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Genetic and Environmental Influences on Global Family Conflict

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Abstract

This study examined genetic and environmental influences on global family conflict. The sample comprised 872 same-sex pairs of twin parents, their spouses/partners and one adolescent child per twin from the Twin and Offspring Study in Sweden (TOSS). The twins, spouses and child each reported on the degree of family conflict, and there was significant agreement among the family members' ratings. These shared perspectives were explained by one common factor, indexing global family conflict. Genetic influences explained 36% of the variance in this common factor, suggesting that twins' heritable characteristics contribute to family conflict, via genotype-environment correlation. Nonshared environmental effects explained the remaining 64% of this variance, indicating that twins' unique childhood and/or current family experiences also play an important role.

Keywords

family conflict; heritable characteristics; TOSS; FES; multi-rater assessment

Conflict, as indexed by the Family Environment Scale (FES) assesses global levels of hostility and aggression in the home (Moos & Moos, 1981). This may encompass an array of family relationships, including discord in family dyads or triads (Cox & Paley, 1997). Studies have documented both genetic and environmental influences on individuals' reports on the FES, with genetic effects explaining 18% to 30% of this variance (e.g., Kendler & Baker, 2007). These previous studies have used ratings by the twins, not other family members; thus, the meaning of the genetic variance is unclear. It may reflect single informant bias or twins' heritable characteristics shaping actual family conflict, as in genotype-environment correlation (*r*GE) (e.g., Scarr & McCartney, 1983). One solution for differentiating between these two explanations is to use a multi-rater assessment of FES as a proxy of family conflict (Deal, 1995). In this study, a twin, spouse and child from the same family each completed the FES,

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and we assessed whether the family members' shared perspectives were captured by a single common factor (representing global family conflict) and whether this common factor was explained by genetic and environmental influences. (Cox & Paley, 1997)

Different explanations have been proposed to describe how family conflict is shaped. According to the spillover model, conflict originating in one family dyad carries over to another family dyad, facilitating a continual pattern of conflict in these relationships (e.g., Margolin, Christensen, & John, 1996). The triangulation model focuses on conflict in the family triad (e.g., Minuchin, 1974). From this perspective, feuding spouses attempt to reduce their distress by involving the child in their marital conflict. For example, parents may form coalitions with or against the child, with the adverse consequence of fueling conflict of two family members against a third (e.g., Minuchin, 1974). A common theme in both of these models is that conflict arising from a single family dyad negatively impacts other family relationships, resulting in global family conflict.

A complementary explanation is that parents' heritable characteristics contribute to discord in family dyads and the triad, resulting in global family conflict. Research has shown that parental genetic variance contributes to parenting behaviors (e.g., Perusse, Neale, Heath, & Eaves, 1994), marital satisfaction (e.g., Spotts et al., 2004) and the covariation between parenting and marital satisfaction (Ganiban et al., 2007; Ganiban et al., 2009). These studies indicate that parents' heritable characteristics contribute to family dyads, but they do not account for triadic family relationships. Measuring FES from the perspective of the twin, spouse and child is a novel approach for examining global family conflict, which encompasses both dyadic and triadic family relationships (Cox & Paley, 1997).

We hypothesized the twins', spouses' and child's FES reports would be accounted for by a common factor (global family conflict), which would be explained by both genetic and environmental effects. Because genetic and environmental information comes from the twins, only their genetic and environmental influences can be discussed. Genetic influences would reflect the presence of *rGE* by which the twins' heritable characteristics lead to conflict in the home. Shared environmental effects would, in contrast, indicate that family conflict arises from shared conflict in the twins' rearing household and/or their current contact. Finally, nonshared environmental effects would illustrate that the twins' unique childhood and/or current family experiences contribute to global conflict in the home.

Method

Participants and Procedure

Participants were from the Twin and Offspring Study in Sweden (TOSS), including 909 same-sex monozygotic (MZ) and dizygotic (DZ) twin pairs (Neiderhiser & Lichtenstein, 2008). TOSS also included data from the twin's spouse/partner and adolescent child (see Ganiban et al., 2007 for details). TOSS was reviewed by the Institutional Review Board (IRB) in Sweden and the United States, and all participants provided informed consent before participating in the study. The current study included a subset of 876 twin pairs, including 327 paternal pairs and 549 maternal pairs. Zygosity was determined through the use of DNA for most twins with a subset being assigned zygosity based on questionnaires (see Ganiban et al., 2007 for details). Moreover, family conflict was assessed using the conflict sub-scale of the well validated and widely used FES (Moos & Moos, 1981). On a 5 item scale ranging from 1 (*not at all correct*) to 5 (*exactly correct*), twins, spouses and child each rated the extent of conflict in their home (e.g., "in our family we fight quite a lot").

Analytical Approach

Biometric model fitting estimated simultaneously the relative additive genetic (A), shared environmental (C) and nonshared environmental (E) variance components using the maximum-likelihood estimation of the raw data in Mx (Neale, 1997). See Plomin, DeFries, McClearn, & McGuffin (2008) for further details about twin modeling. Of note, biometric models assume that A, C and E act independently and a lack of assortative mating. Therefore, A, C and E estimates can be distorted in systematic ways in the presence of gene-environment interplay, including *r*GE (e.g., Jinks & Fulker, 1970) or when assortative mating is involved (Krueger, Moffitt, Caspi, Bleske, & Silva, 1998).

Multivariate biometric models estimated the degree to which genetic and environmental variance components explain covariation between phenotypes (in this case, the family members' FES ratings). Specifically, three different multivariate models were fit to the data, including the Cholesky, independent pathway and common pathway models. To determine which of these three models provided preferable fits, we compared the fit statistics of Akaike Information Criteria (AIC) and Bayesian Information Criteria (BIC) for each model. Lower AIC and BIC values indicated better fitting models. The common pathway model was selected as the model that fit the data best because the AIC and BIC values (AIC = 8224.50, BIC = -8110.30) were lower than the Cholesky (AIC = 8230.82, BIC = -8097.59) and independent pathway model fits (AIC = 8225.37, BIC = -8107.48). The common pathway model specified ACE latent factors on the family member's FES ratings that were mediated through one common latent factor and those specific to each FES rating (Figure 1). The A, C and E contributions to the latent factor were computed as: $(A1^2) + (C1^2) + (E1^2)$. The remaining variance specific to the FES report was parsed into A (a_1, a_2, a_3), C (c_1, c_2, c_3) and E (e_1, e_2, e_3) influences. In this model, measurement error was accounted for by the E variance specific to each phenotype.

Results

The family members' FES reports were all positively associated ($r = .38$ to $.47, p \leq .01$). In addition, twins' sex was related to the twins' FES reports ($r = .16$; twin mothers reported greater conflict) and child's sex was related to the child's FES reports ($r = .14$; daughters reported greater conflict). For these reasons, effects of twin's sex and child's sex were residualized from the twin's and child's FES reports, respectively (McGue & Bouchard, 1984). Regarding the common pathway model, a reduced model eliminating all C pathways and those A pathways specific to each FES measure provided a better fit as indicated by an insignificant increase in χ^2 and lower AIC and BIC values (reduced model: AIC = 8219.77, BIC = -8129.38, $\Delta\chi^2 = 9.27, p = .23$). A and E factors explained 36% and 64% the variance in this common latent factor, respectively. Also, the common factor explained 54%, 46% and 33%, of the variance in the twins', spouses' and child's FES reports, respectively. Remaining variance specific to each FES report was entirely attributable to E effects. Figure 1 display path estimates of full model.

Discussion

In this study, both genetic and nonshared environmental influences contributed to global family conflict. Genetic influences accounted for each individual's FES ratings, suggesting that this genetic variance cannot simply be attributed to single informant bias. Rather it may reflect that the twin's heritable characteristics (e.g., aggression or antisocial behavior) contribute to family conflict, via *r*GE. Consistent with evocative *r*GE, a twin's genetically-influenced characteristics may evoke negative responses from his/her spouse or child leading to spillover and/or triangulation and in turn, to family wide conflict (Margolin et al., 1996; Minuchin, 1974). Moreover, the spouse and child may have heritable characteristics similar to the twin

due to assortative mating and passive rGE respectively, causing overlapping perspectives of family conflict (Rutter & Silberg, 2002). In line with assortative mating, a twin may actively select a partner who is compatible with his/her genetically-influenced characteristics, although this explanation is more applicable to antisocial behavior than to personality traits (Krueger et al., 1998). The twin's heritable characteristics may also be genetically transmitted to the child and shape the conflict to which the child is exposed, as in passive rGE . Future research should delineate specific heritable characteristics in the twin parents and rGE processes explaining conflict in the home.

Nonshared environmental influences explained the largest component of variance in family conflict, perhaps reflecting the importance of the parent's actual experiences not shared with his/her co-twin. This nonshared environmental variance may include unique early rearing experiences (e.g. receiving less parental affection than the co-twin) that have been previously linked to psychopathology in adulthood (Kendler & Gardner, 2001). Distinct experiences occurring inside the current home may also be involved, including the twins' marriages to different spouses and stressful life events such as child drug abuse (Spotts et al., 2004; Towers, 2003). Each of these unique experiences may cause negative emotions in the twin that may, in turn, lead to family conflict via spillover and/or triangulation (Margolin et al., 1996; Minuchin, 1974). Nonshared environmental influences further contributed to the individual specific family conflict ratings, which include error variance and may also reflect unique experiences (e.g., role-related perspectives in the family) shaping individual specific perceptions family conflict. Future studies should employ the MZ discordant twin design to identify specific nonshared environmental factors shaping global family conflict and the individual-specific perceptions.

A limitation is that findings did not inform our understanding of the etiologies of different kinds of family conflict, such as daily hassles and severe arguments. Nor did findings inform our understanding of how genetic, shared environmental and nonshared environmental factors may vary at different levels of family conflict. At the same time, this was the first investigation to assess global family conflict using three family members' FES ratings. Results suggest that a parent's heritable characteristics and unique experiences contribute to family conflict, illuminating novel avenues for future research. In particular, research should specify which parental heritable characteristics account for the genetic variance in global family conflict. Another priority should be to identify the specific rGE mechanisms and nonshared environmental factors that are involved. This research will be crucial for continuing to delineate how genetic and environmental influences shape global conflict in the home.

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References

- Cox MJ, Paley B. Families as systems. *Annu Rev Psychol* 1997;48:243–267. [PubMed: 9046561]
- Deal JE. Utilizing data from multiple family members: A within-family approach. *Journal of Marriage and the Family* 1995;57:1109–1121.
- Ganiban JM, Spotts EL, Lichtenstein P, Khera GS, Reiss D, Neiderhiser JM. Can genetic factors explain the spillover of warmth and negativity across family relationships? *Twin Res Hum Genet* 2007;10(2): 299–313. [PubMed: 17564519]

- Ganiban JM, Ulbricht JA, Spotts EL, Lichtenstein P, Reiss D, Hansson K, et al. Understanding the role of personality in explaining associations between marital quality and parenting. *J Fam Psychol* 2009;23(5):646–660. [PubMed: 19803601]
- Jinks JL, Fulker DW. Comparison of the biometrical genetical, MAVA, and classical approaches to the analysis of human behavior. *Psychol Bull* 1970;73(5):311–349. [PubMed: 5528333]
- Kendler KS, Baker JH. Genetic influences on measures of the environment: a systematic review. *Psychol Med* 2007;37(5):615–626. [PubMed: 17176502]
- Kendler KS, Gardner CO. Monozygotic twins discordant for major depression: a preliminary exploration of the role of environmental experiences in the aetiology and course of illness. *Psychol Med* 2001;31(3):411–423. [PubMed: 11305849]
- Krueger RF, Moffitt TE, Caspi A, Bleske A, Silva PA. Assortative mating for antisocial behavior: developmental and methodological implications. *Behav Genet* 1998;28(3):173–186. [PubMed: 9670593]
- Margolin G, Christensen A, John RS. The continuance and spillover of everyday tensions in distressed and nondistressed families. *Journal of Family Psychology* 1996;10:304–321.
- McGue M, Bouchard TJ Jr. Adjustment of twin data for the effects of age and sex. *Behav Genet* 1984;14(4):325–343. [PubMed: 6542356]
- Minuchin, P. *Families and family therapy*. Harvard University Press; Cambridge, MA: 1974.
- Moos, RH.; Moos, BS. *Family Environmental Scale Manual*. Consulting Psychologists Press; Palo Alto, CA: 1981.
- Neale, MC. *Mx: Statistical modeling*. 3rd ed.. Virginia Commonwealth University; Richmond, VA: 1997.
- Neiderhiser JM, Lichtenstein P. The Twin and Offspring Study in Sweden: Advancing our understanding of genotype-environment interplay by studying twins and their families. *Acta Psychologica Sinica* 2008;40:1116–1123.
- Perusse D, Neale MC, Heath AC, Eaves LJ. Human parental behavior: evidence for genetic influence and potential implication for gene-culture transmission. *Behav Genet* 1994;24(4):327–335. [PubMed: 7993310]
- Plomin, R.; DeFries, J.; McClearn, GE.; McGuffin, P. *Behavioral Genetics*. 5th edition. New York: 2008.
- Rutter M, Silberg J. Gene-environment interplay in relation to emotional and behavioral disturbance. *Annu Rev Psychol* 2002;53:463–490. [PubMed: 11752493]
- Scarr S, McCartney K. How people make their own environments: a theory of genotype greater than environment effects. *Child Dev* 1983;54(2):424–435. [PubMed: 6683622]
- Spotts EL, Neiderhiser JM, Towers H, Hansson K, Lichtenstein P, Cederblad M, et al. Genetic and environmental influences on marital relationships. *J Fam Psychol* 2004;18(1):107–119. [PubMed: 14992614]
- Towers H. Contributions of current family factors and life events to women's adjustment: Nonshared environmental pathways (Doctoral dissertation, The George Washington University, 2003). *Dissertation Abstracts International* 2003;63(12-B):6123.

Figure 1.

Path estimates and 95% confidence intervals for full model. * = significant pathway estimates. A = latent additive genetic factor; C = latent shared environmental factor; E = latent nonshared environmental factor; a1, a2 and a3 = unique additive genetic contributions to each FES report; c1, c2 and c3 = unique shared environmental contributions to each FES report; e1, e2 and e3 = unique nonshared environmental contributions to each FES report; F₁, F₂ and E₃ = pathways linking the common latent factor to each FES report.