G2 SES, G1 Antisocial Personality Disorder, G1 alcoholism diagnosis and G1 affective disorder diagnosis (including both Major Depressive Disorder and Dysthymia). It is important to pay particular attention to the effects of G1 alcoholism diagnosis, as the sample used in the current study is a sample of G2 children of alcoholic parents (COAs) and matched controls (Chassin, Rogosch, & Barrera, 1991). This high-risk data set is advantageous for testing the current hypotheses given that patterns of family conflict and high depressive symptoms in G2s and G2-partners may be more prevalent given the comorbidity of alcoholism and depression.

However, caution must also be taken in examining intergenerational family conflict in the current sample. It may be that such conflict is qualitatively different in COA families versus those in matched controls (i.e., perhaps such families experience more physical aggression and less verbal aggression, or vice-a-versa). It also may be that the magnitude of the depressive pathway to intergenerational continuity in family conflict exhibits differences in COA families and matched controls. Therefore, to address these concerns, sensitivity analysis will be conducted to determine if family conflict has the same meaning across COA and non-COA families and to determine if the magnitude of the depressive pathway differs across COA and non-COA families. The present study will further the current science on intergenerational family conflict by identifying one etiological pathway through which such conflict is transmitted and by identifying points of risk or resilience along the depressive pathway that could guide future intervention development.

METHOD

Data from the Adolescent and Family Development Project (AFDP; Chassin, Pitts, DeLucia, & Todd, 1999; Chassin, Rogosch, & Barrera, 1991) was used for this study. AFDP is an ongoing longitudinal study of children of alcoholic parents and matched controls assessed from adolescence into adulthood. AFDP uses a multi-generational design involving assessments of parents (G1s), target adolescents who were followed over time (G2s) and the children of these targets (G3s). AFDP presently consists of 6 waves of data collected annually for waves 1 through 3 (where data were collected on G1s and G2s) and then at 5 year-intervals through wave 6 (where data were collected on G2s, G2 partners and eventually G3s).

Participants

At wave 1, the AFDP sample consisted of 246 adolescents with at least one alcoholic parent and 208 matched adolescents with no biological or custodial alcoholic parent (Chassin et al., 1999) for a total of 454 G2 adolescents and their parents in G1-G2 families. COA families were recruited using court arrest records for driving under the influence, health maintenance organization wellness questionnaires and community telephone screenings (see Chassin et. al, 1999; Chassin et al., 1991). To be included in the current study, COA families had to have parents who reported being either Hispanic or non-Hispanic Caucasian, had to be Arizona residents, had to have a child aged 10.5-15.5 years at wave 1, had to be English-speaking and had to have parents and children with no cognitive limitations that would preclude interview. Further, direct interview data had to confirm that at least one parent met *Diagnostic and*

Statistical Manual for Mental Disorders, third edition (DSM-III) criteria for alcohol abuse or dependence.

When a COA family was identified, reverse directories were used to locate families living in the same neighborhood and matched controls were recruited from this match. Controls were screened to match COA participants in ethnicity, family structure, socioeconomic status, and target child's age and gender. Direct parent interview data were used to confirm that neither biological nor custodial parents of controls met DSM-III criteria. Attrition biases are minimal as 409 of the original 454 families were retained at wave 6 (90.1% of original sample).

To be included in the current analysis, G2s needed to have at least one child by wave 6 ($N = 273$ of 409 interviewed at wave 6) and complete data on the family conflict measure at wave 6 ($N = 246$ of 273 G2s with children, with 27 having missing data because they contacted their child less than once a week). The decision was made to drop families without children from study analysis in order to investigate conflict in families at similar stages of development, in line with theory and existing work (e.g., Conger et al., 2009; Kovan et al., 2009). However, families without children did not differ from included families on levels of family conflict reported by G1 mothers ($t(323) = -0.60, p = 0.55$), G1 fathers ($t(392) = -1.07, p = 0.29$), or G2 targets ($t(408) = 1.61, p = 0.11$). Missing data among the remaining 246 G2-G3 families was addressed using full information maximum likelihood procedures (see missing data) such that all 246 G2-G3 families were retained in study analyses. G2-G3 families ranged in size from 1 to 4 children ($M = 1.75$ children), though only the oldest G3 child was included in the present analyses. Indicators of G2-G3 family environment were based on G2 reports and on available G2 partner and G3 reporters who were present at the time of the G2 interview. Demographic characteristics of the sample can be found in Table 1.

Procedure

At each wave, data were primarily collected via in-person computer-assisted interviews (Chassin et al., 1999). Family members were typically interviewed simultaneously and in separate rooms to avoid contamination and to increase privacy. In waves 1-3 of data collection, at least one biological and custodial G1 caregiver and one G2 adolescent between the ages of 10 to 15 years old completed interviews. In wave 6 of data collection, only G2 targets were required to complete interviews. However, G2 partners and any G3s who were 7 years old or older were also invited to complete interviews if they were available at the time the G2 was interviewed. Interviews typically lasted from 1 to 3 hours and participants were paid up to \$70 per wave.

Measures

Control variables. Potential confounds were controlled for in study analyses by including wave 2 covariates for G1 antisocial behavior, G1 affective disorder and alcoholism diagnoses and G2 age, as well as wave 6 covariates for G2 ethnicity, G2 gender and G2 educational attainment. G1 mother and G1 father antisocial behavior, affective disorder and alcoholism were measured via self-reported lifetime *DSM-III* diagnoses of antisocial personality disorder, major depressive disorder, dysthymia and alcohol abuse or dependence. These diagnoses were obtained using a computerized version of the DIS interview (Version 3; Robins, Helzer, Croughan & Ratcliff, 1981; Robins, Helzer, Ratcliff, & Seyfried, 1982). Although all reports of antisocial personality disorder and affective disorder were based solely on self-report by mother or father and missing otherwise, alcoholism diagnoses were based on self-report as well as spousal report for non-participating parents using Research Diagnostic Criteria (FH-RDC; Andreasen, Endicott, & Spitzer, 1977). In current analyses, family-level diagnoses were dichotomized as either present (at least one G1 parent meet lifetime criteria) or absent (both

participating G1 parents did not meet lifetime criteria). At wave 6, G2s and their partners reported their gender, ethnicity and highest education level obtained, with education assessed using an 11-point scale ranging from 1=8th grade or less to 11=completed graduate/professional school. Socioeconomic status was indexed as the highest education level obtained by either parent in the G2-G3 family. Other studies using the AFDP data set have accounted for socioeconomic status by controlling for education level in similar ways (Chassin, Flora, & King, 2004; Hussong, Huang, Serrano, Curran, & Chassin, 2012).

G2 and G2-partner depressive symptoms. G2 depressive symptoms were measured at waves 3 ($M_{G2Age} = 15.33$ years, $SD = 1.42$ years, Range: 12.55-18.01 years), 4 ($M_{G2Age} = 20.54$ years, $SD = 1.33$ years, Range: 17.48 - 23.61 years) and 5 ($M_{G2Age} = 25.96$ years, $SD = 1.61$ years, Range: 22.48 - 29.87 years) using the same 5 self-report items (e.g., "felt lonely", "cried a lot", "unhappy/sad/depressed") from the Anxious/Depressed and Withdrawn/Depressed subscales of the Achenbach Childhood Behavior Checklist (CBCL; Achenbach & Edelbrock, 1981). G2 partners completed self-reports on these same items at wave 6. Participants rated how often an item was true for them within the past 3 months on a scale ranging from 1= almost always to 5 = almost never. A mean of items served as the indicator of the depressive symptoms endorsed by subjects within each wave ($\alpha = .76 - .77$ across waves for G2s and $\alpha = .78$ for G2 partners).

Family conflict. Family conflict was measured using the 5-item family conflict subscale derived from Bloom's Family Processes Scale (Bloom, 1985). Participants rated the extent to which they agreed that a statement reflected their family life in the past 3 months using a five-point response scale ranging from 1= strongly agree to 5 = strongly disagree. Items included "We fought a lot in our family", "Family members sometimes hit each other", "Family members

rarely criticized each other", "Family members hardly ever lost their tempers" and "Family members sometimes got so angry they threw things". Bloom found the family conflict subscale to have adequate internal reliability in previous studies ($\alpha=.76$ to $\alpha=.85$) and to demonstrate discriminate validity in distinguishing levels of family conflict before and after marital disruptions (Bloom, 1985). In the present study, G1 mothers, G1 fathers and early adolescent G2s (aged 12-16) completed the family conflict scale at wave 2 in reference to G1-G2 families. In wave 6, G2s, G2 partners and all participating G3 children (aged 7-17) completed the family conflict subscale in reference to G2-G3 families. Items were reverse scored so that higher scores indicated higher family conflict. In the present study, internal reliability estimates were as follows: wave 2 G1 father-reports ($\alpha=.69$), G1 mother-reports ($\alpha=.65$) and G2 reports ($\alpha=.73$); and wave 6 G2 reports ($\alpha=.70$), G2 partner reports ($\alpha=.67$) and G3 reports ($\alpha=.65$). The somewhat low reliability of some family conflict reporters was addressed by combining reports and estimating both G1-G2 family conflict and G2-G3 family conflict as latent variables that by design are free of measurement error.

As reported in Rothenberg et al. (2016), these G1-G2 family conflict and G2-G3 family conflict latent variables were created in a several step process. Initially, parceling procedures were used to integrate G1 mother, G1 father and G2 target reports of G1-G2 family conflict as well as G2 target, G2 partner and all available G3 child reports of G2-G3 family conflict. This technique was used to create latent factor indicators in other intergenerational longitudinal studies (e.g., Lohman, Neppl, Senia, & Schofield, 2013) and is appropriate for this investigation because analyses are focused on associations between latent constructs and because in each generation, every item loads onto the same single factor (Williams & O'Boyle, 2008). For this step, family members' responses to the family conflict scale were averaged at the item level for

both G1-G2 and G2-G3 families (i.e., G1 mother, G1 father and G2 adolescent responses to item 1 of the family conflict scale were averaged to create a single indicator of G1-G2 family conflict for item 1). Item-level correlations between reporters ranged from 0.17-0.41 in the G1-G2 family and from 0.07-.40 in the G2-G3 family. Although these correlations are low, this method provided a data reduction approach collapsing across the diverse perspectives offered by these reporters while equally weighting the perspective of each reporter. An alternative approach drawing on the multi-trait (i.e., multi-item), multi-method (i.e., multi-reporter) literature where latent variables representing mother, father and child reports of family conflict were estimated from the family conflict item indicators and then a second-order factor representing total family conflict was estimated from mother, father and child family conflict latent variables in both G1-G2 and G2-G3 families was examined. However, due to its complexity, this alternative model could not be estimated and was deemed analytically too demanding within the current data structure.

Then, maximum likelihood confirmatory factor analyses using Mplus Version 5.2 (Muthen & Muthen, 2007) were conducted to estimate latent variables representing underlying conflict in the family environment following Bollen and Bauldy (2011). Separate analyses for G1-G2 and G2-G3 families used the five family-averaged item indicators of conflict as depicted in Figure 2. Skewness and kurtosis estimates for all indicators fell in acceptable ranges (skew < 2.0, kurtosis < 3.0), suggesting no violation of the assumption of normally distributed indicators. Additionally, no problematic heteroscedasticity of residuals in indicators was observed. Evaluation of model fit was based upon recommended fit index cut-off values that indicate excellent model fit (CFI/TLI cut-off values > 0.95, RMSEA cut-off value < 0.05, SRMR cut-off value <.08; Schreiber, Stage, King, Nora, & Barlow, 2006).

Initial model fit for G1-G2 family conflict was not acceptable ($\chi^2(5) = 26.84$, $p < .01$, CFI = 0.93, TLI = 0.86, RMSEA = 0.13, SRMR = 0.05). Two correlated errors were added to the model based on modification indices (item 2 and 4 that both involved acts of physical aggression and items 3 and 5 that were both reverse scored), resulting in significantly improved model fit ($\chi^2(2) = 22.73$, $p < .05$). Fit indices showed that the revised model fit the data well ($\chi^2(3) = 4.11$, $p = 0.25$, CFI = 0.99, TLI = 0.99, RMSEA = 0.04, SRMR = 0.02), indicating that the model was appropriate to estimate a latent variable for G1-G2 family conflict.

Similarly, initial model fit for G2-G3 family conflict was not acceptable ($\chi^2(5) = 38.78$, $p < .01$, CFI = 0.89, TLI = 0.77, RMSEA = 0.17, SRMR = 0.06). The same correlated errors were added to the model as for G1-G2 family conflict, once again resulting in significantly improved model fit ($\chi^2(2) = 36.53$, $p < .05$). Fit indices showed that the model fit the data well ($\chi^2(3) = 2.18$, $p = 0.53$, CFI = 1.00, TLI = 1.00, RMSEA = 0.00, SRMR = 0.01), indicating that the model was appropriate to estimate a latent variable for G2-G3 family conflict. Final measurement models estimating both the G1-G2 family conflict and G2-G3 family conflict latent variables are depicted in Figure 2.

Missing Data

The analysis sample consists of 246 target G2s, however there was modest to moderate missingness on key variables. Specifically, some G1-G2 families were missing mother reports (11 families) and father reports (56 families) of family conflict and some G2-G3 families were missing G2 partner reports (144 families) and G3 child reports (123 families) of family conflict. Missingness among G2 partner reports was due to the fact that some G2 partners declined to participate in the study or were unavailable at the time of the G2 target interview. Missingness among G3 child reports was due to the fact that G3 children could not participate in the study

unless they were over the age of 7 or also were unavailable at the time of the interview. Additionally, the number of G2s who failed to report on their depressive symptoms in any particular wave ranged from 3 to 21. However, every G2 reported on depressive symptoms on at least one of waves 3, 4 and 5. Notably, G2-G3 families with versus without missing data did not significantly differ on G2 depressive behavior at wave 3 ($t(241) = -1.40, p = 0.16$), wave 4 ($t(241) = -0.31, p = 0.76$), or wave 5 ($t(228) = 0.40, p = 0.68$). Moreover, G2-G3 families with versus without missing data did not significantly differ on any of the 5 items measuring G1-G2 family conflict ($t(242)$ range from -1.81 to 0.80, $p > .05$), or 4 of the 5 items measuring G2-G3 family conflict ($t(242)$ range from -1.81 to 0.80, $p > .05$). G2-G3 families with ($M = 2.33, SD = 0.88$) versus without ($M = 2.01, SD = 0.80$) missing data did score significantly higher on a single G2-G3 family conflict item, "We fought a lot in our family". However, mean scores for both of these groups fell into the "disagree" or "neutral" range of response options and no other conflict items differed between groups. Consequently, this statistically significant difference did not seem to be a truly meaningful indicator that the entire construct of G2-G3 family conflict differs significantly in families with versus without missing data. Moreover, this difference did not appear to be a result of any variable not already captured and controlled for in the data set. Therefore, because data appeared to be missing at random, full information-maximum likelihood procedures were used in Mplus to account for missing data in subsequent analyses following Kline (2005).

Additionally, it could be argued that G2s who come from the highest conflict G1-G2 homes would be less likely to have children and therefore would be excluded from the study, perhaps resulting in a restriction of range on the family conflict variable. Therefore, I examined whether there were any systematic differences in G1-G2 family conflict in those 246 G2s

included in the study who had G2-G3 families and the 208 G2s excluded from the study. There was no significant differences between these two groups on any of the 5 family conflict items as reported on by G1 mothers, G1 fathers, or G2 adolescents. Consequently, it does not appear that the present sample is missing a group of G2s with high G1-G2 family conflict who have not had children.

RESULTS

Covariate Baseline Model

Once family conflict latent variables were estimated, path modeling within a structural equation model (SEM) framework using a Full Information Maximum Likelihood (FIML) estimator was utilized to examine the unique effects of study covariates (i.e., G2 age (at wave 2), G2 ethnicity, G2 educational attainment at wave 6 and G1 antisocial behavior, affective disorder and alcoholism diagnoses) on G2-G3 family conflict. Results revealed that only G2 age ($\beta = 0.17, p = .04$) and G2 ethnicity ($\beta = 0.13, p = .08$) were moderately significant predictors of G2-G3 family conflict, such that G2s who self-identified as a race other than White and older G2s experienced higher G2-G3 family conflict. Additionally, fit indices revealed that this model fit the data well ($\chi^2(41) = 60.48, p = 0.03, CFI = 0.97, TLI = 0.95, RMSEA = 0.04, SRMR = 0.04$). In the interest of parsimony, all paths from covariates to G2-G3 family conflict that were not significant at $p < .10$ were cut from further analyses, so only G2 age and G2 ethnicity were carried forward as covariates in hypothesis testing.

Testing a Depressive Pathway

Next, hypothesis 1 (that a G2 depressive pathway will mediate intergenerational continuities in family conflict) and hypothesis 2 (that G2 depressive symptoms in adolescence and young adulthood will be developmentally specific mediators of intergenerational family conflict) were tested. To test these hypotheses, a series of path models were iteratively fit within a structural equation model (SEM) framework using a Full Information Maximum Likelihood (FIML) estimator. In the initial model (depicted by the paths labeled 1 in Figure 1), G2-G3

family conflict was regressed on G1-G2 family conflict and G2 depressive symptoms at wave 5, as well as G2 age and G2 ethnicity. Additionally, autoregressive paths were added to the model between the three indicators of G2 depression, such that G2 wave 5 depressive symptoms were regressed on G2 wave 4 depressive symptoms, which were regressed on G2 wave 3 depressive symptoms. Finally, G2 wave 3 depressive symptoms were regressed on G1-G2 family conflict. Omnibus tests of model fit indicated that this initial model did not fit the data well ($\chi^2(76) = 141.40, p < .01, CFI = 0.92, TLI = 0.89, RMSEA = 0.06, SRMR = 0.07$). Because hypothesis 1 may reflect poor model fit due to model misspecification given pathways omitted from hypothesis 1 but posited for hypothesis 2 and because three of the largest MIs were associated with these pathways, I next estimated the proposed model for hypothesis 2. Specifically, three pathways were added to estimate the hypothesis 2 model, including a pathway regressing G2 wave 4 depression on G1-G2 family conflict, a pathway regressing G2-G3 family conflict on G2 wave 3 depression and a pathway regressing G2-G3 family conflict on G2 wave 4 depression. This hypothesis 2 model is depicted in Figure 1 by all the paths labeled 1 and 2.

The revised model fit the data well ($\chi^2(73) = 114.35, p < .01, CFI = 0.95, TLI = 0.93, RMSEA = 0.05, SRMR = 0.05$). Additionally, a χ^2 difference test revealed that this revised model fit the data significantly better than the initial model, ($\chi^2(3) = 27.05, p < .01$), so this model was retained for further analysis. The model explained a significant amount of variance in G2-G3 family conflict ($R^2 = 0.32, p < .01$).

Results (depicted in Figure 3) revealed that the total effect of G1-G2 family conflict on G2-G3 family conflict was significant ($\beta = 0.29, p < .01$). The total indirect effects of G1-G2 family conflict on G2-G3 family conflict were also significant ($\beta = 0.10, p < .01$), indicating significant mediation. Decomposition of specific indirect effects showed two significant indirect

pathways mediated the association between G1-G2 family conflict and G2-G3 family conflict. First, the indirect effect of G1-G2 family conflict through G2 depressive symptoms at wave 3 (when G2s were in adolescence), and G2 depressive symptoms at wave 4 (when G2s were in young adulthood) to G2-G3 family conflict was significant ($\beta = 0.02, p = .05$). In other words, this mediating pathway indicated that higher G1-G2 family conflict was associated with higher G2 depressive symptoms in adolescence which were associated with higher G2 depressive symptoms in young adulthood, which were associated with higher G2-G3 family conflict. A second indirect effect of G1-G2 family conflict on G2-G3 family conflict occurred through the mediating effect of G2 depressive symptoms at wave 4 (in young adulthood; $\beta = 0.06, p = .03$). This mediating pathway indicated that higher G1-G2 family conflict was associated with higher G2 depressive symptoms in young adulthood that were associated with higher G2-G3 family conflict.

It is important to note that these two indirect effects are independent and unique effects. Consequently, these results seem to support both Hypothesis 1 and Hypothesis 2. The first indirect effect indicates that there is a depressive pathway that includes stability in depressive symptoms from adolescence to young adulthood that mediates intergenerational continuity in family conflict from G1-G2 to G2-G3 families. The second indirect effect indicates that, even after controlling for the underlying depressive pathway, G2 depressive symptoms in young adulthood may represent a developmentally-specific mediator of intergenerational continuity in family conflict from G1-G2 to G2-G3 families. Notably, the direct effect of G1-G2 family conflict on G2-G3 family conflict was also significant ($\beta = 0.20, p = .03$), indicating that G2 depressive symptoms partially, but not fully, mediated the association between G1-G2 and G2-G3 family conflict.

Importantly, G2 wave 5 depressive symptoms were not a significant predictor of G2-G3 family conflict ($\beta = 0.02, p = .44$) and there was no significant indirect effect that included G2 wave 5 depressive symptoms. Additionally, a sensitivity analysis was performed to determine if adding a direct path from G1-G2 family conflict to G2 wave 5 depressive symptoms (and thus allowing the specific indirect effect of G2 wave 5 depression to be measured) changed model results and may represent model misspecification. This additional pathway did not substantively change model results. Therefore, it does not appear that G2 depressive symptoms at wave 5 play a significant mediating role in the intergenerational transmission of family conflict in this sample above and beyond depressive symptoms at wave 3 or wave 4.

Gender as a Moderator of the Depressive Pathway

Next, a multiple group analyses within a structural equation modeling framework was used to test hypothesis 3 (i.e. that G2 gender would moderate the entire depressive pathway such that the pathway will be significant for G2 women but not for G2 men). This analysis proceeded in several steps. First, I examined whether the latent G1-G2 and G2-G3 family conflict variables had the same meaning and metric (i.e., were invariant) across genders by following analytic strategies utilized in prior work (Rothenberg et al., 2016). Model invariance of each measurement model was tested within the scope of the larger SEM. Results indicated that when factor loadings and intercept were constrained to be equal across gender, there was no significant decrement in model fit ($\chi^2(8) = 8.30, p > .05$) and that the model fit the data well ($\chi^2(73) = 114.35, p < .01, CFI = 0.95, TLI = 0.93, RMSEA = 0.05, SRMR = 0.05$). Therefore, the G1-G2 and G2-G3 family conflict latent variables demonstrated invariance across G2 men and women indicating that differences in factor variances, covariances and means across G2 gender could be compared in the full model.

Second, to compare structural differences in the model between G2 women and G2 men, all of the pathways in the model labeled with the numbers 1 and 2 in Figure 1 were constrained to be equal across gender (but all other pathways were allowed to freely vary across gender). A χ^2 difference test revealed that the freely estimated model fit the data significantly better than the constrained model ($\chi^2(8) = 29.11, p < .01$). This result held, as expected, even when covariate pathways were also constrained to be equal.

The multiple group model that was freely estimated within gender fit the data well ($\chi^2(162) = 213.13, p < .01$, CFI = 0.94, TLI = 0.92, RMSEA = 0.05, SRMR = 0.07). In women, results revealed a significant total effect ($\beta = 0.44, p < .01$), direct effect ($\beta = 0.28, p = .02$) and indirect effect ($\beta = 0.16, p < .01$) of G1-G2 family conflict on G2-G3 family conflict and explained a significant amount of variance in G2-G3 family conflict scores ($R^2 = 0.37, p < .01$; see Figure 4). However, neither the total effect ($\beta = 0.01, p = .97$), direct effect ($\beta = 0.01, p = .95$), nor indirect effect ($\beta = 0.00, p = .86$) of G1-G2 family conflict on G2-G3 family conflict were significant in men and the model did not explain a significant amount of variance in G2 men's G2-G3 family conflict scores ($R^2 = 0.10, p = .15$).

Decomposition of specific indirect effects revealed that the mediating effects of depression differed from those seen in the aforementioned hypothesis 2 model that pooled both genders together. To begin with, the mediating pathway from G2 depressive symptoms at wave 3 (when G2s were in adolescence) to G2 depressive symptoms at wave 4 (when G2s were in young adulthood) to G2-G3 family conflict was moderately significant for both G2 women ($\beta = 0.01, p = .09$) and G2 men ($\beta = 0.01, p = .09$). This moderately significant mediating pathway indicated that higher G1-G2 family conflict was associated with higher G2 depressive symptoms in adolescence which were associated with higher G2 depressive symptoms in young adulthood,

which were associated with higher G2-G3 family conflict. Notably, this mediating pathway was significant at the $p < .05$ level in the hypothesis 2 model that pooled genders together, but was but only moderately significant at the $p < .10$ in the current model where effects for men and women were calculated separately.

Additionally, as in the pooled model, the developmentally specific mediating effects of G2 depressive symptoms in young adulthood were evaluated. In the current model, the indirect effect of G1-G2 family conflict on G2-G3 family conflict through G2 women's ($\beta = 0.09, p = .04$), but not men's ($\beta = -0.01, p = .75$) depressive symptoms at wave 4 (when G2 women were in young adulthood) was found significant. This mediating pathway indicates that higher G1-G2 family conflict is associated with higher G2 women's depressive symptoms in young adulthood which are associated with higher G2-G3 family conflict. This finding also further justifies the need to examine G2 women and men in separate models. It reveals that the significant mediating effect through G2 young adult depressive symptoms uncovered in the model that pooled genders together is, in fact, found only in G2 women.

Finally, the model separating G2s by gender also revealed an additional gender-specific mediating effect that was not found in the pooled model. Specifically, the indirect effect of G1-G2 family conflict on G2-G3 family conflict through G2 women's ($\beta = 0.07, p = .03$), but not men's ($\beta = -0.01, p = .86$) depressive symptoms at wave 3 (when G2 women were in adolescence) was significant. This mediating pathway indicates that higher G1-G2 family conflict is associated with higher G2 women's depressive symptoms in adolescence which are associated with higher G2-G3 family conflict.

Taken together, these multiple group analyses results support hypothesis 3. Even after controlling for an underlying depressive pathway, G2 women's depressive symptoms in

adolescence and young adulthood were each found to uniquely mediate paths from G1-G2 family conflict to G2-G3 family conflict. Therefore, G2 women's depressive symptoms experienced during these time periods may represent developmentally specific mediators of intergenerational continuity in family conflict from G1-G2 to G2-G3 families.

Partner Depressive Symptoms as a Moderator

Interaction terms were created and added to the unconstrained multiple groups model to test whether G2 partner depressive symptoms (measured at wave 6) moderated the relation between G2 depressive symptoms and G2-G3 high conflict family environment. Three separate multiple group models were run, where interaction terms between G2 partner depressive symptoms and G2 depressive symptoms at wave 3, wave 4 and wave 5 were investigated. In all three models, the interactions terms were not significant in either women or men. Moreover, as a sensitivity analysis, interaction terms were also created in the model that combined both men and women. Again, three separate models were run and interaction terms between G2 partner depressive symptoms and G2 depressive symptoms at wave 3, wave 4 and wave 5 were all not significant. Therefore, hypothesis 4 was not supported.

Sensitivity Analyses

To further understand the robustness of findings across different types of families, sensitivity analyses were also performed to compare families where G2s were children of alcoholics (COAs) versus non-COAs. Analyses proceeded in an analogous manner as with tests of gender differences. The final model indicated that the G1-G2 and G2-G3 family conflict latent variables demonstrated invariance across COA and non-COA families and that a freely estimated model was not a significantly better fit to the data as compared to the model where parameters were constrained to be equal across COA status ($\chi^2(10) = 12.37, p > .10$). This model fit the

data well ($\chi^2(66) = 66.27, p = .47, CFI = .99, TLI = 0.99, RMSEA = 0.01, SRMR = 0.07$).

Therefore, the family conflict latent variables estimated in the present study do not differ in meaning or metric as a result of COA status and the direct and indirect effects of G1-G2 family conflict on G2-G3 family conflict do not significantly differ in magnitude depending on COA status. Taken together, these findings suggest that findings in the present study are not likely to be accounted for by COA status.

DISCUSSION

In the current study, I examined whether G2 depressive symptoms measured at multiple time points across development explained continuity in family conflict from one generation to the next. Three key findings emerged. First, adolescence and young adulthood were periods of vulnerability in which G2 women's depressive symptoms mediated the intergenerational continuity in family conflict. Second, in both men and women, higher depressive symptoms that persisted from adolescence into young adulthood mediated the association between G1-G2 and G2-G3 family conflict. Third, G2 partners' depressive symptoms did not moderate the relation between G2 depressive symptoms and G2-G3 family conflict. Further consideration of how these three findings explain continuity in family conflict from one generation to the next are considered below.

G2 Developmental Sensitivity for Women

Study results confirmed the hypothesis that both adolescence and young adulthood were periods of vulnerability in which G2 women's depressive symptoms mediated intergenerational continuities in family conflict. Adolescence may serve as such a period of vulnerability for G2 women both because family conflict is more likely to give rise to depressive symptoms in G2 girls, compared to boys and because it is more likely that these depressive symptoms inform the development of patterns of family interaction over time in G2 women, compared to men.

First, family conflict is more likely to give rise to depressive symptoms in G2 girls than boys. In high conflict families, normative adolescent negotiations of autonomy and separation from one's family of origin can be especially intense and argumentative, leading to increases in

adolescent perceptions of family conflict (Friedman et al., 2009; Hock et al., 2001; Wray-Lake et al., 2010). Adolescent girls may be particularly vulnerable to experiencing depressive symptoms as a result of high family conflict around issues of adolescent autonomy seeking, because from an early age girls are socialized to invest more in familial relationships (Constantine, 2006; Noller, 1994). As a result, adolescent girls often have more difficulty gaining independence from their families than boys (Constantine et al., 2005; Huston & Alvarez, 1990; Sheeber et al., 1997) and are more likely to experience depressive symptoms and associated social withdrawal while doing so (Hops, 1995; Sheeber et al., 1997). Therefore, the association between family of origin conflict and adolescent depressive symptoms may be stronger in women than men.

Second, it is more likely that G2 girls', as opposed to boys' adolescent depressive symptoms inform patterns of family interaction over time. Multiple investigations have found adolescence to be a time period in which patterns of social interaction that influence adult functioning are likely to be established and internalized (Jaffee, Belsky, Harrington, Caspi & Moffitt, 2006; Thornberry et al., 2003). This may be especially true of adolescent girls, where developmental goals such as the establishment of social support networks and relationships that endure across time are more greatly emphasized than in boys (Triosi, 2001). Therefore, G1-G2 family conflict may make it more likely that adolescent girls establish and internalize social withdrawal and associated depressive symptoms that eventually lead to conflict in their G2-G3 families. In sum, results suggest that adolescence represents a period of vulnerability in which G2 women's depressive symptoms mediate the association between G1-G2 and G2-G3 family conflict. This may be due to the increased likelihood that conflict-withdrawal patterns of interaction and associated depressive symptoms emerge in adolescence and become less malleable after adolescence, in G2 women.

Current results also indicate that young adulthood represents an additional period of vulnerability in which G2 women's depressive symptoms mediated intergenerational continuities in family conflict. Young adulthood may represent a period of vulnerability because young adulthood is characterized by major life transitions (i.e., marriage or the birth of a child) that lead one's family of origin to remerge as heavily involved in one's life (Cowan & Cowan, 2012). For instance, it is likely that a woman will experience extensive recontact with her family of origin around the period of pregnancy and childbirth (Cowan & Cowan, 2012). Additionally, many investigations also reveal that the transition to parenthood leads to husbands' increased focus on work outside the home (Cowan & Cowan, 2012; Dew & Wilcox, 2011), to less quality time spent between women and their partners (Castellano, Velotti, Crowell, & Zavattini, 2014; Dew & Wilcox, 2011) and to a reduction in communication between women and their partners (Castellano et al., 2014; Cowan & Cowan, 2012). Therefore, young adult women from high conflict families of origin likely interact extensively with their families of origin during the same time period (i.e., the transition to parenthood) when they are least likely to receive social support from their parenting partners to cope with experiencing such high conflict. This recontact with one's family of origin and lack of spousal support makes it likely that depressive symptoms are maintained or increase in these young adult women. Furthermore, because women spend more time than men, on average, providing childcare and parenting in their family of destination (Cowan & Cowan, 2012; Craig & Mullan, 2011; Dew & Wilcox, 2011), their depressive symptoms are more likely to manifest in the family context (Elder et al., 1986; Thornberry et al., 2003). These depressive symptoms subsequently lead to high family conflict in women's family of destination (Castellano et al., 2014; Cowan & Cowan, 2012). These high conflict patterns of interaction, once established, show stability over time (Cowan & Cowan, 2012). In sum, in

young adulthood, women from high conflict family environment are more likely to experience extensive recontact with their family of origin as they transition to parenthood, less likely to receive adequate social support from their parenting partners and more likely to have consequent depressive symptoms manifest in family interactions than men.

Consideration of the timing of the transition to parenthood could explain some study findings. Notably, in the present study, G2 women's depressive symptoms at wave 4 (when they were on average 20.5 years old), but not G2 women's depressive symptoms at wave 5 (when they were on average 26 years old), were uniquely predictive of subsequent G2-G3 family conflict. However, in the current sample, over 99% of G2s had children between waves 3 and 4 and the average age of G3 children at wave 4 was 2.14 years, as opposed to 7.14 years at wave 5. Therefore, it appears that most G2 women experienced the transition to parenthood (and accompanying recontact with their family of origin and resultant depressive symptoms) at wave 4, as opposed to wave 5. Therefore, non-significant effects of G2 depressive symptoms on G2-G3 family conflict at wave 5 could be explained because the effects of G2 depressive symptoms on family conflict had already been wrought during the transition to parenthood at wave 4.

Taken together, current results indicate that women from high conflict families may be at greater risk for traversing the depressive pathway to intergenerational continuity in conflict than men from such families. Current findings indicate that there are multiple risk periods throughout development (i.e. adolescence and young adulthood) wherein women can be launched back on the depressive pathway from high conflict in one's family of origin to high conflict in one's family of destination, whereas no such developmentally-specific periods of vulnerability were found for men. These multiple developmental snares may exist for women, but not men, because even in contemporary society, one's family of origin is expected to teach women, more so than

men, how to shape family interactions and be the primary caregiver in their family of destination (Craig & Mullan, 2011; Powell & Greenhaus, 2010; Triosi, 2001). This socialization into the caregiver role by families of origin begins in early childhood play (Triosi, 2001), is further emphasized during autonomy-seeking and relationship formation in adolescence (Constantine, 2006; Triosi, 2001) and continues during the transition to parenthood in young adulthood (Cowan & Cowan, 2012). Because families of origin prioritize this socialization goal for their daughters across ontogeny, women are more likely to come into frequent contact with their family of origin across multiple developmental stages than men (Cowan & Cowan, 2012). For women from high conflict families of origin, greater exposure to this high conflict family environment increases the likelihood that at any particular developmental period, women experience consequent depressive symptoms that launch them on the depressive pathway.

Considering a Continuous Depressive Pathway

Current results provide support for the hypothesis that G2 women's adolescence and young adulthood do represent periods of unique developmental vulnerability where intergenerational family conflict may be at least partially transmitted through G2 depressive symptoms. However, I also hypothesized that a continuous depressive pathway, comprised of G2 experiences of depressive symptoms from adolescence to adulthood, would mediate the intergenerational transmission of family conflict. Evidence supporting this hypothesis from the current study is equivocal. On the one hand, in the model comprised of both G2 women and men, I found that a depressive pathway through G2 symptoms in adolescence and young adulthood partially mediated the association between G1-G2 and G2-G3 family conflict, even after controlling for the developmentally specific mediating effects of depressive symptoms at each of these time points. Additionally, in the model separating G2s by gender, this pathway was found

to be moderately significant in both men and women. On the other hand, none of the models revealed a significant pathway where G2 depressive symptoms experienced across all three time points (i.e., a pathway through wave 3, 4 and 5 depressive symptoms) mediated intergenerational associations in family conflict. Therefore, in the present sample, a depressive pathway through G2s' adolescence and early twenties (i.e., waves 3 and 4), but not through G2s' later twenties (i.e., wave 5) partially accounts for intergenerational continuity in family conflict.

I suspect that the severity of the depressive symptoms that I measured may account for why, in both men and women, the depressive pathway did not continue into G2s' late twenties (i.e., wave 5). In the current sample, even when G2 depressive symptoms were at their highest (when reported at wave 4), G2 mean depressive symptoms were still only a 1.66 on a 5 point scale (i.e., somewhere between "once in awhile" and "almost never" experienced in the past 3 months). While prior research indicates that clinically significant levels of depression are likely to show continuity across ontogeny and affect family environments in adulthood (Hammen & Rudolph, 2003), subthreshold depressive symptoms are likely to exhibit statistically significant, but much more modest continuity (Hammen & Rudolph, 2003). This pattern of findings is reflected in the current results, as autoregressive paths among G2 depressive symptoms across time were statistically significant but modest in size (ranging from $\beta = .25$ to $.54$ across models). These modest pathways indicates less rank-order stability among individuals in depressive symptoms over time and consequently make it less likely that the same individuals rank the highest in depressive symptoms as more time points are added to the pathway. Thus, whereas enough individuals demonstrate rank-order stability in depressive symptoms through adolescence and young adulthood (i.e., waves 3 and 4) for the depressive pathway to be significant at these time points, by the time G2s reach their mid 20s (i.e., wave 5), stability in

rank-order has decayed to the point that no specific indirect effect is found significant. Ultimately, this modest continuity in depressive symptoms makes it less likely a single continuous pathway from G2 adolescent depressive symptoms to G2 adult depressive symptoms 10 years later mediates intergenerational continuity in family conflict. I expect that if I were to investigate this same phenomenon in a sample that included clinically depressed participants, it would be more likely that I would find a contiguous depressive pathway over ontogeny because more severe depression is more likely to demonstrate continuity and rank-order stability over time (Hammen & Rudolph, 2003).

Yet, it is worth noting that the mediating pathway from G1-G2 family conflict to G2-G3 family conflict through G2 depressive symptoms in adolescence and young adulthood was at least moderately significant across models in the present study. In this sample, childbirth is a common event experienced by G2 young adults. Existing evidence indicates in both men and women who already are experiencing depressive symptoms, childbirth could serve as a risk factor for increased family conflict (Cowan & Cowan, 2012; Hammen & Rudolph, 2003). Indeed, longitudinal evidence indicates that existing depressive symptoms are likely to be exacerbated in both new fathers and mothers in the first several years of parenting (Cowan & Cowan, 2012). Consequently, spouses are less likely to provide social support to one another and less likely to feel as feel connected or close to one another (Cowan & Cowan, 2012) and both partners are more likely to exhibit frustration and conflict as they seek confirmation that the other parenting partner still cares for them (Baucom et al., 2010; Holley et al., 2010). Once these conflictual behaviors emerge in the interparental relationship, they often spill over into the family context (Cummings & Schatz, 2012) and are associated with similar patterns of coercive behavior between parents and children (Dishion & Patterson, 2006). These high conflict patterns

of family interaction are less malleable to change once developed in the few years after childbirth (Cowan & Cowan, 2012). In sum, G2 depressive symptoms that persist from adolescence to young adulthood are likely to lead to increased conflict between parenting partners that spills over into the family environment as these young adult parents are faced with the challenges of the transition to parenthood. This high conflict family environment, once established, is not easily changed over time.

Therefore, the contiguous pathway found in both G2 genders from G1-G2 family conflict through G2 adolescent and young adult depressive symptoms to G2-G3 family conflict and the developmentally specific pathway found in G2 women from G1-G2 family conflict through young adult depression, to G2-G3 family conflict demonstrate equifinality. Specifically, both mediating paths lead to high G2-G3 family conflict resulting as a result of young adult depressive symptoms. Additionally, I posit that the transition to parenthood and associated lack of spousal support and connection is a key factor that facilitates the link between G2 depressive symptoms and G2-G3 family conflict in both pathways. However, I posit that these two paths differ in the timing of G1-G2 family conflict that triggers such young adult depressive symptoms. In the developmentally-specific path I believe such depressive symptoms may emerge *in young adulthood* from recontact with one's family of origin in *young adulthood*, and lead to high family conflict in one's family of destination as they transition to parenthood. Since new mothers are likely to spend greater amounts of time recontacting their family of origin as compared to new fathers (Hyun et al., 2002), the developmentally-specific mediating effect in young adulthood is likely to be gender-specific. In contrast, for those G2s on the contiguous path, I posit that G1-G2 family conflict *in adolescence* influences the emergence of depressive symptoms and associated styles of interaction *in adolescence* that are then internalized and

persist *into young adulthood* to inform spousal conflict that spills over into the larger environment as G2s form their own families. In other words, along the developmentally-specific path, G2 recontact with their family of origin and subsequent depressive symptoms may make intergenerational transmission of family conflict more likely. Along the contiguous path, persistence of G2 adolescent depressive symptoms and subsequent interparental conflict and lack of support may make intergenerational transmission of family conflict more likely.

Considering G2 Partner Behavior as Moderator

Unexpectedly, I found no support that G2 partner depressive symptoms moderated the association between G2 depressive symptoms and G2-G3 family conflict. This result differs from prior work demonstrating that the highest levels G2-G3 family conflict occur when both G2s and their parenting partners exhibit high externalizing behavior (Rothenberg et al., 2016). I suspect that these somewhat disparate results may be accounted for by the way in which I have measured family conflict in the current study. The family conflict measure included items asking how often family members got so angry they threw things, lost their tempers, fought and hit each other. I posit that each of these items may be much more sensitive to the effects of parenting partners expressing externalizing behavior. Indeed, adults demonstrating externalizing behavior often initiate coercive, argumentative exchanges within the family environment, resulting in both these adults and their interaction partners learning that the more they yell or argue and the less they back down, the more often they get their way (Rothenberg et al., 2016; Dishion & Patterson, 2006).

Adult depressive symptoms can certainly cause similar types of coercive exchanges, especially between parents and their attention-seeking children (McMahon & Forehand, 2003) and I suspect such parent-child interactions are part of the reason why G2 depressive symptoms

are predictive of G2-G3 family conflict in this sample. However, I also suspect that when two G2 partners with depressive symptoms interact with one another, such interaction patterns are less likely to produce the explosive family interactions well captured by this study's family conflict measure simply because both partners may tend to withdraw from such aversive interactions. If this study's family conflict measure included items such as "Family often walked away instead of solving problems", or "Family members were cold and uncaring with one another" it may have been able to better capture the deleterious family effects of interactions between two partners both experiencing depressive symptoms. Notably, this hypothesis, to my knowledge, has never been directly tested in the literature because most measures of family conflict and parenting behavior either focus exclusively on aggression and hostility in family interactions, or combine hostility and neglect/withdrawal into one measure of family or parenting behavior.

Strengths, Limitations and Future Directions

The present study has numerous strengths, including its multigenerational longitudinal design, incorporation of multiple reporters on family conflict in each generation and measurement of family conflict and G2 depressive symptoms at multiple developmentally salient time points over a 17 year period. Additionally, the present study is one of the first to move beyond examination of associations in family functioning across generations to actually test novel etiological mechanisms that might account for such associations. The study does so by utilizing analytic techniques capable of separating the unique effects of these mechanisms at different points in development. Finally, the study is also unique in its ability to capture families in both generations at similar points in development (i.e., when children in each family are adolescents), as called for by intergenerational researchers (e.g., Conger et al., 2009).

However, there are also several notable limitations. Family conflict in each generation was self-reported, as opposed to observed, making it possible that reporter bias affected estimates of conflict. On the other hand, it should be noted that multiple family members reported on family conflict in each generation, mitigating the risk of reporter bias affecting results. Moreover, intergenerational continuity in several different family processes has been observed across both self-report and observational measures (Conger et al., 2009).

Additionally, the use of parceling techniques in aggregating reports of family conflict means that each family member's report of family conflict was weighted equally in my estimation of latent family conflict variables. However, reporter agreement on family conflict, though statistically significant, was modest at best, suggesting that each family member had a somewhat unique perspective on family conflict and that perhaps some family members perceived higher conflict than others. Equal weighting of reports may not account for the outsized significance one family members' perspective has on shaping family conflict. Furthermore, G2 partner and G3 adolescent reports of family conflict were not available for all families. Consequently, some estimates of G2-G3 family conflict incorporated fewer perspectives than others.

It should also be noted that, as in all intergenerational studies of family processes to date, the present study was only able to measure G1-G2 family conflict for one partner in the G2-G3 family. As a result, the extent to which the mediating depressive pathway and associated moderators apply to the "other" G2 parenting partner in this study is unknown.

Additionally, I attempted to control for genetic effects in the current study by including measures of G1 antisocial personality disorder diagnoses, affective disorder diagnoses and alcoholism diagnoses in all study analyses. However, emerging evidence suggests that measures

that directly account for genetic liability, such as polygenic risk scores, more effectively control for such effects (Beaver & Belsky, 2012; Rice et al., 2006). As such, I cannot rule out that underlying genetic transmission of depressive symptoms cause both continuities in depressive symptoms over time and continuities in family conflict across generation. However, other intergenerational models of family functioning incorporating these state of the art genetic modeling techniques do not find intergenerational processes to be wholly explained by underlying genetic effects (Beaver & Belsky, 2012).

Finally, this work suggests several future directions for the investigation of intergenerational family conflict. The depressive pathway examined in the present study complements earlier work examining an externalizing pathway that accounts for intergenerational family conflict (e.g., Rothenberg et al., 2016). Taken together, these studies indicate that both externalizing and depressive symptoms can mediate intergenerational family conflict, that experiencing these behaviors during specific developmental time frames (i.e., adolescence for externalizing behavior, adolescence and young adulthood for depressive symptoms) may make intergenerational continuity in family conflict especially likely, and that women, as opposed to men, may be more likely to traverse these pathways. However, it is unclear how these externalizing and internalizing pathways interact with one another, how each of these pathways predict G3 psychopathology and whether specific interventions at specific points in development can be used to facilitate discontinuity in intergenerational family conflict.

It will be important for future investigations of intergenerational family conflict to examine G2 externalizing and internalizing symptoms simultaneously. Indeed, systematic reviews of both externalizing (Hinshaw & Lee, 2003) and depressive symptoms (Hammen & Rudolph, 2003) suggest high comorbidity among these different symptoms and at least modest

continuity in both symptom clusters over time. It may be that the presence of both externalizing and internalizing symptoms in an individual may make intergenerational continuity in conflict more likely. If an individual reacts to family conflict by engaging in both coercive, aggressive exchanges and by withdrawing from the family environment, patterns of family conflict in one's family of origin may become more intense and frequent. Such increased intensity and frequency of family conflict may make it even more likely that patterns of family and social interaction related to both externalizing and internalizing problems endure across time into adulthood. Moreover, it could be that an individual could "jump" from externalizing to depressive pathways, or vice-versa across time. For instance, an individual who learns externalizing behavior as a result of high conflict in their family of origin, may employ such behavior in social interactions in adolescence. However, by young adulthood they may experience social rejection as a result of such behavior and ultimately develop depressive symptoms that subsequently inform the development of conflict in their family of destination. Analyzing both externalizing and depressive pathways to intergenerational family conflict simultaneously in one model would help identify the unique effects of each pathway and the interactive effects of both pathways on family conflict over time.

Relatedly, it will be important to integrate measures of G3 child psychopathology into existing work on both G2 externalizing and depressive pathways to intergenerational continuity in conflict. Though it is known that G3 children from families with high conflict across generation show elevations in symptoms of psychopathology and impairment (Rothenberg, Solis, et al., 2016), it is not known how such symptoms relate to underlying etiological pathways to intergenerational family conflict. For example, it could be that G2s traversing a pathway to intergenerational family conflict have G3 children at elevated risk for experiencing depressive

symptoms specifically, or elevated risk for experiencing multiple types of psychopathology more generally.

It would also be interesting for future investigators to examine whether therapeutic treatments that target family processes (e.g., family therapy, behavioral parent training programs) alter pathways to intergenerational continuity in family conflict. For instance, it may be possible that intervening to ameliorate G1-G2 family conflict and prevent the emergence of preadolescent depressive symptoms in G2s could prevent the emergence of G2-G3 family conflict and associated G3 psychopathology years later when G2s start their own families. If family-based therapy can indeed prevent deleterious outcomes across generations, this finding would be vital for informing funding priorities about where and how to invest in preventive interventions.

As these important but unstudied questions indicate, much work remains to be done in investigating intergenerational family conflict. Nonetheless, the present study represents a significant step in considering how depressive symptoms drive intergenerational family conflict and in identifying the time points and family members through which such mechanisms operate to threaten adaptive family functioning.

Table 1

Sample Demographic Characteristics

Demographic Variable	G2 % or <i>M (SD)</i> (<i>N</i> = 246)	G2 Partner % or <i>M (SD)</i> (<i>N</i> = 102)	G3 % or <i>M (SD)</i> (<i>N</i> = 123)
Gender	57% female	43% female	47% female
Ethnicity			
Caucasian	71%	61%	51%
Hispanic	26%	33%	33%
Other	3%	6%	12%
Age (Wave 6)	31.8 (1.76)	33.2 (1.70)	12.14 (2.39)
Age (Wave 2)	14.3 (1.41)	--	--
Highest Level of Education Obtained in G2-G3 Family			
GED	30%	--	--
Completed Some College	31%	--	--
Associates, Bachelor's, or beyond	32%	--	--
G2 Child of Alcoholic (COA) Status	53% COA	--	--

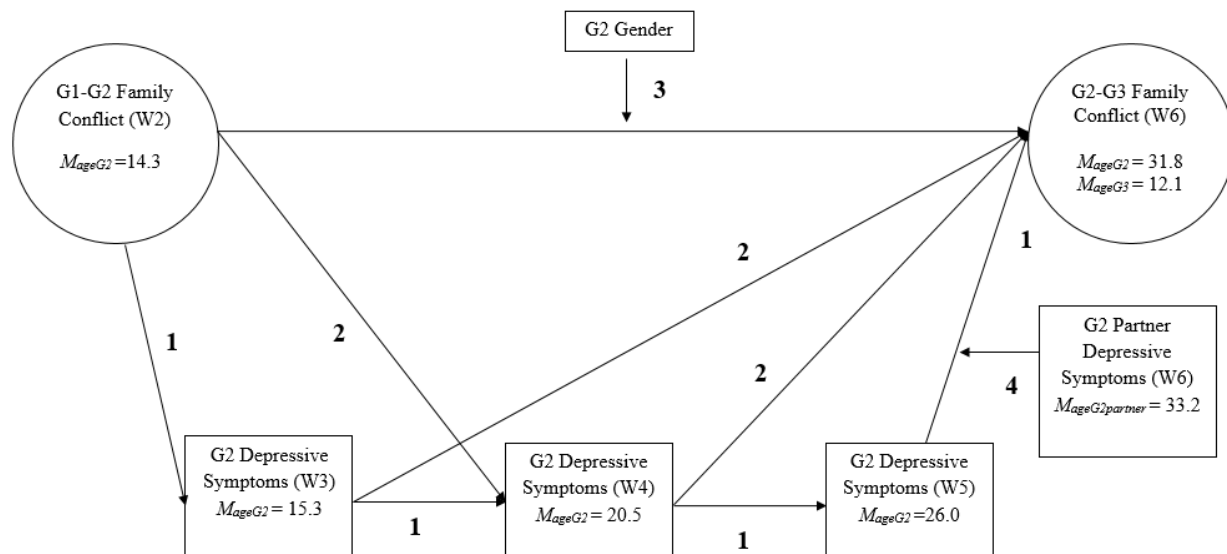


Figure 1. A conceptual model depicting study hypotheses testing a depressive pathway to intergenerational continuity in high conflict family environments. Each number corresponds to a pathway that must be significant for the corresponding hypothesis to be supported. Note that for hypothesis 3, G2 gender is predicted to moderate the entire model, not just the specific direct pathway from G1-G2 family conflict to G2-G3 family environment. Space restrictions necessitated this simplistic depiction of hypothesis 3.

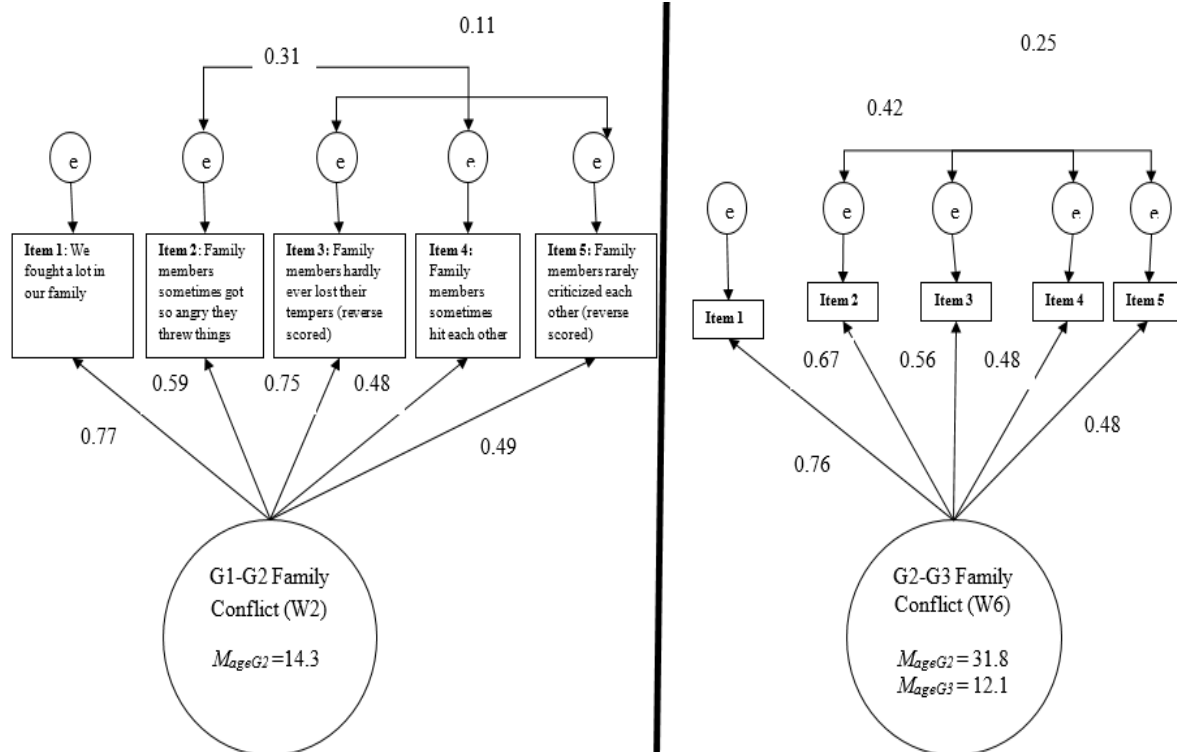


Figure 2. Results of confirmatory factor analyses estimating G1-G2 and G2-G3 latent family conflict variables. Item content is identical in both G1-G2 and G2-G3 latent variables. All paths, loadings and correlations depicted in the diagram are significant at $p < .05$, all estimates are standardized.

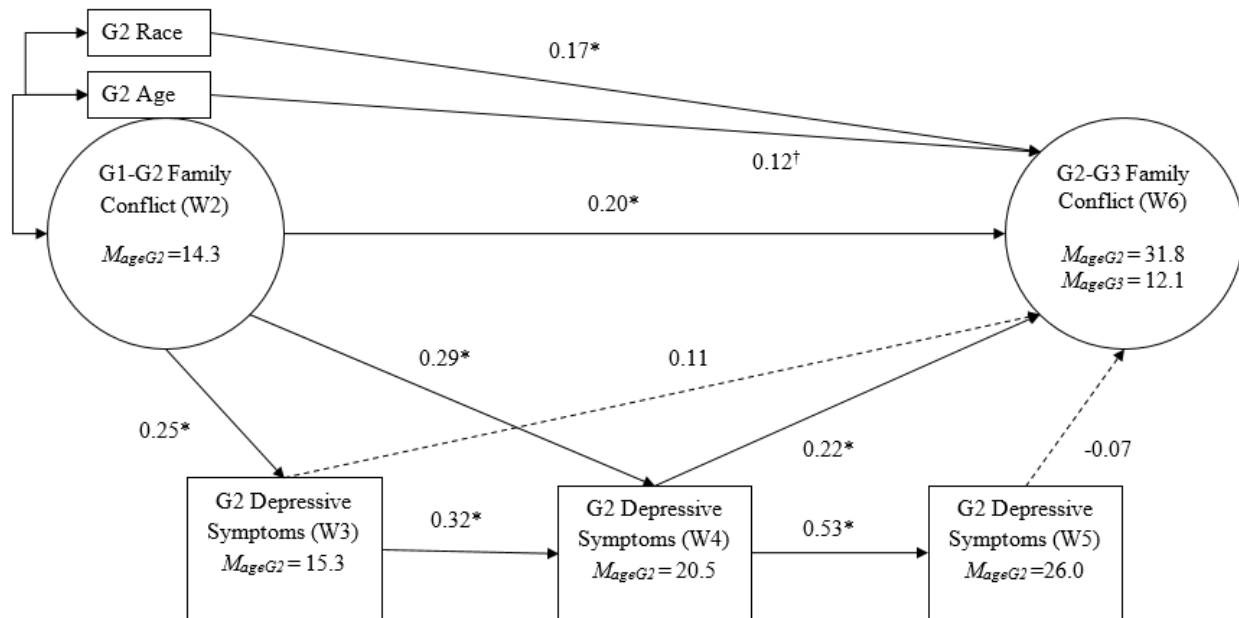


Figure 3. Model of intergenerational family conflict depicting associations between G2 depressive symptoms, G1-G2 family conflict and G2-G3 family conflict when both men and women are combined in the same structural equation model. Solid paths are significant, broken paths are non-significant, all estimates are standardized. $*p < .05$, $^\dagger p < .10$

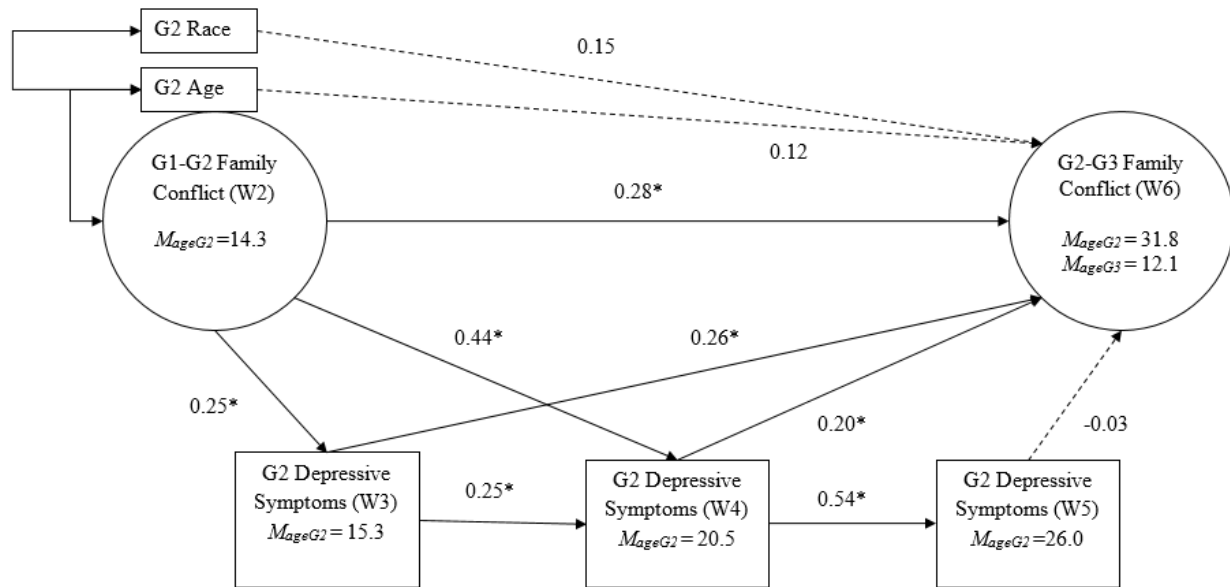


Figure 4. Model of intergenerational family conflict in G2 women depicting associations between G2 women's depressive symptoms, G2 women's G1-G2 family conflict and G2 women's G2-G3 family conflict within a structural equation model. Solid paths are significant, broken paths are non-significant, all estimates are standardized. $*p < .05$.

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