

INTERGENERATIONAL TRANSMISSION OF HIGH CONFLICT FAMILY
ENVIRONMENT

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ABSTRACT

William Andrew Rothenberg: Intergenerational transmission of high conflict family environment
(Under the direction of Andrea Hussong)

The current study examined whether family conflict is passed from one generation to the next within families, and explored potential mediators and moderators which could explain this continuity. The study utilized a multigenerational longitudinal data set to examine family conflict as reported by multiple reporters from three successive generations in 246 families. Results showed that conflict in the current family was strongly correlated with that of the family of origin in women but not in men. Continuity in family conflict across generations was mediated by patterns of elevated adolescent antisocial behavior in members of the second generation (G2). Additionally, analyses revealed an interaction between G2 and G2 partner antisocial behavior such that even if just one partner in the G2 family demonstrated high levels of antisocial behavior, elevated levels of family conflict resulted. Potential explanations and implications of these findings are considered.

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INTRODUCTION

In the 1980s and early 1990s, several landmark longitudinal studies of adolescents and their families began to track how individual development was impacted by family context (see Chassin, Rogosch & Barrera, 1991; Conger & Elder, 1994; Capaldi & Patterson, 1989 for examples). For some of these studies, follow-up assessments are ongoing as these adolescents have grown into adults with families of their own. These three-generation studies provide prospective assessments capable of tracking how the family environments of parents (generation 1 respondents, or G1s) and their adolescents (generation 2 respondents, or G2s) come to shape the family environment of these same G2s later in development when they become parents of their own children (generation 3 respondents or G3s).

Continuity in family environments across generations is a particular concern for understanding the intergenerational transmission of individual risk behaviors (Conger, Capaldi, & Belsky, 2009, Belsky, Conger & Capaldi, 2009, Bailey, Hill, Oesterle, & Hawkins, 2009). Behavioral genetic studies have long shown familial aggregation (Bailey et. al, 2009; Moffitt, 2006) as well as cross-generational continuities in maladaptive behaviors (Silberg, Maes, & Eaves, 2012; Moffitt, 2006; Hines & Saudino, 2002), including antisocial behavior (Silberg et. al, 2012; Dionne, Tremblay, Boivin, Laplante, Perusse, 2003; Viding, Blair, Moffitt, & Plomin, 2005; Moffitt, 2006). Although behavioral genetics studies show that genetic influences account for as much as 75% of the variance in antisocial behavior (Arseneault, Moffitt, Caspi, Taylor, Rijdsdijk, Jaffee et. al, 2003; Moffitt, 2006), these same studies also indicate that environmental factors play a substantial role in influencing the persistence of antisocial behavior from one generation to the next (Arseneault et. al, 2003; Silberg et. al, 2012; Moffitt & the E-Risk Study

Team, 2002; Moffitt 2006; Caspi, McClay, Moffitt, Mill, Martin, Craig, et. al, 2002; Foley, Wormley, Silberg, Mae, Hewitt, Eaves, et. al, 2004). An important factor impacting the development of antisocial behavior in children is the family environment (Dishion & Patterson 2006; Moffitt, 2006).

Studies of individual parenting behaviors suggest that some aspects of the family environment, such as parent hostility, show significant continuity across generations (Neppel et. al, 2009; Hops, Davis, Lave, & Sheeber, 2003; Thornberry et. al, 2003; Belsky & Jaffee, 2006). Additionally, extant research indicates that continuities in family environment may be linked to cross-generational continuities in antisocial behavior, though findings are mixed and limited by reliance on narrow indicators of the family environment and antisocial behavior (Sidberg, 2012; Capaldi et. al, 2003, Conger et. al, 2003; Hops et. al, 2003; Thornberry et. al, 2003; Bailey et. al, 2009). Together, these studies suggest that family environments and individual child development inform each other in a recurrent cycle across generations. Testing this cycle, the current study uses a three-generation study of high-risk families (with an alcoholic parent) and matched controls to evaluate cross-generational continuities in family environment with attention to broad indicators of poor family functioning as reported by multiple family members across generations. In addition, the current study assesses how antisocial behavior may mediate these cross-generational consistencies and what factors may make such consistencies more likely to occur.

Mechanisms underlying cross-generational continuities in family context

According to Social Interactional Theory (SIT), children's behavior is shaped by the quality of their interactions with specific environments, including the family environment (Scaramella, Conger, Spoth, & Simons, 2002; Dishion & Patterson, 2006). Theorists have

utilized the SIT framework to posit that maladaptive functioning in G1-G2 families will result in the development of maladaptive behavioral styles in G2s (Scaramella et. al, 2002; Dishion & Patterson, 2006; Patterson, Reid, & Dishion, 1992). Other investigators have extended SIT to speculate that G2s' maladaptive behavioral styles will negatively impact subsequent family functioning in G2-G3 families (Hops et. al, 2003; Neppl et. al, 2009). More specifically, SIT posits that a high conflict family environment will promote harsh, coercive parent-child interactions in G1-G2 families (Dishion & Patterson, 2007; Patterson, 1982). It is through these coercive interactions that G2 children initially learn that employing aggressive, harsh, antisocial behaviors serve as effective strategies which can be used to help them obtain their goals and avoid parental demands in the short-term (Dishion & Patterson, 2007; Patterson, 1982). Over the long term, these repetitive coercive parent-child interactions are posited to teach children that they can gain control in a hostile, unstable family environment by employing antisocial behaviors (Dishion & Patterson, 2006). According to SIT, once this pattern of antisocial behavior is learned, it is then generalized by children to shape their interactions in other social environments. As a result, these youth are at risk for engaging in antisocial behaviors across development to the extent that antisocial behavior in the larger social environment is reinforced and effective in allowing attainment of social goals (e.g., avoiding authority figure demands, associating with other deviant peers, and winning popularity; Dishion & Patterson, 2006; Scaramella et. al, 2002). SIT predicts that repeated negative reinforcement of antisocial behavior in the family environment which is then extended to the larger social context is a learned behavior likely to endure into adulthood.

Recently, investigators who are interested in studying continuities in parenting behavior have extended elements of the SIT model to explain how aspects of maladaptive family

environments are passed from one generation to the next (Hops et. al, 2003; Capaldi, Pears, Kerr, & Owen, 2008). Specifically, they hypothesize that the same patterns of high, stable antisocial behavior that are learned by G2s as a strategy for goal attainment in social situations in childhood and adolescence are applied by G2s to attain their parenting goals as they form their own families. Consequently, the same parent-child coercive cycles that G2s participated in as children are repeated in their own G2-G3 families where they participate as parents (Neppl et. al, 2009; Bailey et. al, 2009; Brook, Lee, Finch, & Brown, 2012). Thus, SIT makes two predictions about cross-generation family processes: first, there is intergenerational continuity in high conflict family environments; and second, this continuity is mediated by persistent antisocial behavior in members of the second generation.

Although no studies have tested these hypotheses for high conflict family environments more broadly, the SIT framework has been tested for one component of a high conflict family environment, namely harsh parenting behavior. Prospective, longitudinal studies have found that levels of G1 harsh parenting behavior prospectively predict levels of G2 harsh parenting behavior (Conger, Neppl, Kim, & Sacramella, 2003; Capaldi, Pears, Patterson & Owen, 2008; Capaldi, Pears, Patterson, & Owen, 2003; Sacramella & Conger, 2003), with significant positive correlations between the harsh parenting practices of G1 parents and their G2 offspring found in the range of .15 to .40 (e.g., Neppl et al., 2009; Bailey et. al, 2009; Conger, Schofield, & Neppl, 2012). Moreover, several investigations have found the association between G1 parenting practices and G2 parenting practices to be robust across both urban (Bailey et. al, 2009; Smith & Farrington, 2004) and rural (Neppl et. al, 2009; Conger et. al, 2008) geographic locations, across five year (Conger et. al, 2003; Hops et. al, 2003) to fourteen year (Bailey et. al, 2009) gaps between G1 and G2 parenting assessments, and across both parent self-reports (Capaldi et. al,

2008) and independent observations (Conger et. al, 2012; Hops et. al, 2003) of harsh parenting behavior. Overall, there is little doubt that harsh parenting behaviors show at least moderate intergenerational continuity (Conger et. al, 2009).

Somewhat less consistent, however, is evidence regarding the mediating role of antisocial behavior in these cross-generation continuities in harsh parenting. Antisocial behavior observed in G2's as young adults has been shown to mediate observed intergenerational continuities in harsh parenting behaviors (Neppel et. al, 2009; Conger et. al, 2009; Capaldi et al., 2008; Smith & Farrington, 2004). However, the harsh parenting literature seems to be divided in its support of whether antisocial behavior seen in G2 adolescents mediates the relationship between G1 and G2 harsh parenting behaviors (Conger, Belsky, & Capaldi, 2009). In some studies, adolescent antisocial behavior has been found to directly mediate the relationship between G1 and G2 parenting behaviors (Hops et. al, 2003; Capaldi et. al, 2003), whereas in other investigations no such significant relationship was supported (Bailey et. al, 2009; Kerr, Capaldi, Pears, & Owen, 2009; Conger et al., 2003). Though several researchers have tentatively concluded that G2 antisocial behavior could serve to link G1 and G2 harsh parenting practices (Conger, Belsky, & Capaldi, 2009), the question of whether G2s' adolescent antisocial behavior in particular serves as a mediator of intergenerational continuities in harsh parenting behavior remains unclear.

Although the harsh parenting literature provides an exciting test of SIT as a way of understanding cross-generation continuities in high conflict family environments, it is limited in several ways. First, the harsh parenting literature often relies on single reporters of family environment. Specifically, the harsh parenting literature often only includes G1 mother reports of harsh parenting practices in G1-G2 families, and only includes G2 target reports of harsh parenting practices in G2-G3 families (though see Capaldi et. al, 2009 and Kerr et. al, 2009 for

exceptions). Over reliance on single reporters can be problematic, because these single reporters may be biased (i.e., underreport their own harsh parenting behaviors) or have limited validity (i.e., present patterns of family functioning from only one perspective).

A second limitation is that many current studies testing antisocial behavior as a mediator of intergenerational continuity in harsh parenting behavior do not effectively account for temporal precedence. Specifically, many of these investigations measure G1 harsh parenting behavior and G2 antisocial behavior at the same time point, making it difficult to delineate whether G1 harsh parenting practices lead to increases in G2 antisocial behavior or whether increased G2 antisocial behavior actually leads to greater G1 harsh parenting practices. The inability to establish temporal precedence casts doubt on the finding that antisocial behavior mediates the relationship between G1 and G2 harsh parenting practices. For instance, one could alternatively argue that child antisocial behavior elicits similar harsh parenting practices in G1 and G2 parents, and that perceived continuity between G1 and G2 harsh parenting is an artifact of this recurring parent-child interaction pattern.

Perhaps the most important limitation of the harsh parenting literature in testing the SIT model is its narrow conceptualization of both the family environment and mediating antisocial behaviors. Harsh parenting behaviors demonstrated by a single parent are only one component of the larger high conflict family environment which impacts child outcomes (Patterson & Dishion, 2002; Patterson, 1997). Conflict between parenting partners (Cummings & Schatz, 2012), between parents and children (Lam, Solmeyer, & McHale, 2012), and between siblings (Campion-Barr, Greer, & Kruse, 2013) each contribute to the development of conflict in the family environment. Further evidence indicates that each of these different forms of dyadic conflict reciprocally interact with one another to inform the development of high conflict family

environments (Cummings & Davies, 2010; Lam et al., 2012). Measures of harsh parenting behaviors are unable to account for how the behaviors of multiple family members interact to influence intergenerational continuities in high conflict family environments. To address this issue, studies are needed that consider family-level, rather than individual-parent level conflict within the family environment (Emery, 1993). Thus, *the first objective of the present investigation is to determine if high conflict family environments demonstrate continuity across generations.*

A final limitation of the harsh parenting literature is that tests of G2 antisocial behavior as a mediator have relied on single time point assessments of antisocial behavior or have aggregated antisocial behavior observed at different time points across development into a single point estimate of antisocial behavior. Yet, SIT posits that elevated levels of antisocial behavior which persist across development, rather than single time-point elevations in antisocial behavior, mediate continuities in high conflict family environments. No study has tested whether elevated levels of G2 antisocial behavior which persists from adolescence through young adulthood mediates continuities from G1-G2 to G2-G3 high conflict family environments. Rather, studies of harsh parenting show that antisocial behavior exhibited by G2's in young adulthood mediates intergenerational continuities in harsh parenting behaviors (Capaldi et al., 2008; Smith & Farrington, 2004), but evidence of antisocial behavior exhibited by G2's in adolescence as a mediator of these continuities is mixed (Capalid et al., 2003; Kerr et al., 2009).

These findings may not be surprising in light of developmental theory and supporting research indicating that antisocial behavior may have different meaning across development. Developmentally-informed theories of antisocial behavior indicate that patterns of antisocial behavior that extend from adolescence into adulthood may be a better indicator of risk for long-

term negative outcomes. For example, Moffitt's developmental taxonomy of antisocial behavior (Moffitt, 1993; Moffitt, 2006) proposes that there are two primary patterns of antisocial behavior; a life-course-persistent pattern and an adolescent-limited pattern (Moffitt, 1993). The life-course-persistent pattern of antisocial behavior originates early in life, when neuropsychological and genetic predispositions toward antisocial behaviors within a child interact with the maladaptive environments in which a child is placed to produce a high, stable pattern of antisocial behavior which persists across the life-course (Moffitt, 2006). In Moffitt's developmental taxonomy, as in SIT, high family conflict plays a prominent role in the development of life-course persistent antisocial behavior (Moffitt, 2006). In contrast, the adolescent-limited pattern of antisocial behavior originates in early adolescence, when increasing numbers of otherwise well-adjusted youth begin to turn to antisocial behavior to seek autonomy from their parents and earn praise from their peers (Moffitt, 2006). For adolescent-limited antisocial youth, antisocial behavior tapers off soon after the social goals of young adulthood (such as getting a job and finding a partner) necessitate the adoption of prosocial behavior (Moffitt, 2006).

Many prospective longitudinal studies have identified a life-course persistent pattern of antisocial behavior as posited by Moffitt. Nagin and colleagues (Nagin et. al, 1995) discovered a "high-level chronic" group of individuals who exhibited elevated antisocial behavior in comparison to peers which comprised 12% of London males followed from ages 10 to 32 years. Raine et. al (2005) labeled a "life-course persistent" path which characterized 13% of Pittsburgh youth followed from ages 7 to 17 years. Other longitudinal investigations detected similar patterns of stable, chronic levels of antisocial behavior which characterized anywhere between 7% and 16% of sampled participants (Broidy, Broidy, Nagin, Tremblay, Brame, Dodge, et. al,

2003; Brame, Nagin, & Tremblay, 2001; Raine, Moffitt, Caspi, Loeber, Stouthamer-Lober, & Lyman, 2005; Chung, Hill, Hawkins, Gilchrist, & Nagin 2002; Capaldi et al, 2003; Moffitt, 2006). Moreover, several investigations have supported Moffitt's assertion that, in addition to neurophysiological and genetic dispositions, family environments characterized by high family conflict are associated with the development of life-course-persistent, but not adolescent-limited, antisocial behavior (Moffitt, 2006; Brennan, Hall, Bor, Najman, & Williams, 2003; Moffitt & Caspi, 2001).

However, studies testing antisocial behavior as a mediator of cross-generational consistency in harsh parenting do not consider this important distinction in Moffitt's developmental taxonomy of antisocial behavior. Some studies use a single time point indicator of antisocial behavior that is unable to consider course of antisocial behavior. Other studies using repeated assessments of antisocial behavior aggregated across multiple reports in adolescence to form a single measure of antisocial behavior. This aggregation combines antisocial behavior perpetrated by both the life-course-persistent and adolescent-limited proportions of the sample. Studies that use this aggregation approach with either two (e.g., Bailey et. al, 2009; Conger et. al, 2003) or three (e.g., Kerr et al, 2009) time points in adolescence did not support the mediation hypothesis. Thus, the mixing of life-course persistent and adolescent-limited proportions of the sample in these investigations could have led to these non-significant results.

To address this issue, the current study uses repeated measures of antisocial behavior (one in adolescence and two in young adulthood) to separate life course persistent and adolescent limited patterns of antisocial behavior and uses that information to understand how patterns of antisocial behavior mediate intergenerational continuities in family environments. Thus, *the second objective of the present investigation is to determine if repeated elevation in*

antisocial behavior over time mediates continuity in high conflict family environments seen across generations.

The Influence of Partners

One challenge of three-generation studies is the lack of G1-G2 family history information on an important member of the G2-G3 family environment, namely G2-partners. G2-partners are important for two reasons. First, just as high, stable patterns of antisocial behavior may create continuities in high conflict family environments over time in target G2s, the same process may occur for G2-partners. As a result, G2-G3 high-conflict family environments may represent continuity over time from both the G1-G2 and G1-G2-partner family histories. According to SIT, if G2-partners also come from high conflict family environments then G2 partners are also at risk for developing patterns of antisocial behavior which endure into adulthood.

These patterns of antisocial behavior are the second reason it is important to consider G2-partners in conceptualizing how high conflict family environments show intergenerational continuity from one generation to the next. If a G2 with high levels of antisocial behavior finds a partner who exhibits a similarly high level of antisocial behavior, then the coercive interactions among the two partners are likely to be volatile and damaging, with negative effects for children and the family environment (Humbad, Donellan, Iacono & Burt, 2010). As a result, the same patterns of high family conflict that antisocial partners grow up with will be perpetuated in their new family environments (Patterson, 1998; Rutter, 1998; Capaldi et. al, 2003). Therefore, G2's selection of a partner high in antisocial behavior may be an additional mechanism that underlies continuities in high conflict family environments over time (Conger et. al, 2012; Capaldi et. al, 2008; Patterson, 1998; Rutter, 1998).

Indeed, antisocial individuals are more likely than their peers to select antisocial partners, a phenomenon known as "assortative mating" (Burt & Klump, 2012; Rhule-Louie & McMahon, 2007; Krueger, Moffitt, Caspi, Bleskey, & Silva, 1998). Assortative mating describes findings showing that individuals choose partners whose attributes and behaviors are similar to their own (Rhule-Louie & McMahon, 2007). Several studies have found correlations for partner similarity in antisocial behavior in young adulthood to exceed .4 (Capaldi & Crosby, 1997; Kim and Capaldi, 2004). Other work has also shown that, in young adulthood, one partner's antisocial behavior contributes to continuity in another partner's antisocial behavior (Moffitt & Caspi, 2001).

To the extent that G2-partners show elevated antisocial behavior, G2's own antisocial behavior is expected to predict higher conflict in G2-G3 family environments, and due to assortative mating this may be a common occurrence for G2s with high levels of antisocial behavior. This hypothesis has never been directly tested in the literature, though two studies from the harsh parenting literature provide indirect support for it. Capaldi et al. (2008) showed that G2 mothers' history of antisocial behavior was predictive of G2 fathers' harsh discipline toward their children. Though G2 father's history of antisocial behavior was also measured in the study, no direct test of the interaction of G2 father's and G2 mother's antisocial behavior on G2 father harsh parenting was conducted. Additionally, Conger et al. (2012) found that G2 parents with a history of harsh parenting were likely to select partners who demonstrated poor relationships with children. Yet, the same study found that if a parent with a history of harsh parenting was able to marry a spouse who demonstrated a warm, supportive parenting style, then intergenerational continuity in harsh parenting was broken (Conger et. al, 2012). However, the study did not directly measure G2 parents' antisocial behaviors. Therefore, despite their

promising results, these studies do not directly test the moderating effects of G2-partner antisocial behavior on the relation between G2's antisocial behavior and G2-G3 family environment and they still focus only on a single parent behavior (harsh parenting), rather than the broader family environment. *Thus, the third objective of the present investigation is to investigate whether G2-partners' antisocial behavior moderates the relationship between G2 targets' antisocial behavior and G2-G3 high conflict family environments.*

The influence of G2 Gender

Whether continuities in family environment across generations are more likely to occur for women or men is still unclear (Conger et. al, 2009). In one longitudinal study, intergenerational transmission of warm, sensitive parenting, from G1-G2 to G2-G3 homes occurred for G2 mothers but not for G2 fathers (Belsky, Jaffee, Silgo, Woodward, & Silva, 2005). In another longitudinal investigation, continuities in harsh parenting in the G1-G2 and G2-G3 families were stronger for women and non-significant for men (Thornberry et. al, 2003). However, these studies have relied on maternal reports of parenting behavior, which may account for stronger effects for women (Belsky et. al, 2005) and no studies have addressed this question with a focus on the broader family environment. Other studies have found no moderating effect for G2 gender on continuities in harsh parenting (Neppl et. al, 2009) or parenting quality (Shaffer, Burt, Obradovic, Herbers, & Masten, 2009). However, some prospective longitudinal investigations that have investigated intergenerational continuities in parenting behaviors have only examined male samples (e.g., Smith & Farrington, 2004; Capaldi et. al, 2003; Capaldi et. al, 2008). The current study adds to existing literature on the subject by examining whether G2 gender moderates continuities in family environment across generations in a prospective, longitudinal sample that includes both men and women. Therefore, *the fourth*

objective of the current study is to examine whether G2 gender is a moderator of the intergenerational transmission of high conflict family environments.

The Current Study

The current study builds on existing literature examining intergenerational continuities in high conflict family environments in several ways. First, it is the first prospective longitudinal study that goes beyond considering intergenerational continuities in parenting practices to examine intergenerational continuities in the larger high conflict family environments within which such parenting practices are embedded. To achieve this, multiple reporters of family environment are considered. Second, the present study permits control for temporal precedence of mediators and outcomes. Third, it considers multiple time points to investigate the mediating role of antisocial behavior in the intergenerational transmission of conflict in family environments. Fourth, it examines the role G2-partner antisocial behavior plays in moderating the relationship between G2 antisocial behavior and G2-G3 high conflict family environment. Fifth, it utilizes a high-risk data set where patterns of high antisocial behavior in G2s and G2-partners may be more prevalent. Sixth, it examines the moderating role G2 gender plays in the transmission of high conflict family environments across generations. The present study tests four specific hypotheses which align with these unique contributions and are depicted in an overarching conceptual model in Figure 1.

Hypothesis 1: High conflict family environments will show moderate but significant levels of continuity across generations.

Hypothesis 2: A life-course-persistent pattern of antisocial behavior, characterized by high, stable levels of antisocial behavior measured across one time point in adolescence and two

time points in adulthood, will mediate intergenerational continuities in high conflict family environments.

Hypothesis 3: Antisocial behavior in a G2 parenting partner will moderate the relationship between G2 antisocial behavior and conflict in the G2-G3 family environment. Higher levels of antisocial behavior in G2 partners will be associated with a stronger association between G2's antisocial behavior and high conflict family environments.

Hypothesis 4: Moderating effects of G2 gender on the relation between G1-G2 and G2-G3 family environment will be explored.

In testing these hypotheses, several important statistical controls were considered based on previous literature which suggests that other mediating mechanisms may also explain continuity in parenting behaviors across generations. For instance, several studies have found that intergenerational continuities in socioeconomic status at least partially account for intergenerational continuities in harsh parenting behaviors (Scaramella, Neppl, Ontai, & Conger, 2008; Simons, Whitbeck, Conger, & Wu, 1991), though other results indicate that intergenerational continuities in parenting behavior are still seen after controlling for socioeconomic status (Conger et. al, 2009; Bailey et. al, 2009; Neppl et. al, 2009; Kovan, Chung, & Sroufe, 2009; Shaffer et. al, 2009). In addition, patterns of antisocial behavior that also show intergenerational continuity (Bailey et. al, 2009; Smith & Farrington, 2004; Thornberry et. al, 2003) may actually drive these findings. For example, similar styles of harsh parenting seen across generation could be a result of underlying genetic predispositions to antisocial behavior which are passed from one generation to the next and manifested as both antisocial behavior outside of the family context and within the family context (as contributors to a high conflict

family environment). To partially account for these alternative explanations in the current study, socioeconomic status and parental (G1) antisocial behavior were controlled for in all analyses.

METHODS

Data from the Adolescent & Family Development Project (AFDP; Chassin, Lee, Cho, Wang, Agrawal, Sher, & Lynskey, 2012; Chassin, Rogosch, & Barrera, 1991; Chassin, Pitts, Delucia, & Todd, 1998) were used for this study. AFDP is an ongoing longitudinal study of children of alcoholic parents (COAs) and matched controls assessed from adolescence into adulthood. AFDP is 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 for wave 4 (where data were collected on G1s and G2s), as well as wave 5 and wave 6 (where data were collected on G2s, G2 partners, and G3s).

Participants

At wave 1, the AFDP sample consists of 246 adolescents with at least one alcoholic parent and 208 matched adolescents with no biological or custodial alcoholic parent (Chassin et al., 1998) 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., 1991; Chassin et. al, 1998). COA families had to meet the following criteria: parents reported being either Hispanic or non-Hispanic Caucasian, Arizona residency, a child aged 10.5-15.5 years at wave 1, English-speaking, and 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.

Matched controls were recruited using telephone interviews. 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 (single versus two-parent household), target child's age and gender, and socioeconomic status. 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 the original sample).

To be included in the current study, G2's needed to have at least one child by wave 6 and needed to have completed the family conflict subscale of Bloom's Family Processes Scale at wave 2 and wave 6 (Bloom, 1985). Of the 409 G2 participants assessed at wave 6, 273 had children. Of these 273 G2s, 246 completed the family conflict subscale, while 27 G2s did not complete the family conflict subscale because they contacted their child less than once a week. These G2s were subsequently dropped from the analysis sample, though they did not significantly differ from retained G2s on levels of mother-reported G1-G2 family conflict ($t(257) = 1.75, p = 0.08$), father-reported G1-G2 family conflict ($t(209) = 1.14, p = 0.25$) or antisocial behavior at wave 3 ($t(267) = 1.50, p = 0.13$), wave 4 ($t(244) = 0.64, p = 0.52$), or wave 5 ($t(250) = -0.24, p = 0.81$). However, these groups did differ on G2 adolescent reported G1-G2 family conflict ($t(268) = 2.08, p = 0.04$) such that G2s who contacted their G3 child less than once a week reported higher levels of G1-G2 conflict. Missing data among the remaining 246 G2-G3 families was addressed using full information maximum likelihood procedures (see results) such

that all 246 G2-G3 families were retained in analyses of hypotheses 1, 2 and 4. These G2-G3 families had G2s that were 57% female, 53% COAs, 71% Caucasian, 26% Hispanic, and 3% other, ranging in age from 11-17 at wave 2 ($M=14.3$, $SD=1.41$) to 27-36 at wave 6 ($M=31.8$, $SD=1.76$). G2 partners in these 246 G2-G3 families were 43% female, 61% Caucasian, 33% Hispanic, and 6% Other, ranging in age from 27-40 at wave 6 ($M=33.2$, $SD=2.78$). G3 children in these G2-G3 families were 47% female, 51% Caucasian, 33% Hispanic, and 12% other, ranging in age from 7 to 17, ($M=12.14$, $SD=2.39$). The highest education level obtained by anyone in these G2-G3 families included 30% of families in which a family member earned a GED or high school degree, 31% in which a family member completed some college, and 32% in which a family member completed an associate's degree, bachelor's degree, or entered graduate training. G2-G3 families ranged in size from 1 to 4 children ($M=1.75$ children). Indicators of family environment were based on G2 reports but also on available G2 partner and G3 reporters who were present at the time of the G2 interview.

Of these 246 G2's, a subsample of 102 G2s whose parenting partners also provided self-reports of antisocial behavior and family conflict at wave 6 was used to evaluate hypothesis 3. G2s in this subsample were 63% female, 55% COAs, 63% Caucasian, 34% Hispanic, and 3% other, ranging in age from 27-36 at wave 6 ($M=32.5$, $SD=1.70$), and G2 partners in this subsample were 43% female, 61% Caucasian, 33% Hispanic, and 6% Other, ranging in age from 27-40 ($M=33.2$, $SD=2.78$). This subsample did not significantly differ from the full sample of 246 G2-G3 families on any characteristic except G2 age at wave 6 ($t(244) = -5.48$, $p < .01$). G2s in this subsample were significantly older than G2s in the full sample.

Procedure

At each wave, data were primarily collected via in-person computer-assisted interviews (Chassin et. al, 2012). Family members were typically interviewed simultaneously and in separate rooms to avoid contamination and to increase privacy. In wave 6 of data collection, only G2 parents were required to complete computer-assisted interviews. However, other members of the G2-G3 family were also invited to complete computer-assisted interviews if they were available. When a family moved out of state, an interviewer from a nearby university administered a shortened version of the battery; the shortened version of the battery was completed via telephone if no nearby interviewer was available. Mail-in surveys augmenting the primary battery were added in later waves. Interviews typically lasted from 1 to 3 hours and participants were paid up to \$70 per wave. Confidentiality was reinforced with a Department of Health and Human Services Certificate of Confidentiality.

Measures

Demographic variables. At wave 6, G2's and their partners reported their gender, ethnicity, and highest education level obtained, with the 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 (indicated by G2 and G2 partner reports of educational attainment at wave 6). Other studies using the AFDP data set have accounted for socioeconomic status by controlling for education level in similar ways (Hussong, Huange, Serrano, Curran, & Chassin, 2012; Chassin et. al, 2004).

G1 antisocial behavior & alcoholism. G1 mother and G1 father antisocial behavior and alcoholism were measured via self-reported lifetime *DSM-III* diagnoses of antisocial personality

disorder 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, & Seyefried, 1982). Although all reports of antisocial personality disorder were based solely on the direct reports of mothers and fathers, alcoholism diagnoses were based on direct 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 met lifetime criteria) or absent (neither G1 parent met lifetime criteria).

G2 and G2-partner antisocial behavior. G2 antisocial behavior was measured at waves 3, 4, and 5 using 12 self-report items from the antisocial scale of The Achenbach Childhood Behavior Checklist (CBCL; Achenbach & Edelbrock, 1981). G2-partners completed 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. For each G2 at each wave of data collection, a summary score for antisocial behavior was created by calculating the mean of the 12 items, with higher scores indicating higher levels of antisocial behavior. Internal reliability at Waves 3, 4, and 5 for G2 self-reported antisocial behavior ranged from $\alpha=.65$ to $\alpha=.82$. Internal reliability at Wave 6 for G2 partners' self-reports was $\alpha=.80$.

Conflict in Family Environment: 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. Examples of items include, "We fought a lot in our family" 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 Subscale at Wave 2 on G1-G2 families. In wave 6, G2s, G2 partners, and G3 children (Aged 7-17) also completed the family conflict subscale in reference to G2-G3 families. Items were reverse scored so that higher scores indicated higher levels of family conflict. In the present study, internal reliability estimates were as follows: wave 2 for 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 ($\alpha=.65$).

RESULTS

Zero-order correlations between key observed variables included in the study model were calculated (Table 1). The analysis sample consists of 246 target G2s, however there is modest to moderate missingness on key variables. Table 2 provides descriptions of missingness and reasons for missingness for each variable. Notably, G2s who were missing on one or more study variables did not significantly differ from G2s who had no missing data on levels of G2-G3 family conflict ($t(244) = -1.21, p = 0.23$), G1-G2 family conflict ($t(244) = -1.06, p = 0.29$), or levels of antisocial behavior at wave 3 ($t(241) = -1.22, p = 0.22$), wave 4 ($t(222) = -1.83, p = 0.07$), or wave 5 ($t(227) = -1.48, p = 0.14$). Full information-maximum likelihood procedures were used in Mplus to account for missing data following Kline (2005).

Measurement Models

A measure of conflict within the family environment was created following several steps. First, family members' responses to the family conflict scale were averaged at the item level for both G1-G2 and G2-G3 families. For instance, in the G1-G2 family, mother, father, and G2 adolescent responses to item 1 of the family conflict subscale ("we fight a lot in our family") were averaged together to produce one score for item1 for the whole G1-G2 family. G2-G3 family-wide item scores were calculated using the same method. In G2-G3 families with multiple G3 children, all G3 scores were included in the calculation of item-level averages. Family conflict scores were averaged across reporters in this manner in order to equally weight each reporter's perception of conflict within a family. 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. An alternative approach drawing on the multi-trait (i.e., multi-item), multi-method (i.e., multi-reporter) literature was also examined but deemed analytically too demanding within the current data structure.

A confirmatory factor analysis implemented in Mplus Version 5.2 (Muthen & Muthen, 2007) was used to estimate a latent variable representing underlying conflict in the family environment using techniques developed by Bollen and Bauldy (2011). This underlying latent variable was estimated with maximum likelihood separately in both G1-G2 and G2-G3 families using the six 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 which 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).

First, the measurement model estimating G1-G2 family conflict was fit. Initial model fit was not acceptable ($\chi^2(5) = 26.84$, $p < .01$, CFI = 0.93, TLI = 0.86, RMSEA = 0.13, SRMR = 0.05). Two item correlations were added to the model based on modification indices (item 3 and 7 which both involved acts of physical aggression and items 5 and 9 which were both reverse scored). A χ^2 difference test revealed that the addition of these two inter-item correlations significantly improved model fit ($\chi^2(2) = 22.73$, $p < .05$). Resulting fit indices showed that the revised model fit the data well ($\chi^2(3) = 4.11$, $p = 0.25$, CFI = 0.99, TLI = 0.988, RMSEA = 0.04,

SRMR = 0.02), indicating that the model was appropriate to estimate a latent variable for G1-G2 family conflict. This model was retained in subsequent analyses.

Next, the measurement model estimating G2-G3 family conflict was fit. Initial model fit was not acceptable ($\chi^2(5) = 38.78$, $p < .01$, CFI = 0.89, TLI = 0.77, RMSEA = 0.17, SRMR = 0.06). Once again, inter-item correlations between items 3 and 7, and between items 5 and 9, were added to the model. A χ^2 difference test revealed that the addition of these two inter-item correlations significantly improved model fit ($\chi^2(2) = 36.53$, $p < .05$). Fit indices showed that that 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. This model was retained in subsequent analyses.

Hypothesis 1: A path analysis estimated in Mplus Version 5.2 was used to test the hypothesis that high conflict family environments show moderate but significant levels of continuity across generations. To test this model, the latent G2-G3 family conflict variable was regressed on the latent G1-G2 family conflict variable along with the following covariates: G1 parent antisocial behavior, G2 educational attainment, G2 ethnicity, G2 COA status, G2 gender, and G2 age at wave 2. To control for across-time inter-item correlations in the family conflict measurement models all identical items were correlated over time (i.e., item 1 in the G1-G2 family was correlated with item 1 in the G2-G3 family). The resulting structural path between G1-G2 family conflict and G2-G3 family conflict tested for continuity in family conflict over time while accounting for over time consistency in item response. A χ^2 difference test revealed that the addition of these inter-item correlations significantly improved model fit as compared to a model in which they were omitted ($\chi^2(5) = 17.36$, $p < .05$).

The resulting model fit the data well ($\chi^2(73) = 98.27, p=.03, CFI = .96, TLI = .95, RMSEA = 0.04, SRMR = 0.04$) and explained a significant amount of variance in G2-G3 family conflict ($R^2 = 0.17, p = .002$; see Table 3). Two covariates significantly predicted greater G2-G3 family conflict after controlling for levels of G1-G2 family conflict; namely, G2 age (standardized $\beta = 0.14, p = .04$) and G2 race (standardized $\beta = 0.16, p = .05$), indicating that older G2's at wave 2 and Hispanic as compared to non-Hispanic Caucasian G2's had higher levels of G2-G3 family conflict. Moreover, the direct path from G1-G2 family conflict to G2-G3 family conflict was significant even after controlling for covariates (standardized $\beta = 0.25, p<.01$). This result supports hypothesis 1 and shows that high levels of family conflict in the G1-G2 family predict high levels of family conflict in the G2-G3 family.

Hypothesis 2: Structural equation modeling was used to test the hypothesis that three repeated indicators of antisocial behavior (G2 self-reported antisocial behavior at waves 3, 4, and 5) mediate the relationship between G1-G2 and G2-G3 high conflict family environment. To test this model, these three mediating variables were added to the model testing Hypothesis 1. Covariates in this model predicted both G2-G3 family conflict and G2 antisocial behavior at wave 3 and included G1 parent antisocial behavior, G2 educational attainment, G2 ethnicity, G2 COA status, G2 gender, and G2 age at wave 2. Additionally, G2-G3 family conflict was regressed directly on G2 antisocial behavior at wave 5 and the auto-regressive parameters among the G2 antisocial behavior variables were estimated.

This model did not fit the data very well, ($\chi^2(117) = 194.85, p<.01, CFI = .91, TLI = .88, RMSEA = 0.06, SRMR = 0.07$). Consequently, a new model was fit to explore the unique mediational effects of G2 antisocial behavior at each of waves 3, 4, and 5. To create this model, structural paths were added so that G1-G2 family conflict predicted G2 antisocial behavior at

waves 3, 4, and 5 (as opposed to just wave 3). Additionally, G2-G3 family conflict was regressed directly on G2 antisocial behavior at waves 3, 4, and 5 (as opposed to just wave 5).

This new model fit the data much better, ($\chi^2(113) = 164.04, p < .01, CFI = .94, TLI = .92, RMSEA = 0.04, SRMR = 0.05$). Additionally, the χ^2 difference revealed that this new model fit the data significantly better than the first model, ($\chi^2(4) = 30.81, p < .01$), so this model was retained for further analysis. The model explained a significant amount of variance in G2-G3 family conflict ($R^2 = 0.23, p < .01$), and explained an additional 5.6% of the variance in G2-G3 conflict beyond G1-G2 family conflict and covariates alone. No covariates were significant predictors of G2-G3 family conflict. Figure 3 depicts key model results, and parameter estimates produced by this model can be found in Table 4.

The total effect of G1-G2 family conflict on G2-G3 family conflict continued to be significant (standardized $\beta = 0.27, p < .01$) and total indirect effects of G1-G2 family conflict on G2-G3 family conflict were also significant (standardized $\beta = 0.14, p < .02$). Decomposition of specific indirect effects showed that only the indirect effect of G1-G2 family conflict on G2-G3 family conflict through G2's antisocial behavior at wave 3 (when G2's were in adolescence) was significant (standardized $\beta = 0.08, p = .02$); non-significant effects were found for the mediator at wave 4 (standardized $\beta = 0.03, p = 0.24$) and wave 5 (standardized $\beta = 0.02, p = 0.44$). Moreover, the specific path from G1-G2 family conflict to G2-G3 family conflict was no longer significant (standardized $\beta = 0.13, p = 0.25$), indicating that the effect of G1-G2 family conflict on G2-G3 family conflict was fully mediated.

Given the specificity of these mediational results to the wave 3 antisocial behavior indicator, a series of sensitivity analyses were conducted to determine the relative contributions of developmental timing to this mediational process. The first sensitivity analysis tested whether

G2 antisocial behavior at wave 5 (in young adulthood) mediated the relationship between G1-G2 and G2-G3 high conflict family environment when the high stability in antisocial behavior (modeled through including wave 3 and 4 antisocial mediators) was omitted from the model. The same model depicted in figure 3 to test hypothesis two was re-estimated with the omission of waves 3 and 4 G2 antisocial behavior. The resulting model fit the data well ($\chi^2(84) = 110.94$, $p < .01$, CFI = .96, TLI = .94, RMSEA = 0.04, SRMR = 0.04). However, no indirect effect of G1-G2 family conflict on G2-G3 family conflict through G2 antisocial behavior at wave 5 was found (standardized $\beta = 0.03$, $p = .16$) and G2 antisocial behavior at wave 5 was only a marginally significant predictor of family conflict after controlling for covariates (standardized $\beta = 0.13$, $p = .10$). This model indicated that measures of antisocial behavior in adulthood alone are not sufficient to capture this mediational process.

A second sensitivity analysis tested whether antisocial behavior beginning in early adulthood (G2 antisocial behavior at waves 4 and 5) mediated the relationship between G1-G2 and G2-G3 high conflict family environment. Wave 4 G2 antisocial behavior was added to the model used for the first sensitivity analysis. The resulting model fit the data well, ($\chi^2(98) = 129.90$, $p = .02$, CFI = .96, TLI = .94, RMSEA = 0.04, SRMR = 0.04). Total (standardized $\beta = 0.25$, $p < .01$) and indirect effects of G1-G2 family conflict on G2-G3 family conflict (standardized $\beta = 0.06$, $p = 0.04$) were significant. Though the total indirect effect was significant, neither the specific indirect effect of G2 antisocial behavior at Wave 4 (standardized $\beta = 0.04$, $p = 0.10$) nor the specific indirect effect of G2 antisocial behavior at Wave 5 (standardized $\beta = 0.01$, $p = 0.34$) was significant in the model. Moreover, the specific path from G1-G2 family conflict to G2-G3 family conflict was marginally significant (standardized $\beta = 0.19$, $p = 0.07$), indicating that the effect of G1-G2 family conflict on G2-G3 family conflict was

partially mediated. These results indicate that G2 antisocial behavior measured at waves 4 and 5 did together partially mediate the relationship between G1-G2 and G2-G3 family conflict. However, the total indirect effects accounted for by this pathway (standardized $\beta = 0.06$) were smaller than those that included G2 antisocial behavior at wave 3 in the model.

A final sensitivity analysis was performed to determine if G2 antisocial behavior in adolescence (at wave 3) solely mediated the relationship between G1-G2 and G2-G3 high conflict family environment. The same mediational model was estimated with only G2 antisocial behavior at wave 3 as a mediator. The model fit the data well, ($\chi^2(84) = 120.17, p < .01, CFI = .95, TLI = .93, RMSEA = 0.04, SRMR = 0.04$). Total effects of G1-G2 family conflict on G2-G3 family conflict (standardized $\beta = 0.25, p < .01$), and indirect effects of G1-G2 family conflict on G2-G3 family conflict via G2 antisocial behavior in adolescence (standardized $\beta = 0.10, p < .01$) were significant. Moreover, the specific path from G1-G2 family conflict to G2-G3 family conflict was no longer significant (standardized $\beta = 0.16, p = 0.13$) after accounting for G2 antisocial behavior at wave 3, indicating that the effect of G1-G2 family conflict on G2-G3 family conflict was fully mediated.

Taken together, these results suggest that the relationship between G1-G2 and G2-G3 family conflict is primarily mediated by elevated rates of antisocial behavior in G2s evident in early adolescence and that patterns of G2 antisocial behavior that persist into adulthood add only marginally to this prediction.

Hypothesis 3: A SEM model with latent variables was used to test whether G2 partner antisocial behavior (measured at wave 6) moderated the relation between G2 antisocial behavior in adulthood (measured at wave 5) and G2-G3 high conflict family environment. Covariates predicting G2-G3 family conflict included G1 parent antisocial behavior, G2 educational

attainment, G2 ethnicity, G2 partner ethnicity, G2 COA status, G2 gender, and G2 age at wave 2. Predictor variables included the latent variable for G1-G2 family conflict, G2 partner's antisocial behavior at wave 6, G2's antisocial behavior at wave 5 and the interaction between these two antisocial variables. All continuous manifest predictor variables and covariates were centered at their mean, and an interaction term was created by multiplying G2 antisocial behavior at wave 5 by G2 partner antisocial behavior at wave 6.

The resulting model fit the data adequately, ($\chi^2(105) = 142.25, p < .01, CFI = .95, TLI = .93, RMSEA = 0.04, SRMR = 0.04$), and explained a significant amount of variance in G2-G3 family conflict ($R^2 = 0.47, p < .01$). As depicted in Table 5, G1-G2 family-conflict (standardized $\beta = .30, p < .04$), G2 partner antisocial behavior at wave 6 (standardized $\beta = 0.54, p < .01$), G1 wave 1 antisocial behavior (standardized $\beta = -0.21, p < .01$), and G2 age at wave 2 (standardized $\beta = 0.15, p < .01$) each significantly predict G2-G3 family conflict. A significant interaction between G2 antisocial behavior at wave 5 and G2 partner antisocial behavior at wave 6 was found (standardized $\beta = -0.39, p < .01$). Probing of the simple slopes for this interaction followed procedures outlined by Aiken and West (1991). The model was re-estimated to obtain parameters depicting the association between G2's antisocial behavior and G2-G3 family conflict at 1 standard deviation below mean levels of G2 partners' antisocial behavior, at mean levels of G2 partners' antisocial behavior, and at 1 standard deviation above mean levels of G2 partners' antisocial behavior (see Figure 4). The pattern of findings show that G2s' antisocial behavior predicted higher levels of G2-G3 family conflict at low ($\beta = 0.96, p < .01$) but not high ($\beta = -0.26, p = 0.32$) or moderate ($\beta = 0.35, p = 0.10$) levels of G2 partners' antisocial behavior. In other words, the unique impact of G2 antisocial behavior on G2-G3 family conflict was only evident when levels of G2 partner antisocial behavior were low. However, the highest levels of

G2-G3 family conflict were associated with high levels of G2 and G2 partner antisocial behavior.

Hypothesis 4: Multiple group analysis in a SEM framework was used to explore the moderating effects of G2 gender on the relationship between G1-G2 and G2-G3 family conflict. The same model estimated for hypothesis 1 was re-estimated constraining the structural path between G1-G2 and G2-G3 family conflict to be equal for men and women but allowing all other parameters to freely vary over gender and then again estimated allowing this path to be free. The χ^2 difference test revealed that the multiple group model in which the effect of G1-G2 family conflict on G2-G3 was freely estimated across both male and female groups fit the data significantly better than the multiple group model in which the effect of G1-G2 family conflict was constrained to be equal for men and women ($\chi^2(1) = 5.39, p < .05$). In the freed model, greater G1-G2 family conflict predicted greater G2-G3 family conflict in G2 women (standardized $\beta = 0.44, p < .01$) but not in G2 men (standardized $\beta = -0.04, p = .79$).

DISCUSSION

The current study examined whether family conflict is passed from one generation to the next and explored potential mediators and moderators which could explain this continuity. Results of the study showed that conflict in the current family was strongly correlated with that of the family of origin in women but not in men. Continuity in family conflict was also mediated by patterns of elevated G2 antisocial behavior and G2 antisocial behavior in adolescence played a key role in this mediational process. Additionally, analyses revealed that there is an interaction between G2 and G2 partner antisocial behavior such that even if just one partner in the G2-G3 family demonstrates high levels of antisocial behavior, elevated levels of family conflict in the G2-G3 family can result. Several design features of the study represent methodological strengths which provide confidence that study findings are valid. For instance, the present study utilized longitudinal data from a high-risk sample where patterns of high antisocial behavior and high family conflict may be more prevalent, utilized multiple reporters of family conflict in each generation, and surveyed G2 antisocial behavior at multiple time points. Additionally, temporal precedence was established such that measurement of G1-G2 family conflict preceded measurement of G2 antisocial behavior. Furthermore, the present study included both mothers and fathers in each generation, and considered the roles of G2s and their partners in the development of G2-G3 family conflict. The roles that G2 gender, G2 antisocial behavior, and G2 partner antisocial behavior play in the transmission of family conflict from one generation to the next as well as study limitations and future directions are considered below.

G2 Gender as a Moderator

The current study is one of the very first multi-generational longitudinal investigations to recruit and follow large numbers of both G2 mothers and G2 fathers and is uniquely designed and powered to explore the moderating effects of gender. As a result, the finding that family conflict persists across generations only in women is novel. Although no one has tested gender as a moderator of intergenerational family conflict, previous studies have found this effect in the literature examining intergenerational transmission of individual parenting behaviors (Belsky et al, 2005; Thornberry et. al, 2003). Although some studies found no gender differences in continuities in parenting behavior (Neppl et. al, 2009; Shaffer et. al, 2009), others found greater continuities in warm, sensitive, and stimulating parenting for G2 mothers but not fathers (Belsky et. al, 2005; Thornberry et. al, 2003). The gender effect found in these studies and in the current investigation may be the result of the differing social roles undertaken by men and women in American culture. Even in contemporary society, women are still the primary caretakers of children (Craig & Mullan, 2011), and are more likely than men to place greater importance on their family roles (Powell & Greenhaus, 2010). For this reason, women may define and create many more of the interaction patterns in families than men do. Consequently, family interaction patterns may look a lot more like interaction patterns from the mother's family of origin as opposed to the father's family of origin.

G2 Antisocial Behavior as a Mediator

Findings from the current investigation support G2 antisocial behavior as one mechanism by which family conflict is passed from one generation to the next. However, this mediational process appears to be developmentally sensitive. G2 antisocial behavior in adolescence is a stronger mediator of the relation between G1-G2 and G2-G3 family conflict than is G2 antisocial behavior in adulthood. The effect of G1-G2 family conflict on G2-G3 conflict accounted for by

G2 antisocial behavior in adolescence was twice as large as the effect accounted for by G2 antisocial behavior at either time point in adulthood.

G2 antisocial behavior in adolescence could play such a prominent role in the continuity of family conflict from one generation to the next because adolescence is a developmental period when the coercive interactional patterns that G2 children learn in high-conflict G1-G2 families can be successfully implemented and reinforced in a broader social context. Other investigations have found that adolescence is a unique time period when individuals who demonstrate high levels of antisocial behavior actually become more popular and more accepted among their peers (Moffitt, 2006; Cillessen & Mayeux, 2004; Rodkin, Farmer, Pearl, & Van Acker, 2000). Thus, in adolescence, coercive, antisocial interaction patterns can be used to successfully achieve social goals, and are reinforced as effective interactional strategies. In this way, patterns of coercive, antisocial behavior that are learned by G2s as adaptive strategies in high-conflict families in childhood are internalized as effective and normative patterns of social interaction in adolescence, and subsequently influence adult functioning.

The manner in which high levels of G2 adolescent antisocial behavior influence adult functioning varies across individuals (Dishion & Patterson, 2006; Moffitt, 2006). In many adolescents with high antisocial behavior in comparison to peers, these elevated levels of coercive, antisocial behavior continue to persist and be reinforced into adulthood (Dishion & Patterson, 2006; Moffitt, 2006) where they may create high conflict with G2 partners and in G2-G3 families. However, in up to 45% of G2 adolescents who display high levels of antisocial behavior in comparison to their peers, elevated levels of antisocial behavior in adolescence leads to increasing internalizing problems and social skill deficits in adulthood (Dishion & Patterson, 2006; Moffitt, 2006). This shift in symptomatology results when adolescents carry the antisocial

interactional patterns that are socially rewarding in adolescence into young adult environments where such behavior causes social rejection and isolation (Dishion & Patterson, 2006; Moffitt, 2006). For these adult G2s, conflict in G2-G3 families could arise from their social skill deficits and internalizing symptomatology in adulthood as opposed to antisocial behavioral patterns in adolescence. In prior investigations, about 15% of all adolescents seemed to traverse the antisocial pathway in which adolescent antisocial behavior leads to adult antisocial behavior (Rane et al., 2005; Broidy et. al, 2003; Brame et. al, 2001) whereas about 8% of all adolescents seem to traverse the second antisocial pathway, where adolescent antisocial behavior leads to adult internalizing behavior and social skill deficits (Dishion & Patterson, 2006; Moffitt, 2006, Moffitt, Caspi, Harrington & Milne, 2002). It is possible that in the present investigation, elevated levels of antisocial behavior in adolescence mediate intergenerational continuity in family conflict so strongly because both the 'externalizing' and 'internalizing' adult antisocial groups are captured by the adolescent measure of antisocial behavior and because both groups ultimately experience conflict in the families that they start in adulthood. However, elevated levels of antisocial behavior in adulthood do not have as strong a mediational effect on intergenerational continuity in family conflict because some adolescents who are high in externalizing behavior become adults who are high in internalizing behavior and social skill deficits and those G2s are ultimately not captured by measures of antisocial behavior in adulthood.

An alternative explanation for why G2 antisocial behaviors serves as a mediator between G1-G2 and G2-G3 family conflict could be that antisocial behavior passed from one generation to the next may actually be driving these findings. It is well known that genetic predispositions to antisocial behavior can be passed across generations (Bailey et. al, 2009; Smith & Farrington,

2004; Thornberry et. al, 2003) and it is possible that continuity in family conflict across generations is merely a by-product of repeated patterns of antisocial behavior that are passed from one generation to the next. The current investigation accounted somewhat for this possibility by controlling for G1 antisocial behavior in all analyses. Study results were found to be significant above and beyond G1 antisocial behavior and G1 antisocial behavior was not a significant predictor of G2-G3 family conflict in the mediational analysis, suggesting that an intergenerational pattern of antisocial behavior does not explain the whole process by which family conflict is passed from one generation to the next. However, genetic mechanisms of risk were not part of the present investigation, so the role of genetic effects in these findings cannot be discerned.

It is notable that the finding that G2 antisocial behavior in adolescence plays the most prominent mediating role in the transmission of family conflict from one generation to the next runs somewhat contrary to what has been found in other studies which have examined maladaptive family interaction patterns at the dyadic level. These studies have found that G2 antisocial behavior in adolescence is not a significant predictor of continuity in parenting behaviors across generation (Conger et. al, 2009; Neppl et. al, 2009; Conger et. al, 2003), though none of these studies simultaneously measured adolescent and adult antisocial behavior as potential mediators of continuity in parent behaviors across generation. Several different explanations could account for the difference between the present study findings and these prior results. First, many previous studies aggregated antisocial behavior across several time points in adolescence and young adulthood into one measure of antisocial behavior (Bailey et. al, 2009; Conger et. al, 2003) used to predict family conflict, whereas the present study measured antisocial behavior separately across multiple time points in adolescence and adulthood.

Consequently, the present investigation was the first to evaluate the unique mediating effects of G2 antisocial behavior at different time points in adolescence and young adulthood. In parsing these unique effects, the present study may have been able to more precisely measure the effects of adolescent antisocial behavior on family conflict than previous studies.

A second explanation for this difference in findings could arise from the fact that previous studies on transmission of parenting behaviors were focused on individual parent-child dyads, whereas the current investigation studied transmission of conflict in the family as a whole, across multiple dyads. Conflict within individual parent-adolescent dyads is a normative experience in families with adolescents (Ehrlich, Dykas, & Cassidy, 2012), whereas high levels of conflict across the entire family (across multiple dyads) is less normative in families with adolescents (Habib et. al, 2013; Emery, 1993). Thus, ultimately, both the dyadic parenting studies and the present investigation may be capturing the same population of adolescents who grow up in high conflict family environments, who exhibit high levels of antisocial behavior compared to their peers, and who experience high levels of G2-G3 family conflict in adulthood as a result. However, because dyadic parent-child conflict is much more prevalent in the lives of adolescents than family-level conflict, harsh parenting studies may also capture a large portion of adolescents who experience both parent-child conflict (a normative adolescent experience), and elevated levels of antisocial behavior (a normative adolescent experience), but who do not carry that behavior forward into adulthood. In contrast, in the present study because family conflict (a much less normative adolescent experience) is measured and used to predict adolescent antisocial behavior, those G2s who will carry their antisocial behavior forward to adulthood and experience higher levels of conflict in G2-G3 families are much more easily separated from "typical" adolescents and are able to be identified in adolescence.

Effect of the G2 Partner

Evidence from the current study indicates that elevated levels of G2 antisocial behavior uniquely predict elevated levels of G2-G3 family conflict only when G2 partners demonstrates low antisocial behavior. Since interactions are symmetrical, this also means that G2 partner antisocial behavior uniquely predicts elevated levels of G2-G3 family conflict only when G2 antisocial behavior is low. Together, these results suggest that even if only one parent in a G2-G3 family demonstrates elevated levels of antisocial behavior, elevated levels of conflict in the G2-G3 family conflict can result. This finding is consistent with previous work that has examined the relationship between parent antisocial behavior and family disruption within a single family generation. Studies have found that couples with at least one antisocial partner report more problematic marriages, less relational satisfaction, and less family cohesion (Bornovalova, Blazei, Malone, McGue, & Iacono, 2013), as well as greater likelihood for partner violence (Kim & Capaldi, 2004).

Additionally, present study findings demonstrate that the highest levels of family conflict are experienced in families where both partners possess elevated levels of antisocial behavior. Yet, the direction of the significant interaction indicated that high levels of antisocial behavior in one partner did not significantly predict high levels of family conflict in the presence of high levels of antisocial behavior in the other partner. One explanation for these apparently paradoxical findings is that high antisocial behavior in just one partner is so disruptive to the family environment that it accounts for much of the variance in family conflict scores. As a result, the addition of the second partner's antisocial behavior to the model adds little to the prediction of high family conflict scores that was not already being accounted for by the high antisocial behavior scores of the first partner.

Limitations

Though study findings present a new perspective on the processes by which family conflict can be passed from one generation to the next, several limitations of the current study should be noted. First, in the present study, item-level parent and child reports of family conflict were aggregated to estimate both G1-G2 and G2-G3 latent family conflict variables. As a result, each available family members' report was equally weighted in the estimation of latent family conflict. However, the range of correlations for reporter agreement on family conflict items demonstrated that each family member had a somewhat unique perspective on conflict within their family. It is possible that one family member's perception of family conflict may play a larger role in how conflict within a family is shaped, and thus each family members' perspective should not have been equally weighted in the calculation of family conflict. Additionally, G2 partner and G3 adolescent reports of family conflict were not available for all G3 families. Thus, some estimates of conflict in G2-G3 families incorporated less perspectives than others. Second, only one self-report questionnaire was used to measure family conflict throughout the study. However, previous investigations of continuity in parenting behaviors across generation find no significant difference in strength of parenting behaviors obtained by self-report versus observational measures (Conger et. al, 2009). Furthermore, concern over use of a single self-report questionnaire to measure family conflict is somewhat mitigated by the fact that the same questionnaire was used by multiple reporters to assess family conflict across generation. Finally, as in all studies which measure intergenerational parenting and family processes, the present investigation was only able to collect data on G1-G2 family conflict and adolescent externalizing behavior for one of the two parents in the G2-G3 family environment. As a result,

the extent to which the mediating and moderating processes implicated in the current study apply to the "other" G2 partner in the G2-G3 family is unknown.

Future Directions

In conclusion, despite its limitations the current study provides evidence for the intergenerational transmission of family conflict and implicates key moderators and mediators which facilitate such intergenerational continuity in family conflict. Future research should expand this exploration of intergenerational family conflict in several ways. First, continued examination of how gender roles moderate the transmission of family conflict is warranted. Social roles of G2s can be measured to determine why family conflict is passed across generations in G2 women rather than G2 men. For instance, future research could investigate whether taking on certain roles in the G2-G3 family (primary caretaker, primary family activity planner) determines which G2 transmits their G1-G2 family environment to their G2-G3 family.

Second, future studies should incorporate G1, G2, and G3 genetic data to investigate how family conflict is passed from one generation to the next. It is already well-established in extant literature that genetic factors influence antisocial behavior (Silberg et. al, 2012; Dionne et. al, 2003) and are exacerbated by maladaptive environments in one's family of origin (Moffitt, 2006; Moffitt, 1993). Understanding how gene-environment interactions inform the development and persistence of G1-G2 family conflict and G2 antisocial behavior could lead to a deeper understanding of how G2 antisocial behavior serves as a mediating mechanism in the process by which conflict is passed from G1-G2 to G2-G3 families.

Finally, a search for additional mediating and moderating mechanisms which may help explain how family conflict is passed from one generation to the next is warranted. Substance misuse could be one such mediating mechanism; high levels of G1-G2 family conflict could lead

G2s' to drink as a coping strategy in adolescence and continue their problematic drinking into adulthood, resulting in high levels of conflict in G2-G3 families. Additionally, the quality of the relationship between parents in the G2-G3 family could be a moderating mechanism; high levels of family conflict could be passed from G1-G2 to G2-G3 families only in G2-G3 families where parenting partners report poor, conflictual interaction patterns between one another.

In summary, this study represents a significant first step in investigating how conflict can be passed across generation in families. The current investigation reveals that family conflict is transmitted from one generation to the next for women but not men. G2 antisocial behavior, especially in adolescence, seems to mediate this transmission of family conflict, and it appears that it only takes one G2 parenting partner to be high in antisocial behavior for levels of G2-G3 family conflict to increase. Study findings suggest that in planning prevention and intervention programming to treat family conflict, levels of antisocial behavior and family histories of conflict for both G2 partners may need to be assessed and considered.

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Table 1: Correlation matrix of key predictor and outcome variables

	<i>% or</i>													
	<i>M (SD)</i>	<i>1</i>	<i>2</i>	<i>3</i>	<i>4</i>	<i>5</i>	<i>6</i>	<i>7</i>	<i>8</i>	<i>9</i>	<i>10</i>	<i>11</i>	<i>12</i>	
1. G1 Antisocial Behavior (% Diagnosed Antisocial)	6.5%	1.00												
2. G2 Age at Wave 2	14.33 (1.41)	0.04	1.00											
3. G2 Race (% Caucasian)	71.4%	0.05	0.13	1.00										
4. Parent Education in G2-G3 Family	7.05 (2.37)	-0.14*	0.09	-0.01	1.00									
5. COA (% COA)	53.2%	-0.25*	-0.06	0.21*	-0.15*	1.00								
6. G2 Gender (% female)	57 %	-0.03	-0.07	-0.07	-0.07	-0.07	1.00							
7. G1-G2 Family Conflict	0 (0.59)	0.18*	-0.09	0.19*	-0.19*	0.38*	-0.13*	1.00						
8. G2-G3 Family Conflict	0 (0.55)	0.01	0.10	0.21*	-0.12	0.22*	-0.02	0.34*	1.00					
9. G2 Antisocial Behavior at Wave 3	1.68 (0.49)	0.14*	0.04	0.07	-0.20*	0.25*	0.07	0.36*	0.32*	1.00				
10. G2 Antisocial Behavior at Wave 4	1.27 (0.30)	0.22*	-0.02	0.21*	-0.05	0.25*	0.01	0.31*	0.25*	0.40*	1.00			
11. G2 Antisocial Behavior at Wave 5	1.18 (0.24)	0.19*	-0.02	0.15*	-0.21*	0.23*	0.04	0.27*	0.21*	0.32*	0.39*	1.00		
12. G2 Partner Antisocial Behavior at Wave 6	1.27 (0.35)	-0.11	-0.04	0.02	-0.14	0.11	-0.17	0.09	0.33*	0.21*	0.09	0.12*	1.00	

* Denotes significant correlation at $p < .05$

Table 2: Missing data

Reporter	Percent of Missingness (of 246 families)		Reason for Missingness
	Family Conflict	Self-reported Antisocial Behavior	
G1 Mother	4.4% (11 cases)	--	G1 mother reports of family conflict were missing in some families because those families were single-parent families (n = 2), were missing in other families because the mother was not interviewed at the time of data collection (n = 6), and were missing in other families because the family did not participate in data collection at wave 2, but did participate in data collection at wave 1 and in subsequent waves (n = 3).
G1 Father	22.7% (56 cases)	--	G1 father reports of conflict were missing in some families because those families were single parent families (n = 26), were missing in other families because the father was not interviewed at the time of data collection (n = 27), and were missing in other families because the family did not participate in data collection at wave 2, but did participate in data collection at wave 1 and in subsequent waves (n = 3).
G2 Target Adolescent (reporting on antisocial behavior across waves)	0.81% (2 cases)	1.2% (3 Cases)- 8.9% (22 Cases)	Some G2 targets were unable to be located to report on antisocial behavior at wave 3 (n = 3), wave 4 (n = 22), and wave 5 (n = 17). However, no G2 targets were missing reports of antisocial behavior across all three waves of data collection.
G2 Target Adolescent (reporting on G1-G2 family environment)	1.1% (3 cases)	--	These three adolescents were members of families who did not participate in data collection at wave 2, but who did participate in data collection at wave 1 and in subsequent waves (n = 3).
G2 Target Adult (reporting on G2-G3 family environment)	0%	--	
G2 Partner	0%	0%	
At least one G3 report	0%	--	

Table 3: Hypothesis 1 path analysis predicting G2-G3 family conflict from G1-G2 family conflict and covariates

Predictors	β (SE)
G1-G2 Family Conflict	0.25 (.09)**
G1 Antisocial Behavior at Wave 1	-0.10 (0.07)
G2 Ethnicity	0.16 (0.08)*
G2 Educational Attainment at Wave 6	-0.07 (0.08)
COA	0.13 (0.08)
G2 Gender	0.04 (0.07)
G2 Age at Wave 2	0.14 (0.07)*
R²	0.17 (0.06)**

Note: * $p < .05$, ** $p < .01$, all coefficients are standardized estimates

Table 4: Hypothesis 2 path analysis predicting G2-G3 family conflict from G1-G2 family conflict and G2 antisocial behavior at waves 3, 4, and 5

Predictors	β (SE)
G1-G2 Family Conflict	0.13 (0.11)
G2 Antisocial Behavior at Wave 3	0.22 (0.08)**
G2 Antisocial Behavior at Wave 4	0.11 (0.08)
G2 Antisocial Behavior at Wave 5	0.07 (0.08)
R²	0.23 (0.06)**

Note: * $p < .05$, ** $p < .01$, all coefficients are standardized estimates

Table 5: Hypothesis 3 structural equation model predicting G2-G3 family conflict at mean levels of G2 partner antisocial behavior

Predictors	β (SE)
G1-G2 Family Conflict	0.30 (0.10)**
G2 Antisocial Behavior at wave 5	0.13 (0.08)
G2 Partner Antisocial Behavior at wave 6	0.54 (0.10)**
Interaction term	-0.39 (0.15)**
G1 Antisocial Behavior at wave 1	-0.22 (0.10)*
G2 Ethnicity	-0.01 (0.12)
G2 Partner Ethnicity	0.20 (0.13)
G2 Educational Attainment at Wave 6	-0.04 (0.08)
COA	0.05 (0.08)
G2 Gender	0.06 (0.07)
G2 Age at Wave 2	0.15 (0.07)*
R²	0.47 (0.10)**

Note: * $p < .05$, ** $p < .01$, all coefficients are standardized estimates

Figure 1: A model for intergenerational continuity in high conflict family environment

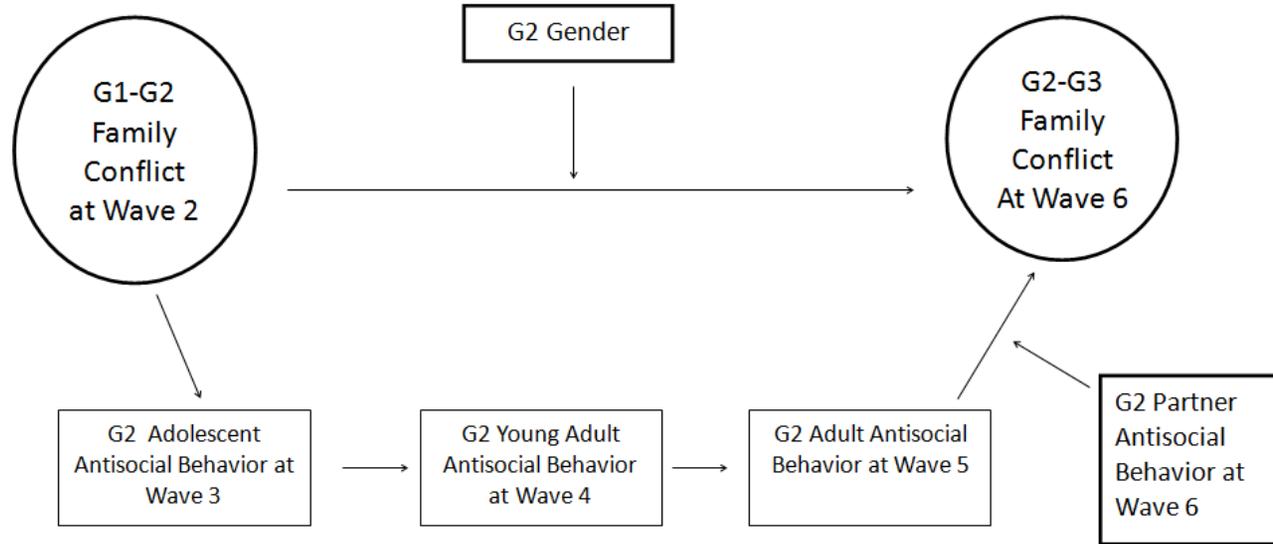


Figure 2: Family conflict measurement models

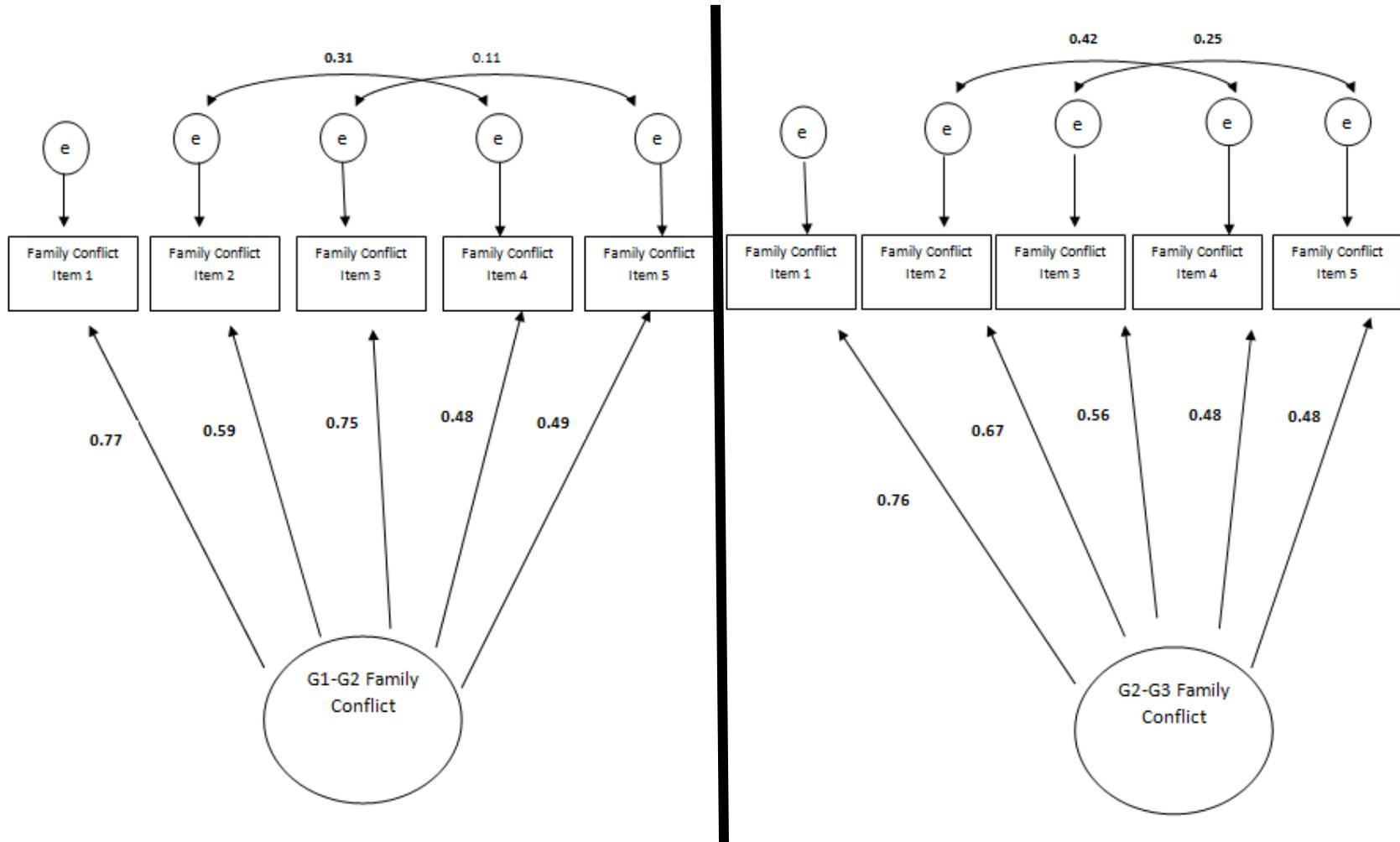
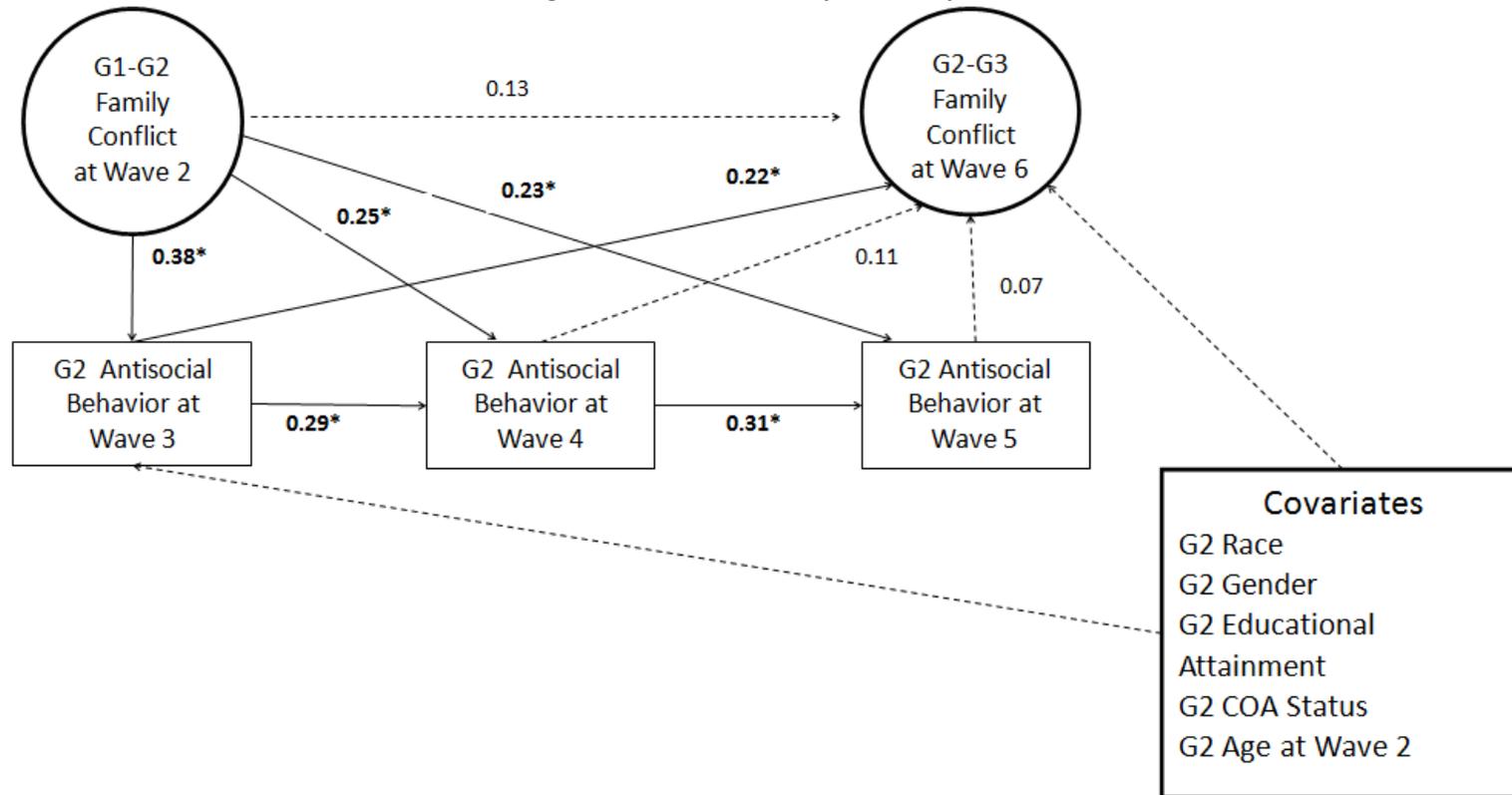
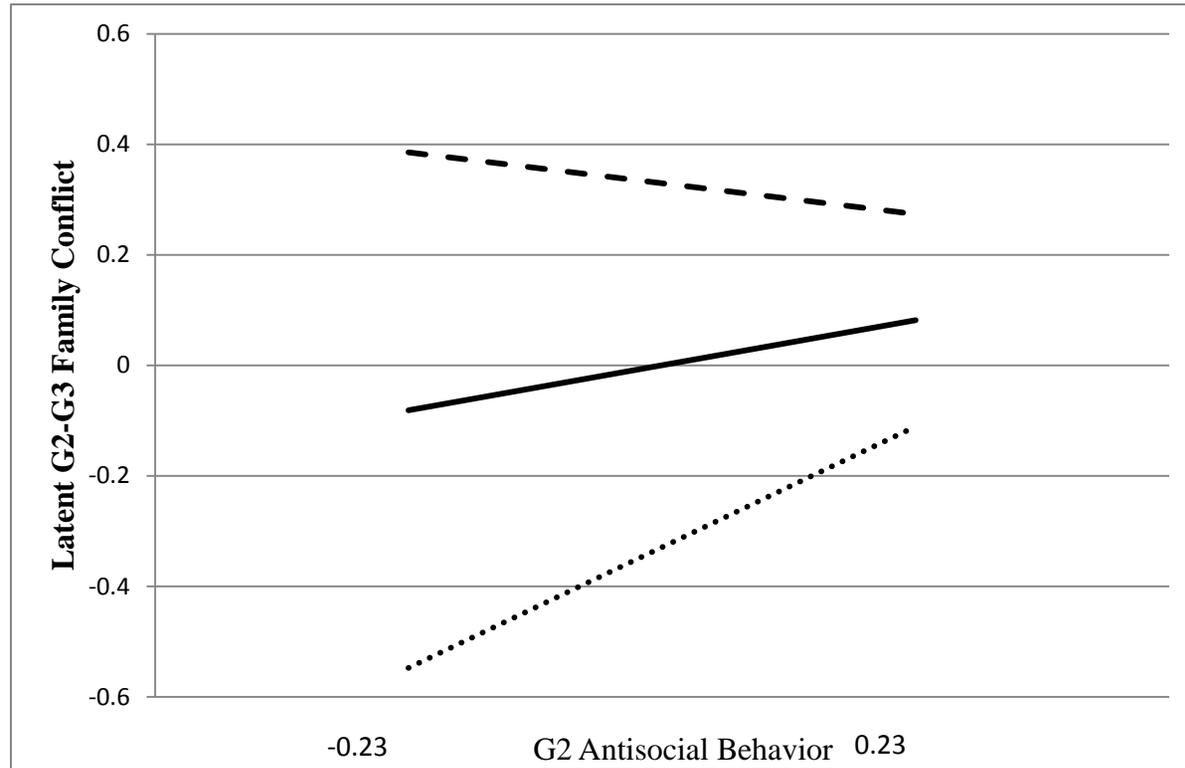


Figure 3: G2 antisocial behavior mediates intergenerational continuity in family conflict



Note: * $p < .05$, ** $p < .01$, all coefficients are standardized estimates

Figure 4: G2 partner antisocial behavior moderates relationship between G2 antisocial behavior and G2-G3 family conflict



- 1 SD Above Mean G2 Partner Antisocial Behavior
- Mean G2 Partner Antisocial Behavior
- 1 SD Below Mean G2 Partner Antisocial Behavior

Note: In this graph, G2-G3 family conflict is centered so that zero represents mean levels of G2-G3 family conflict.