A Genetically Informed Study of Associations between Family Functioning and Child Psychosocial Adjustment

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Abstract

Research has documented associations between family functioning and offspring psychosocial adjustment, but questions remain regarding whether these associations are partly due to confounding genetic factors and other environmental factors. The current study used a genetically informed approach, the Children of Twins design, to explore the associations between family functioning (family conflict, marital quality, and agreement about parenting) and offspring psychopathology. Participants were 867 twin pairs (388 MZ; 479 DZ) from the Twin and Offspring Study in Sweden (TOSS), their spouses, and children (51.7% female; *M* = 15.75 years). The results suggested associations between exposure to family conflict (assessed by the mother, father, and child) and child adjustment were independent of genetic factors and other environmental factors. However, when family conflict was assessed using only children's reports, the results indicated that genetic factors also influenced these associations. In addition, the analyses indicated that exposure to low marital quality and agreement about parenting was associated with children's internalizing and externalizing problems, and that genetic factors also

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contributed to the associations of marital quality and agreement about parenting with offspring externalizing problems.

Keywords

Family Relations; Child Adjustment; Genetics; Offspring; Twins

Research has demonstrated that family functioning is associated with child well-being. There have been calls to examine these associations at a process-oriented level (Cummings, Davies, & Campbell, 2000) and to disentangle the mechanisms underlying these associations (Rutter, 1994). Recent research has examined the roles of genetic and environmental factors in relations between some areas of family functioning and child functioning. Notably, research has examined these factors in associations between parental divorce and offspring functioning (Amato & Cheadle, 2008; D'Onofrio et al., 2005; D'Onofrio et al., 2006; D'Onofrio, Turkheimer, Emery, Harden, et al., 2007; D'Onofrio, Turkheimer, Emery, Maes, et al., 2007; O'Connor, Caspi, DeFries, & Plomin, 2000), and findings from these studies suggest that the experience of parental divorce elevates offspring adjustment problems. However, most children live in nondivorced families. Thus, one important question is whether experiencing more common family relationship problems also increases adjustment problems. In the current study, we used a genetically informed approach to examine the contributions of genetic and environmental factors to the associations of family conflict, marital quality, and marital agreement about parenting with child psychosocial adjustment.

Family Relationships and Child Adjustment Problems

Research has examined family functioning, including conflict between family members (*family conflict*), marital cohesion, marital warmth, and marital satisfaction (*marital quality*), and spouses' agreement with each other about child discipline (*agreement about parenting*), as it relates to child development and psychopathology. Previous work indicates that family conflict is associated with child internalizing and externalizing problems (Handal, Tschannen, & Searight, 1998; Harachi et al., 2006; Jaycox & Repetti, 1993), and family aggression is associated with child psychopathology (McCloskey, Figueredo, & Koss, 1995; Richmond & Stocker, 2006).

In terms of implications for child adjustment problems, the marital relationship has also been the focus of considerable research (Cummings & Davies, 2002). Destructive marital conflict has been linked with children's adjustment problems (Cummings, Goeke-Morey, & Papp, 2003), and mothers' marital satisfaction and marital conflict resolution are associated with fewer child adjustment problems (Goeke-Morey, Cummings, & Papp, 2007). In addition, whereas child-rearing disagreements are associated with higher levels of internalizing and externalizing problems (Lee, Beauregard, & Bax, 2005), spouses' cooperation with one another in the parenting of their children is associated with fewer externalizing problems (Schoppe, Mangelsdorf, & Frosch, 2001).

Thus, considerable progress has been made in advancing knowledge of associations between family functioning and child adjustment. However, many questions remain regarding these associations. One commonly held view is that exposure to family relationship problems increases child adjustment problems. In support of this notion, increases in couples' conflict resolution and constructiveness following participation in a program to prevent marital problems were associated with decreases in child adjustment problems (Cummings, Faircloth, Mitchell, Cummings, & Schermerhorn, 2008). Moreover, preschoolers exposed to simulated interadult expressions of anger in the laboratory showed more negative responses

(Cummings, 1987) and increases in blood pressure (El-Shiekh, Cummings, & Goetsch, 1989) than when exposed to positive or neutral interadult expressions. Relatedly, family conflict explains 40% of the similarity between siblings in ADHD symptomatology, and parental psychopathology does not explain unique variance in ADHD symptomatology once family conflict is accounted for (Pressman et al., 2006). These findings are consistent with notions that exposure to family dysfunction elevates child adjustment problems.

In addition to effects of exposure, there may be third variables—other environmental factors or genetic factors—that contribute to both family relationship problems and child adjustment problems. For example, marital and family functioning, parental psychopathology, and child adjustment have been found to interrelate (Nomura, Wickramaratne, Warner, Mufson, & Weissman, 2002; Papp, Cummings, & Schermerhorn, 2004). Thus, it may be that the association between family relationship problems and offspring functioning is partially due to the mutual associations of family and offspring functioning with parental psychopathology. Moreover, low socioeconomic status has been linked with both family functioning and with child adjustment (Brody & Flor, 1997). Thus, these potential third variables may underlie part of the association between family relationship problems and child functioning.

Genetic factors that influence parental characteristics can be inherited by offspring, thereby conferring risk on the offspring; genetic confounds of this type are due to passive genotype-environment correlation (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). Although there is no gene that directly causes family conflict, genes contribute to phenotypic differences between people. Genetic factors contribute to parenting (Bakermans-Kranenburg & van IJzendoorn, 2008; Neiderhiser, Reiss, Lichtenstein, Spotts, & Ganiban, 2007), as well as to numerous other dimensions of family and social relationships (Kendler & Baker, 2007; Reiss, Neiderhiser, Hetherington, & Plomin, 2000; Ulbricht & Neiderhiser, in press). Of particular relevance to the current study, both genetic and environmental factors contribute to marital quality (Spotts, et al., 2004). Genetic factors also contribute to associations between marital negativity and mother-to-child negativity (Ganiban et al., 2007). These findings suggest the importance of examining the possible role of genes in associations between family functioning and offspring adjustment.

Previous studies have also examined the influence of exposure to parent-child conflict and negativity on child adjustment, as well as the contributions of genetic factors to this association (see Ulbricht & Neiderhiser, in press). For example, one study found that genetic and environmental influences both explain covariation among parental negativity and child depression and antisocial behavior (Pike, McGuire, Hetherington, Reiss, & Plomin, 1996). Other studies have found genetic factors to account for most of the association between parent-child conflict and child adjustment problems (Narusyte, Andershed, Neiderhiser, & Lichtenstein, 2006; Neiderhiser, Reiss, Hetherington, & Plomin, 1999). Interestingly, whereas the association between parent-child conflict in early adolescence and subsequent adolescent externalizing problems is accounted for by both genetic and environmental factors, the association between externalizing problems in early adolescence and subsequent parent-child conflict is largely accounted for by genetic factors (Burt, McGue, Krueger, & Iacono, 2005). Thus, findings regarding associations between parent-child conflict and child adjustment have been somewhat mixed. The current study builds on this literature and extends it by examining the relations of child adjustment to the constructs family conflict, marital quality, and agreement about parenting.

Findings from studies on environmental influence on early development in non-human animals, in which genetic and environmental factors can be controlled, are also informative. Research on rats suggests that maternal behavior influences gene expression and stress

responses in offspring (Weaver et al., 2004). Further, Suomi and his colleagues have separated genetic and environmental factors by comparing infant monkeys reared by peers or by biological mothers. Monkeys reared by their peers tend to be very fearful and timid, and also more impulsive, compared with monkeys reared by their mothers (Suomi, 1991). However, infant monkeys who are initially highly fearful, and who are raised by especially nurturing mothers, have particularly adaptive outcomes (Suomi, 1987). In a separate line of research on rhesus monkeys, Maestripieri (2004) found a genetic association between the conflict behavior of mothers and that of their children. However, the extent to which findings from research on non-human animals can be generalized to humans is unclear, and thus, studies with humans using multiple designs are needed.

Genetically Informed Research Designs

Researchers have used several genetically informed designs to examine offspring adjustment, including adoption, sibling comparison, and co-twin control approaches. The sibling comparison approach involves comparing siblings who are differentially exposed to risk factors. Sibling comparison and co-twin control designs can be used to examine environmental risk factors on which siblings/twins in the same family differ (Lahey, D'Onofrio, & Waldman, in press; Rutter, 2007). Although these designs can be used to examine the influence of putative risk factors for individual family members (e.g., maternal age at childbearing), they cannot be used to examine putative family-wide risk factors (e.g., divorce). Recent studies have also used the COT design (see D'Onofrio et al., 2003 and Silberg & Eaves, 2004). Because the COT design is based on the twin status of parents, rather than children, it allows the underlying processes related to shared environmental risks to be disentangled. The COT design controls for environmental factors that differ between extended twin families (i.e., both twins of a pair and their nuclear families).

The COT design also helps account for shared genetic factors. The design compares the children of monozygotic (MZ) and dizygotic (DZ) twins who differ from one another with regard to a risk factor of interest—family relationship problems (family conflict, low marital quality, and interparental disagreement about childrearing) in the current study. Because offspring of MZ twins share 50% of their genes with each of their parents and with their parent's co-twin, but the parent's co-twin does not provide the offspring's environment, this design can be used to disentangle the effects of genetic and environmental factors. Comparing cousins in MZ families, if the cousin with more family problems has more adjustment problems, that suggests exposure to family problems increases adjustment problems. This is because children of MZ twins differ in environmental risk, but share similar genetic risk for adjustment problems. Conversely, if the cousins from MZ twin families have equivalent levels of adjustment problems, the results would suggest that other factors (i.e., genetic factors or other environmental influences) are at least partly responsible for the association between the family and child functioning.

Offspring of MZ twins share on average 25% of their genes with the co-twin's offspring; however, offspring of DZ twins share on average 12.5% of their genes with the twin's offspring. By comparing children of MZ and DZ twins discordant for relationship problems, the COT design can help disentangle genetic and environmental influences on child outcomes and thus begin to test the role of specific potential selection factors in associations between family and child functioning. Finding that cousins in MZ families have more similar levels of adjustment problems than cousins in DZ families would suggest that genetic factors are important. But, finding that cousins' similarity in MZ families is equivalent to cousins' similarity in DZ families would suggest that other environmental factors are important (because associations with outcomes would be unrelated to genetic risk). These other environmental factors would be ones that influence all cousins in an

extended family, like poverty, which could contribute to both family relationship problems and offspring adjustment problems.

Importantly, however, the design does not control for unmeasured environmental factors that influence only one of the co-twin nuclear families, including characteristics of the twins' spouses (Eaves, Silberg, & Maes, 2005). Measured characteristics can be included in the statistical analyses to help address these potential confounds (D'Onofrio et al., 2005; Jacob et al., 2003). In the current study, we control for socioeconomic factors that differ between extended families (both co-twins and their offspring) and between nuclear families (one twin of a pair and his/her offspring).

Recent work has used the COT design to examine the unique environmental and genetic contributions to associations between parent and offspring generations. Findings from one such study are consistent with the hypothesis that exposure to parental harsh physical discipline increases externalizing problems and substance use in young adults (Lynch et al., 2006). Research on parental divorce has also generally supported the view that divorce increases offspring psychopathology, including substance use, internalizing, and externalizing problems in adolescence (D'Onofrio et al., 2005; 2006). These findings are consistent with studies of divorce using adoption designs (Amato & Cheadle, 2008; Burt, Barnes, McGue, & Iacono, 2008; O'Connor et al., 2000). However, findings have been mixed for some outcomes in adolescence through middle adulthood, with some COT studies suggesting that environmental factors other than divorce, such as SES and parental psychopathology, and genetic factors also contribute in important ways to these associations (D'Onofrio et al., 2006; D'Onofrio, Turkheimer, Emery, Harden, et al., 2007; D'Onofrio, Turkheimer, Emery, Maes, et al., 2007).

Although a fairly consistent picture has emerged from these studies of divorce, few COT studies have examined genetic and environmental contributions to associations between child adjustment and less severe forms of marital problems, such as low marital quality and marital agreement about parenting. One of the few studies to address this gap examined associations between marital conflict and children's conduct problems using a sample of Australian twin families with conduct problems prior to age 18 assessed retrospectively (Harden et al., 2007). The results were inconsistent with the notion that exposure to marital conflict increases child conduct problems, suggesting instead that the association is explained by genetic factors that contribute to both marital conflict in the parent generation and conduct problems in the child generation.

The Current Study

Given the methodological limits inherent in phenotypic studies and in genetically informed designs, no single study can provide definitive answers regarding the development of psychopathology (Rutter et al., 2001). Multiple studies, using different designs, are needed. In the current study, we used the COT design to test genetic and environmental contributions to associations between child adjustment problems and three dimensions of family relationships: family conflict, marital quality, and marital agreement about parenting.

In addition, because previous work suggests children's unique perceptions of family functioning merit separate examination (Neiderhiser, Pike, Hetherington, & Reiss, 1998; Powers, Welsh, & Wright, 1994), we conducted additional tests, examining children's perceptions separately from those of their parents. Adolescents' perceptions of interparental conflict and parent-to-child hostility mediate the associations of interparental conflict and parent-to-child hostility with adolescents' internalizing problems (Harold, Fincham, Osborne, & Conger, 1997). In addition, children's perceptions of parenting account for genetic influences on associations between parenting and child adjustment, which might be

because the same genetic factors that contribute to children's perceptions of parenting might also contribute to the association between actual parenting behavior and child adjustment (Neiderhiser et al., 1998). Because of these findings of the importance of examining children's unique perceptions of family functioning, we conducted the analyses of family conflict both with and without parents' reports of family conflict (using child report in all of these models). Children did not report on either marital quality or marital agreement about parenting, so we were unable to carry out separate tests for those constructs.

For all of the tests, we anticipated that the underlying processes would be complex, involving influence of both exposure to family functioning problems and genetic factors. Specifically, we hypothesized that exposure to family relationship problems contributes to the development of adjustment problems in children, and that genetic factors also contribute to associations between family relationship problems and child adjustment problems.

Method

Participants

Participants were recruited through the Swedish Twin Registry (Lichtenstein et al., 2002) to participate in the Twin and Offspring Study in Sweden (TOSS), which included 909 same-sex adult twin pairs. Details of the research methods are described in Reiss et al. (2001) and Neiderhiser & Lichtenstein (2008). The current study included 1734 twins (867 pairs: 259 female MZ; 129 male MZ; 286 female DZ; 193 male DZ) for whom zygosity, family, marital, and child psychosocial adjustment data were available. There were 1734 nuclear families (i.e., twin families), nested within 867 extended families (i.e., twin and co-twin families). Twins' spouses and one adolescent child of each twin also participated. Twins' mean age was 44.88 years (SD = 4.88); their spouses' mean age was 45.56 years (SD = 5.36). Cousins were selected if they were of the same sex and within 4 years of each others' age. Children's ages ranged from 11–22 (M = 15.75 years, SD = 2.42). Of the 867 cousin pairs, 51.7% (448 pairs) were female.

Zygosity was assessed using self-report methods described by Nichols and Bilbro (1966). Twins who described themselves as similar as "two berries on a bush," were classified as MZ twins. Twins who perceived themselves as different and indicated that others have little difficulty distinguishing between them were classified as DZ twins. DNA was used to confirm zygosity for a subset of the sample; agreement between the questionnaire and DNA assignments was 96%. The current paper used only zygosity data from self-report methods.

Each household's highest status occupation was used as the index of household occupation status. This index was *unskilled/semi-skilled employment* for 10.5% of the households; *skilled/assistant non-manual employment* for 25.5%; *non-manual employment or self-employment with no employees* for 32.8%; and *professional employment, higher civil servant employment, or executive level or self-employment* for 31.2%. Regarding educational attainment, 14.5% of twins and 15.6% of spouses had completed only elementary school; 1.4% of twins and 1.9% of spouses had completed only junior high school/middle school; 28.7% of twins and 26% of spouses had completed two years of senior high school; 11.4% of twins and 15.4% of spouses had completed three or four years of senior high school; 34.1% of twins and 31.7% of spouses had completed college; and 9.9% of twins and 9.4% of spouses reported other levels of educational attainment. There were no differences between MZ and DZ twins in the study in terms of age, highest household occupation level, education, or age of partners (Ganiban et al., 2009).

Procedures

Twins, spouses, and children completed questionnaires via postal mail and during in-home visits. All family relationship questionnaires were completed during the home visit in order to avoid the possibilities of families responding to these relationship questions together. When possible, questionnaires that had previously been used in Sweden were used in the current study; when that was not possible, measures were translated and back-translated (Reiss et al., 2001).

Measures

Family conflict—Twins, spouses, and children completed the family conflict subscale of the Family Environment Scale (FES, Moos & Moos, 1981). This widely used measure has well-established psychometric properties. A sample item from the family conflict subscale is "We fight a lot in our family." This subscale consists of 5 items answered using a 5-point Likert scale ranging from *Fits not at all* (1) to *Fits exactly* (5). Cronbach's alphas for the family conflict subscale in the current study were .74 for twin report, .74 for spouse report, and .68 for child report.

Marital quality—Twins and spouses completed the Dyadic Adjustment Scale (DAS, Spanier, 1976), a widely used measure, which includes the dyadic consensus (13 items), dyadic satisfaction (10 items), dyadic cohesion (5 items), and affectional expression (4 items) subscales. The items were presented using two formats: Items about partners' agreement with one another on a range of topics (e.g., religion, time together) are answered using a 6-point Likert scale, with response options ranging from *Always disagree* (0) to *Always agree* (5); items about frequency of positive (e.g., laughing together) and negative events (e.g., discussing divorce or separation) are answered using a 6-point Likert scale, with response options ranging from *Never* (0) to *Always* (5). Cronbach's alphas for the DAS in the current study ranged from .77 to .91 for twin- and spouse-reported subscales.

Marital agreement about parenting—Twins and spouses completed the Agreement on Parenting measure, which was developed for the Nonshared Environment in Adolescent Development project (Reiss et al., 2000). This 12-item measure assesses agreement between spouses about a variety of parenting issues, such as "Your children's religious training." Items are answered using a 6-point Likert scale, with response options ranging from *Always disagree* (0) to *Always agree* (5). Cronbach's alphas for the current sample were .91 for twin and spouse report. Using husbands' and wives' individual reports of agreement about parenting would have necessitated creating a latent variable with only two (husband report, wife report) indicators. Instead, to facilitate model testing, since husbands' and wives' reports were significantly correlated (r = .35, $p < .001$), we composited husbands' and wives' reports and used the composite as a manifest variable in the SEMs.

Child internalizing and externalizing problems—Children completed the Child Behavior Checklist (CBCL, Achenbach & Edelbrock, 1979), reporting on their internalizing and externalizing problems. The child-report version of the CBCL consists of 110 items, such as "I can't sit still" and "I feel overly tired." The items are answered using a 3-point Likert scale, with answers ranging from *Not True* (0) to *Very True or Often True* (2). Cronbach's alphas for the current sample were .86 for internalizing and .81 for externalizing.

Data Analyses

We conducted a series of structural equation models (SEM), after standardizing the variables for ease of interpretation. Structural equation modeling was conducted using Mplus Version

5 (Muthen & Muthen, 1998–2007) using full information maximum likelihood (FIML) to accommodate missing data. In Mplus, this is based on the assumption of ignorable missingness (missing at random; MAR) (Muthen, 1998–2004). Mplus uses Little and Rubin's (1987) Expectation-Maximization (EM) algorithm.

We report multiple fit indices to facilitate evaluation of the degree to which our models fit the sample data and for comparison of related models. Values of the root mean square error of approximation (RMSEA; Browne & Cudeck, 1993) should be less than or equal to .06 (Hu & Bentler, 1999), although Browne and Cudeck have indicated that RMSEA values less than or equal to .08 also indicate reasonable fit. Values of the standardized root mean squared residual (SRMR; Hu & Bentler, 1998) should also be less than or equal to .08 (Hu & Bentler, 1999). We also report the χ^2 discrepancy test and the Bayesian Information Criterion (BIC, Schwarz, 1978), for comparison of different models; smaller values for these indices indicate better fit.

For the primary analyses, each family relationship construct was modeled separately. Our structural equation model has three main parts (see Figure 1). First is a simple confirmatory common factor model of mother, father and child reports of family functioning. We fixed the variance of the latent variable by setting the loading for mother report to 1.0.

The second part of the model is a standard twin model of the latent common variable for family functioning, with the common variance in family relationship functioning decomposed into three variance components: an additive genetic component (A), an environmental component that makes twins similar to one another (C), and an environmental component that makes twins different from each other (E). Because MZ twins share 100% of their genes and DZ twins share 50% of their genes, the covariance for the A components in MZ co-twins was set equal to the MZ variance of A (resulting in a correlation=1.00), the covariance of the A components in DZ co-twins was set equal to half the DZ variance of A (resulting in a correlation=.50), and the paths from A, C, and E to family functioning were set equal to 1. This is equivalent to models specifying MZ correlations of 1.0 and DZ correlations of .50, but parameterized in terms of the unstandardized variances of the components. By definition, C is completely shared by twins (correlation of 1.00) and E is unshared (correlation of 0.00).

For each of the relationship constructs, we first ran an SEM for the associations between the co-twin families' latent variables (e.g., family conflict), without modeling the offspring adjustment outcomes. For each of these SEMs, the magnitudes of the correlations were small to medium, indicating the importance of nonshared environmental influences. The MZ correlations were larger than the DZ correlations (family conflict: $r_{MZ} = 0.24$, $p < .001$, $r_{DZ} = 0.05$, $p > .10$; marital quality: $r_{MZ} = 0.15$, $p < .001$, $r_{DZ} = 0.04$, $p > .10$; agreement about parenting: $r_{MZ} = 0.28$, $p < .001$, $r_{DZ} = 0.10$, $p < .05$), suggesting genetic factors also influence family conflict, marital quality, and agreement about parenting. We found that the variance of the C component was negligible, indicating it could not account for the association between family conflict and child functioning. Thus, component C was dropped from all subsequent models.

The third part of the structural equation model consisted of regressions of child functioning variables on the family functioning latent variables. The child functioning variables have residual variation not associated with family functioning, and a residual covariance not explained by their common relation with family function. To allow associations between cousins to differ for MZ vs. DZ families, we estimated the residual offspring correlation separately for MZ and DZ families. In addition, we decomposed the regression of child functioning and family functioning into a direct path from latent family phenotype to child

functioning (path *b* in Figure 1) and an indirect path mediated by the A component of family functioning (path *a* in Figure 1).

The logic of our analysis follows from a method sometimes referred to as the co-twin control design. Suppose one is interested in the association between exposure to a particular environmental factor and outcomes. People are not exposed to this environment at random, it is not possible to randomize exposure for ethical reasons, and there is reason to believe that certain genetic make-ups might predispose people to exposure—a gene-environment correlation. In a study that compares exposed and non-exposed individuals in the general population, an association might be observed between the exposure and the outcome because some people have a genetic predisposition to exposure, which is then correlated genetically with the outcome. If this is the case, the exposure itself might have no influence on the outcome at all.

In order to test this, one useful approach is to collect data from a sample of discordant identical twins (one of whom has been exposed and the other of whom has not) and compare outcomes. This approach "controls for" the possibility that genetic predisposition to exposure is responsible for the population association between the exposure and the outcome; identical twins are identical genetically, so differences between identical twins cannot be the result of genetic differences. A relationship between identical twin exposure differences and identical twin outcomes, therefore, cannot be the result of genetic differences either. Thus, the possibility that exposure influences outcomes is supported, because the genetic confound has been ruled out via the fixed effect of the co-twin (Rutter et al., 2001).

Next, we describe a variation that is an expansion of the description above; it is expanded especially in terms of the statistical analyses. In the following, we use a parameterization described by Carlin et al. (2005). The twins in this sample are included regardless of their exposure status; some pairs are concordant for exposure, some are concordant for non-exposure, and some are discordant. A multilevel regression analysis is conducted, regressing outcome on exposure between and within twin pairs. So for twin$_i$ in family$_f$, outcome y_{if} is predicted by exposure x_{if}. Exposure has been partitioned into a grand mean, and variances u_f between families, and σ_{if} within families. The outcome, y_{if}, is regressed on the between and within portions of *x*.

$$y_{if}=b_0+b_B u_f+b_W \sigma_{if}$$

The between (b_B) and within (b_W) regressions are then compared. Since the hypothetical genetic confound is entirely between families (because they are all MZ pairs), to the extent the between regression is much larger than the within regression it is an indication that background family factors (genetic or shared environmental confounds) underlie the phenotypic relationship. If, however, the within-pair regression remains substantial (especially, as shown by Carlin et al. [2005], if it is equal to the between-families regression) it means that *within MZ pairs*, the twin with the highest level of exposure is also the twin with the worst outcome; this result would suggest that exposure influences outcomes. Notably, this interpretation holds despite the fact that u_f and σ_{if} are independent; the control has accrued from the way the variation in *x* was partitioned.

The actual analysis employed in our paper differs from the presentation above in a couple of ways. First and least important, the analysis was conducted on a latent common factor rather than on a single observed *x*. Second, we are studying characteristics of the offspring of the twins, not the twins themselves. The children of twins design is an extension of the co-twin

control design, in which the outcome measure is a characteristic of the offspring. Third, we use structural equation modeling instead of random effects modeling to decompose the variance in *x*. Multivariate behavior genetic models can be analyzed using mixed-effects multi-level models or using latent-variable approaches (McArdle & Prescott, 2005). In fact, the mixed-effects multi-level models can be seen as variance component models (a specific parameterization of latent-variable models) for behavior genetic analyses. McArdle and Prescott (2005) demonstrated that both approaches yield the same results.

Fourth, instead of decomposing *x* into between- and within-pair portions as in Carlin et al. (2005), we use the between- and within-families variances in combination with zygosity to decompose *x* into ACE components. The ACE components are simply a reparameterization of the between and within components. Notably, this reparameterization of between and within variances into ACE variances represents the significant computational advantage of a structural equation approach to the problem, as compared to a multilevel model approach. Per the classical model, in MZ twins,

$$u_f = A + C$$
$$\sigma_{if} = E$$

while in DZ twins,

$$u_f = \tfrac{1}{2}A + C$$
$$\sigma_{if} = \tfrac{1}{2}A + E$$

Thus, one can regress the child functioning score, y_{if}, separately onto the Ax_{if} and Ex_{if} terms, with a residual Z_{if} (McArdle and Prescott, 2005).

$$Y_{if} = \mu_f + \beta_A A_{x\,if} + \beta_E E_{x\,if} + z_{if} \tag{1}$$

The residuals Z_{if} can be further decomposed into genetic and environmental portions, but we do not do so here, instead simply fitting a covariance between the residuals for the members of a pair, with separate residual covariances estimated for the identical and fraternal pairs.

It is important to consider the substantive interpretation of the unstandardized genetic and unique environmental regression coefficients β_A and β_E. The genetic regression coefficient β_A estimates the regression of *Y* onto genetic differences among twins, whether between families or within DZ pairs (in the classical twin model, the genetic effects between and within pairs are assumed to be equal). The β_E coefficient, which as we will show below is the key quantity of interest, estimates the regression of *y* on unique environmental differences in *x*. Within identical twin pairs, for example, differences are entirely defined by E. Suppose the relationship between *x* and *y* were entirely phenotypic, that is,

$$Y_{if} = \mu_f + \beta_P X_{if} + Z_{if} \tag{2}$$

with β_P the ordinary phenotypic regression of *y* on *x*. We can nevertheless still decompose *x* into the genetic and unique environmental components A_{xif} and E_{xif}.

Despite our assumption that the regression of *y* on *x* is phenotypic, suppose we fit Equation (1); that is, we fit a model with separate genetic and environmental regressions to data generated by a true model in which the regression is phenotypic. That is to say, given the phenotypic model in Equation (2), with $x_{if} = A_{if} + E_{if}$, we have

$$y_{if} = \mu_f + \beta_P(A_{if} + E_{if}) + Z_{if}, \tag{3}$$

which simplifies into

$$y_{if} = \mu_f + \beta_P A_{if} + \beta_P E_{if} + Z_{if} \tag{4}$$

Given Equation (4), a direct exposure model would result in

$$\beta_A = \beta_E = \beta_P$$

So when the true model is phenotypic (as, for example, when exposure to *x* is influencing *y* via phenotypic pathways) the genetic and environmental regression coefficients will be equal to each other, and equal to the phenotypic regression coefficient that underlies the model. This simply means that to the extent a unit change in the phenotype *x* produces β_p units of change in the phenotype of *y*, it does so regardless of the genetic or environmental origins of *x* (the interpretation of genetically informed unstandardized regressions is discussed in more detail in Turkheimer and Waldron, 2000).

Finally, instead of regressing *y* on the independent components of *x* (*u* and σ, or A and E) we regress *y* on one of the components (A) and on the full phenotype. This is just a reparameterization, which we find easier to interpret because our substantive hypotheses involve phenotypic effects. In the reparameterization, as noted by Carlin et al. (2005, p. 1096), the regression on the phenotype is equal to the within-pair regression. Again, this estimate reflects the influence of exposure. In this parameterization, the regression on A is equal to the difference between the A regression and the within-pair regression (the E regression in a standard Cholesky decomposition). Thus, the parameter associated with the regression on the *a* path in our model is interpreted as the influence of shared genetic factors that is above and beyond what would be expected given the influence of exposure. Carlin et al. (2005) also provide justification for the approach.

The overall approach is similar to econometric analyses using fixed-effects modeling to study variations within families as a way of examining the influence of exposure to environmental factors (e.g., Lin & Liu, 2009). With fixed-effects modeling, the outcome is regressed on the individual twin's score on the predictor and on the co-twin average on the predictor, which is equivalent to u_f in our approach.

In summary, our approach separates the predictor variable (e.g., family conflict) into separate components, based on the between and within variation across MZ and DZ twins. Offspring characteristics are then regressed on these components (and the parental measure) using a model that provides within-pair effects, as well as estimates of genetic confounding.

The substantive difference between the direct and indirect effects of family functioning on child functioning is manifest in the cross-covariation between the family functioning of one child of an identical twin and the childhood outcome of his or her cousin, as illustrated in

Figure 2, which depicts the associations for MZ families. The top panel of the figure illustrates a result in which the effect of family functioning on child outcome is purely phenotypic, with no association via common genes. In this case, the cross-covariance is equal to $b\sigma_A$, the direct effect multiplied by the covariance between the two family environments, which, without C in the model can only arise from the genetic correlation between the environments. The bottom panel of the Figure illustrates the result in which the phenotypic relation is entirely mediated by genetic effects in common with family functioning and child outcomes. In this case, b=0, and the regression of child outcome on A is equal to b/σ_A. Now, the cross-covariance between a child of an MZ twin and the family environment of his or her cousin, is the same as the covariance with his or her own family environment, or b.

Regarding the interpretation of the model, path b tests the contribution of exposure to family relationship problems on child adjustment, and path a tests the contributions of genetic factors to both family and child functioning. With path a included in the model, path b is estimated controlling for genetic factors (i.e. the b path estimates the independent association when accounting for shared genetic liability). Because MZ twins are identical in terms of both genes and shared environment (i.e., the parent generation, not the offspring generation), their only differences lie in the nonshared environment. Within an MZ co-twin family, if the child of the twin with more family problems has more adjustment problems than the cousin, path b will be relatively large. In contrast, if the cousins in MZ families have more similar levels of adjustment problems than cousins in DZ families, path a will be relatively large.

Notably, path a does not test whether there are genetic influences on child adjustment problems, but rather, whether the parents' genetic makeup underlies the association of family relationship problems with child adjustment problems. The model, however, does not control for environmental factors that differ between co-twin families (aside from the measured family functioning variables). For that reason, we also tested a model in which we controlled for several putative environmental risk factors. The outcome variables in this model were the residualized scores resulting from regressing child externalizing and internalizing scores on twin education, spouse education, and highest household occupation status, to control for differences in socioeconomic status (SES). This model assessed whether these factors—factors that can differ between co-twin families—are responsible for the associations between family functioning and child functioning.

In addition, because of the possibility that children perceive family relationships differently than their parents, we conducted two separate sets of tests for family conflict. One set included all three family members' reports of family conflict, and one set included only the child's reports of family conflict. Results of all model tests, including fit indices, unstandardized regression coefficients, variances of A and E, and residual offspring (cousin) correlations, are presented in Tables 3–6.

Results

Descriptive Statistics and Phenotypic Associations

Table 1 shows the means, standard deviations, and correlations among the variables, ignoring the nested nature of the data, to examine associations between variables at the phenotypic level. As shown in Table 1, all phenotypic correlations were significant in the expected direction.

Structural Equation Models

To calculate cross-twin-family and cross-trait correlations (shown in Table 2), we computed an SEM allowing all of the latent variables to correlate with one another, with corresponding pairs of correlations constrained to equality across twin families, and allowing MZ and DZ correlations to differ. In MZ families, offspring share 50% of their genes with their parent's co-twin, whereas in DZ families, offspring share only 25% of their genes with their parent's co-twin. Therefore, a larger cross-twin-family and cross-trait correlation in MZ families than in DZ families would indicate that genetic factors are responsible for associations between family relationship functioning and child adjustment, whereas equal correlations in MZ and DZ families would indicate that environmental factors are responsible for the associations (D'Onofrio et al., 2003). As indicated in Table 2, these cross-twin-family, cross-trait correlations, were similar for MZ and DZ families, implying that these associations are best explained by environmental factors. For example, in MZ families, correlations between family conflict in one twin family and offspring externalizing problems in the other twin family ($r_{MZ} = 0.10$, $p < .01$) were comparable to those in DZ families ($r_{DZ} = 0.08$, $p < .01$).

Family conflict models—We began by testing a model with only a path from family conflict to child functioning (see Table 3, Model 1). The model for externalizing showed acceptable fit to the data (RMSEA = 0.06, SRMR = 0.06), and path *b* was significant ($b = 0.61$, $p < .001$). Next, we added a path from the A component to externalizing (Table 3, Model 2). The model fit changed only slightly relative to Model 1; path *b* remained significant ($b = 0.58$, $p < .001$) and path *a* was not significant ($b = 0.05$, $p > .10$). Results for the residualized outcome variables were similar (Table 3, Model 3), both in terms of fit and in terms of regression coefficients. These results suggest that shared genetic liability and the measured covariates do not account for the association; rather, environmental factors specifically related to family conflict are responsible for elevated externalizing problems.

For internalizing, the model that included path *b* but not path *a* (Table 3, Model 1) showed acceptable fit to the data (RMSEA = 0.06, SRMR = 0.07), and path *b* was significant ($b = 0.38$, $p < .001$). Adding path *a* to the model (Table 3, Model 2) resulted in slight changes in model fit and a small decrease in the magnitude of path *b* ($b = 0.35$, $p < .001$), but the magnitude of path *a* was non-significant ($b = 0.09$, $p > .10$). Results were again similar for the residualized outcome variables (Table 3, Model 3) in model fit and in regression coefficients; see Figure 3 for comparison of Models 1–3. Thus, as with externalizing problems, the findings for internalizing problems suggest that genetic factors and the measured covariates do not account for the association with family conflict; the findings are instead consistent with the notion that exposure to family conflict is responsible for higher levels of internalizing problems.

Because of the possibility that children and parents perceive family conflict differently, we reran each of these models without parent reports of family conflict (i.e., using child report only). For the test with only a path from family conflict to child externalizing (see Table 4, Model 1), the model showed acceptable fit to the data (RMSEA = 0.00, SRMR = 0.04), and path *b* was significant ($b = 0.41$, $p < .001$). For the model with a path from the A component to externalizing (Table 4, Model 2), the model fit changed little relative to Model 1; path *b* remained significant ($b = 0.35$, $p < .001$), and there was a trend for path *a* ($b = 0.27$, $p < .10$). Results for the residualized outcome variables were similar (Table 4, Model 3) in terms of regression coefficients and model fit. These results suggest that environmental factors specifically related to family conflict play an especially important role in externalizing problems, and they also provide some evidence suggesting that shared genetic liability might also contribute to the association, although path *a* was only marginally significant.

For internalizing, the model that included only a path from family conflict to adjustment problems (Table 4, Model 1) showed acceptable fit to the data (RMSEA = 0.06, SRMR = 0.07), and path *b* was significant (*b* = 0.31, *p* < .001). Adding path *a* to the model (Table 4, Model 2) resulted in slight changes in model fit, but the magnitude of path *b* also decreased somewhat (*b* = 0.18, *p* < .001), and path *a* was significant (*b* = 0.56, *p* < .01). Results were again similar for the residualized outcome variables (Table 4, Model 3); see Figure 3 for comparison of Models 1–3. Thus, the findings for internalizing problems suggest that exposure to family conflict predicts higher levels of internalizing problems, and that genetic factors help account for part of this association.

Marital quality models—As with family conflict, for marital quality we first tested a model with only a path to child functioning (see Table 5, Model 1). The model for externalizing showed marginal fit to the data (RMSEA = 0.11, SRMR = 0.07), and path *b* was significant (*b* = −0.16, *p* < .001). Next, we added a path from A to externalizing (Table 5, Model 2), which resulted in minimal change in model fit. The magnitude of path *b* decreased considerably, becoming non-significant (*b* = −0.05, *p* > .10), and path *a* was marginally significant (*b* = −0.44, *p* < .10). Results were consistent with this for the residualized outcome variables (Table 5, Model 3); see Figure 3 for comparison of Models 1–3. This pattern of results suggests that exposure to low levels of marital quality has a modest relationship with externalizing problems. The results also suggest that genetic factors partially underlie the association between low marital quality and high levels of externalizing problems. However, given the similarity in fit between the models, and given the marginal significance of path *a*, the model that does not include path *a* is in some ways preferable.

For internalizing, the model that included only a path from marital quality to child adjustment problems (Table 5, Model 1) showed marginal fit to the data, and path *b* was significant. Adding path *a* to the model produced minimal change in model fit (Table 5, Model 2), and results were again fairly consistent for the residualized outcome variables (Table 5, Model 3). The results suggest that shared genetic liability does not account for the association between low marital quality and internalizing problems, suggesting instead that environmental factors specifically related to low levels of marital quality are responsible for elevated internalizing problems. Notably, none of the models for marital quality fit the data well.

Agreement about parenting models—We first tested a model with only a path from agreement about parenting to child functioning (see Table 6, Model 1). The model for externalizing fit the data very well (RMSEA = 0.00, SRMR = 0.05), and path *b* was significant (*b* = −0.21, *p* < .001). Next, we added a path from A to externalizing (Table 6, Model 2), which changed the model fit only slightly. The magnitude of path *b* decreased somewhat, but remained significant (*b* = −0.11, *p* < .05), and path *a* was also significant (*b* = −0.40, *p* < .05). Results were consistent with this for the residualized outcome variables (Table 6, Model 3); see Figure 3 for comparison. This pattern of results suggests that both genetic factors and exposure to (dis)agreement about parenting are important in the association with externalizing problems, although for reasons of parsimony, the simpler model (excluding path *a*) may be preferable.

For internalizing, the model that included only a path from agreement about parenting to child adjustment problems (Table 6, Model 1) fit the data well (RMSEA = 0.04, SRMR = 0.07), and path *b* was significant (*b* = −0.13, *p* < .001). Adding path *a* to the model (Table 6, Model 2) produced only slight changes in model fit, and the magnitude of path *a* was non-significant. Results were again fairly consistent for the residualized outcome variables (Table 6, Model 6). These findings suggest that genetic liabilities do not underlie the

association, and suggest instead that environmental factors specifically related to low marital agreement about parenting elevate internalizing problems.

Sensitivity tests—Because of the possibility of differences in family members' perceptions of child adjustment, we conducted additional analyses using parents' reports of child adjustment, in addition to children's reports. Bivariate correlations among all family members' reports of child internalizing and externalizing problems were positive and significant at the $p < .001$ level. We reran all of the SEM analyses using mother, father, and child reports of child adjustment as manifest indicators of a latent child adjustment variable. Results were essentially the same as those using only child report, in indicating the importance of exposure to family conflict, marital quality, and agreement about parenting for child internalizing and externalizing problems. The models also indicated the importance of examining the influence of genetic factors on associations between child-reported family conflict and externalizing and internalizing problems, and for the marital quality–externalizing association and the agreement about parenting–externalizing association. Details of these results are available from the authors upon request.

We also reran all of the SEM analyses separately for mothers' and fathers' individual reports of each of the family relationship constructs (details available upon request). Again, the results were essentially the same as the models using both parents' reports together; all of the models underscored the influence of exposure to family relationship problems on child adjustment problems. Further, the models showed patterns of influence of genetic factors on these associations that were consistent with those shown in Tables 3, 5, and 6.

Discussion

Consistent with calls for advancing knowledge of family and child functioning at a process level (Cummings et al., 2000), we used a genetically informative research design to separate out the influence of exposure to family relationship problems from the influence of genetic factors and from the influence of other environmental factors (Rutter et al., 2001). As a whole, the findings for family conflict, marital quality, and agreement about parenting highlight the importance of exposure to family relationship problems in explaining associations with child adjustment problems. The results also indicate that genetic factors contribute to some of these associations, illustrating the importance of considering shared genetic liability when studying family relations.

Specifically, for family conflict, results based on mother-, father-, and child-reported family conflict suggested exposure to family conflict is associated with elevated internalizing and externalizing problems. This also holds for models in which SES, a putative environmental risk factor, was statistically controlled. These findings are consistent with recent work suggesting that exposure to parental harsh physical discipline (Caspi et al., 2004; Lynch et al., 2006) and to parental divorce (Amato & Cheadle, 2008; Burt et al., 2008; D'Onofrio et al., 2005; 2006; O'Connor et al., 2000) may increase child psychopathology.

However, results based on only child-reported family conflict suggested that exposure, and to some extent, genetic factors, explain associations with externalizing problems, and that both exposure and genetic factors play important roles in associations with internalizing problems. That is, when only using child report, genetic factors appear to play a larger role than if parent reports are also used. Importantly, child report indexes children's perceptions of family conflict, as opposed to children's actual experiences with family conflict. Thus, one interpretation is that children's perceptions of family conflict might be particularly influenced by genetic factors. Our results might be tapping into genetically influenced ways of perceiving family functioning. Our findings build on previous work examining

differences in the extent to which genetic and shared and nonshared environmental factors contribute to different reporters' perceptions. For example, whereas father-reported positivity, negativity, monitoring, and control were all influenced by evocative genotype-environment correlation and father-reported negativity and monitoring were also influenced by passive genotype-environment correlation, adolescent-reported paternal positivity, negativity, monitoring, and control were all influenced by passive genotype-environment correlation and paternal positivity and negativity were also influenced by evocative genotype-environment correlation (Neiderhiser et al., 2007). Relatedly, Feinberg, Neiderhiser, Howe, and Hetherington (2001) found that the variance shared between parents' and children's reports of parental warmth and negativity was largely influenced by genetic factors, but that the unique variance in parents' perceptions of their own parenting was influenced primarily by shared environmental factors, and the unique variance in children's perceptions of parenting was influenced by nonshared environmental factors. Feinberg and colleagues suggested these findings might be due to parents' bias toward reporting that they treat their children similarly, and children's bias toward reporting more differential treatment.

Low marital quality appears to be associated with more child internalizing problems, independent of child and parent genotype. In contrast, genetic factors appear to explain associations between marital quality and externalizing problems, although this regression coefficient was only marginally significant. That is, genetic factors appear to underlie both low marital quality and elevated externalizing problems. The externalizing results are consistent with a recent study in which genetic factors explained the association between marital conflict and child conduct problems (Harden et al., 2007). Thus, the current study yields converging evidence regarding the role of genetic factors in associations between marital functioning and externalizing.

Examination of agreement about parenting highlighted the roles of both exposure and genetic factors. The models suggested that exposure to interparental disagreement about parenting is associated with internalizing problems, independent of genetic factors. The models also suggested that exposure to interparental disagreement about parenting is associated with higher levels of adolescent externalizing problems, and that genetic factors are also important in this association. As with marital quality, this finding is consistent with the findings of Harden and colleagues (2007). The findings for associations between the marital constructs and externalizing problems are consistent with those of Harden et al.'s (2007) study in pointing to the role of genetic factors in associations between marital functioning and child behavior problems. This consistency is particularly noteworthy given that these studies used different data collection methods, different reporters of marital functioning, samples from different countries, and different marital constructs and child behavior problems (marital conflict and conduct problems in the study by Harden and colleagues; marital quality, agreement about parenting, and externalizing problems in the current study). At the same time, our findings for marital quality and agreement about parenting diverge from findings in the literature on associations between divorce and child outcomes, which are more consistent with the notion that divorce increases child adjustment problems. However, our findings for associations with internalizing problems highlight the role of exposure, while also providing some evidence that genetic factors help account for associations between family conflict and outcomes when examining only children's reports of family conflict.

There may be important differences between family conflict (at least as parents perceive it) and marital relationship functioning that could partially explain these findings. For example, previous work suggests that family conflict is more strongly associated with child maladjustment than is poor marital functioning (Jaycox & Repetti, 1993). Jaycox and

Repetti speculated that the association between marital conflict and child adjustment in other studies may be partially explained by spillover of hostility from the marital relationship. However, David, Steele, Forehand, and Armistead (1996) found that, whereas marital dissatisfaction showed weaker associations with child maladjustment than family conflict did, marital conflict witnessed by the child was as strongly associated with child maladjustment as family conflict was. Fincham and Osborne (1993) speculated that effects of marital conflict on children may be particularly strong when children become involved in marital conflict (i.e., family conflict). Because our measure of marital quality included neither marital conflict per se, nor child involvement in marital conflict, it may have tapped only aspects of marital functioning that are less salient to children (and therefore less likely to impact child adjustment). In contrast, the marital agreement about parenting construct does represent a dimension of marital conflict, and it is a dimension of conflict that has implications for the child, or at least for the child's siblings. However, compared with marital conflict or other aspects of the marital relationship, family conflict presumably more directly involves children as participants in conflict. Relatedly, the shared environment has been found to explain most of the covariance between mother-adolescent and sibling-sibling relationships, suggesting that interaction processes in one relationship may spill over into other family relationships and/or serve as a model for interaction in these other relationships (Bussell et al., 1999).

Moreover, family conflict is a more severe form of family relationship dysfunction than are (low) marital quality and agreement about parenting. Thus, our findings suggest that the more problematic the exposure (i.e., family conflict) the more direct the effect on child behavior. For more benign dimensions of family functioning (i.e., marital quality, agreement about parenting), genetic factors are more important. That is, our results suggest that more severe, stressful family relationship problems have a greater impact on the development of adjustment problems in children. This conceptualization is consistent with findings from behavior genetic studies of divorce (e.g., Amato & Cheadle, 2008), which suggest that experiencing divorce—an especially severe and stressful relationship problem—is associated with elevated adjustment problems in offspring. This conceptualization is also consistent with findings suggesting that genetic factors contribute to associations between children's antisocial behavior and corporal punishment, but do not explain associations between children's antisocial behavior and physical abuse (Jaffee et al., 2004).

In the present study, we have focused on child behavior problems, but temperament-related characteristics also merit consideration, as they predict the development of behavior problems (see Rothbart & Bates, 2006 for a review). For example, because of temperament-related individual differences in reactivity, some children might be especially reactive (e.g., fearful and anxious) in response to a variety of environmental events, including family relationship problems, which in turn, might contribute to the development of internalizing problems. Similarly, temperament-related individual differences in self-regulation might mean that some children have more difficulty regulating their behavior in the context of stressors, including family-related ones, and these difficulties might, over time, contribute to the development of externalizing problems. A few recent behavior genetic studies have examined associations between temperament or personality and family relationships (see Loehlin, Neiderhiser, & Reiss, 2005, for example). Personality has been found to explain a moderate portion (26–42%) of the covariance between marital quality and parenting (Ganiban et al., 2009). Additional work is needed using genetically informed approaches to examine interrelations among family relationship problems and child temperament in the development of internalizing and externalizing problems.

Child age might partly explain associations between family relationship problems and child adjustment. Previous work has found that child age at time of parental divorce has

implications for adjustment. Parental divorce during elementary school is more strongly associated with both internalizing and externalizing problems than divorce during middle school (Lansford et al., 2006). Similarly, in a sample of children ranging in age from 9–18 years, the relationship between marital conflict and externalizing problems was stronger for younger children than for older children (Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings, 2006). Given that children in the current study were primarily middle-school-age or older (11–22 years), genetic and environmental contributions to adjustment problems could differ as a function of child age. Although a sample including younger children would be helpful in addressing this possibility, genetically informed longitudinal studies would provide a clearer answer, allowing questions regarding intraindividual change in children's sensitivity to family relationship problems to be addressed.

We could have used fixed effects regression (see Allison, 2009) instead of our SEM tests to address our research questions. Fixed effects regression has much in common with the approach used in the current paper and has been used with samples of twins (see for example Miller, Mulvey, and Martin, 1995). For example, as with our models, fixed effects regression can be used to examine whether differences between twin nuclear families can be accounted for by a key risk factor of interest on which the twins differ from each other. Further, both approaches can control for environmental factors that differ between twin extended families, and neither approach can control for factors that differ between twin nuclear families, and both approaches produce the same results. However, the approach used in the current study is more elegant, because it calculates the discrepancies between MZ twin pairs and the discrepancies between DZ twin pairs, and then, within the same model, compares the MZ discrepancies with the DZ discrepancies to determine the degree to which genetic factors influence the association between family functioning and child outcomes (indicated in path a).

Several limitations have implications for the conclusions that can be drawn from the current study. Because the data are cross-sectional the direction of effects cannot be disentangled. That is, it is possible that our findings suggesting family relationship problems influence child adjustment could instead reflect the influence of child adjustment on relationships. Recent research suggests that children influence various dimensions of family life, including parenting (Tucker, McHale, & Crouter, 2003) and marital conflict (Jenkins, Simpson, Dunn, Rasbash, & O'Connor, 2005; Schermerhorn, Chow, & Cummings, in press; Schermerhorn, Cummings, DeCarlo, & Davies, 2007), so there is a basis for conceptualizing our results in terms of both parent-to-child and child-to-parent effects. Moreover, findings from behavior genetics studies are consistent with evocative effects of children's genetically influenced characteristics on the environment. For example, research indicates that adopted children whose biological parents experienced psychological problems (indicating these children have genetic risk for psychological problems) received poorer parenting from their adoptive parents than adopted children not at genetic risk, and this association is mediated by the externalizing problems of children at genetic risk (Ge et al., 1996; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). In addition, more recent studies suggest child antisocial behavior may evoke corporal punishment (Jaffee et al., 2004) and parental negative feelings (Larsson, Viding, Rijsdijk, & Plomin, 2008), and child internalizing problems evoke maternal emotional over-involvement (Narusyte et al., 2008).

However, longitudinal studies demonstrating that marital difficulties precede offspring internalizing problems (e.g., Cummings et al., 2006) support the theory that marital difficulties increase adolescent internalizing problems. These findings, in combination with the findings in the current study (suggesting genetic factors do not account for associations between marital functioning and internalizing problems), support the view that the direction of effects is (at least in part) marital-to-child. Both family conflict and marital quality could

be evoked by unmeasured child heritable characteristics—neither phenotypic designs, nor the children-as-twin design, nor the COT design can rule out this possibility, but it could be assessed in an adoption design. Although a combination of longitudinal phenotypic data (that indicate temporal precedence for marital variables) and the current COT findings weight the parent-to-child direction more heavily, a more complete understanding of the association awaits the estimation of genetically-based child effects. Because the data in the current study are not longitudinal, they do not allow us to examine possible child effects. However, this paper does address other important issues that traditional family studies cannot address.

Our findings also do not account for characteristics of twins' spouses that may be inherited by their children (Eaves, Silberg, & Maes, 2005). In addition, there may also be aspects of the environment that influence only one twin family of a yoked pair (D'Onofrio et al., 2005). In the current study, we examined the possibility that the observed associations are actually caused by family SES. However, other factors, such as parental psychopathology or personality, could be the true source of the associations. Future work should investigate this possibility. Further, assortative mating, or the tendency to select a spouse who is similar to oneself, could have confounded the results; however, assortative mating does not typically represent a confound. In addition, the analyses do not account for differences in frequency of contact between MZ and DZ families (D'Onofrio et al., 2003). However, in this sample, although MZ families have more contact with each other than DZ families, frequency of contact is unrelated to outcomes (Pedersen et al., 1999). Finally, selection bias could have affected our results (Hernan, Hernandez-Diaz, & Robins, 2004). For example, we restricted the offspring sample to cousin pairs within 4 years of each others' age. However, these sorts of restrictions strengthen the paper in other ways. By excluding cousins who were not within 4 years of each others' age, we were able to decrease the influence of developmental factors on our results. Although all of these limitations have implications for the conclusions we can draw from this study, there are tradeoffs between internal validity and external validity (Shadish, Cook & Campbell, 2002). When converging evidence is obtained from studies utilizing different methods—with different strengths and weaknesses—that is particularly informative. Divergent findings are also important, as they provide clues about which methodological differences are important, which can also lead to advances in knowledge. Thus, in the current paper, we have endeavored to attend to both consistencies and inconsistencies in results across different studies.

Thus, the current study represents an important step toward a clearer understanding of the effects of exposure and of the contributions of genetic factors and environmental factors other than family relationship problems. We sought to examine whether findings from previous work, suggesting that experiencing parental divorce increases child adjustment problems would also apply to more typical forms of family relationship problems. Rather than downplaying either genetic or environmental processes, our findings point to the importance of *both* genetic factors and exposure to family relationship problems in associations between family and child functioning.

Acknowledgments

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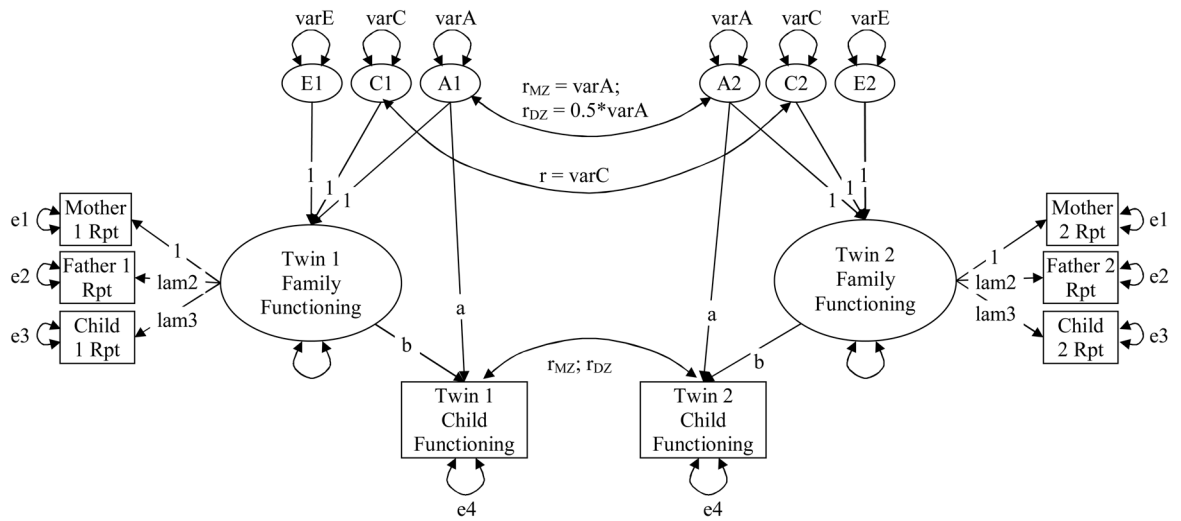
Figure 1.
Double-headed curved arrows represent variances. The direct path is labeled "b" and the path from the A component is labeled "a." r_{MZ} = cross-twin family correlation in MZ families; r_{DZ} = cross-twin family correlation in DZ families; Rpt = report; 1 = twin family 1 and 2 = twin family 2. Intercepts of the A, C, E, Family Functioning, and Child Functioning components were fixed equal to zero. Child Functioning consisted of children's self-reports.
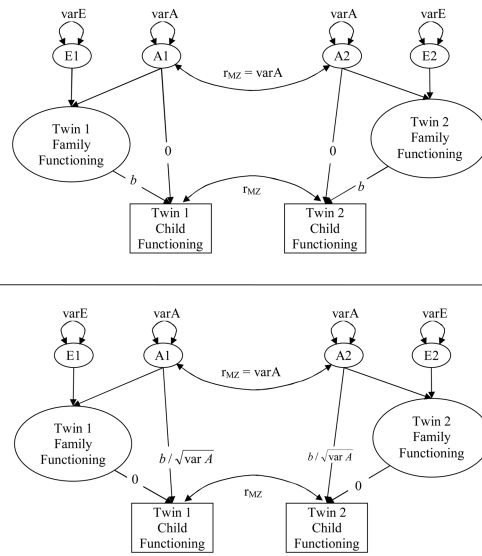
Figure 2.
Depiction of purely phenotypic association (top panel) and of purely genetic association (bottom panel). Some details that are included in Figure 1 (e.g., error terms) have been omitted from this figure, to simplify the presentation.
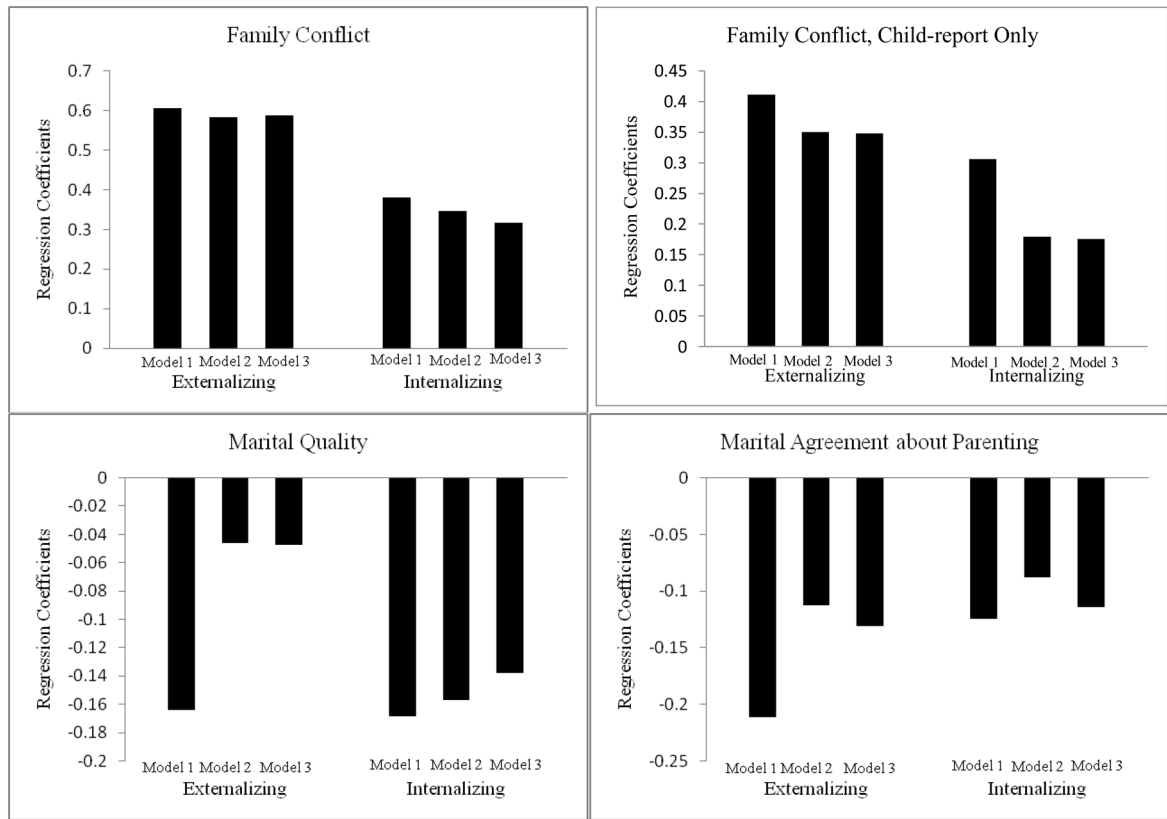
Figure 3.
Associations between family functioning and child functioning, using different controls. The bars represent the regression coefficients for path *b*. Model 1 depicts the association at the phenotypic level, Model 2 depicts the association controlling for unmeasured genetic and environmental factors, and Model 3 depicts the association controlling for unmeasured genetic and environmental factors, as well as measured SES. The regression coefficients in the graphs for marital quality and marital agreement about parenting are negative in sign because high levels of these constructs reflect positive relationship functioning; the relevant comparison for each relationship construct is the absolute length of the bars for Models 1–3 for the same outcome.

Table 1

Means, Standard Deviations, and Intercorrelations among the Variables at the Phenotypic Level

	FW	FH	FC	DCNW	DCNH	DCHW	DCHH	DSW	DSH	DAW	DAH	AP	EC	IC
FH	0.49	--												
FC	0.44	0.38	--											
DCNW	−0.34	−0.28	−0.18	--										
DCNH	−0.20	−0.38	−0.15	0.45	--									
DCHW	−0.20	−0.20	−0.13	0.50	0.31	--								
DCHH	−0.14	−0.22	−0.09	0.33	0.48	0.46	--							
DSW	−0.43	−0.37	−0.26	0.70	0.43	0.56	0.38	--						
DSH	−0.28	−0.47	−0.22	0.46	0.66	0.41	0.53	0.62	--					
DAW	−0.33	−0.25	−0.16	0.62	0.35	0.44	0.31	0.67	0.47	--				
DAH	−0.23	−0.37	−0.15	0.35	0.65	0.33	0.47	0.45	0.68	0.50	--			
AP	−0.31	−0.36	−0.24	0.57	0.54	0.31	0.30	0.47	0.41	0.39	0.37	--		
EC	0.20	0.19	0.41	−0.08[a]	−0.07[a]	−0.05[b]	−0.05[c]	−0.13	−0.10	−0.08	−.06[b]	−.22	--	
IC	0.12	0.13	0.31	−0.07[a]	−0.10	−0.06[b]	−0.05[c]	−0.10	−0.10	−0.09	−0.08[a]	−.13	0.43	--
M	14.25	13.40	13.95	64.92	64.13	18.97	18.84	48.95	49.90	17.98	17.91	−0.01	11.97	8.70
SD	3.56	3.28	3.58	7.74	7.40	4.15	3.96	5.57	5.31	2.88	2.84	1.66	6.23	6.62

Note. F = Family Environment Scale Conflict Subscale; DCN = Dyadic Adjustment Scale-Consensus, DCH = Dyadic Adjustment Scale-Cohesion, DS = Dyadic Adjustment Scale-Satisfaction, and DA = Dyadic Adjustment Scale-Affection Subscales; AP = Marital Adjustment Scale Agreement about Parenting Subscale; E = Child Behavior Checklist Externalizing Scale; I = Child Behavior Checklist Internalizing Scale; W = wife; H = husband; C = child.

Due to missing data, ns for the intercorrelations range from 1622 to 1725.

All correlations are significant at the $p < .001$ level, except as follows:

[a] $= p < .01$,

[b] $= p < .05$,

[c] $= p < .10$.

Table 2

Cross-twin Family Correlations

	FES Conflict 2	Marital Quality 2	Agree - Parenting 2	Externalizing 2	Internalizing 2
Monozygotic Twin Families					
FES Conflict 1	0.204***				
Marital Quality 1	−0.121***	0.163***			
Agree - Parenting 1	−0.124***	0.161***	0.288***		
Externalizing 1	0.100**	−0.064*	−0.112**	0.146**	
Internalizing 1	0.066*	−0.015	−0.041	0.086*	0.187***
Dizygotic Twin Families					
FES Conflict 1	0.042				
Marital Quality 1	−0.004	0.039			
Agree - Parenting 1	−0.035	0.054*	0.101*		
Externalizing 1	0.075**	−0.039	−0.089**	0.148**	
Internalizing 1	0.069*	−0.015	−0.021	0.082*	0.108*

Note. FES Conflict = Family Environment Scale Conflict Subscale; Marital Quality = Dyadic Adjustment Scale Consensus, Cohesion, Satisfaction, and Affection Subscales; Agree - Parenting = Marital Adjustment Scale Agreement about Parenting Subscale; Externalizing = Child Behavior Checklist Externalizing Scale; Internalizing = Child Behavior Checklist Internalizing Scale; 1 = Twin Family 1; 2 = Twin Family 2.

*
p < .05,

**
p < .01,

***
p < .001.

Table 3

Estimated Parameters of Structural Equation Models for Family Conflict (Husbands', Wives', and Children's Reports)

	Child Externalizing Problems		
	Model 1 (path *b*)	**Model 2 (paths *b* & *a*)**	**Model 3 (residualized)**
Fit indices			
BIC	18267.852	18274.553	17901.440
RMSEA	0.064	0.065	0.063
SRMR	0.056	0.056	0.056
χ^2	198.445	198.381	191.895
df	71	70	70
Parameters			
$\sigma^2(A)$	0.196^{***}	0.194^{***}	0.195^{***}
$\sigma^2(E)$	0.266^{***}	0.268^{***}	0.269^{***}
a	= 0	0.054	0.066
b	0.607^{***}	0.583^{***}	0.588^{***}
r_{MZ}	$0.085^{\dagger}$	$0.083^{\dagger}$	$0.087^{\dagger}$
r_{DZ}	0.091^{*}	0.090^{*}	0.068
	Child Internalizing Problems		
	Model 1 (path *b*)	Model 2 (paths *b* & *a*)	Model 3 (residualized)
Fit indices			
BIC	18177.710	18184.302	17842.968
RMSEA	0.062	0.063	0.062
SRMR	0.065	0.065	0.065
χ^2	189.553	189.380	184.918
df	71	70	70
Parameters			
$\sigma^2(A)$	0.210^{***}	0.208^{***}	0.208^{***}
$\sigma^2(E)$	0.298^{***}	0.300^{***}	0.299^{***}
a	= 0	0.087	0.162
b	0.381^{***}	0.346^{***}	0.318^{**}
r_{MZ}	0.165^{***}	0.162^{**}	0.125^{*}
r_{DZ}	0.071	0.070	$0.087^{\dagger}$

Note. $\sigma^2(A)$ = variance of A; $\sigma^2(E)$ = variance of E; a = unstandardized regression coefficient for path a; b = unstandardized regression coefficient for direct path; r_{MZ} = residual offspring (cousin) correlation for MZ twins; r_{DZ} = residual offspring (cousin) correlation for DZ twins.

$^{\dagger}p < 10$,

$^{*}p < .05$,

$^{**}p < .01$,

$^{***}p < .001$.

Table 4

Child Report Only: Estimated Parameters of Structural Equation Models for Family Conflict

	Child Externalizing Problems		
	Model 1 (path *b*)	Model 2 (paths *b* & *a*)	Model 3 (residualized)
Fit indices			
BIC	9320.356	9324.068	8960.780
RMSEA	0.000	0.000	0.000
SRMR	0.036	0.034	0.033
χ^2	14.009	10.956	10.972
df	22	21	21
Parameters			
$\sigma^2(A)$	0.243***	0.246***	0.247***
$\sigma^2(E)$	0.757***	0.755***	0.754***
a	= 0	0.266†	0.286†
b	0.411***	0.350***	0.348***
r_{MZ}	0.066	0.060	0.058
r_{DZ}	0.107**	0.100*	0.078†

	Child Internalizing Problems		
	Model 1 (path *b*)	Model 2 (paths *b* & *a*)	Model 3 (residualized)
Fit indices			
BIC	9277.139	9272.468	8930.323
RMSEA	0.040	0.022	0.009
SRMR	0.065	0.057	0.054
χ^2	36.910	25.474	21.686
df	22	21	21
Parameters			
$\sigma^2(A)$	0.246***	0.241***	0.246***
$\sigma^2(E)$	0.754***	0.759***	0.755***
a	= 0	0.555**	0.584**
b	0.306***	0.179***	0.175***
r_{MZ}	0.112*	0.060	0.029
r_{DZ}	0.053	0.033	0.041

Note. $\sigma^2(A)$ = variance of A; $\sigma^2(E)$ = variance of E; a = unstandardized regression coefficient for path a; b = unstandardized regression coefficient for direct path; r_{MZ} = residual offspring (cousin) correlation for MZ twins; r_{DZ} = residual offspring (cousin) correlation for DZ twins.

† $p < 10$,

* $p < .05$,

** $p < .01$,

*** $p < .001$.

Table 5

Estimated Parameters of Structural Equation Models for Marital Quality (Consensus, Cohesion, Satisfaction and Affection)

	Child Externalizing Problems		
	Model 1 (path *b*)	**Model 2 (paths *b* & *a*)**	**Model 3 (residualized)**
Fit indices			
BIC	37965.730	37968.725	37595.009
RMSEA	0.107	0.107	0.107
SRMR	0.069	0.069	0.069
χ^2	2036.046	2032.276	2037.021
df	340	339	339
Parameters			
$\sigma^2(A)$	0.141***	0.142***	0.141***
$\sigma^2(E)$	0.358***	0.357***	0.357***
a	= 0	−0.441†	−0.462†
b	−0.164***	−0.046	−0.047
r_{MZ}	0.129*	0.117*	0.120*
r_{DZ}	0.136**	0.127**	0.100*
	Child Internalizing Problems		
	Model 1 (path *b*)	Model 2 (paths *b* & *a*)	Model 3 (residualized)
Fit indices			
BIC	37769.163	37775.882	37433.453
RMSEA	0.108	0.108	0.108
SRMR	0.071	0.071	0.071
χ^2	2044.608	2044.562	2053.667
df	340	339	339
Parameters			
$\sigma^2(A)$	0.140***	0.140***	0.141***
$\sigma^2(E)$	0.358***	0.358***	0.358***
a	= 0	−0.049	−0.110
b	−0.169***	−0.157*	−0.138†
r_{MZ}	0.185***	0.185***	0.149**
r_{DZ}	0.107*	0.106*	0.132**

Note. σ (A) = variance of A; σ (E) = variance of E; a = unstandardized regression coefficient for path a; b = unstandardized regression coefficient for direct path; r_{MZ} = residual offspring (cousin) correlation for MZ twins; r_{DZ} = residual offspring (cousin) correlation for DZ twins.

†
p < 10,

*
p < .05,

**
p < .01,

***
p < .001.

Table 6

Estimated Parameters of Structural Equation Models for Marital Agreement about Parenting

	Child Externalizing Problems		
	Model 1 (path *b*)	**Model 2 (paths *b* & *a*)**	**Model 3 (residualized)**
Fit indices			
BIC	9645.712	9645.824	9274.596
RMSEA	0.000	0.000	0.000
SRMR	0.046	0.041	0.039
χ^2	20.078	13.425	10.828
df	22	21	21
Parameters			
$\sigma^2(A)$	0.263***	0.266***	0.265***
$\sigma^2(E)$	0.738***	0.735***	0.736***
a	= 0	−0.399*	−0.357*
b	−0.212***	−0.113*	−0.131**
r_{MZ}	0.108*	0.090†	0.103†
r_{DZ}	0.117**	0.102*	0.077†
	Child Internalizing Problems		
	Model 1 (path *b*)	Model 2 (paths *b* & *a*)	Model 3 (residualized)
Fit indices			
BIC	9501.262	9507.103	9159.060
RMSEA	0.035	0.036	0.035
SRMR	0.065	0.065	0.066
χ^2	33.521	32.596	32.166
df	22	21	21
Parameters			
$\sigma^2(A)$	0.261***	0.265***	0.264***
$\sigma^2(E)$	0.739***	0.737***	0.737***
a	= 0	−0.153	−0.110
b	−0.125***	−0.088†	−0.114*
r_{MZ}	0.176***	0.178***	0.143**
r_{DZ}	0.111*	0.107*	0.136**

Note. $\sigma^2(A)$ = variance of A; a = unstandardized regression coefficient for path a; b = unstandardized regression coefficient for direct path; $\sigma^2(E)$ = variance of E; r_{MZ} = residual offspring (cousin) correlation for MZ twins; r_{DZ} = residual offspring (cousin) correlation for DZ twins.

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