Longitudinal Pathways From Marital Hostility to Child Anger During Toddlerhood: Genetic Susceptibility and Indirect Effects via Harsh Parenting

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We examined direct and indirect pathways from marital hostility to toddler anger/frustration via harsh parenting and parental depressive symptoms, with an additional focus on the moderating role of genetic influences as inferred from birth parent anger/frustration. Participants were 361 linked triads of birth mothers, adoptive parents, and adopted children who were 9 (T1) and 18 (T2) months old across the study period. Results indicated an indirect effect from T1 marital hostility to T2 toddler anger/frustration via T2 parental harsh discipline. Results also indicated that the association between marital hostility and toddler anger was moderated by birth mother anger/frustration. For children whose birth mothers reported high levels of anger/frustration, adoptive parents’ marital hostility at T1 predicted toddler anger/frustration at T2. This relation did not hold for children whose birth mothers reported low levels of anger/frustration. The results suggest that children whose birth mothers report elevated frustration might inherit an emotional lability that makes them more sensitive to the effects of marital hostility.

Keywords: marital hostility, parenting, parental depression, temperament, gene-environment interaction

The association between marital hostility and child adjustment is clearly established (Barletta & O’Mara, 2006; Buehler, Anthony, Krishnakumar, & Stone, 1997). Associations between marital conflict and child negative emotionality has been shown as early as 8 months of age (Pauli-Pott & Beckmann, 2007). Moreover, marital conflict is associated, most likely reciprocally, with dysfunctional parenting behaviors (Krishnakumar & Buehler, 2000) and parental depressive symptoms (Kouros, Papp, & Cummings, 2008), which are both associated with child adjustment (Kaczynski, Lindahl, Malik, & Laurenceau, 2006; Shaw, Gilliom, Ingoldsby, & Nagin, 2003). Although the pathways from marital hostility to child adjustment have been well studied, research, to date, has predominantly been conducted in biologically related families with school-age children and adolescents (Harold, Shelton, Goeko-Morey, & Cummings, 2004; Kaczynski et al., 2006). The current study addresses prior gaps in this area by examining relations between

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marital hostility and toddler anger/frustration from 9 to 18 months of age in a sample of adoptive families. The adoption design has the methodological strength of eliminating potentially confounding passive genetic influences, because the rearing parents and child are genetically unrelated. In addition, genetic moderation of environmental influences can be examined through associations with birth parent characteristics.

Because the association between marital hostility and child adjustment has been consistently demonstrated, recent investigations are increasingly focusing on examining the processes by which hostility within the marital subsystem affects children. Individuals and subsystems within families are inherently interconnected. The behaviors and affect of individuals and dyads within families influence, and are influenced by, the behaviors and affect of other individuals and dyads within the family system (Cox & Paley, 1997). Both intraparental characteristics and parent-child dyadic processes have been shown to affect, and to be affected by, child adjustment (Low & Stocker, 2005; Pardini, Fite, & Burke, 2008; Schofield et al., 2009). The current study focuses on two influential, frequently studied family processes: harsh parenting and parental depressive symptoms.

The spillover hypothesis (Erel & Burman, 1995) suggests that parents’ negative thoughts and emotions during marital conflict affect subsequent parent-child interactions. In support of this hypothesis, marital hostility is associated with parent-child hostility and parental rejection (Harold et al., 2004; Shelton & Harold, 2008). These parenting behaviors partially mediate the relations between marital conflict and child adjustment (Kaczynski et al., 2006; Schoppe-Sullivan, Schermerhorn, & Cummings, 2007), indicating that marital hostility has both direct and indirect associations with child and adolescent adjustment in biologically related families. To our knowledge, research, to date, has not examined indirect effects of marital hostility on child adjustment via parenting with children younger than age 5, or in biologically unrelated families. Thus, although marital conflict has been associated with dysfunctional parenting behaviors and negative outcomes in infancy and toddlerhood, it is unknown whether the indirect effects via parenting that have been found for older children are also present during infancy and early toddlerhood. In the current study, we examined harsh parenting based on its associations with child adjustment problems from very early childhood through adolescence (Arnold, O’Leary, Wolff, & Acker, 1993; Irvine, Biglan, Smolkowski, & Ary, 1999) and its similarity to parental rejection and hostility as examined in previous research (Shelton & Harold, 2008).

Marital hostility has additionally been shown to longitudinally affect depressive symptoms (Kouroš et al., 2008; Low & Stocker, 2005). If parents are not receiving adequate support from the couple relationship, or are experiencing high levels of hostility from their partners, they may be more prone to developing or worsening their depressive symptoms over time. Parental depression, in turn, is associated with a difficult/fussy temperament (Bridgett et al., 2009; McGrath, Records, & Rice, 2008). These effects appear to proceed from mothers’ depression to infant temperament (Sugawara, Kitamura, Toda, & Shima, 1999), although, again, associations are likely bidirectional. Previous studies have found indirect effects from marital distress to adolescent depression via maternal depression (Davies, Dumenci, & Windle, 1999). Prior research on this topic, however, has been conducted predominately within biologically related families, and work examining indirect effects of marital hostility on child adjustment via parental depression has not examined these processes in infants or toddlers. The current study addresses these gaps in the literature.

The Adoption Design

In biological families where children share 50% of their genes with each parent, the same genes that influence marital hostility might also affect the expression of child anger/frustration, thereby creating an association between marital hostility and child anger/frustration that results from genetic influences shared between parent and child, rather than from family environmental influences (passive rGE; Ulbricht & Neiderhiser, 2009). In the present study, we had the opportunity to examine associations between family processes and toddler anger/frustration without the potential confounding influence of shared genes. Our aim, therefore, was to examine the environmental effects of family processes on child adjustment free from the confounding of shared genes. In addition, we aimed to test whether genetic influences, as indexed by birth mother anger/frustration, moderate the association between marital hostility and toddler anger/frustration (i.e., GxE). We chose birth mother anger/frustration as the index of genetic effects because it is conceptually similar to child anger/frustration and would, therefore, likely have similar genetic underpinnings.

Prior studies with the current data set found evidence for GxE influences on negative emotionality and behavior problems in young children (Leve et al., 2009; Leve et al., 2010; Natsuaki et al., 2010). Because these studies focused exclusively on parenting and parental depressive symptoms, it is unknown if these effects extend to associations between child adjustment and other family processes, such as the marital relationship.

Hypotheses

The current study evaluated a model in which marital hostility is hypothesized to be both directly related to toddler anger/frustration and indirectly related via harsh parenting and parental depressive symptoms. Hypotheses are as follows (see Figure 1 for the hypothesized model):

1. Marital hostility at T1 will be directly associated with toddler anger/frustration at T2, while controlling for harsh parenting and parental depressive symptoms.

2. Marital hostility at T1 will be significantly indirectly associated with toddler anger/frustration at T2 via harsh parenting and parental depressive symptoms.
3. Relations among marital hostility, harsh parenting, parental depressive symptoms, and toddler anger/frustration will be moderated by birth mother anger/frustration, such that children whose birth mothers report higher levels of anger/frustration will demonstrate stronger relations between the marital and adoptive parent variables and toddler anger/frustration than children whose birth mothers report lower levels of anger/frustration.

Method

Participants and Procedures

Participants were 361 linked sets of adopted children, adoptive mothers and fathers, and birth mothers from the Early Growth and Development Study (EGDS). Eligibility criteria included (a) domestic adoption placement, (b) placement occurred within 3 months postpartum, (c) non-relative placement, (d) no known major medical conditions, such as extreme prematurity or extensive medical surgeries, and (e) birth and adoptive parents able to read and understand English at the eighth-grade level. Participating agencies included both those that favored closed adoptions and those that favored open adoptions. Recruitment and assessment staff for the birth parents and adoptive parents did not overlap. The children were 9 months old during the first assessment \((M = 8.8, SD = .97)\) and 18 months old during the second assessment \((M = 17.95, SD = .96)\). Forty-two percent of the children were female. The median child age at adoption placement was 2 days. The adoptive parents were typically college educated, middle- to upper-class families. The adoptive mother (AM) and adoptive father (AF) mean ages were 37 \((SD = 5.46)\) and 38 \((SD = 5.82)\), respectively. Ninety-three percent of the AMs and 92% of the AFs were Caucasian. Birth mothers (BMs) typically had less than a college education and had household annual incomes less than $25,000. BM mean age was 24 \((SD = 5.89)\). Seventy-eight percent of the BMs were Caucasian. This was a relatively high-risk sample of birth mothers; 43% of birth mothers met diagnostic criteria for lifetime substance abuse, 29% met criteria for lifetime major depressive disorder, and 32% met the adult criteria for antisocial personality disorder. For full demographic information, refer to Leve, Neiderhiser, Scaramella, & Reiss (2010).

In the present study, BMs were assessed in person at 18 months postpartum (T2) and adoptive parents were assessed in person when the child was 9 months (T1) and 18 months (T2). Retention rates remained high throughout the course of the study (97% for adoptive families and 95% for BMs); sample sizes for each variable in the analyses are presented in Table 1. There was one significant difference in demographic characteristics at T1 between families who had complete data and those who were missing data at T2. Families with missing data reported slightly higher BM household income \((p < .05)\).

Measures

Marital hostility in the adoptive family. AM and AF marital hostility was assessed at T1 using the 22-item Behavior Affect Rating Scale (Melby, Conger, Ge, & Warner, 1995). Adoptive parents reported on their partners’ hostility...
Table 1

Means, Standard Deviations, and Bivariate Correlations

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*p < .05. **p < .01. ***p < .001.

toward them on a 7-point scale ranging from never to always, where high scores indicated more hostility. Example items were as follows: How often did your partner (1) get angry at you; (2) hit, push, grab, or shove you; (3) criticize you or your ideas; or (4) call you bad names. Scores from the 13-item hostility subscale were summed to create a marital hostility score for AMs (α = .89) and AFs (α = .90). AM and AF reports of marital hostility were significantly correlated (r = .49, p < .001) and were thus averaged to create a composite measure, reflecting the overall degree of perceived marital hostility in the family.

**AM and AF harsh parenting.** AM and AF harsh discipline practices were assessed using the Overreactive subscale of the Parenting Scale (Arnold et al, 1993) at T1 and T2. The Parenting Scale is a 30-item measure designed to assess parental discipline strategies. Parents rate their likelihood of using specific discipline strategies in response to child misbehaviors on 7-point scales anchored by one effective and one ineffective discipline strategy. A score of 1 indicates effective discipline, and a score of 7 indicates dysfunctional discipline. We computed the Overreactive subscale (Rhoades & O’Leary, 2007; T1 AM Ovr α = .61; T1 AF Ovr α = .59; T2 AM Ovr α = .69; T2 AF Ovr α = .62). At T1, two Overreactive items (“When my child misbehaves . . . I usually get upset and under stress . . . I’m picky and on my child’s back/I am no more picky than normal”; “When my child misbehaves . . . I raise my voice or yell/I speak to my child calmly”; and “When there’s a problem with my child . . . things build up and I do things I don’t mean to do/things don’t get out of hand”), and the scale at T2 contains five items (the T1 items plus the two additional items). Although the internal consistencies of these subscales are moderately low, they are reasonable given the small number of items (3 to 5) included in each subscale and similar to those found in other studies using the same factors (e.g. Rhoades & O’Leary, 2007).

**AM and AF depressive symptoms.** Adoptive parent depressive symptoms were measured at T1 and T2 using a 20-item version of the Beck Depression Inventory (BDI; Beck & Steer, 1993). The item related to suicidal ideation was omitted from the scale at both time points. Depressive symptom scores were calculated by summing the ratings across the 20 items (AM: T1 α = .71; T2 α = .79; AF: T1 α = .75; T2 α = .81). Higher scores indicate higher levels of depressive symptoms.

**Child anger/frustration.** Child frustration and anger proneness was measured at T2 using parent report on the 111-item Toddler Behavior Assessment Questionnaire (TBAQ; Goldsmith, 1996). The Anger Proneness (Anger/Frustration) subscale (AM α = .87; AF α = .87) consists of 28 items that assess crying, hitting, protesting, pouting, or other signs of anger during conflict with adults or other children. AM and AF reports were significantly correlated (rs = .39, p < .001). To reduce the effects of rater bias, AM and AF reports were combined (i.e., averaged).
BM anger/frustration

A parallel dimension of BM temperament (anger/frustration) was assessed at T2 using the 77-item short form of the Adult Temperament Questionnaire (ATQ; Rothbart, Ahadi, & Evans, 2000). The six-item Frustration subscale (α = .66; example item: “Whenever I have to sit and wait for something [e.g., a waiting room], I become agitated”) was used in the current analyses. Items were scored on a 1 to 7 scale, with higher scores indicating more anger/frustration.

Covariates

Adoption openness. To control for similarities between birth and adoptive parents that might result from contact between parents, we included the T1 level of openness in the adoption as a covariate in all analyses. Openness in the adoption was measured using a composite of BM, AM, and AF ratings of perceived adoption openness (Ge et al., 2008). Interrater agreement was high (r range = .72–.85, all ps < .001).

Obstetric complications. Perinatal obstetric complications can confound genetic influence estimates and was, therefore, included as a covariate. Perinatal obstetric complications were assessed at T1 using BM report of her (a) Maternal/Pregnancy Complications (e.g., illness, exposure to drugs); (b) Labor and Delivery Complications (e.g., prolonged labor, cord complications); and (c) Neonatal Complications (e.g., prematurity, low birth weight), using a pregnancy screener and a pregnancy calendar method. Scoring was derived from the McNeil-Sjostrom Scale for Obstetric Complications (McNeil & Sjostrom, 1995), with item scores ranging from 1 (not harmful or relevant) to 6 (very great harm to or deviation in offspring). The obstetric complications total was created by calculating the frequency of scores greater than or equal to 3, indicating risk that is at least “potentially, but not clearly, harmful or relevant.”

Measures Used in Supplemental Analyses: Infant Distress to Limitations

Infant distress to limitations was measured at T1 with the Infant Behavior Questionnaire (IBQ; Rothbart, 1981). The IBQ was designed to measure temperament in children between the ages of 3 and 12 months. We used the 20-item distress to limitations subscale (AM α = .85; AF α = .85), which measures infants’ fussing, crying, or showing distress while (a) in a confining place or position, (b) involved in caretaking activities, and (c) unable to perform a desired action.

Data Analytic Plan

Hypothesis testing proceeded in three steps: evaluation of the hypothesized model (see Figure 1), examination of differences between mothers and fathers, and examination of genetic moderation. An overview of each step is described here. First, we evaluated the fit of the hypothesized model to the data using Mplus 5.2 (Muthén & Muthén, 2007), which uses full information maximum likelihood (FIML) to estimate parameters when data are missing. FIML produces unbiased estimates when data are missing at random (MAR). For all variables included in the current study, there were less than 7% data missing. The model was deemed to have adequate fit if either the chi-square was non-significant or the chi-square/df ratio was < 2, and if the comparative fit index (CFI) was > .95 and the root mean square error of approximation (RMSEA) was < .06 (Hu & Bentler, 1999). All indirect effects were estimated with bias-corrected bootstrapping and were considered statistically significant if the corresponding 95% confidence interval (CI) did not include zero (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Preacher & Hayes, 2008). We examined both specific indirect effects and total indirect effects of AM and AF harsh parenting and AM and AF depressive symptoms. In addition to the hypothesized paths, the model also controlled for the influence of adoption openness and obstetric complications on all endogenous variables.

Second, we evaluated whether paths from harsh parenting and parent depressive symptoms to child anger/frustration were significantly different for AMs and AFs by comparing two models: one where paths from harsh parenting to toddler anger/frustration for AMs and AFs were allowed to be freely estimated to one in which these paths were constrained to be equal, and one where paths from parent depressive symptoms to toddler anger/frustration for AMs and AFs were allowed to be freely estimated to models in which these paths were constrained to be equal. A significant difference in the chi-square values of the two models would indicate that the path estimates are not equal for AMs and AFs.

Third, we ran a multigroup analysis to test for genetic moderation. We first split the sample at the median of BM anger/frustration and ran one model, allowing all paths to be freely estimated for each group. We then constrained the paths from T1 marital hostility, T2 AM and AF harsh parenting, and T2 AM and AF depressive symptoms to T2 toddler anger/frustration to be equal across the two groups and compared the chi-square values of the two models. We then followed up this omnibus test with specific path comparisons for the five paths predicting toddler anger/frustration.

Results

Descriptive Statistics and Correlations

Means, standard deviations, correlations among primary study variables, and sample sizes are summarized in Table 1. AF depressive symptoms at T1 had a kurtosis value greater than 8.0 (Kline, 2005) and was thus square root transformed. The transformed variable did not have significant skew or kurtosis and was used in all subsequent analyses. All other variables were approximately normally distributed (skew < 2.0 and kurtosis < 8.0). Marital hostility and all primary study variables were positively associated. In addition, AM and AF harsh parenting and AM
depressive symptoms were significantly related to toddler anger/frustration at all time points, and T2 AF depressive symptoms were associated with toddler anger/frustration. There was only one significant correlation involving the two control variables: BM anger/frustration was positively associated with obstetric complications ($r = .13$, $p < .05$).

Path Models Predicting Toddler Anger/Frustration

The hypothesized model provided a good fit to the data. $\chi^2(31) = 41.73$, $ns$, $CFI = .98$, RMSEA = .03. Marital hostility at T1 significantly predicted AM and AF harsh parenting and AM depressive symptoms at T2. AM and AF harsh parenting at T2 significantly predicted toddler anger/frustration. The control path from adoption openness to T2 AM harsh parenting was also significant and negative, indicating that adoptive mothers with more open adoptions were less harsh. No other control paths were significant. Bias-corrected bootstrapped CIs of the indirect effects indicated a significant total indirect path through AM and AF harsh parenting (95% CI = .02-.09), a significant specific indirect effect through AF harsh parenting (95% CI = .01-.05), and a marginally significant indirect effect through AM harsh parenting (95% CI = .00-.05). All other indirect effects were nonsignificant. The model explained 10% of the variance in T2 toddler anger/frustration. Path estimates from harsh parenting and depressive symptoms to toddler anger/frustration for AMs and AFs were not significantly different from one another; all $\Delta \chi^2(5) < 3.84$, indicating that adoptive mothers’ and adoptive fathers’ harsh parenting and depressive symptoms are similarly associated with toddler anger/frustration.

Moderation by BM Anger/Frustration

The model in which the paths from marital hostility, harsh parenting, and parental depressive symptoms to toddler anger/frustration were constrained to be equal for the two groups (high BM anger/frustration vs. low BM anger/frustration) provided a significantly worse fit to the data than the model in which those paths were allowed to be freely estimated; $\Delta \chi^2(1) = 11.46$, $p < .05$, indicating that, as a whole, these paths in the model were significantly moderated by BM anger/frustration. We then evaluated moderation on a path-by-path basis by comparing models with all paths freely estimated to those in which one of the five hypothesized paths was constrained to be equal in the two groups. This set of analyses indicated that the path from T1 marital hostility to T2 toddler anger/frustration was significantly moderated by BM anger/frustration ($\Delta \chi^2(1) = 5.52$, $p < .05$; see Figure 1), such that marital hostility was only positively associated with toddler anger/frustration for children whose BMs reported higher anger/frustration. None of the other four paths predicting toddler anger were significantly moderated by birth mother anger/frustration.

Supplemental Analyses

To test the robustness of our model, we ran a series of additional analyses, which are each briefly described here. Although only one variable in our model demonstrated significant skew and/or kurtosis, we re-ran the model using a robust estimator (MLR in Mplus), thus eliminating the assumption that variables are normally distributed. The model fit was comparable, $\chi^2(31) = 39.04$, $ns$, CFI = .98, RMSEA = .03, and all path estimates were of similar magnitude and significance. We next evaluated the fit of the model controlling for the effects of infant distress to limitations at T1 on toddler anger/frustration at T2. The overall model fit was comparable, $\chi^2(35) = 47.04$, $ns$, CFI = .98, RMSEA = .03, and path estimates were of similar magnitude and significance. In this model, the total indirect effect through AM and AF harsh parenting remained significant. The specific indirect effect for AFs was reduced to marginal significance. Last, we controlled for the effect of BM anger/frustration on T2 toddler anger/frustration. The model fit was again comparable, $\chi^2(35) = 44.73$, $ns$, CFI = .98, RMSEA = .03, and path estimates were of similar magnitude and significance. The significance of all indirect effects remained the same. The path from BM anger/frustration to T2 toddler anger/frustration was nonsignificant.

Discussion

The current study examined both direct and indirect associations between marital hostility and subsequent toddler anger/frustration in an adoption sample. Results indicated a significant indirect effect of marital hostility on later toddler anger/frustration via harsh parenting. Relations between adoptive mother and adoptive father depressive symptoms and toddler anger/frustration, controlling for harsh parenting, were not statistically significant. We also found partial support for our third hypothesis: that birth mother anger/frustration would moderate associations among marital hostility, harsh parenting, and parental depressive symptoms and toddler anger/frustration. The direct path from T1 marital hostility to T2 toddler anger/frustration was significant only in the subgroup of families where birth mothers reported higher levels of anger/frustration.

This is the first study, to our knowledge, to examine these aspects of family relations in a nonbiological sample during very early childhood. Our results largely replicate previous findings that marital conflict is associated with parenting (Krishnakumar & Buehler, 2000), which is then associated with child outcomes (Kaczynski et al., 2006; Schoppe-Sullivan et al., 2007). Previous studies, however, were conducted in biologically related families and were thus unable to differentiate between two alternatives: that the covariance among marriage, parenting, and child outcomes is due to genetic influences common to all three, versus environmental mechanisms where the effects of shared genes are removed. In the present adoption sample, the effects of shared genes were eliminated because adoptive parents and their children share no genetic relationship. Our results therefore confirm that there are significant environmental effects of marital hostility on children’s anger/frustration via harsh parenting in very early childhood.

This contribution is essential, as prevention programs are designed to impact environmental factors that influence
child adjustment and thus depend on our ability to identify and target modifiable environmental factors. The family factors identified in this study, particularly harsh parenting and, for some children, marital hostility, are thus prime targets for prevention efforts. This may be especially so for children who are temperamentally at higher risk for developing conduct problems, as evidenced by our finding that the association between marital hostility and toddler anger/frustration was significant only in children whose birth mothers had high levels of anger/frustration, suggesting the moderating role of genetic influences. Recent evidence suggests that temperament is potentially modifiable (Rothbart & Derryberry, 2002); as such, improving family functioning in marital and parenting domains might have the potential to reduce the expression of genetically influenced temperamental anger/frustration before it progresses to later conduct problems.

In the context of the larger model, parental depressive symptoms were neither associated with toddler anger/frustration nor were there significant indirect effects from marital hostility to toddler anger/frustration via parental depression. Overall, this sample of adoptive mothers and adoptive fathers reported fairly modest levels of depressive symptoms ($M = 3.57$ and $3.80$ at T1 and T2, and $2.86$ and $2.81$ at T1 and T2, respectively). In comparison, BDI scores between 0 and 9 indicate normal or asymptomatic depressive symptoms (Beck & Steer, 1993). It is possible that the potential effects of parental depressive symptoms on child anger/frustration might only be apparent at moderate to clinical levels of depressive symptoms; this is a question for future research to investigate.

Although there was no direct association between birth mother anger/frustration and toddler anger/frustration, as noted above, birth mother anger/frustration significantly moderated the relation between adoptive parent marital hostility and later toddler anger/frustration. This genetic moderation is consistent with the premise that children whose birth mothers report higher levels of anger/frustration inherit an emotional lability, making them more susceptible to the negative impact of marital hostility. It was unexpected that birth mother anger/frustration would moderate the association between marital hostility and toddler anger/frustration, but not the association between harsh parenting and toddler anger/frustration. Perhaps harsh parenting, as a potentially stronger proximal effect (because marital hostility does not necessarily occur in front of the child), affects children similarly regardless of genetic susceptibility, whereas there is more variability in children’s responses to marital hostility. This may be especially true for younger children who may not yet be able to interpret the meaning of marital conflict as it relates to the stability of the family system.

Belsky and a colleague (Belsky, 2005; Belsky & Pleuss, 2009) have proposed that genetic factors might predispose individuals to be more sensitive to their environments. This differential susceptibility hypothesis states that individuals with particular genetic backgrounds may be more vulnerable to environmental stressors, but also most likely to benefit from advantageous or supportive environments. Consistent with this hypothesis, toddlers whose birth mothers report elevated anger/frustration appear to be more vulnerable to the effects of marital hostility than toddlers whose birth mothers do not report elevated frustration. Such children may also benefit more from marital warmth or the absence of marital hostility. To directly address this question, one would need to examine relations between marital harmony/warmth and child anger/frustration to confirm that children inherit both sensitivity toward positive environments and sensitivity toward negative environments; this hypothesis was beyond the intended scope of this article, but an area where future research is needed.

The current study is notable for its inclusion of fathers. Interestingly, there were no significant differences in the associations among marital hostility, harsh parenting, parental depressive symptoms, and toddler anger/frustration for mothers versus fathers. This result is striking, especially considering that in very early childhood, mothers spend disproportionally more time with their infants, even in couples where both parents work (Pleck & Masciadrelli, 2004), and considering that fathers have been more likely to show evidence of spillover of negativity from the marital relationship to the parent-child relationship than mothers (Kerig, Cowan, & Cowan, 1993; Schofield et al., 2009). Thus, although fathering and mothering may be qualitatively different with each contributing uniquely to children’s development (Parke, 2002; Stolz, Barber, & Olsen, 2005), changes in harsh parenting during very early childhood are similarly associated with toddler anger/frustration for both mothers and fathers. This result may be due, in part, to the nature of our sample. For example, Holditch-Davis, Sandelowskki, & Harris (1999) found that adoptive mothers and adoptive fathers were more similar in the amount of time they spent interacting with their infants than were biological parents, suggesting that adoptive fathers might be more involved in interacting with their infants than biological fathers.

Some caveats must be noted. First, our analyses and hypotheses were specific to two time points, limiting inferences about the direction of effects from harsh parenting to toddler anger/frustration. Previous research, however, has indicated that parenting impacts later child behavior (Ary, Duncan, Duncan, & Hops, 1999), and that improving parenting practices through intervention programs results in decreased child behavior problems (Chamberlain et al., 2008; Dishion et al., 2008). Further, when we include a T1 measure of child frustration, the present results were essentially unchanged. This noted, it is likely that the relations between marital hostility and child adjustment are bidirectional (Pardini, Fite, & Burke, 2008).

Second, because the analyses only contained two time points, the present study was not ideally suited for addressing mediation per se due to the causal assumptions inherent in mediation analyses (MacKinnon et al., 2002). Our aim in the current study was to examine the relations, both direct and indirect, of marital hostility on subsequent toddler anger/frustration. As such, our analyses purposefully did not examine potential child effects (i.e., reverse causality), although we recognize that such effects are likely present,
important, and prime targets for future research. The current results, similar to results from any nonexperimental study, should not be interpreted as conclusive causal evidence that harsh discipline fully mediates the effects of marital hostility on toddler anger. A more accurate interpretation would be that marital hostility is longitudinally associated with toddler anger partially via harsh parenting.

Third, our sample had limited ethnic and sociodemographic diversity. Caution should be used in generalizing these results to more diverse or at-risk populations. In addition, all included measures were parent-report questionnaires. Although self-reports are vulnerable to self-presentation and other biases, the measure of harsh parenting has been shown to be significantly associated with observed parenting behavior in the laboratory (Arnold et al., 1993). Moreover, parents’ responses on the overreactivity subscale of the Parenting Scale are significantly different than their responses on a parallel measure that asks parents to report what they should do when interacting with their children, indicating that social desirability does not fully drive parents’ responses on the measure (Rhoades & O’Leary, 2007). Nonetheless, the self-report nature of this measure is a limitation. Last, we employed composite measures of adoptive mother and adoptive father marital hostility and toddler anger/frustration to decrease single-rater bias of these constructs. Although we employed composite measures of adoptive mother and adoptive father reported toddler anger/frustration, there is still the possibility that the depressive symptoms of one or both of the adoptive parents influenced their toddler temperament ratings (see Orhon, Ulukol, & Soykan, 2007). There are discrepant findings in the literature as to whether this is a reporting bias or indicative of actual differences in child temperament (see Baumann, Pelham, Lang, Jacob, & Blumenthal, 2004). In all cases where potential bias is indicated, however, mothers with more depressive symptoms rate their children as more problematic. In relation to our results, this bias would thus potentially increase the magnitude of the associations among adoptive mother and adoptive father depressive symptoms and toddler anger/frustration. In our model, these paths are small and nonsignificant. Any potential bias would simply further reduce the already nonsignificant associations. Including adoptive mother and adoptive father depressive symptoms in the overall model additionally increases our confidence in the robustness of the associations between harsh parenting and toddler anger/frustration, as these analyses control for parental depressive symptoms and the potential reporting biases that parental depression could introduce into our model.

As a whole, the results of this study confirm and extend prior research with biologically related families with older children: marital hostility is related to child outcomes partially through its effects on parenting. Our results suggest that these relations begin very early in development and cannot be explained by the effects of shared genes. Finally, our results indicate that the detrimental environmental effects of marital hostility on toddler anger/frustration are moderated by the child’s genotype, such that the direct relation is only apparent for toddlers whose birth mothers reported relatively higher levels of anger/frustration. These results strengthen the assertion that interventions designed to decrease marital hostility may have additional benefits for children in families with high biological risk.

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