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Intergenerational continuity in high conflict family environments: Investigating a mediating depressive pathway

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Abstract

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. Addressing this limitation, we sampled 246 families from a multigenerational, high-risk, longitudinal study of parents (G1s) and their children (G2s), followed from adolescence to adulthood as well as the children (G3s) of G2 targets. Specifically, the current study examined whether G2s' depressive symptoms measured at multiple time points across development explained continuity in family conflict from one generation (G1–G2) to the next (G2–G3). Results revealed that after controlling for externalizing symptoms, depressive symptoms served as mediators of intergenerational family conflict in both men and women, but in different ways. Specifically, G2 women's young adulthood represented a period 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 intergenerational transmission of family conflict are discussed.

Keywords

family conflict; intergenerational; depression; externalizing; multigenerational; gender

Family conflict is a prospective predictor of myriad maladaptive behaviors including internalizing symptoms, externalizing symptoms, substance misuse and impairments in social role functioning across the life-course (Cummings & Schatz, 2012; Herrenkohl, Lee, Kosterman & Hawkins, 2012; Horowitz et al., 2011; Kimonis, Frick, & McMahon, 2014, Kouros & Garber, 2014; Rothenberg, Hussong, & Chassin, 2017a; Rothenberg, Hussong, & Chassin, 2017b). Emerging evidence suggests that family conflict can persist across

Ideas and analyses presented in this manuscript have never been disseminated elsewhere.

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, their spouse and their G3 (or third generation) children (Rothenberg, Hussong, & Chassin, 2016). Furthermore, conflict that persists from G1–G2 homes to G2–G3 homes is associated with high G2 and G3 psychopathology symptoms and deficits in social role functioning (Rothenberg et al., 2017a). These deleterious associations 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 pernicious intergenerational cycles of conflict.

Therefore, the present investigation explored one mediating mechanism that may explain intergenerational continuity in conflict; namely, a depressive pathway that persists across the life-course. We posited 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. We further delineate the purported mechanisms by which this pathway could emerge and the potential moderators that might alter progression along this pathway below.

Theoretical Models of Intergenerational Family Conflict

In accordance with other investigators (Cummings, Koss, & Davies, 2015; Emery, 1993; Horwitz et al., 2011), we believe that family conflict cannot be inferred from assessments of individual dyads only but must also be assessed as a broader, family-level construct. Therefore, the present investigation defined family conflict as the experience of aggression, criticism, anger, or arguments within the overall family climate (i.e., across multiple family relationships). Notably, the measure of family conflict used in the present study included items measuring both general family conflict (e.g., “We fought a lot in our family”) and family violence (e.g., “Family members sometimes hit each other”). Though both constructs are measured, for the remainder of this manuscript we described our measure of family conflict and violence as a measure of “family conflict” (due to space limitations).

Few studies have investigated intergenerational continuity in family conflict. Our own prior work investigated an externalizing pathway that might mediate intergenerational continuity in family conflict (Rothenberg et al., 2016). Specifically, utilizing principles from Social Interactional Theory and Coercion Theory (Dishion & Patterson, 2006; Scaramella, Conger, Spoth, & Simons, 2002), we posited that G2s from high conflict G1–G2 family environments learn to increase their own externalizing behavior (e.g., shouting, noncompliance) to obtain social goals (e.g., receiving parental attention, avoiding chores) when such interaction patterns are normative and reinforced in the family. These G2 reactions to conflict may then be applied to obtain social goals in a variety of settings across the lifespan, including in G2s’ own G2–G3 families. Thus, this pattern of externalizing

behavior may ultimately give rise to similar conflict in the G2–G3 family as seen in the G1–G2 family (Rothenberg et al., 2016).

To investigate this hypothesis, we tested whether the association between G1–G2 family conflict when G2s were 14 years old and G2–G3 family conflict when G2s were 32 years old (and G3s were 12 years old) was mediated by G2 externalizing behavior at three time points; when G2s were 15, 21, and 25 years old. Four major findings emerged from this investigation. First, we did not find evidence that a *contiguous externalizing pathway* mediated intergenerational continuity in family conflict (i.e., G1–G2 family conflict was not indirectly associated with G2–G3 family conflict via an autoregressive pathway comprised of all 3 measures of G2 externalizing behavior). Second, we did find evidence that a *developmentally-specific* externalizing pathway mediated intergenerational continuity in family conflict. Yet, third, we also discovered that this developmentally-specific pathway was *gender moderated*. Specifically, we found that G2 externalizing symptoms at age 15 mediated the association between G1–G2 and G2–G3 family conflict in G2 women but not men. Fourth, we found that *G2 partner externalizing behavior* moderated these associations, such that the highest levels of G2–G3 family conflict resulted when a G2 high in externalizing behavior partnered with someone who also demonstrated elevations in externalizing behavior (Rothenberg et al., 2016).

Yet, extant literature demonstrates that externalizing symptoms are only one set of child reactions to family conflict that may be present, and that depressive symptoms are another set of reactions that may emerge due to high family conflict (e.g., Cummings & Schatz, 2012; Hammen et al., 2014). Therefore, we posited that a depressive pathway might also mediate intergenerational continuity in family conflict. Specifically, we hypothesized that some G2s may respond to high-conflict interactions within the G1–G2 family environment by withdrawing to avoid the aversive consequences of family conflict. This withdrawing behavior may serve as a negative reinforcer because it allows G2s to avoid aversive stimuli within the family environment (Auerbach & Ho, 2012; Cummings et al., 2015). However, such withdrawal may also result in a functional impairment that is associated with depression more broadly (Hammen & Rudolph, 2003). Due to the negatively reinforcing nature of behavioral withdrawal within the G1–G2 family environment, G2s may generalize withdrawal as a way of coping with conflict in social interactions beyond the family, a strategy that may persist throughout ontogeny. This withdrawn-depressed constellation of behaviors may then lead to the emergence of conflict in the G2–G3 family as partners and children perceive G2s as cold, unengaged, lethargic, irritable, and unable to complete household chores and duties.

Because no existing studies had investigated such a depressive pathway, we decided to do so by extending each of the four findings that had emerged from our investigation of an externalizing pathway. Specifically, we tested whether a contiguous depressive pathway existed (paths labeled 1 in Figure 1), whether developmentally-specific depressive paths existed (paths labeled 2 in Figure 1), whether gender moderated this depressive pathway (path labeled 3 in Figure 1), and whether G2 partner internalizing symptoms moderated this pathway (path labeled 4 in Figure 1). Most importantly, due to the high co-occurrence of

externalizing and depressive symptoms (Hinshaw & Lee, 2003), we controlled for the externalizing pathway (see paths labeled 5 in Figure 1) in all study analyses.

A Contiguous Depressive Pathway

Due to the negatively reinforcing nature of behavioral withdrawal, we expect that high G1–G2 family conflict may lead to the emergence of G2 depressive symptoms that persist across ontogeny and predict high conflict in G2s' G2–G3 families in adulthood. Though not yet studied as an intergenerational process, numerous single generation studies provide evidence consistent with such a contiguous depressive pathway. For instance, a large body of literature demonstrates that high-conflict family interactions predict symptoms related to depression in children including withdrawal, emotion dysregulation, insecurity, uncertainty, and negative affect (Auerbach & Ho, 2012; Cummings & Schatz, 2012; Habib et al., 2014). Moreover, once such depressive symptoms emerge, they demonstrate relative stability into adulthood (e.g., Hammen & Rudolph, 2003; Hammen, Rudolph, & Abaied, 2014), where they can deleteriously affect multiple family interactions. For instance, research demonstrates that depression and withdrawal in one romantic partner can lead to frustration, confusion and increased demandingness within the other romantic partner (Baucom, McFarland, & Christensen, 2010). When these interactions happen within the family context, such interparental conflict often spills over into the family environment (Cummings & Schatz, 2012) where it is linked to frustrated, defiant and attention-seeking behaviors in the children of depressed parents (e.g., Dishion & Patterson, 2006; McMahon & Forehand, 2003). Therefore, we posit that high conflict in one's G1–G2 family will contribute to a contiguous depressive pathway characterized by the development and reinforcement of depressive symptoms in G2s that persist into adulthood and contribute to high G2–G3 family conflict.

Developmental Specificity in the Depressive Pathway

Prior studies show that G2 psychopathology at certain points in development, such as adolescence (e.g., Rothenberg et al., 2016) and young adulthood (Conger et al., 2009), may be an especially strong mediator of intergenerational continuity in family conflict. Notably, our own work investigating the externalizing pathway showed that G2 externalizing behavior experienced in adolescence but not young adulthood mediated continuity in intergenerational family conflict (Rothenberg et al., 2016). Because the extent to which such adolescence-specific mediation can be generalized to depressive symptoms is unknown, we tested whether G2 depressive symptoms in adolescence were specific mediators of family conflict.

Several existing lines of research provide evidence that adolescent-specific mediation may occur. Adolescents often experience emotional distress and behavioral disengagement as they seek to individuate from their families of origin (Friedman, Holmbeck, DeLucia, Jandased & Zebracki, 2009; Wray-Lake, Crouter, & McHale, 2010). However, within high-conflict families these hallmark parent-adolescent negotiations can lead to coercive family interactions that, for some adolescents, increase risk for subsequent withdrawal, insecurity and depression (Auerbach & Ho, 2012; Constantine, 2006; Vivona, 2000). Withdrawal

patterns of responding to high conflict interactions that develop for G2 adolescents may become entrenched and less malleable at later points in development (Jaffee, Belsky, Harrington, Caspi & Moffitt, 2006; Thornberry et al., 2003). The extent to which resulting depressive symptoms are present for G2s in adolescence then may be a particularly salient signal of a long term pattern of withdrawal from social interactions and related symptomatology that can impact later G2–G3 family interactions.

G2 depressive symptoms experienced in young adulthood may also represent a salient risk period for intergenerational continuity in family conflict. Developmental scientists have found that as young adults undergo major life transitions 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). If G2s are from high conflict family environments, such interactions are likely to continue to be high in conflict during these life transitions and associated with elevated G2 depressive symptoms (Cowan & Cowan, 2012). These depressive symptoms are associated with increased conflict in one's adult family during the first years of marriage and parenthood (Hammen et al., 2014; Cowan & Cowan, 2012). Such conflict shows stability for many years after a transition occurs (e.g., Cowan & Cowan, 2012; Cowan, Cowan, & Barry, 2011). In sum, we explore whether G2 adolescent and young adult depressive symptoms may be especially salient mediators of intergenerational family conflict.

Gender Specificity in the Depressive Pathway

In our prior work, we found the externalizing pathway to be gender-moderated (Rothenberg et al., 2016). We 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 for women but not for men. Extant research supports this hypothesis. Adolescent girls are more likely to experience depression due to family conflict (Hops, 1995; Mazza et al., 2009; Sheeber, Hops, Alpert, Davis, & Andrews 1997), perhaps because 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; Noller, 1994; Sheeber et al., 1997). Additionally, in their adult families, 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). Therefore, G2 gender may moderate the depressive pathway because G2 women are more likely to be launched onto the pathway due to high family conflict faced in adolescence, and are also more likely to have their depressive symptoms manifest in the adult family environment (Elder, Caspi, & Downey, 1986; Thornberry et al., 2003; Hammen et al., 2014).

The Role of Parenting Partners

Our prior work indicated partner effects on the externalizing pathway (Rothenberg et al., 2016). Extending this work, the current study explores partner effects on the depressive pathway. Individuals with depressive symptoms are more likely to partner with other depressed individuals (e.g., Desai, Schimmack, Jidkova, & Bracke, 2012; Mathews & Reus,

2001). If G2s high in depressive symptoms select similar romantic partners, then both partners may be more likely to engage in associated withdrawn, uninvolved interaction styles that result in greater G2–G3 family conflict. As a result, the high family conflict that a G2 experienced across childhood may be more likely to be perpetuated in their G2–G3 family. Therefore, to the extent that G2 partners show elevated depressive symptoms, we posit that G2s' own depressive symptoms are expected to predict greater conflict in the family environment.

Considering the Externalizing Pathway

Systematic reviews of both externalizing (Hinshaw & Lee, 2003) and depressive symptoms (Hammen et al., 2014) suggest high comorbidity among these different symptoms and at least modest continuity in both symptom clusters over time. Moreover, other literatures exploring multiple externalizing and internalizing symptom developmental pathways to dysfunction (e.g., the substance use disorder literature) have found that, after controlling for externalizing symptoms, internalizing symptoms may not predict deleterious outcomes (Chassin, Sher, Hussong, & Curran, 2013). Taking these issues into account and building on our existing work, we ensured that we controlled for the externalizing pathway in analyses exploring the depressive pathway (Figure 1, paths labeled "5).

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, we tested five hypotheses using a multigenerational longitudinal study assessing families across a twenty-year period (see Figure 1). First, we predicted that a G2 depressive pathway measured across ontogeny would mediate intergenerational continuities in family conflict (i.e., paths labeled 1 in Figure 1). Second, we predicted that G2 depressive symptoms in adolescence and young adulthood would 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). Third, we predicted that G2 gender would moderate the entire depressive pathway, such that the pathway would be significant for G2 women but not for G2 men (i.e., path labeled 3 in Figure 1). Fourth, we predicted that a stronger association between G2 depressive symptoms and high G2–G3 family conflict would occur for families where G2 partners have greater depressive symptoms (i.e., path labeled 4 in Figure 1). Fifth, we predicted that the depressive pathway would persist even after controlling for the externalizing pathway (i.e., paths labeled 5 in Figure 1). In investigating all study hypotheses, we controlled for other variables that could be associated with family conflict or G2 depression, including G2 age, G2 ethnicity, G2 SES, G1 Antisocial Personality Disorder, G1 alcoholism diagnosis and G1 affective disorder diagnoses. Notably, we controlled for these G1 diagnoses to ensure that intergenerational transmission of family conflict persisted above and beyond genetically-mediated intergenerational psychopathology. 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 along the depressive pathway that could guide future intervention development.

Methods

Data from the Adolescent and Family Development Project (AFDP; Chassin, Pitts, DeLucia, & Todd, 1999) were used for this study. AFDP is an ongoing longitudinal study of children of alcoholic parents (COAs) and matched controls assessed into adulthood. AFDP uses a multi-generational design involving assessments of parents (G1s), target adolescents followed over time (G2s) and the children of these targets (G3s). AFDP presently consists of 6 waves of data collected annually for waves 1–3 (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 G3s).

Participants

At wave 1, the AFDP sample consisted of 246 adolescents with at least one alcoholic parent and 208 matched adolescents with no 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, HMO wellness questionnaires and community telephone screenings (see Chassin et. al, 1999). To be included in the current study, COA families had to have parents who reported being either Hispanic or non-Hispanic Caucasian, be Arizona residents, have a child aged 10.5–15.5 years at wave 1, be English-speaking, have parents and children with no cognitive limitations that would preclude interview, and have at least one parent meet 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 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 for alcohol use or dependence. 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) and complete data on the family conflict measure at wave 6 (N = 246 of 273 G2s with children, with 27 missing data because they contacted their child less than once a week). All remaining 246 G2–G3 families were retained in study analyses. G2–G3 families ranged in size from 1–4 children (M =1.75 children), though only the oldest G3 child was included in the present analyses. Sample demographic characteristics can be found in Table 1.

Procedure

At each wave, data were collected via in-person interviews (Chassin et al., 1999). Family members were interviewed simultaneously and in separate rooms to increase privacy. In waves 1–3, at least one G1 caregiver and one G2 adolescent between the ages of 10 to 15

years old completed interviews. In wave 6, only G2 targets were required to complete interviews. However, G2 partners and any G3s 7 years old or older were also invited to interview if they were available at the time the G2 was interviewed. Interviews lasted from 1–3 hours. All study procedures were approved by the Arizona State University IRB (Project Title: Adult and Family Development Project; Protocol Number: 0506000017)

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 psychopathology was 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 the computerized DIS interview (Version 3; Robins, Helzer, Ratcliff, & Seyfried, 1982). In current analyses, family-level diagnoses were dichotomized as either present (at least one G1 parent met lifetime criteria) or absent (neither participating G1 parent met lifetime criteria). At wave 6, G2s and their partners reported their gender, ethnicity and highest education level obtained (using a scale ranging from 1=8th grade or less to 11=completed graduate/professional school).

G2 and G2-partner depressive symptoms—G2 depressive symptoms were measured at waves 2 (M_{G2Age} = 14.33 years, SD = 1.41 years, Range: 11.57–17.05 years), 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 Childhood Behavior Checklist (Achenbach & Edelbrock, 1981). Note that hereafter, each of these timepoints will be referred as G2 age “14”, “15”, “21” and “26” time points for the sake of parsimony, but it should be noted that each of these time points actually represents the range of ages referenced above. G2 partners self-reported 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 (α = .71 – .77 across waves for G2s and α = .78 for G2 partners).

Family conflict—Family conflict was measured using the 5-item family conflict subscale of 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”. This subscale was found to have adequate validity and internal reliability in previous studies (Bloom, 1985). In the present study, G1 mothers, G1 fathers and adolescent G2s completed the family conflict scale at wave 2 in reference to G1–G2 families. In wave 6, G2s, G2 partners and participating G3

children completed the family conflict subscale in reference to G2–G3 families. Items were scored so that higher scores indicated higher family conflict. In the present study, internal reliability estimates were as follows: wave 2 G1 father-reports (α=.69), G1 mother-reports (α=.65) and G2 reports (α=.73); and wave 6 G2 reports (α=.70), G2 partner reports (α=.67) and G3 reports (α=.65). The somewhat low reliability of some 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 several steps. Initially, domain-representative parceling procedures (Kishton & Widaman, 1994) 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 G3 child reports of G2–G3 family conflict. For this step, family members' responses to the family conflict scale were averaged at the item level for G1–G2 and G2–G3 families (i.e., G1 mother, G1 father and G2 adolescent responses to item 1 of the scale were averaged to create a single indicator of G1–G2 family conflict for item 1). Then, maximum likelihood confirmatory factor analyses using Mplus Version 7 (Muthen & Muthen, 2015) were conducted to estimate latent variables representing conflict in the family environment. All indicators loaded satisfactorily on their respective latent factors ($\lambda > .45$ for all indicators). Fit indices showed that that both the G1–G2 family conflict (χ^2 (3) = 4.11, p = 0.25, CFI = 0.99, TLI = 0.99, RMSEA = 0.04, SRMR =0.02), and G2–G3 family conflict (χ^2 (3) = 2.18, p = 0.53, CFI = 1.00, TLI = 1.00, RMSEA = 0.00, SRMR = 0.01) latent variables fit the data well, indicating that it was appropriate to estimate latent variables for both G1–G2 and G2–G3 family conflict.

Missing Data and Power Analysis

The analysis sample consists of 246 target G2s, however there was missingness on key variables. Specifically, some G1-G2 families were missing mother reports (4.47%, $N = 11$) and father reports (22.76%, $N = 56$) of family conflict and G2–G3 families were missing G2 partner reports (58.54%, $N = 144$) and G3 child reports (50%, $N = 123$) of family conflict. Missingness among G2 partner reports and G3 reports was due to partner or child unavailability at the time of the G2 target interview (the study was originally designed to primarily obtain G2 reports of functioning over time). Additionally, the number of G2s who failed to report on their depressive symptoms in any particular wave ranged from 0.81% to 8.54% ($N = 3$ to 21). However, every G2 reported their depressive symptoms in at least one of waves 3–5. Notably, G2–G3 families with versus without missing data did not significantly differ on G2 depressive behavior at waves 2–5 ($t(241)$ range from −1.40 to 0.40, $p > .05$), or G1–G2 family conflict items ($t(242)$ range from −1.81 to 0.80, $p > .05$). Additionally, mean levels of G2 target, G2 spouse, and G3 child report of conflict did not differ from one another ($t(102$ to $123)$ range from 0.99 to 1.73, $p > .05$), indicating that extensive missingness in G2 partner or G3 reports was unlikely to affect parcel integrity in G2–G3 family conflict model estimation. Finally, following best practice guidelines (e.g., Schlomer, Bauman, & Card, 2010), we utilized Little's Missing Completely at Random Test (Little, 1988) to ensure that missing values were distributed at random throughout the data set and not missing as a function of any other observed or unobserved variable. Results

revealed that missingness in all primary study variables could be considered missing completely at random (χ^2 (1049) = 1040.29, p = 0.57). Therefore, full information-maximum likelihood (FIML) procedures were used to account for missing data in subsequent analyses following Kline (2005). Additionally, utilizing power analysis procedures established by Kline (2005), we calculated that our sample was well above the minimum required sample size of 200 needed to detect medium effects (d = 0.3) at 0.8 power within a structural equation modeling (SEM) framework. Given that our prior work investigating intergenerational family conflict yielded medium to large effect sizes (Rothenberg et al., 2016), our study was adequately powered to test proposed hypotheses.

Results

Path modeling within a SEM framework using a FIML estimator was utilized. Following recommendations offered by Fritz & MacKinnon (2007) we generated bias-corrected bootstrapped errors and confidence intervals from 2000 bootstrap iterations to estimate all significant paths and indirect effects.

Covariate Baseline Model

First, the unique associations of study covariates (i.e., G2 age, G2 ethnicity, G2 educational attainment and G1 antisocial behavior, affective disorder and alcoholism diagnoses) with G2–G3 family conflict were examined. Results revealed that only G2 age (β = 0.17, SE = 0.08, p = .04) and G2 ethnicity (β = 0.13, SE = 0.07, p = .08) were significantly associated with G2–G3 family conflict at p < .10, such that G2s who self-identified as a race other than non-Hispanic Caucasian and older G2s experienced higher G2–G3 family conflict. Additionally, fit indices revealed that this model fit the data well (χ^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 retained in hypothesis testing.

Testing a Depressive Pathway

Primary hypothesis testing was conducted utilizing multiple group structural equation modeling to ensure that gender differences in the depressive pathway were captured (see Figure 2). This analysis proceeded in several steps. We initially examined whether the latent G1–G2 and G2–G3 family conflict variables were invariant across gender by following analytic strategies utilized in prior work (Rothenberg et al., 2016). Results indicated that when factor loadings and intercepts were constrained to be equal across gender, there was no significant decrement in model fit (χ^2 (8) = 8.30, p > .05) and the model fit the data well (χ^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 family conflict held the same meaning and metric across gender.

Next, structural differences in the model between G2 women and G2 men were investigated. First, a model was estimated in which all paths related to primary study hypotheses (i.e., all paths labeled 1, 2, and 5 in Figure 1) were constrained to be equal across gender in the

model. Then, one at a time, each path was freed to vary across gender. A path was allowed to freely vary across gender if a 1 degree of freedom χ^2 difference test revealed that the model fit significantly better with the path freed. Once a path was allowed to freely vary, separate estimates for G2 women and men were calculated. Analyzing the data in this way was advantageous for answering our study questions, as it allowed us to identify with precision the specific paths which vary across gender over development. Our final model revealed four specific paths that varied over gender (see Figure 2 and below for further discussion). A χ^2 difference test revealed that our final estimated model fit the data significantly better than the constrained model (χ^2 (4) = 22.93, $p < .01$). An additional chi-square test revealed that no additional improvement in model fit resulted if all other parameters were freed to vary across gender (χ^2 (9) = 7.35, $p > .05$), indicating that the final model depicted in Figure 2 was the most appropriate fit to the data. The model fit the data well (χ^2 (219) = 298.78, $p < .01$, CFI = 0.93, TLI = 0.91, RMSEA = 0.05, SRMR = 0.08).

For women, results revealed a significant total effect ($B = 0.33$, $p < .01$, 95% CI [0.16, 0.51]) and total indirect effect ($B = 0.22$, $p < .01$, 95% CI [0.09, 0.41]) of G1–G2 family conflict on G2–G3 family conflict and explained a significant amount of variance in G2–G3 family conflict scores ($R^2 = 0.38$, $p < .01$; see Figure 2). However, neither the total ($B = 0.15$, $p = .14$, 95% CI [−0.05, 0.36]) nor total indirect ($B = 0.05$, $p = .42$, 95% CI [−0.05, 0.19]) effect of G1–G2 family conflict on G2–G3 family conflict was significant for men and the model did not explain a significant amount of variance in G2 men's G2–G3 family conflict scores ($R^2 = 0.14$, $p > .05$).

Results revealed support for the existence of a contiguous pathway to intergenerational family conflict for men and women. Specifically, the indirect effect of G1–G2 family conflict on G2–G3 family conflict through G2 depressive symptoms at ages 14, 15, and 21 ($B = 0.01$, $p < .05$, 95% CI [0.001, 0.02]) was significant. This mediating pathway indicated that, in both men and women, higher G1–G2 family conflict was associated with higher G2 depressive symptoms at age 14, that were associated with higher G2 depressive symptoms at age 15, that were associated with higher G2 depressive symptoms at age 21 that were, in turn, associated with higher G2–G3 family conflict. Results supported our hypothesis that, even after controlling for the externalizing pathway a contiguous pathway that includes stability in depressive symptoms from ages 14 to 21 mediates intergenerational continuity in family conflict. Importantly, G2 age 26 depressive symptoms were not a significant predictor of G2–G3 family conflict in either women or men ($B = −0.10$, $p = .63$, 95% CI [−0.52, 0.30]) and there was no significant indirect effect that included G2 age 26 depressive symptoms. Therefore, it does not appear that G2 age 26 depressive symptoms significantly mediated the intergenerational transmission of family conflict in this sample above and beyond depressive symptoms at ages 14, 15, or 21.

Additionally, the developmentally-specific indirect effects of depressive symptoms were evaluated. Results revealed a significant indirect effect of G1–G2 family conflict on G2–G3 family conflict for G2 women's ($B = 0.08$, $p < .05$, 95% CI [0.02, 0.19]), but not men's ($B = 0.01$, $p > .05$, 95% CI [−0.04, 0.07]) age 21 depressive symptoms, even after controlling for the externalizing pathway. Notably, gender differences in this effect seem to be driven by the path associating G1–G2 family conflict with age 21 depressive symptoms. This path was

allowed to freely vary across gender and was the only path in the age 21 mediating pathway significant for women ($B = 0.22$, $p < .05$, 95% CI [0.13, 0.31]), but not men ($B = 0.01$, $p > .05$, 95% CI [−0.10, 0.15]). Taken together, these results support our hypothesis that G2 depressive symptoms experienced at age 21(i.e., young-adulthood) uniquely mediate associations between G1–G2 and G2–G3 family conflict, even after controlling for the externalizing pathway and prior depressive symptoms (e.g., at ages 14, and 15).

Importantly, after controlling for the externalizing pathway and prior depressive symptoms at age 14, G2 age 15 depressive symptoms were not found to be a significant mediator of the association between G1–G2 and G2–G3 family conflict in either women ($B = 0.02$, $p > .05$, 95% CI [−0.01, 0.07]) or men ($B = -0.02$, $p > .05$, 95% CI [−0.09, 0.08]). Therefore, we did not find support for our hypothesis that G2 depressive symptoms at age 15 (i.e., adolescence) are a unique mediator of intergenerational family conflict. We chose to evaluate symptoms at age 15, as opposed to age 14, for two reasons. First, evaluating symptoms at age 15 allowed us to control for age 14 depressive symptoms and thus evaluate these symptoms as a developmentally-specific mediator above-and-beyond previous depressive symptoms. Second, in prior work we evaluated externalizing symptoms at age 15, and wanted to conduct an analogous evaluation of depressive symptoms in the present study.

Notably, current results replicate our prior findings concerning the externalizing pathway, as G2 women's ($B = 0.13$, $p < .05$, 95% CI [0.02, 0.26]), but not men's ($B = 0.06$, $p > .05$, 95% CI [−0.06, 0.18]) age 15 externalizing symptoms were found to mediate the association between G1–G2 and G2–G3 family conflict. Additionally, the current analyses added to this finding by demonstrating its persistence even after controlling for G2 depressive symptoms. The current analyses also extended this finding by demonstrating that gender differences seem to be driven by the path associating age 15 externalizing symptoms with G2–G3 family conflict. This path was allowed to freely vary across gender and was the only path in the age 15 mediating pathway significant for women ($B = 0.39$, $p < .05$, 95% CI [0.03, 0.72]), but not men ($B = 0.17$, $p > .05$, 95% CI [−0.20, 0.46]).

After accounting for mediating paths, the direct path associating G1–G2 with G2–G3 family conflict was not found to be significant in either women or men ($B = 0.10$, $p > .05$, 95% CI [−0.14, 0.30]). Therefore, the significant indirect pathways described above fully mediated the association between G1–G2 and G2–G3 family conflict.

Partner Depressive Symptoms as a Moderator of Intergenerational Family Conflict

Interaction terms were created and added to the 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. Four separate multiple-group models were estimated, where interaction terms between G2 partner depressive symptoms and G2 depressive symptoms at ages 14, 15, 21, and 26 were investigated. In all four models, the interactions terms were not significant in either gender. Therefore, hypothesis 4 was not supported.

Sensitivity Analyses

To further test the robustness of these findings, we conducted sensitivity analyses. First, we added additional assessments of externalizing symptoms to the model to test whether the depressive pathway maintained significance after controlling for externalizing symptoms later in development. We added to the model in Figure 2(a) G2 externalizing symptoms at ages 21 and 26, (b) an autoregressive path from G1–G2 family conflict through G2 externalizing symptoms at ages 15, 21, and 26 to G2–G3 family conflict and (c) correlations between G2 depressive and externalizing symptoms ages 21 and 26. Model results did not substantively vary when these two time points were added, but (as expected) model fit degraded with the addition of these two nonsignificant predictors.

Second, to test robustness of findings across different types of families, we compared families where G2s were children of alcoholics (COAs) versus non-COAs. Analyses proceeded in an analogous manner as with tests of gender differences for the model in Figure 2. 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 (χ^2 (13) = 10.20, $p > .50$). 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.

Discussion

We examined whether G2 depressive symptoms measured at multiple time points across development explained continuity in family conflict from one generation to the next even after accounting for externalizing symptoms. Three key findings emerged. First, in both men and women, there was evidence of a contiguous depressive pathway, as higher depressive symptoms that persisted from age 14 to age 21 mediated the association between G1–G2 and G2–G3 family conflict. Second, there was some evidence for developmental specificity in the depressive and externalizing pathways, as G2 women's externalizing symptoms at age 15 and depressive symptoms at age 21 mediated intergenerational continuity in family conflict. Third, partner influences on the depressive pathway were minimal, as G2 partners' depressive symptoms did not moderate the relation between G2 depressive symptoms and G2–G3 family conflict. Interpretation of these findings and their implications for understanding intergenerational continuity in family conflict is discussed below.

Considering a Contiguous Depressive Pathway

We hypothesized that a contiguous depressive pathway, comprised of G2 experiences of depressive symptoms from adolescence to adulthood, would mediate the intergenerational transmission of family conflict. The current study partially supported this hypothesis; results indicated that a depressive pathway stretching from when G2s were age 14 to 21 partially accounted for intergenerational continuity in family conflict, even after controlling for a G2 externalizing pathway and for developmentally-specific mediational pathways. We suspect

this pathway might endure in both genders because it might capture the underlying, gender-invariant process through which depressive symptoms facilitate intergenerational transmission of family conflict. Indeed, even after controlling for gender-specific associations, evidence suggests that both boys and girls often withdraw to avoid high family conflict in childhood (Auerbach & Ho, 2012; Cummings et al., 2015), and that in both girls and boys withdrawn/depressed styles of interaction show stability into young adulthood (Auerbach & Ho, 2012; Hammen et al., 2014) where they predict the emergence of maladaptive conflict as families form (Baucom et al., 2010; Cowan & Cowan, 2012; McMahon & Forehand, 2003). This process may be more likely to emerge for young adult women due to gender-specific contextual conditions faced in young adulthood (e.g., greater recontact with one's family of origin, less social support from one's family of origin, and greater caregiving responsibilities). However, we posit that in both genders, this same process (i.e., withdrawn/depressed styles of interaction being reinforced and applied from adolescence into young adulthood) might underlie the depressive pathway. Therefore, whereas our gender-specific findings may capture the greater *contextual* risks faced by women, the continuous pathway we found in both genders may capture the shared underlying *process* by which the depressive pathway confers intergenerational risk for family conflict in both men and women.

Gender-Specific Developmental Pathways

Results indicated G2 women's depressive symptoms at age 21 mediated intergenerational continuities in family conflict, even after controlling for prior depressive symptoms at ages 14 and 15, and the larger depressive and externalizing pathways. Additionally, analyses revealed that the path associating G1–G2 family conflict with age 21 depressive symptoms was the specific path that varied across gender, and consequently led to such mediating effects. We posit two reasons why this pathway in particular may have varied across gender. First, it is more likely that a woman will experience extensive recontact with her family of origin around young adult major life transitions, such as the period of pregnancy and childbirth (Cowan & Cowan, 2012). Therefore, women from high-conflict families of origin might be especially likely to develop or redevelop depressive symptoms in the young adult years (e.g., around age 21) when such transitions occur. Second, many investigations reveal that the transition to parenthood is especially difficult for women who do not receive adequate social support from their extended family environment (e.g., Castellano, Velotti, Crowell, & Zavattini, 2014; Dew & Wilcox, 2011). Young adult women from high conflict families may have withdrawn from their family environments in adolescence, subsequently been unable to draw on the support of their family of origin during young-adult life transitions, and therefore be at greater risk for the emergence of depressive symptoms during such life transitions. In sum, we posit that the continued presence of high conflict families of origin, or the absence of social support due to growing up in a high conflict family of origin, may account for the association between G1–G2 family conflict and age 21 depression for women.

Consideration of the timing of the transition to parenthood could explain why G2 women's age 21, but not age 26, depressive symptoms predicted subsequent G2–G3 family conflict. Specifically, in the current sample, over 86% of G2s were either pregnant or had a child by

age 21 and the average age of G3 children was 2.14 years when G2 women were age 21. Therefore, it appears that most G2 women experienced the transition to parenthood (and accompanying recontact with their family-of-origin and risk for depressive symptoms) at age 21 as opposed to age 26.

Furthermore, given that G2 women were 21 years old when their children were 2 years old, many of these G2s became parents in their late teens or early 20s. Other scholars who have reviewed the literature note that the associations between the transition to parenthood and the emergence of depression is especially high for parents in their late teens and early 20s because they have fewer resources to buffer against the social and emotional stressors associated with parenthood (Pearlman, Schieman, Fazio, & Meersman, 2005). Young parents who emerge from high conflict families lack familial support and therefore may possess even fewer social and emotional supports, making the emergence of depression even more likely (Pearlman et al., 2005). Thus, the age 21time point may be a specific period of vulnerability for the emergence of depression and subsequent G2–G3 family conflict in the current study because it captures the developmental time point where most G2s in the sample transition to parenthood, and where such a transition is most likely to be associated with stressors that lead to parental depression in young parents from high conflict families.

The current results also replicated our prior findings that G2 women's externalizing behavior at age 15 mediated the association between G1–G2 and G2–G3 family conflict (Rothenberg et al., 2016). Yet, the current results also extended these findings in two ways. First, present findings demonstrated that G2 women's age 15 externalizing behavior mediates intergenerational family conflict even after controlling for age 15 depressive symptoms and a depressive pathway that extends across ontogeny. The robustness of this age 15 pathway may indicate it as an especially important target for future preventive interventions. Such intervention could be based upon family communication or behavioral parent training strategies that have already shown efficacy in decreasing family conflict and externalizing behavior in families with adolescents (Cummings & Schatz, 2012; McMahon & Forehand, 2003). Second, the current investigation also isolated the pathway associating women's externalizing behavior at age 15 with G2–G3 family conflict as the specific pathway that varied across gender, and consequently led to the gender differences in the age 15 mediating effect seen in this and prior studies. We suspect this specific pathway may have varied across gender because women spend more time than men, on average, providing childcare and parenting in their adult family (Cowan & Cowan, 2012; Craig & Mullan, 2011). Consequently, women's externalizing symptoms, once learned and reinforced in adolescence may be more likely to manifest in the family context (Elder et al., 1986; Thornberry et al., 2003).

Contrary to our hypotheses, neither men nor women's age 15 depressive symptoms served as a unique mediator of intergenerational conflict. We suspected that such symptoms could serve as unique mediators because negotiation of adolescent autonomy may be especially intense in high conflict families, and may consequently lead to greater adolescent withdrawal and depression. However, it may be that the arguments, emotional distress, and coercive interaction processes that often characterize adolescent autonomy-seeking in high conflict families may be best captured by externalizing, as opposed to internalizing,

symptoms (Dishion & Patterson, 2006). That seems to be the case in the present sample, as the association between G1–G2 family conflict and G2 externalizing, but not internalizing, behavior at age 15 was significant in both genders.

Considering G2 Partner Behavior as Moderator

Unexpectedly, we 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 examining G2 and G2 partner externalizing behavior (Rothenberg et al., 2016). These null results may be accounted for by the measure of family conflict used in the current study. The family conflict measure included multiple items that may better capture the effects of parenting partners expressing externalizing behavior (e.g., asking how often family members threw things, lost tempers, fought, etc.). We suspect that when two G2 partners with depressive symptoms interact with one another, such interaction patterns are less likely to produce the explosive externalizing family interactions well captured by this study's family conflict measure simply because both partners may tend to withdraw from such aversive interactions.

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 study constructs at multiple time points over a 17 year period. Additionally, the present study is one of the first to move beyond examination of associations in intergenerational family functioning to actually test novel etiological mechanisms that might account for such associations. It does so by simultaneously testing multiple potential pathways to intergenerational continuity in family conflict and by utilizing analytic methods capable of isolating gender-specific associations within such pathways. 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 limitations. First, family conflict 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. Second, 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. Third, it should also be noted that in the present study, both G1–G2 family conflict and G2 depressive symptoms were measured contemporaneously when G2s were 14. Therefore, directionality of associations at the start of the depressive pathway are unclear; we cannot determine whether G1–G2 family conflict leads to the emergence of age 14 depressive symptoms or vice-a-versa. Fourth, we attempted to control for genetic associations in the current study by including measures of G1 psychopathology diagnoses in study analyses. However, emerging evidence suggests that measures that directly account for genetic liability, such as polygenic risk scores, more effectively control for such associations (Beaver & Belsky, 2012). As such,

we do not fully capture the effects of underlying genetic transmission on either depressive symptoms or family conflict.

Additionally, this work suggests several future directions for the investigation of intergenerational family conflict. 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. For example, it is unknown whether G2s traversing a depressive pathway to intergenerational family conflict have G3 children at elevated risk for experiencing depressive symptoms specifically, or elevated risk for experiencing heterogeneous psychopathology more generally. Additionally, future investigators should examine whether therapeutic treatments that target family processes alter pathways to intergenerational continuity in family conflict. Interventions in G1–G2 families could prevent the emergence of both depressive and externalizing pathways to G2–G3 family conflict. Finally, future investigations should examine how the timing of family transitions (e.g., marriage, parenthood) may increase risk for intergenerational continuity in family conflict. As our age 21 findings may preliminarily demonstrate, it may not just be the transition to parenthood, but also when it occurs in the life-course (i.e., late teens and early 20s for our sample) that makes intergenerational family conflict more likely.

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.

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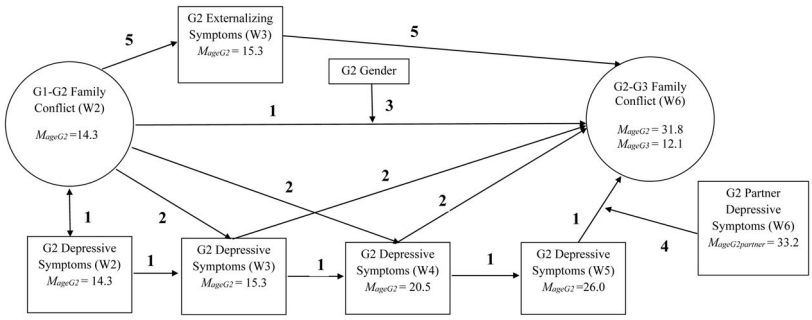

Figure 1.

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.

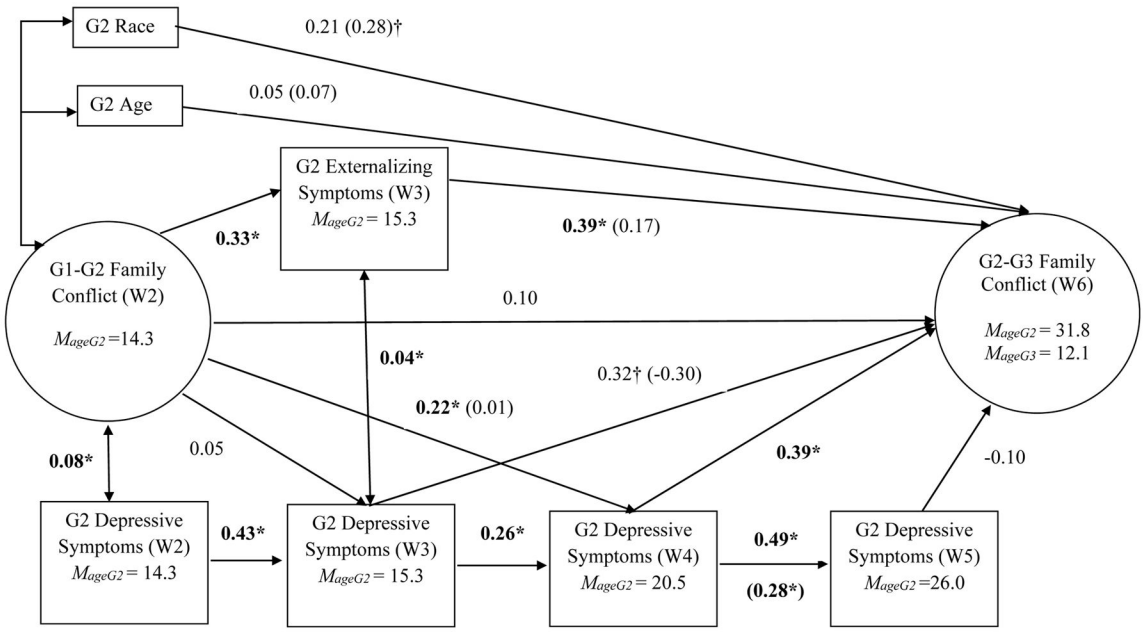

Figure 2.

Model of intergenerational family conflict depicting associations between G2 depressive symptoms, G2 externalizing symptoms, G1–G2 family conflict and G2–G3 family conflict within a multiple group structural equation model. Paths that were freed to varied by gender include estimates for women first, and then estimates for men second in parentheses. Paths that were constrained to be equal by gender just include a single estimate (since such paths, by definition, are equivalent for men and women). Significant effects are bolded. *$p < .05$, †$p < .10$

Table 1

Sample Demographic Characteristics

Demographic Variable	**G2** *% or M (SD) (N= 246)*	**G2 Partner** *% or M (SD) (N= 102)*	**G3** *% or M (SD) (N = 123)*
Gender	57% female	43% female	47% female
Ethnicity			
Non-Hispanic 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	--	--