Moderation of Genetic Factors by Parental Divorce in Adolescents' Evaluations of Family Functioning and Subjective Wellbeing

Niels van der Aa,[1] Dorret I. Boomsma,[1] Irene Rebollo-Mesa,[2,1] James J. Hudziak,[3,1] and Meike Bartels[1,4]

[1] *Department of Biological Psychology, VU University, Amsterdam, The Netherlands*

[2] *Section of General Psychiatry, Institute of Psychiatry, King's College London, London, United Kingdom*

[3] *Departments of Psychiatry, Medicine, and Pediatrics, Vermont Center for Children, Youth and Families, University of Vermont, College of Medicine Burlington, Burlington, Vermont, United States of America*

[4] *EMGO+ Institute, VU University Medical Center, Amsterdam, the Netherlands*

Adolescents' evaluations of family functioning may have a significant impact on their subjective wellbeing and adjustment. The aim of the study was to investigate the degree to which genetic and environmental influences affect variation in evaluations of general family functioning, family conflict, and quality of life and the overlap between them. We assessed whether genetic and environmental influences are moderated by parental divorce by analyzing self-report data from 6,773 adolescent twins and their non-twin siblings. Genetic, shared, and nonshared environmental influences accounted for variation in general family functioning and family conflict, with genetic influences being relatively more important in girls than boys in general family functioning. Genetic and nonshared environmental influences accounted for variation in quality of life, with genetic influences being relatively more important in girls. Evidence was found for interaction between genetic factors and parental divorce: genetic influence on general family functioning was larger in participants from divorced families. The overlap between general family functioning and quality of life, and family conflict and quality of life was accounted for the largest part by genetic effects, with nonshared environmental effects accounting for the remaining part. By examining the data from monozygotic twins, we found evidence for interaction between genotype and nonshared, non-measured, environmental influences on evaluations of general family functioning, family conflict, and quality of life.

Keywords: family functioning, family conflict, subjective wellbeing, parental divorce, gene-environment interaction

The impact of family functioning, conflict, and cohesion on children's and adolescents' wellbeing and behavior has been a focus in research for decades. Positive family functioning characterized by supportive, close, and warm family relationships has been associated with better psychological adjustment in children and adolescents (e.g., Kurdek & Fine, 1994; Leary & Katz, 2004; McHale & Rasmussen, 1998). On the other hand, negative family functioning characterized by, for example, non-supportive family relationships and high levels of conflict has been linked to several negative outcomes such as low subjective wellbeing (McFarlane et al., 1995; Proctor et al., 2009), internalizing problems (e.g., Cui et al., 2007; Formoso et al., 2000; Hughes et al., 2008; Vandewater & Lansford, 2005), and externalizing problems (e.g., Cui et al., 2007; Formoso et al., 2000; Richmond & Stocker, 2006; Vandewater & Lansford, 2005). However, individual differences have been found in how adolescents evaluate their family functioning and subjective wellbeing (SWB) and the association between family functioning and adolescent SWB is presumed to be less due to true family experiences but more to the ways adolescents perceive and interpret these experiences and hence their family functioning (Harold et al., 1997; Millikan et al., 2002; Neiderhiser et al., 1998). In other words, only adolescents' evaluations of how their family functions are assumed to be directly associated with their SWB and adjustment. For example, Millikan et al. (2002) found that adolescents' but not parental evaluations of family relationships are directly associated with their self-reported internalizing symptoms. Neiderhiser et al. (1998) reported that adolescent perceptions of parenting mediate the association between actual observed parental behavior and adolescent adjustment. Harold et al. (1997) found that the adverse influence of parental conflict and hostility on adolescent internalizing and externalizing behavior was completely accounted for by adolescents' evaluations of these parental behaviors.

An increasing body of behavioral genetic research suggests that a substantive part of individual differ-

Received 10 February, 2010; accepted 3 March, 2010.

Address for correspondence: Niels van der Aa, Department of Biological Psychology, VU University, Van der Boechorststraat 1, 1081 BT Amsterdam, The Netherlands. E-mail: n.van.der.aa@psy.vu.nl

ences in the way adolescents evaluate family functioning can be accounted for by genetic factors (e.g., Herndon et al., 2005; Jacobson & Rowe, 1999; Neiderhiser et al., 1998). For example, Herndon et al. (2005) investigated genetic influence on adolescents' ratings of the 10 subscales of the Family Environment Scale (FES) and reported heritability estimates ranging between 20% and 40%, with no evidence for sex-differences. Jacobson and Rowe (1999), however, reported genetic influences on adolescents' evaluations of family connectedness that were twice as large in girls as compared to boys (58% vs. 26% respectively). Shared environmental influences were higher in boys (20% vs. 0% respectively). This finding argues for potential quantitative sex differences in genetic influences on evaluations of family functioning. Genetic factors also have been found to account for individual differences in evaluations of SWB (e.g., Bartels & Boomsma, 2009; Nes et al., 2006; Røysamb et al., 2002). For example, in a sample of Dutch adolescents Bartels and Boomsma (2009) reported that about half of the variation in SWB, including a measure of quality of life, could be accounted for by genetic influences with no evidence for sex differences. However, Røysamb et al. (2002) reported genetic influences on SWB to be larger in females than in males (55% vs. 46%), suggesting that quantitative sex differences in heritability may be present in SWB. In a recent paper, Bartels et al. (in press, this issue) found substantial differences in heritability of subjective happiness between males and females (22% vs. 41%). A variety of family factors, such as parental divorce, could contribute to individual differences in the way family functioning and SWB is evaluated. In a meta-analysis about the effect of parental divorce on wellbeing and adjustment, Amato and Keith (1991) reported that effect sizes are generally small. In general, literature regarding the effect of parental divorce on adolescents' evaluations of family functioning, SWB, and adjustment suggests heterogeneity in the way children and adolescents react to parental divorce. For example, some adolescents from divorced families have more conflicts with their parents (e.g., Dunn et al., 1998; O'Connor et al., 2001) and receive less emotional support, supervision, and involvement from their parents (e.g., Carlson & Corcoran, 2001), which is likely to lead to negative family functioning. Furthermore, some adolescents from divorced families show lower levels of SWB and adjustment compared to those from nondivorced families (Cuffe et al., 2005; Størksen et al., 2006). However, others have found that parental absence may only have deleterious effects on children's and adolescents' adjustment in certain circumstances. Specifically, the presence of the father in the family may only be beneficial to children's adjustment when he does not engage in antisocial behavior (Blazei et al., 2008; Jaffee et al., 2003). Others (McFarlane et al., 1995) have reported no significant differences with regard to evaluations of wellbeing and adjustment between adolescents from families with divorced parents and intact families. Finally, characteristics of the offspring themselves may contribute to the risk of divorce in their parents (Robbers et al., under review).

In addition to a main effect of parental divorce on evaluations of family functioning and SWB, parental divorce may affect evaluations of family functioning and SWB in an indirect way by complex interplays with genetic factors (e.g., Jaffee & Price, 2007; Kendler & Baker, 2007; Price & Jaffee, 2008). One possibility is that genetic factors control the degree of sensitivity to environmental factors such as parental divorce (i.e., gene-environment interaction; Kendler & Eaves, 1986). With respect to the current work, some adolescents may be genetically predisposed towards negative evaluations of family functioning and SWB whereas others will be genetically predisposed towards positive evaluations. Those who are genetically predisposed towards negative evaluations will be more sensitive to negative effects of parental divorce, whereas those who are genetically predisposed towards positive evaluations will be more sensitive to positive effects of parental divorce.

Adolescents' evaluations of family functioning have been associated with their evaluations of SWB and adjustment. Since individual differences in adolescents' evaluation of family functioning and SWB are substantially accounted for by genetic influences it is plausible that these genetic influences are also responsible for at least part of the association between family functioning and SWB (Jacobson & Rowe, 1999; Neiderhiser, 1998, Pike et al., 1996). For example, Jacobson and Rowe (1999) reported high genetic overlap between adolescents' reports of family connectedness and depressed mood for girls. Pike et al., (1996) reported high genetic overlap between adolescents' evaluations of maternal negativity and adolescent depression for boys and girls. Family functioning has many characteristics (e.g., cohesion, level of conflict, warmth) and these characteristics may be perceived in various ways. For example, an adolescent might evaluate the level of family conflict as high, whereas the level of family warmth might be evaluated as high as well. Genetic influences have been reported for different characteristics of family functioning (see Kendler & Baker, 2007; Plomin & Bergeman, 1991). In the present study, we investigate whether variation in the evaluation of SWB and different aspects of family functioning share the same underlying genetic and environmental influences. Since adolescents' evaluations of family functioning and adolescent adjustment are associated, information on the underlying source of this association is crucial for prevention and intervention.

In the current study two measures of family functioning and one measure of subjective wellbeing (SWB) were analyzed in adolescent twins and their non-twin siblings. Family functioning measures con-

sisted of evaluations of general family functioning (GFF; i.e., the overall health/pathology of a family) and evaluations of the level of family conflict (FC; i.e., the amount of openly expressed anger, aggression, and conflict among family members). SWB was assessed by asking adolescents to evaluate their quality of life in general (QLg). Other studies investigating the association between adolescent evaluations of family functioning and adjustment focused solely on psychopathology as an outcome measure (e.g., Harold et al., 1997; Millikan et al., 2002; Neiderhiser et al., 1998). Using QLg as an outcome measure for adolescent adjustment has the important advantage that it is sensitive to the entire spectrum of adjustment, and thus is an indicator of both wellbeing and psychopathology (Proctor et al., 2009). The data on GFF, FC, and QLg were collected in a large population of adolescent twins and their non-twin siblings. We tested whether genetic and environmental influences, including environmental influences specifically shared by twins and not by their siblings, affected variation in evaluations of GFF, FC, and QLg, and the overlap between GFF and QLg, and FC and QLg. The presence of environmental influences specifically shared by twins and not by their siblings implies that twin pairs share more of their environment than non-twin siblings (Eaves et al., 1999). We investigated whether the genetic architecture of GFF, FC, and QLg was different between boys and girls, and between participants from nondivorced and divorced families.

In addition to interaction between genetic and environmental factors and measured parental divorce (i.e., by definition an environmental factor that is shared by all member of a family), interaction between genetic factors and nonshared, nonmeasured, environmental influences was explored. This was done by looking at the association between intrapair sum and difference scores in monozygotic (MZ) twin pairs (Jinks & Fulker, 1970). Genetic and shared environmental influences add to the similarity of MZ twin pairs, whereas nonshared environmental influences add to differences within MZ twin pairs. When individuals who evaluate GFF as negative are more similar than individuals who evaluate family functioning as positive, this indicates that the latter group is more sensitive to environmental influences that are not shared by members of the same family.

Methods

Subjects

Participants were registered with the Netherlands Twin Registry (NTR), kept by the Department of Biological Psychology at the VU University in Amsterdam (Bartels et al., 2007; Boomsma et al., 2006). Parents of twins were first contacted to ask for consent to send their children a survey. If their parents consented, 14-, 16-, and 18-year old twins and their non-twin siblings received an online or paper & pencil self-report survey about the development of behavior, lifestyle, and wellbeing. When twins and siblings did not return the survey on time they were contacted by mail for a first reminder and next they were contacted by phone for a second reminder. A total of 4,912 families with twins born between 1986 and 1991 participated in this ongoing study so far. The overall family response rate was 56%.

Sibling data were included in the analyses if the siblings were older than 13 and younger than 20 years. For this article, data from one additional sibling per family were included. From families with more than one additional participating sibling, we selected the sibling closest in age to the twin. This resulted in exclusion of 401 siblings from the analyses because they were either too young or too old and the exclusion of another 35 siblings from families with more than one additional sibling. Data from 40 twins were excluded from the analyses due to missing data on zygosity. We excluded 261 subjects for whom it was uncertain which family they reported on, because they started a family on their own (i.e., having children themselves or living together with a partner). Another 87 individuals were excluded because one of their parents was deceased. This resulted in a total sample of 6,773 individuals of whom 5,773 were twins (44% male) and 1,000 were non-twin siblings (45% male), coming from 3,185 families. There were data from 441 (14%) incomplete and 2,666 (86%) complete twin pairs. In Table 1, the exact constellation of the participating families is presented. Participants came from intact families (88.5%) and families in which the biological parents were divorced (11.5%). The age of the twins ranged between 13.19 and 19.93 years, with a mean age of 16.05 years (SD = 1.59) and the age of the non-twin siblings ranged between 13.01 and 19.97 years with a mean age of 17.03 years (SD = 1.77). Participants were primarily Caucasian and they came from all regions of the Netherlands, including rural and urban areas.

For 1,089 (36.1%) of the same-sex twin pairs zygosity was determined based on blood group or DNA typing. Zygosity for the remaining same-sex twin pairs was determined by questionnaire items about physical similarities and confusion by family members and strangers. These items allow accurate determination of zygosity in 93% of same-sex twin pairs (Rietveld et al., 2000).

Measures

General Family Functioning (GFF) refers to adolescents' evaluations of the overall health or pathology of the family with regard to problem solving, communication, roles within the household, affection, and control. GFF was assessed with the subscale General Functioning from the McMaster Family Assessment Device (FAD) (Epstein et al., 1983). We used a Dutch translation of the subscale which shows good psychometrical properties (Wenniger et al., 1993). The subscale consists of 12 items which had to be answered on a 4-point scale ranging from 1 = *Strongly*

Table 1
Sample Constellation

	N of individuals	*N* of families	1 twin	2 twins	1 twin + sib	2 twins + sib
MZM	1061	474	28	290	15	141
DZM	917	425	56	232	14	123
MZF	1540	697	54	432	11	200
DZF	1116	512	49	309	13	141
DOS	2061	999	169	566	32	232
Sibs only	78	78	—	—	—	—
Total	6773	3185	356	1829	85	837

Note: *N* = Number; 1 twin = families with data from 1 twin; 2 twins = families with data from both members of the twin pair; 1 twin + sib = families with data from 1 twin and 1 additional sibling; 2 twins + sib = families with data from both members of the twin pair and 1 additional sibling; MZ = Monozygotic twin pair; DZ = Dizygotic twin pair; M = male; F = female; Sibs only = families with data from non-twin siblings only.

agree to 4 = *Strongly disagree*. Six items of the scale had to be recoded, because they were asked in opposite direction. Examples of items are 'planning family activities is difficult because we misunderstand each other' and 'in times of crisis we can turn to each other for support'. Scores on the individual items were summed to get an overall score for GFF which could range from 12 to 48 with high scores indicating high levels of GFF. A factor analysis of the items indicated one single factor that explained 40.51% of the variance. Internal consistency of the scale was good with a Chronbach's Alpha of .86.

Family Conflict (FC) refers to adolescents' evaluations of the amount of openly expressed anger, aggression, and conflict among family members. FC was assessed with the subscale Conflict from the Family Environment Scale (FES) (Moos, 1974). We used a Dutch translation of this subscale, which shows satisfactory psychometric properties (De Coole & Jansma, 1983). The subscale consists of 11 items which had to be answered on a 2-point scale ranging from 1 = *No* to 2 = *Yes*. Examples of items are 'We argue a lot at home' and 'Family members criticize each other frequently'. One item in the scale ('We seldom get openly angry at each other at home') was removed from the scale because it was clearly misunderstood by the participants. Answering 'yes' on this item implies a low level of family conflict, whereas answering 'yes' on the other items implies a high level of family conflict. Many participants appeared not to realize this. Furthermore, the number of missing values on this item was far higher as compared to the other items (458 vs. 46–115). Scores of the remaining 10 items were summed to get an overall score for FC. These scores could range between 10 and 20 with high scores indicating low levels of FC (a positive correlation between the GFF scale and the FC scale therefore means that high levels of GFF are associated with low levels of FC). Factor analysis of the FC scale based on 10 items revealed one single factor that explained 26.40% of the variance. Internal consistency of the scale was acceptable with a Chronbach's Alpha of .68.

Evaluations of quality of life in general (QLg) was assessed with the Cantril Ladder (Cantril, 1965). The ladder has 10 steps: the top indicated the best possible life, and the bottom the worst possible life. Participants had to indicate the step of the ladder at which they place their lives *in general*.

Parental divorce was assessed by asking two questions. Participants were asked whether their parents divorced: 1 = *No*, 2 = *Yes, less than 2 years ago*, 3 = *Yes, more than 2 years ago*. With the second item participants were asked what living situation applied to them: 1 = *Living with both biological parents*, 2 = *Living with biological mother*, 3 = *Living with biological father*, 4 = *Living with biological mother and her new partner*, 5 = *Living with biological father and his new partner*, 6 = *Other living situation*. Reports on both items had to indicate that the biological parents were divorced. If the reports on both items were incongruent, parental reports on their children's behavior at age 12 were examined in order to establish whether or not the biological parents of the twins and siblings were divorced. The resulting divorce measure consisted of two categories 0 = *Intact family* and 1 = *Families in which biological parents were divorced*, and it was equal for all members of the same family.

Univariate Saturated and Genetic Analyses

The data on GFF, FC, and QLg were first analyzed with univariate genetic models. The data were structured into entire family units consisting of two or three individuals (i.e., two twins and one additional sibling) with missing data for families without siblings. In three so-called saturated models, means and variances for GFF, FC, and QLg were estimated conditional on sex and age. In addition, twin and twin-sibling correlations within traits were obtained. All parameters were allowed to differ between nondivorced and divorced families. This was done in the software package Mx (Neale et al., 2006). In the saturated models, we first tested whether sex and age influenced individual differences in mean levels of GFF, FC, and QLg. These variables were included as fixed effects (covariates) in the means model. Under

this model, an individual's score (*Y*) on GFF, FC, and QLg for nondivorced (ND) and divorced (D) families respectively can be expressed as:

$$Y_{i.ND} = \mu_{ND} + \beta_{sex,ND}\,\mathrm{sex}_i + \beta_{age,ND}\,\mathrm{age}_i + \varepsilon_{i,ND} \quad (1)$$

$$Y_{i,D} = \mu_{D} + \beta_{sex,D}\,\mathrm{sex}_i + \beta_{age,D}\,\mathrm{age}_i + \varepsilon_{i,D} \quad (2)$$

where subscript *i* indicates the individual, μ the population mean, and ε the residual. We tested whether constraining each regression weight at zero led to a significant deterioration of model fit. In addition, we assessed whether mean levels of GFF, FC, and QLg were different between nondivorced and divorced families by testing whether constraining means of both family types to be equal led to a significant deterioration of model fit.

Next, we tested whether twin and sibling correlations for GFF, FC, and QLg were equal for DZ twins and siblings and for MZ twins and DZ twins/siblings from nondivorced and divorced families. Estimating correlations for MZ twin, DZ twin, and twin-sibling pairs constitutes a first step in evaluating the relative influence of genetic and environmental factors on trait variances. More specifically, when the MZ correlation is higher for a certain trait than the DZ and the non-twin sibling correlations, it is inferred that genetic variation influences individual differences in the trait under study. DZ/sib correlations higher than half the MZ correlation implies shared environmental effects referring to environmental influences shared by *all* siblings reared in the same family. When the DZ correlation is higher than the twin-sibling correlation, a specific environment might exist which is shared by twins but not by non-twin siblings (Eaves et al., 1999). The remaining variation is attributed to nonshared environment which refers to environmental influences that are not shared by family members. The nonshared environmental variance component also includes measurement error variance. We estimated correlations for MZ twin, DZ, twin, and twin-sibling pairs as a function of sex and parental divorce.

Using structural equation modeling in Mx, genetic models were fitted to the data in which the genetic architecture of GFF, FC, and QLg was specified. A graphical representation of the genetic model for GFF is given in figure 1. This representation also holds for the genetic models of FC and QLg. The amount of variance in a single trait due to additive genetic effects (A), shared environmental effects (C), and nonshared environmental effects (E) can be estimated by considering the different levels of genetic relatedness between MZ and DZ twin pairs and non-twin siblings. MZ twin pairs are genetically identical, whereas DZ twin pairs and non-twin siblings share on average 50% of their genetic material. A variance component comprising twin specific environment was not modeled, because twins and siblings did not differ in correlational structure. The influence of A, C and E is represented by path coefficients *a*, *c*, and *e*. Parameter estimates of *a*, *c*, and *e* were allowed to differ for individuals from nondivorced and divorced families. Significant covariates (sex and/or age) were retained in the means model.

To test for interaction between sex and genetic and environmental influences, the method as proposed by Purcell (2002) was used. The effect of sex was included as a moderator on the path coefficients *a*, *c*, and *e* and it was allowed to be different between nondivorced and divorced families. This can be seen in Figure 1, in which the path coefficients of the latent factors are expressed as linear functions of the moderator, which are different between nondivorced and divorced families. Under this model, for each individual in the family conditional on the individuals' sex, the expected trait variance (ε_i) of GFF, FC, and QLg for nondivorced and divorced families respectively can be expressed as:

$$\varepsilon_{i,ND} = (a_{ND} + \alpha_{ND}\mathrm{sex}_i)^2 + (c_{ND} + \gamma_{ND}\mathrm{sex}_i)^2 + (e_{ND} + \eta_{ND}\mathrm{sex}_i)^2 \quad (3)$$

$$\varepsilon_{i,D} = (a_{D} + \alpha_{D}\mathrm{sex}_i)^2 + (c_{D} + \gamma_{D}\mathrm{sex}_i)^2 + (e_{D} + \eta_{D}\mathrm{sex}_i)^2 \quad (4)$$

For pairs of relatives (i.e., MZ twins, DZ twins, and twin-sibling pairs) the within-trait covariance of GFF, FC, and QLg for nondivorced and divorced families respectively can be expressed as:

$$Cov_{\varepsilon 1ND,\varepsilon 2ND} = r_g(a_{ND} + \alpha_{ND}\mathrm{sex}_1)(a_{ND} + \alpha_{ND}\mathrm{sex}_2) + (c_{ND} + \gamma_{ND}\mathrm{sex}_1)(c_{ND} + \gamma_{ND}\mathrm{sex}_2) \quad (5)$$

$$Cov_{\varepsilon 1D,\varepsilon 2D} = r_g(a_{D} + \alpha_{D}\mathrm{sex}_1)(a_{D} + \alpha_{D}\mathrm{sex}_2) + (c_{D} + \gamma_{D}\mathrm{sex}_1)(c_{D} + \gamma_{D}\mathrm{sex}_2) \quad (6)$$

where r_g is 1.0 for MZ twin pairs, and 0.5 for DZ twin and twin-sibling pairs.

Under this model, *a*, *c*, and *e* represent the unmoderated variance components, and the α-, γ- and η-coefficients represent the moderating effects of sex on the genetic, shared environmental, and nonshared environmental variance components respectively. If for example, under this model, α_{ND} is significantly different from zero, this is evidence for an interaction between the latent genetic factor of GFF, FC, or QLg and sex in nondivorced families. In the same way interactions between sex and both latent environmental factors can be detected as well. We assessed the significance of the sex effects by testing whether fixing the α-, γ- and η-coefficients to zero resulted in a significant deterioration of model fit.

To assess interaction between parental divorce and genetic and environmental influences, we tested whether constraining the genetic ($a_{ND} = a_D$), shared environmental ($c_{ND} = c_D$), and nonshared environmental ($e_{ND} = e_D$) parameter estimate of GFF, FC, and QLg to be equal between nondivorced and divorced families resulted in a significant deterioration of model fit. If for example, the genetic parameter estimate of GFF in nondivorced families (a_{ND}) is significantly different from the genetic

parameter estimate in divorced families (a_D), this is evidence for an interaction between the latent genetic factor of GFF and parental divorce. In the same way interactions between parental divorce and the latent environmental factors can be detected as well. The presence of these interactions implies that genetic, shared environmental, and nonshared environmental influences on GFF, FC, and QLg increase or decrease as a function of parental divorce. A complicating issue is that interaction between parental divorce and genetic and environmental influences can be sex specific as well. To assess if this is the case we tested whether constraining the α-, γ- and η-coefficients between nondivorced and divorced families to be equal ($\alpha_{ND} = \alpha_D$; $\gamma_{ND} = \gamma_D$; $\eta_{ND} = \eta_D$) led to a significant deterioration of model fit. If for example, the α-, γ- and η-coefficients of GFF in nondivorced families are significantly different from the α-, γ- and η-coefficients in divorced families, this indicates that the genetic architecture of GFF is different between nondivorced and divorced families in boys and girls.

Bivariate Saturated and Genetic Analyses

To examine the overlap between both measures of family functioning and quality of live in general, two bivariate models were fitted to the data. First, phenotypic correlations between GFF and QLg and between FC and QLg, and cross-twin cross-trait correlations were obtained. Just as twin and twin-sibling correlations *within* traits provide information on the relative influence of genetic and environmental factors on trait variances, cross-twin cross-traits correlations provide information about the relative influence of these factors on the covariation between traits. Bivariate genetic models were fitted to the data in which genetic and environmental influences on the covariation between GFF and QLg, and between FC and QLg were estimated. The shared environmental covariance component was not modeled, because the

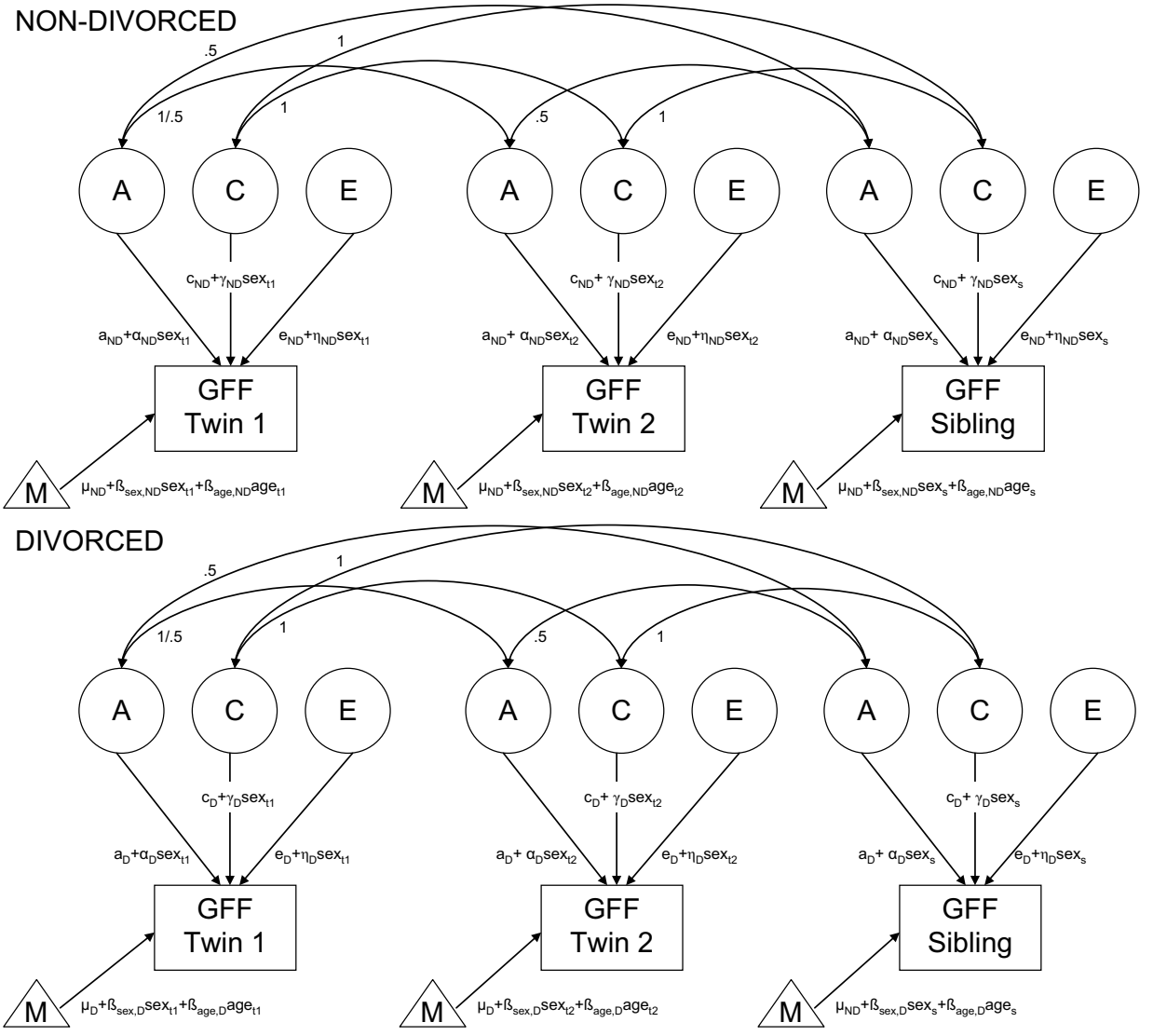

Figure 1

Univariate genetic model for general family functioning (GFF) with moderating effects of sex. Note. GFF = general family functioning; M = moderator.

contribution of shared environmental influences to variation in QLg was negligible. A graphical representation of the bivariate genetic model for the overlap between GFF and QLg is given in figure 2. This representation also holds for the genetic bivariate model for FC and QLg. Constraints on the parameter estimates of trait variances were adopted from the best fitting univariate models. Parameter estimates of the covariation between traits were estimated without constraints. The effect of sex on the covariance between traits was included as a moderator on the path coefficients and all parameter estimates were allowed to differ for individuals from nondivorced and divorced families. Under this model, for each individual (*i*) in the family conditional on the individual's sex, the observed (within-person) cross-trait covariance (*CrossC,* i.e., the covariance of the residuals (ε) for each trait) for nondivorced (ND) and divorced families (D) respectively can be expressed as:

$$CrossC_{i,ND} = (a_{11,ND} + \alpha_{11,ND}\text{sex}_i)\,(a_{21,ND} + \alpha_{21,ND}\text{sex}_i) + (e_{11,ND} + \eta_{11,ND}\text{sex}_i)\,(e_{21,ND} + \eta_{21,ND}\text{sex}_i) \quad (7)$$

$$CrossC_{i,D} = (a_{11,D} + \alpha_{11,D}\text{sex}_i)\,(a_{21,D} + \alpha_{21,D}\text{sex}_i) + (e_{11,D} + \eta_{11,D}\text{sex}_i)\,(e_{21,D} + \eta_{21,D}\text{sex}_i) \quad (8)$$

For pairs of relatives the cross-trait covariance (CrossC) for nondivorced and divorced families respectively can be expressed as:

$$CrossC_{1,2,ND} = r_g(a_{11,ND} + \alpha_{11,ND}\text{sex}_1)\,(a_{21,ND} + \alpha_{21,ND}\text{sex}_2) \quad (9)$$

$$CrossC_{1,2,D} = r_g(a_{11,D} + \alpha_{11,D}\text{sex}_1)\,(a_{21,D} + \alpha_{21,D}\text{sex}_2) \quad (10)$$

Where 1 and 2 refer to different traits in twin 1 and twin 2 (see Figure 2) and r_g is 1.0 for MZ twin pairs, and 0.5 for DZ twin and twin-sibling pairs. We assessed the significance of the sex effects by testing whether fixing the α- and η-coefficients linked to the covariance components to zero resulted in a significant deterioration of model fit.

To assess interaction between parental divorce and genetic and environmental influences on the overlap between GFF and QLg, and FC and QLg we tested whether constraining the genetic ($a_{21,ND} = a_{21,D}$) and nonshared environmental ($e_{21,ND} = e_{21,D}$) parameter estimate to be equal between nondivorced and divorced families resulted in a significant deterioration of model fit. The presence of these interactions implies that genetic and nonshared environmental influences on the overlap between GFF and QLg, and between FC and QLg increase or decrease as a function of parental divorce. A complicating issue is that interaction between parental divorce and genetic and environmental influences on the overlap can be sex specific as well. To assess if this is the case we tested whether constraining the α- and η-coefficients between nondivorced and divorced families to be equal ($\alpha_{ND} = \alpha_D$; $\eta_{ND} = \eta_D$) led to a significant deterioration of model fit. If for example, the α- and η-coefficients of GFF in nondivorced families are significantly different from the α- and η-coefficients in divorced families, this indicates that the genetic architecture of the overlap between GFF and QLg and between FC and QLg is different between nondivorced and divorced families in boys and girls.

The fit of the different models was compared by the log-likelihood ratio test (LRT). The difference in minus two times the log-likelihood (–2LL) between two models has a χ^2 distribution with the degrees of freedom (Δdf) equaling the difference in *df* between the two models. The *df* of a model equals the difference in the number observations and the number of model parameters. If a *p* value higher than 0.05 was obtained from the χ^2-test the fit of the constrained model was not significantly worse than the fit of the more complex model. In this case, the constrained model was kept as the most parsimonious and best fitting model.

Interaction Between Genetic Influences and Nonshared Environment

To explore the interaction between genotype and nonshared, non-measured, environmental influences, correlations between the sum scores and absolute difference scores within MZ twin pairs were computed (Jinks & Fulker, 1970). By looking at the association between intrapair differences and sum scores in MZ twins the assumption of independency of genetic and nonshared environmental influences can be determined. In the present study, 1,171 families with monozygotic twins participated. In each family (*n*) twins have scores s_{n1} and s_{n2} on, for example, GFF. Each absolute intrapair difference score (i.e., $\text{abs}(s_{n1} - s_{n2})$) provides an estimate of the magnitude of nonshared environmental influences within families. There will be variation in these intrapair differences, because twins in some families are likely to react differently from those in other families when exposed to environmental influences, or because twins in some families are exposed to different environments than in other families. On the other hand, variation in the sum of intrapair twin scores (i.e., $s_{n1} + s_{n2}$) appears if twins belonging to different families have different genotypes and/or family environments. When there is a negative correlation between intrapair sum and absolute differences, individuals who evaluate GFF as negative are more different than those who evaluate GFF as positive, and thus individuals who evaluate GFF as positive are less susceptible to unique environmental influences (e.g., Van Leeuwen et al., 2007). Analyses were done in SPSS (version 16.0).

Results

Univariate Saturated Models

In Table 2, means and variances of GFF, FC, and QLg (uncorrected for age) are presented as a function of parental divorce and sex. Sex and age effects on means

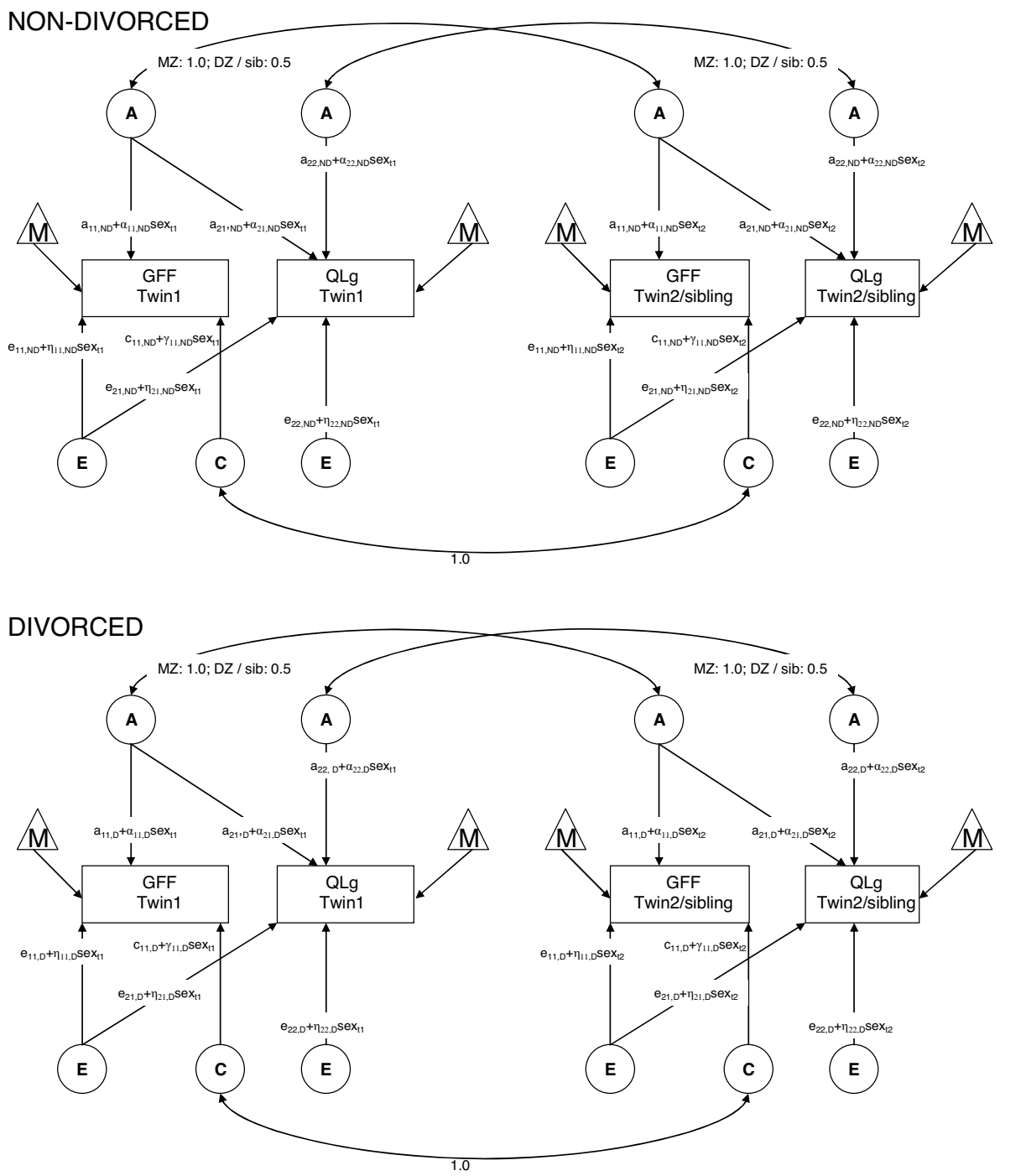

Figure 2

Bivariate genetic model for general family functioning (GFF) and quality of life in general (QLg) with moderating effects of sex. Note. GFF = general family functioning; QLg = quality of life in general; M = moderator.

of GFF (χ^2 (2) = .58, p = .75) and FC (χ^2 (2) = 1.87, p = .39) and the age effect on the mean of QLg (χ^2 (1) = .11, p = .74) were not significantly different between nondivorced and divorced families. The sex effect on the mean of QLg was significantly different between nondivorced and divorced families (χ^2 (1) = 11.54, p < .01). The effect of sex on the means of GFF (χ^2 (1) = .004, p = .95) and FC (χ^2 (1) = 3.67, p = .06) were not significant, whereas the effect of sex on the mean of QLg was significant for nondivorced (χ^2 (1) = 12.09, p <.01) and divorced families (χ^2 (1) = 22.09, p < .01). In nondivorced and divorced families boys reported higher levels of QLg than girls. There was a small but significant effect of age on the mean of GFF (χ^2 (1) = 25.71, p < .01; standardized regression coefficient β = –.07; r^2 = .005) and QLg (χ^2 (1) = 46.50, p < .01; standardized regression

Table 2

Maximum Likelihood Estimates of Means and Variances of GFF, FC, and QLg as a Function of Sex and Parental Divorce

			M	Var
GFF	ND	Boys	38.7	24.1
		Girls	38.6	26.0
	D	Boys	37.5	23.3
		Girls	37.6	32.0
FC	ND	Boys	16.7	4.8
		Girls	16.6	4.9
	D	Boys	16.5	4.9
		Girls	16.3	5.1
QLg	ND	Boys	7.9	1.1
		Girls	7.8	1.2
	D	Boys	7.8	1.1
		Girls	7.3	1.9

Note: GFF = general family functioning; FC = family conflict; QLg = quality of life general; ND = nondivorced families; D = divorced families.

coefficient $\beta = -.09$; $r^2 = .008$), indicating that means were higher for younger participants. No significant age effect was found on the mean of FC. Means of GFF (χ^2 (1) = 18.23, $p < .01$), FC (χ^2 (1) = 4.76, $p < .05$), and QLg (χ^2 (1) = 5.16, $p < .05$) were significantly lower for individuals from divorced families compared to those from nondivorced families. As can be seen in Table 2, variation in GFF, FC, and QLg is the same or higher for participants from divorced families compared to those from nondivorced families, indicating that participants living in divorced families form an equally or even more heterogeneous group regarding scores on GFF, FC, and QLg compared to those living in nondivorced families. This suggests that the effects of living in nondivorced or divorced families have a positive influence on reports of GFF, FC, and QLg for some participants and a negative influence for others.

Twin and twin-sibling correlations for GFF, FC, and QLg are presented in the upper part of Table 3. Constraining the sex effects on the covariance structure for nondivorced and divorced families to be equal led to a significant deterioration of model fit for GFF (χ^2 (1) = 4.78, $p < .05$) and QLg (χ^2 (1) = 19.21, $p < .01$), but not for FC (χ^2 (1) = .004, $p = .95$). The significance of the sex effects on the covariance structure of GFF and QLg was therefore tested for nondivorced and divorced families separately. For GFF, there were significant sex differences on the covariance structure for participants from nondivorced (χ^2 (1) = 4.61, $p < .05$) and divorced families (χ^2 (1) = 9.05, $p < .01$). For QLg, there also were significant sex differences on the covariance structure for participants from nondivorced (χ^2 (1) = 8.40, $p < .01$) and divorced families (χ^2 (1) = 29.42, $p < .01$). No significant sex differences were found on the covariance structure of FC. No indication was found for environmental influences specifically shared by twins explaining variation in GFF, FC, and QLg for nondivorced and divorced families (p values exceeded a 5% significance level). For GFF, FC and QLg in nondivorced families, the MZ twin correlation was larger than the DZ twin and twin–sibling correlation (all p values < .01). For GFF and QLg in divorced families, the MZ twin correlation was equal to the DZ twin and twin–sibling correlation (both p-values > .05). For FC in divorced families, the MZ twin correlation was larger than the DZ twin and twin–sibling correlation (χ^2 (1) = 11.71, $p < .01$). Because the correlational structure of the data was not identical for participants from nondivorced and divorced families and between boys and girls, we started genetic modeling with an ACE model with different parameter estimates for participants from nondivorced and divorced families and

Table 3

Familial and Cross-Twin Cross-Trait Correlation for GFF, FC, and QLg

	MZM	DZM	MZF	DZF	DOS	Brother	Sister	Brother–sister
Twin correlations for GFF								
ND	.44	.37	.53	.43	.33	.34	.31	.32
D	.24	.16	.56	.29	.46	.11	.30	.20
Twin correlations for FC								
ND	.63	.58	.68	.57	.55	.38	.46	.46
D	.69	.58	.76	.56	.49	.61	.66	.61
Twin correlations for QLg								
ND	.38	.20	.46	.36	.24	.04	.18	.19
D	.23	.14	.35	.10	.11	.02	.25	.11
Cross-twin cross-trait correlations for GFF and QLg								
ND	.24	.17	.30	.24	.23	.12	.21	.18
D	.09	.16	.26	.17	.16	–.04	.14	.15
Cross-twin cross-trait correlations for FC and QLg								
ND	.29	.15	.24	.14	.17	.14	.19	.15
D	.23	.28	.20	.01	.05	.04	.13	.08

Note: ND = nondivorced families; D = divorced families; GFF = general family functioning; FC = family conflict; MZ = Monozygotic twin pair; DZ = Dizygotic twin pair; M = male; F = female; DOS = Opposite-sex twin pairs; Brother = Male twin-brother pairs; Sister = Female twin-sister pairs; Brother-sister = Male twin-sister pairs, Female twin-brother pairs

Table 4

Univariate Model Fitting Results for GFF, FC, and QLg

Model	vs.	–2LL	*df*	χ^2	*df*	*p*
GFF						
1. Full model	—	39932.350	6669	—	—	—
2. $\alpha_{ND} = \alpha_D$, $\gamma_{ND} = \gamma_D$, $\eta_{ND} = \eta_D$	1	39936.106	6672	3.76	3	.29
3. $\alpha_{ND} = \alpha_D = 0$, $\gamma_{ND} = \gamma_D = 0$, $\eta_{ND} = \eta_D = 0$	2	39957.131	6675	21.03	3	< .01
4. $a_{ND} = a_D$	2	39940.401	6673	4.30	1	< .05
5. $c_{ND} = c_D$	2	39938.414	6673	2.31	1	.13
6. $e_{ND} = e_D$	2	39936.152	6673	.05	1	.83
7. $c_{ND} = c_D$, $e_{ND} = e_D$	2	39938.599	6674	2.49	2	.29
FC						
1. Full model	—	28231.223	6698	—	—	—
2. $\alpha_{ND} = \alpha_D$, $\gamma_{ND} = \gamma_D$, $\eta_{ND} = \eta_D$	1	28232.124	6701	.90	3	.83
3. $\alpha_{ND} = \alpha_D = 0$, $\gamma_{ND} = \gamma_D = 0$, $\eta_{ND} = \eta_D = 0$	2	28239.099	6704	6.98	3	.07
4. $a_{ND} = a_D$	3	28239.207	6705	.11	1	.74
5. $c_{ND} = c_D$	3	28239.352	6705	.25	1	.61
6. $e_{ND} = e_D$	3	28241.622	6705	2.52	1	.11
7. $a_{ND} = a_D$, $c_{ND} = c_D$, $e_{ND} = e_D$	3	28243.079	6707	3.98	3	.26
QLg						
1. Full model	—	19662.038	6601	—	—	—
2. $\alpha_{ND} = \alpha_D$, $\gamma_{ND} = \gamma_D$, $\eta_{ND} = \eta_D$	1	19674.482	6604	12.44	3	< .01
3. $\alpha_{ND} = 0$, $\gamma_{ND} = 0$, $\eta_{ND} = 0$	1	19676.436	6604	14.40	3	< .01
4. $\alpha_D = 0$, $\gamma_D = 0$, $\eta_D = 0$	1	19688.708	6604	26.67	3	< .01
5. $a_{ND} = a_D$	1	19663.254	6602	1.22	1	.27
6. $c_{ND} = c_D$	1	19662.038	6602	.00	1	> .99
7. $e_{ND} = e_D$	1	19662.711	6602	.67	1	.41
8. $a_{ND} = a_D$, $c_{ND} = c_D$, $e_{ND} = e_D$	1	19663.294	6604	1.26	3	.74

Note: vs. = versus; –2LL = –2 log likelihood; *df* = degrees of freedom; χ^2 = chi-square test statistic; *df* = degrees of freedom of χ^2-test; p = p value; full model = ACE model with differences in parameter estimates for parental divorce and sex.

with sex as a moderator on the genetic and environmental path coefficients.

Univariate Genetic Models

Table 4 presents the univariate model fitting results of the genetic models. In the upper part of the table the model fitting results for GFF are presented. We began with constraining the moderation effects of sex on the path coefficients of GFF to be equal between nondivorced and divorced families, which did not result in a significant deterioration of model fit (model 2). In model 3, the statistical significance of the moderation effects of sex on the path coefficients of GFF was tested, which resulted in a significant deterioration of model fit. This indicates that the magnitude of genetic, shared environmental, and nonshared environmental effects on GFF are different between boys and girls. Models 4 to 6 tested whether constraining the genetic, shared environmental, and nonshared environmental parameter estimate to be equal between nondivorced and divorced families would lead to a significant deterioration of model fit. Additive genetic effects on GFF were significantly different between participants from nondivorced and divorced families, indicating that additive genetic effects on GFF are relatively more important in divorced families compared to nondivorced families: i.e., evidence for an interaction between additive genetic effects and parental divorce. Shared and nonshared environmental effects on GFF were not significantly different between participants from nondivorced and divorced families and therefore they were constrained to be equal in model 7 simultaneously which did not result in a significant deterioration of model fit. Figure 3 presents the unstandardized contributions of genetic, shared environmental and nonshared environmental effects to variation in GFF as a function of sex and parental divorce.

In the middle part of Table 4 the model fitting results for FC are presented. Constraining the moderation effects of sex on the path coefficients of FC to be equal between nondivorced and divorced families did not result in a significant deterioration of model fit (model 2). In model 3, no statistical significant moderation effects of sex on the path coefficients of FC were found, suggesting that the genetic architecture of FC is equal between boys and girls. Additive genetic (model 4), shared environmental (model 5), and nonshared environmental effects (model 6) on FC were not significantly different between participants from nondivorced and divorced families. In model 7, the genetic, shared environmental, and nonshared environmental path coefficient on FC were constrained to be equal between nondivorced and divorced families simultaneously, which did not result in a significant deterioration of model fit.

In the lower part of Table 4 the model fitting results of QLg are presented. With regard to QLg, constraining the moderation effects of sex on the path coefficients to be equal between nondivorced and

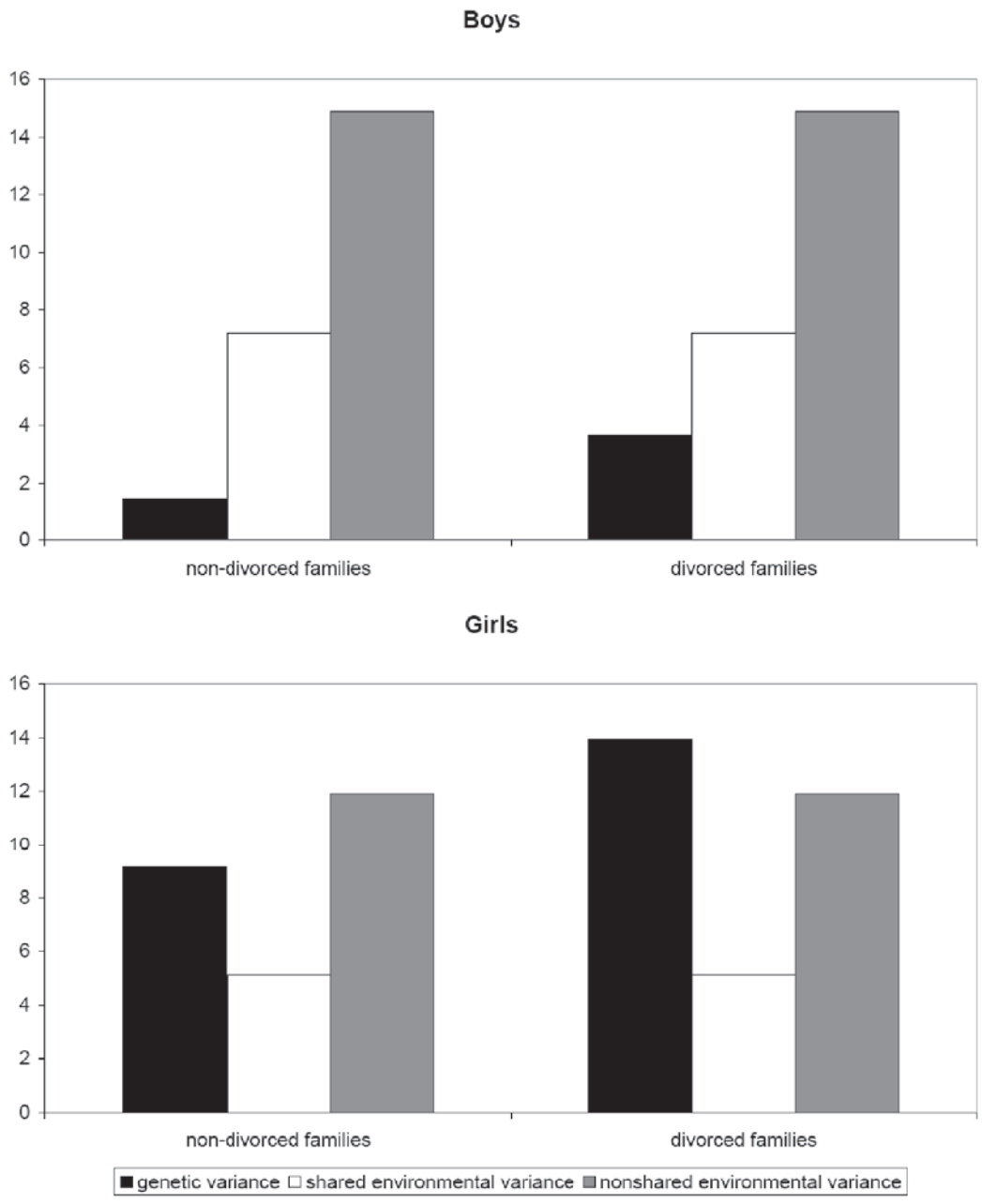

Figure 3

Changes in the absolute contribution of genetic, shared environmental, and nonshared environmental effects to variation in GFF in boys and girls.

divorced families resulted in a significant deterioration of model fit (model 2). This indicates that the effects of sex on the genetic architecture of QLg are different between participants from nondivorced and divorced families. In model 3, the statistical significance of the moderation effect of sex on the genetic architecture of QLg in nondivorced families was tested, which resulted in a significant deterioration of model fit. In model 4, the statistical significance of the moderation effect of sex on the genetic architecture of QLg in divorced families was tested, which also resulted in a significant deterioration of model fit. This indicates that the magnitude of genetic, shared environmental and nonshared environmental effects on QLg is different between boys and girls from nondivorced and divorced families. In model 5 till 7, the genetic, shared environmental, and nonshared environmental parameter estimates were constrained to be equal between nondivorced and divorced families, which did not result in a significant deterioration of model fit. This suggests that, independent of sex, additive genetic, shared environmental, and nonshared environmental effects on QLg were not significantly different between nondivorced and divorced families. However, the results from model 2 till 4 indicate that there is a sex specific interaction

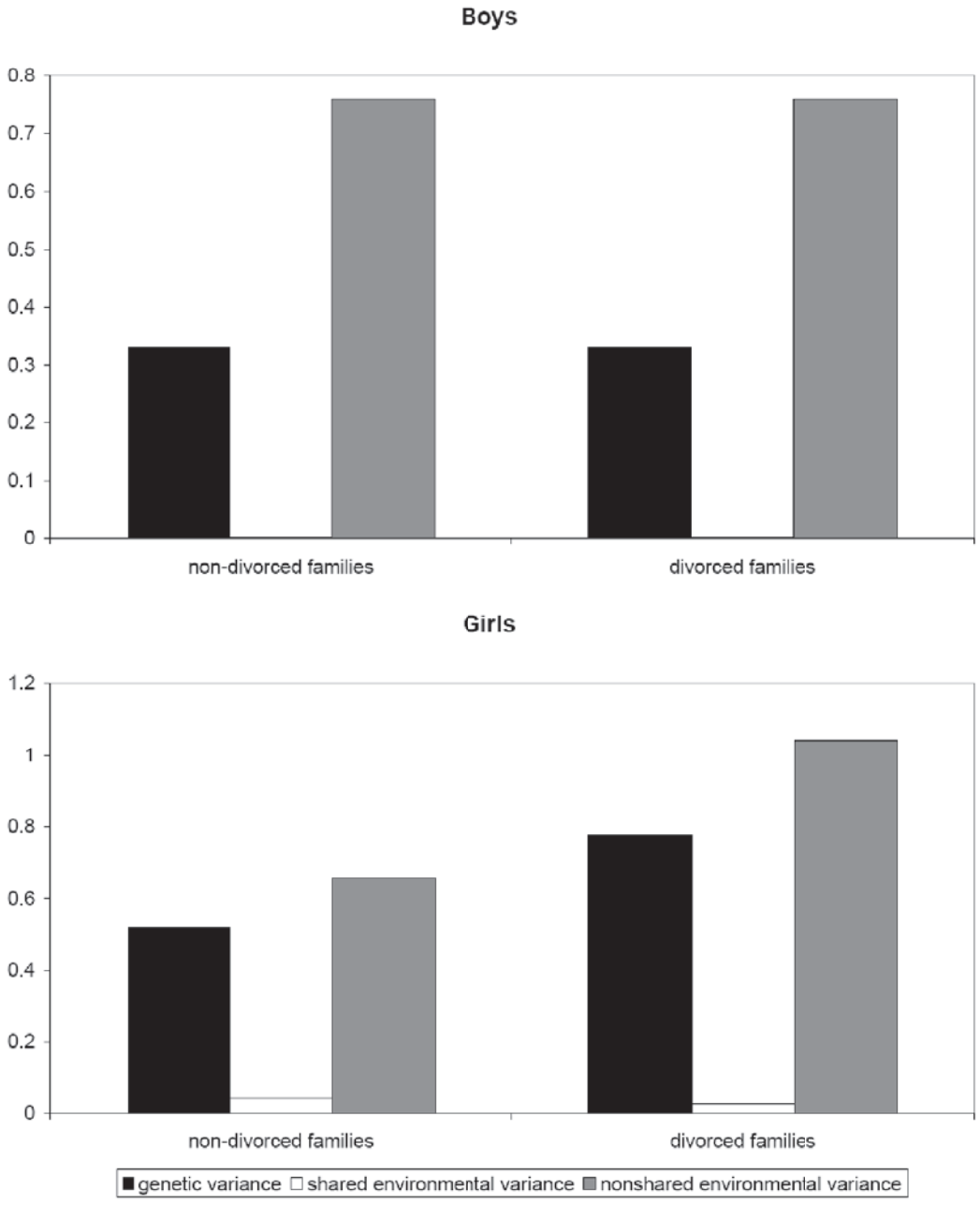

Figure 4

Changes in the absolute contribution of genetic, shared environmental, and nonshared environmental effects to variation in QLg in boys and girls.

between parental divorce and the genetic architecture of QLg. In Figure 4 can be seen that the contribution of genetic and environmental effects to variation in QLg is equal between nondivorced and divorced families in boys, whereas for girls the contribution of genetic and nonshared environmental effects increased in divorced families.

The contributions of A, C, and E to variation in GFF, FC and QLg in nondivorced and divorced families are summarized in Table 5. Variation in GFF was mainly accounted for by shared and nonshared environmental influences in boys, whereas in girls additive genetic influences played a substantive role as well. Heritability of GFF increased from 6% in nondivorced families to 14% in divorced families in boys and from 35% to 45% in girls. With regard to variation in FC, additive genetic, shared environmental and nonshared environmental influences accounted equally in boys and girls from nondivorced and divorced families. Variation in QLg was accounted for by additive genetic and nonshared environmental influences. The influence of shared environmental influences was negligible. Additive genetic influences were relatively more important in girls. The absolute contribution of additive genetic and nonshared environmental influences on QLg increased for girls from divorced

Table 5

Unstandardized and Standardized Parameter Estimates for GFF, FC, and QLg, Including 95% Confidence Intervals Shown in Parentheses

		A		C		E	
		Unstand.	Stand.	Unstand.	Stand.	Unstand.	Stand.
GFF							
Boys	ND	1.47	.06 (.00–.30)	7.20	.31 (.12–.40)	14.88	.63 (.55–.70)
	D	3.67	.14 (.00–.38)	7.20	.28 (.11–.39)	14.88	.58 (.48–.67)
Girls	ND	9.15	.35 (.20–.47)	5.10	.20 (.10–.35)	11.89	.45 (.41–.52)
	D	13.91	.45 (.30–.56)	5.10	.17 (.08–.29)	11.89	.38 (.32–.45)
FC							
Boys	ND	1.54	.31 (.24–.39)	1.75	.36 (.30–.41)	1.61	.33 (.30–.36)
	D	1.54	.31 (.24–.39)	1.75	.36 (.30–.41)	1.61	.33 (.30–.36)
Girls	ND	1.54	.31 (.24–.39)	1.75	.36 (.30–.41)	1.61	.33 (.30–.36)
	D	1.54	.31 (.24–.39)	1.75	.36 (.30–.41)	1.61	.33 (.30–.36)
QLg							
Boys	ND	.33	.30 (.18–.37)	.00	.00 (.00–.09)	.76	.70 (.63–.77)
	D	.33	.30 (.18–.37)	.00	.00 (.00–.09)	.76	.70 (.63–.77)
Girls	ND	.52	.43 (.25–.52)	.04	.03 (.00–.18)	.66	.54 (.48–.60)
	D	.78	.42 (.00–.58)	.03	.01 (.00–.38)	1.04	.56 (.42–.75)

Note: Unstand. = unstandardized variance component; Stand. = standardized variance component; GFF = general family functioning; FC = family conflict; QLg = quality of life general; ND = nondivorced families; D = divorced families.

families compared to girls from nondivorced families, whereas the relative contribution was similar. This is due to an overall increased variation in QLg observed in girls from divorced families (see Table 2). In other words, this indicates that girls from divorced families are more heterogeneous with regard to their reports on QLg as compared to those from nondivorced families.

Bivariate Genetic Models

Phenotypic correlations between GFF and QLg were .36 for boys and .41 for girls (χ^2 (1) = 6.14, $p < .05$). Phenotypic correlations between FC and QLg were .20 for boys and .31 for girls (χ^2 (1) = 27.59, $p < .01$). Cross-twin cross-trait correlations are presented in the lower part of Table 3. Table 6 presents the bivariate model fitting results of the genetic models. Path coefficients and their corresponding beta-weights representing shared environmental influences to covariation between GFF and QLg and FC and QLg were fixed to zero, because of the negligible contribution of shared environmental effects to variation in QLg. In the upper part of Table 6 the model fitting results for the overlap between GFF and QLg are presented. In model 2, constraining the moderation effects of sex on the path coefficients to be equal between nondivorced and divorced families resulted in a significant deterioration of model fit (model 2). This indicates that the effects of sex on the genetic architecture of the overlap between GFF and QLg are different in magnitude between participants from nondivorced and divorced families. In model 3, the statistical significance of the moderation effect of sex on the genetic and nonshared environmental parameter estimate of the covariation between GFF and QLg was tested, which resulted in a significant deterioration of model fit. In model 4 and 5, the genetic and nonshared environmental parameter estimates were constrained to be equal between nondivorced and divorced families, which did not result in a significant deterioration of model fit. This suggests that, independent of sex, the contribution of additive genetic and nonshared environmental effects to the overlap between GFF and QLg was not significantly different between nondivorced and divorced families. However, the results from model 2 and 3 indicate that there is a sex specific interaction between parental divorce and the genetic architecture of the overlap between GFF and QLg. In Figure 5 can be seen that the contribution of genetic and nonshared environmental effects to covariation between GFF and QLg is equal for boys from nondivorced and divorced families, whereas for girls the contribution of genetic and nonshared environmental effects increased in divorced families.

In the lower part of Table 6 the model fitting results for the overlap between FC and QLg are presented. In model 2, constraining the moderation effects of sex on the path coefficients to be equal between nondivorced and divorced families resulted in a significant deterioration of model fit. This indicates that the effects of sex on the genetic architecture of the overlap between FC and QLg are different in magnitude between participants from nondivorced and divorced families. In model 3, the statistical significance of the moderation effect of sex on the genetic and nonshared environmental parameter estimate of the overlap between FC and QLg was tested, which resulted in a significant deterioration of model fit. In model 4 and 5, the genetic and nonshared environmental parameter estimates were constrained to be equal between nondivorced and divorced families, which did not result in a significant deterioration of model fit. This suggests that, independent of sex, the contribution of additive genetic and nonshared environmental effects to the overlap between FC and QLg

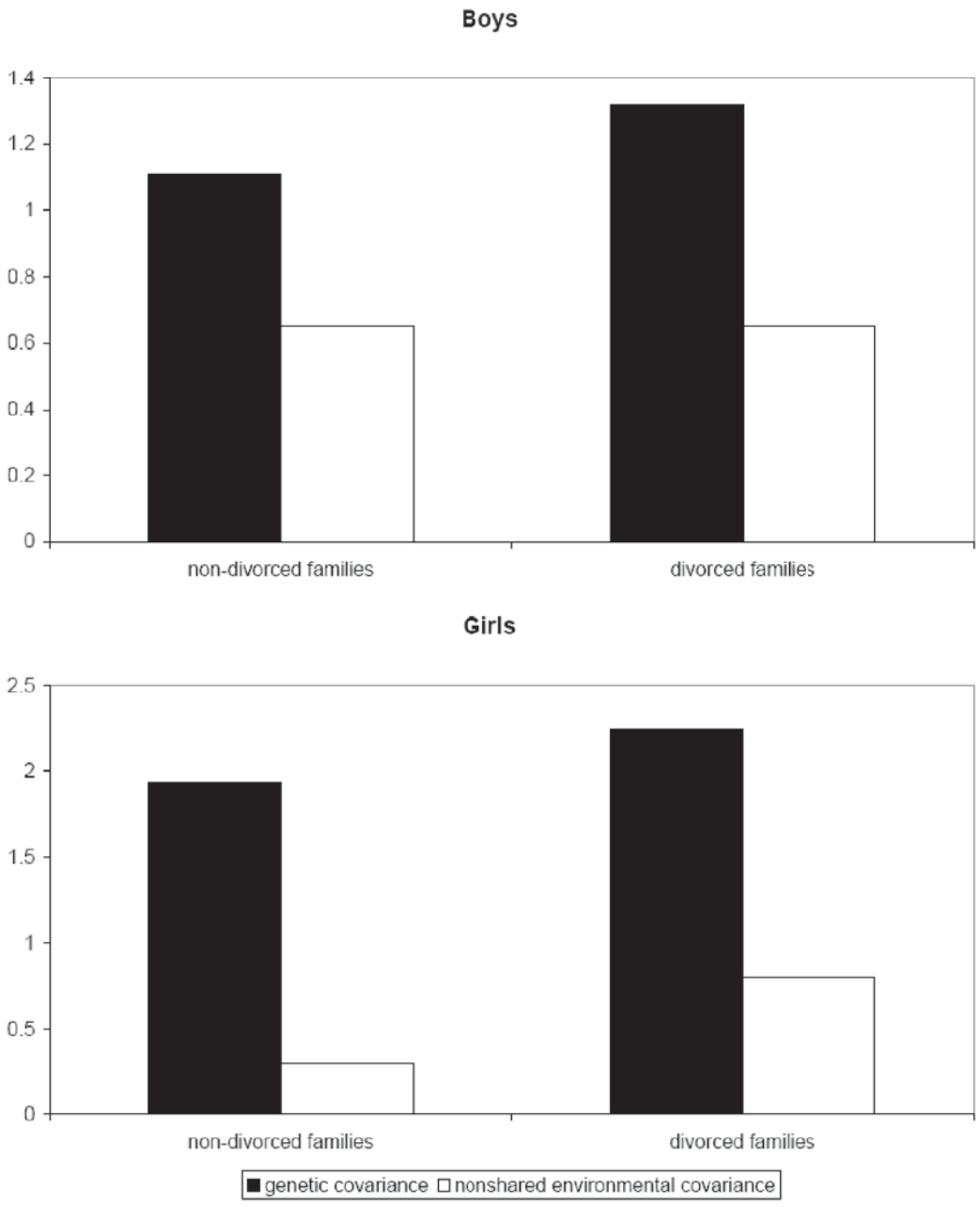

Figure 5

Changes in the absolute contribution of genetic and nonshared environmental covariation between GFF and QLg in boys and girls.

was not significantly different between nondivorced and divorced families. However, the results from model 2 and 3 indicate that there is a sex specific interaction between parental divorce and the genetic architecture of the overlap between FC and QLg. In Figure 6 can be seen that the contribution of genetic and nonshared environmental effects to covariation between FC and QLg is equal for boys from nondivorced and divorced families, whereas for girls the contribution of genetic and nonshared environmental effects increased in divorced families.

The contributions of A and E to overlap between GFF and QLg, and FC and QLg in nondivorced and divorced families are presented in Table 7. Additive genetic influences accounted for the largest part of the covariation between GFF and QLg, whereas nonshared environmental effects accounted for a small part as well. Additive genetic influences were relatively more important in girls. The absolute contribution of additive genetic and nonshared environmental influences to the overlap between GFF and QLg increased for girls from divorced families compared to girls from nondivorced families. This is due to an overall increase in covariation between GFF and QLg for girls from divorced families. The relative contribution of additive genetic influences for girls from divorced fam-

Table 6

Bivariate Model Fitting Results for the Overlap Between GFF and QLg, and FC and QLg

Model	vs.	–2LL	*df*	$\chi 2$	*df*	*p*
GFF – QLg						
1. Full model	—	58667.780	13270	—	—	—
2. $\alpha_{21,ND} = \alpha_{21,D}$; $\eta_{21,ND} = \eta_{21,D}$	1	58674.192	13272	6.41	2	< .05
3. $\alpha_{21,ND} = 0$; $\alpha_{21,D}=0$; $\eta_{21,ND} = 0$; $\eta_{21,D} = 0$	1	58678.026	13274	10.25	4	< .05
4. $a_{21,ND} = a_{21,D}$	1	58668.387	13271	.61	1	.44
5. $e_{21,ND} = e_{21,D}$	1	58668.164	13271	.38	1	.54
6. $a_{21,ND} = a_{21,D}$; $e_{21,ND} = e_{21,D}$	1	58670.231	13272	2.43	2	.30
FC – QLg						
1. Full model	—	47475.630	13303	—	—	—
2. $\alpha_{21,ND} = \alpha_{21,D}$; $\eta_{21,ND} = \eta_{21,D}$	1	47482.365	13305	6.74	2	< .05
3. $\alpha_{21,ND} =0$; $\alpha_{21,D} = 0$; $\eta_{21,ND} = 0$; $\eta_{21,D}=0$	1	47510.240	13307	34.61	4	< .01
4. $a_{21,ND} = a_{21,D}$	1	47476.546	13304	.92	1	.34
5. $e_{21,ND} = e_{21,D}$	1	47476.999	13304	1.37	1	.24
6. $a_{21,ND} = a_{21,D}$; $e_{21,ND} = e_{21,D}$	1	47477.347	13305	1.72	2	.42

Note: vs. = versus; –2LL = –2 log likelihood; *df* = degrees of freedom; χ^2 = chi-square test statistic; *df* = degrees of freedom of χ^2 test; *p* = *p* value; Full model = AE model with differences in parameter estimates for parental divorce and sex.

ilies decreased in favor of nonshared environmental influences. With regard to the covariation between FC and QLg, additive genetic influences accounted (almost) exclusively for it. The absolute contribution of additive genetic and nonshared environmental influences to the overlap between FC and QLg increased for girls from divorced families compared to girls from nondivorced families. This is due to an overall increase in covariation between FC and QLg for girls from divorced families. The relative contribution of additive genetic influences for girls from divorced families decreased in favor of nonshared environmental influences.

With regard to overlap between GFF and QLg, the genetic and nonshared environmental correlations for boys from nondivorced and divorced families were .80 and .20 respectively. For girls from nondivorced families these correlation were .77 and .11, whereas for girls from divorced families they were .79 and .22 respectively. This suggests that adolescents' views of GFF and QLg share most of the underlying genetic influences and part of nonshared environmental influences. Regarding the overlap between FC and QLg, genetic and nonshared environmental correlations for boys from nondivorced and divorced families were .77 and .09 respectively. For girls from nondivorced families these correlations were .68 and .04, whereas for girls from divorced families they were .79 and .29. This suggests that most genetic influences and part of nonshared environmental influences underlying adolescents' views of FC and QLg are common.

Correlation Between Sum and Difference Scores in MZ Twin Pairs

In Table 8, correlations between intrapair difference and sum scores in MZ twins are presented. Significant negative correlations were found for GFF, FC, and QLg in boys and girls, suggesting the presence of interaction between genetic and nonshared, non-measured, environmental influences. The significant correlations

Table 7

Unstandardized and Standardized Parameter Estimates for Covariation Between GFF and QLg, and FC and QLg, Including 95% Confidence Intervals Shown in Parentheses

		A		E	
		Unstand.	Stand.	Unstand.	Stand.
GFF – QLg					
Boys	ND	1.11	.63 (.50–.76)	.65	.37 (.24–.50)
	D	1.32	.67 (.67–.79)	.65	.33 (.21–.44)
Girls	ND	1.93	.86 (.86–.95)	.30	.14 (.05–.23)
	D	2.25	.74 (.63–.95)	.80	.26 (.05–.50)
FC – QLg					
Boys	ND	.56	.85 (.79–1.0)	.10	.15 (.14–.28)
	D	.56	.85 (.79–1.0)	.10	.15 (.14–.28)
Girls	ND	.64	.95 (.67–.98)	.04	.05 (.00–.17)
	D	.81	.67 (.67–.98)	.37	.31 (.09–.54)

Note: Unstand. = unstandardized variance component; Stand. = standardized variance component; GFF – QLg = overlap between general family functioning and quality of life general; FC – QLg = overlap between family conflict and quality of life general; ND = nondivorced families; D = divorced families.

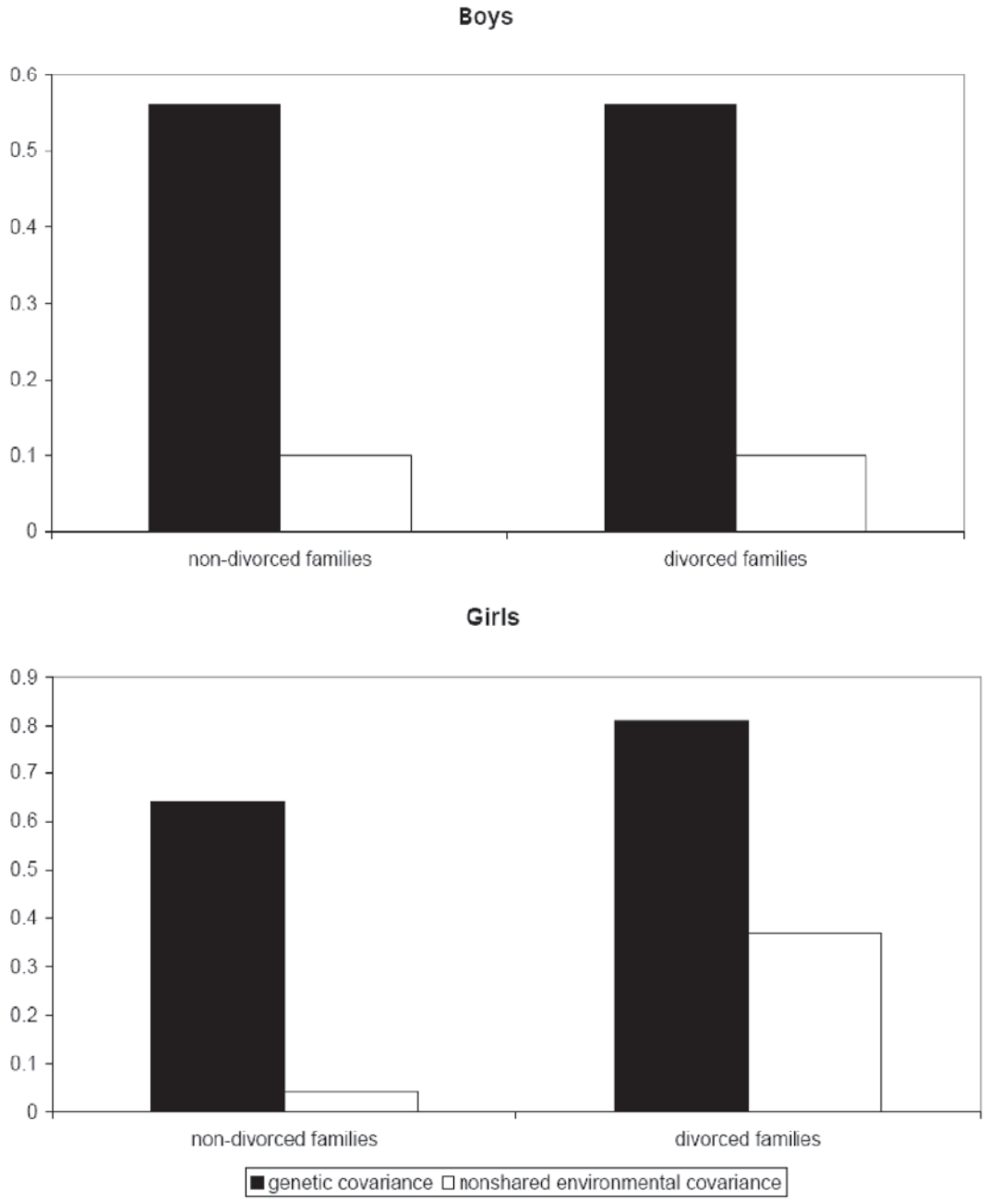

Figure 6

Changes in the absolute contribution of genetic and nonshared environmental covariation between FC and QLg in boys and girls.

indicate that MZ twins reporting low levels of FC and high levels of GFF and QLg are more similar than those reporting high levels of FC and low levels of GFF and QLg. Hence, nonshared environmental influences are relatively more important in explaining variation for those who report low levels of FC, and high levels of GFF and QLg.

Discussion

The present study investigated causes of variation in evaluations of GFF, FC, and QLg in a large sample of Dutch adolescent twins and their non-twin siblings. We tested whether the genetic architecture of GFF, FC, and QLg differed between boys and girls and between those living in nondivorced and divorced families. We found that individual differences in evaluations of GFF and FC could be accounted for by additive genetic, shared environmental and nonshared environmental influences. Heritability of evaluations of GFF was larger in girls and for participants from divorced families. Variation in QLg was accounted for by additive genetic and nonshared environmental influences, with heritability being larger in girls. Furthermore, girls from divorced families showed larger heterogene-

Table 8

Correlations Between Intrapair Difference and Sum Scores in MZ Twins

	MZM	MZF
GFF	–.11*	–.09*
FC	–.24**	–.24**
QLg	–.39**	–.41**

Note: * $p < .05$; ** $p < .001$

ity in their perceptions of QLg as compared to girls from nondivorced families.

We also examined the overlap between GFF and QLg and between FC and QLg by investigating to what extend genetic and environmental influences contribute to the overlap. Again, we tested whether the genetic architecture of the overlap differed between boys and girls and between those living in nondivorced and divorced families. The overlap between GFF and QLg and between FC and QLg could be accounted for by additive genetic and nonshared environmental influences. Additive genetic influences were relatively more important than nonshared environmental influences. Girls from divorced families showed larger phenotypic overlap between GFF and QLg, and FC and QLg compared to girls from nondivorced families and boys. Finally, for evaluations of GFF, FC, and QLg we provided evidence that nonshared, non-measured, environmental influences interact with genetic factors.

With regard to adolescents' evaluations of GFF we found that genetic effects were relatively more important in girls than in boys. This finding is consistent with what was reported by Jacobson and Rowe (1999). Genetic influences on evaluations of QLg were also relatively more important in girls, which is in line with was reported by Røysamb (2003) and Bartels et al. (in press). Genetic effects may be larger in evaluations of family functioning and subjective wellbeing in girls because they also have been found to be larger in traits that are likely to influence evaluations or perceptions of an individual's life. For example, sex differences have been found for depression and neuroticism with higher heritabilities for females (Boomsma et al., 2000; Jardine et al., 1984; Kendler et al., 2001). Furthermore, Jacobson and Rowe (1999) found genetic overlap between evaluations of family connectedness and depressed mood in girls. Thus, it is possible that genetic factors that influence traits such as depression and neuroticism may also influence evaluations of family functioning and SWB in girls.

Parental divorce modified the genetic architecture of evaluations of GFF. Genetic effects were larger in participants from families with divorced parents as compared to those from intact families. The increased impact of genetic factors on evaluations of GFF suggest that participants with a genetic predisposition towards negative evaluations of GFF are more sensitive to negative effects of parental divorce and those with a genetic predisposition towards positive evaluations are more sensitive to positive effects of parental divorce. For example, adolescents with a genetically predisposed tendency towards negative evaluations of family functioning may be disproportionally more likely to evaluate their family functioning as worse if one parent is absent, because they are likely to have more conflicts with their parents (e.g., Dunn et al., 1998; O'Connor et al., 2001) and receive less emotional support, supervision, and involvement from their parents (e.g., Carlson & Corcoran, 2001). On the other hand, participants with a genetically predisposed tendency towards positive evaluations of family functioning are disproportionally more likely to evaluate their family functioning as positive if the absence of one parent leads to a less stressful family situation as is for example likely to be the case when an antisocial father is absent (Blazei et al., 2008; Jaffee et al., 2003).

The genetic architecture of evaluations of the level of FC was, however, not modified by sex and parental divorce. It could be that the evaluation of the level of FC is more dependent on true experiences of conflict and therefore is a more objective evaluation of family functioning. For example, evaluating whether family members criticize each other frequently (i.e., one example of the family conflict scale) might be more objective than the evaluation of family members get sufficient support from each other in times of crisis (i.e., one example of the general family functioning scale). Thus, the evaluation of the level of FC is assumed to be less due to the way adolescents perceive and interpret experiences in the family.

Individual differences in evaluations of QLg were larger for girls from families with divorced parents compared to those from intact families. This indicates that parental divorce has no general main effect on evaluations of subjective wellbeing but rather that there is large heterogeneity in the effects of parental divorce on evaluations of subjective wellbeing in girls. Although to a lesser extent, this also holds for boys, because boys from families with divorced parents also did not become more homogenous with regard to their evaluations of QLg which might be expected if parental divorce had a general negative main effect on the evaluation of SWB and adjustment. Heterogeneity in the effects of parental divorce on SWB is a plausible explanation of general small effect sizes found in research investigating main effects of parental divorce on wellbeing and adjustment (e.g., Amato & Keith, 1991; McFarlane et al., 1995). Similar results were also obtained for evaluations of GFF and FC. An important implication of these results for future research with regard to adolescents' evaluations of family functioning, subjective wellbeing, and adjustment is to focus on etiologies of individual differences in the effects of (negative) family events and life events in general, instead of focusing on general effects. Our results indicated that adolescents react differently to

parental divorce and that heterogeneity in these reactions has different causes (e.g., genetic, factors from an adolescent's personal environment).

Moderate associations were found between GFF and QLg and between FC and QLg, indicating that higher levels of GFF and lower levels of FC are associated with higher levels of QLg, which is consistent with the results of other studies (e.g., Harold et al., 1997; Jacobson & Rowe, 1999; Millikan et al., 2002). We found that genetic factors were responsible for the largest part of the overlap between GFF and QLg and between FC and QLg. High genetic overlap between adolescents' evaluations of family functioning and depressive symptoms have previously been reported by Pike et al. (1996), Neiderhiser et al. (1998) and Jacobson and Rowe (1999), although the latter reported this high genetic overlap only for girls. A possible explanation for the high genetic overlap is that evaluations of family functioning and subjective wellbeing are distinct components of a more general view towards life which is influenced by genetic factors. This implicates that environmental factors, e.g., true family experiences, have a marginal role in explaining the overlap between adolescents' evaluations between family functioning and subjective wellbeing. Rather, the overlap is due to a genetic predisposed way of evaluating one's life in general, which can either be positive or negative. Our results also indicated that the overlap between GFF and QLg and between FC and QLg was larger for girls from families with divorced parents.

The interaction between genetic influences and nonshared, non-measured, environmental influences for GFF, FC, and QLg reflected by the significant negative correlations between intrapair sumscores and difference scores in MZ twins, indicated that genetic influences are relatively more important in those adolescents experiencing high levels of GFF and QLg, and low levels of FC (since they were more similar) as compared to those experiencing lower levels of GFF and QLg, and higher levels of FC. One possible reason for this is that adolescents with a genetic predisposition towards positive evaluations of family functioning and subjective wellbeing (reflected by the experience of high levels of GFF and QLg and low levels of FC) are more sensitive to positive effects of environmental factors that are not shared by members of the same family. Thus, adolescents with a genetic tendency towards positive evaluations of family functioning and subjective wellbeing may be disproportionally more likely to evaluate their family functioning and subjective wellbeing as positive when certain nonshared environmental influences are present. This would imply that certain environmental influences unique to an adolescent play a protective role with regard to evaluations of family functioning and subjective wellbeing. Although these results suggest that nonshared environmental factors can play a protective role, it remains unclear which particular factors are involved because we did not measure these factors.

Research has shown that the way adolescents evaluate their family functions is positively associated with subjective wellbeing and psychopathology. The present study contributed to the literature in several ways. We showed that individual differences in adolescents' evaluations of family functioning and subjective wellbeing can be explained by the combined effect of genetic and environmental effects and that these effects can vary by sex. We found that the genetic architecture of evaluations of family functioning and subjective wellbeing can be modified by influences from the family environment (e.g., parental divorce as was demonstrated in this study) and the personal unique environment of an adolescent. Moreover, our results indicated that genetic factors are the main contributing factors to the association between evaluations of family functioning and subjective wellbeing.

Acknowledgments

Funding was obtained from the following grants: 'Spinozapremie' (NWO/SPI 56-464-14192); 'Twin-family database for behavior genetics and genomics studies' (NWO 480-04-004); 'Genetic and Family influences on Adolescent Psychopathology and Wellness' (NWO 463-06-001); 'A twin-sib study of adolescent wellness' (NWO-VENI 451-04-034). M. Bartels is financially supported by a senior fellowship of the EMGO+ institution.

References

Amato, P. R. & Keith, B. (1991) Parental divorce and the well-being of children: A meta-anlysis. *Psychological Bulletin, 110,* 26–46.

Bartels, M., Beijsterveld van, C. E. M., Derks, E. M., Stroet, T. M., Polderman, T. J. C, Hudziak, J. J., & Boomsma, D. I. (2007). Young Netherlands Twin Register (Y-NTR): A longitudinal multiple informant study of problem behavior. *Twin Research and Human Genetics, 10*, 3–11.

Bartels, M. & Boomsma, D. I., (2009). Born to be happy? The etiology of subjective well-being. Behavior Genetics, 39, 605–615.

Bartels, M., Savyuk, V., de Moor, M. H. M., Willemsen, A. H. M., van Beijsterveldt, C. E. M., Hottenga, J. J., de Geus, E. J., Boomsma, D. I. (in press). Heritability and Genome-wide Linkage Scan for Subjective Happiness. *Twin Research and Human Genetics*.

Boomsma, D. I., Beem, A. L., van den Berg, M., Dolan, C., Koopmans, J. R., Vink, J. M., de Geus, E. J., & Slagboom, P. E. (2000). Netherlands twin family study of anxious depression (NETSAT). *Twin Research, 3,* 323–334.

Boomsma, D. I., De Geus, E. J. C., Vink, J. M., Stubbe, J. H., Distel, M. A., Hottenga, J. J., Posthuma, D., Van Beijsterveldt, T. C. E. M., Hudziak, J. J., Bartels, M., & Willemsen, G. (2006). Netherlands Twin Register: From twins to twin families. *Twin Research and Human Genetics, 9,* 849–857.

Blazei, R. W., Iacono, W. G., & McGue, M. (2008). Father-child transmission of antisocial behavior: the moderating role of father's presence in the home. *Journal of the American Academy for Child and Adolescent Psychiatry, 47*, 406–415.

Cantril, H. (1965). *The pattern of human concerns*. New Brunswick, NJ: Rutgers University Press.

Carlson, M. J., & Corcoran, M. E. (2001). Family structure and children's behavioral and cognitive outcomes. *Journal of Marriage and the Family, 63*, 779–792.

Cuffe, S. P., McKeown, R. E., Addy, C. L., & Garrison, C. Z. (2005). Family and psychosocial risk factors in a longitudinal epidemiological study of adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 44*, 121–129.

Cui, M., Donellan, M. B., & Conger, R. D. (2007). Reciprocal influences between parents' marital problems and adolescents internalizing and externalizing behavior. *Developmental Psychology, 43,* 1544–1552.

De Coole, R. L. & Jansma, J. B. M. (1983). *G.K.S. Gezinsklimaatschaal*. Handleiding. Lisse: Swets & Zeitlinger.

Dunn, J., Deater-Deckard, K., Pickering, K., & O'Connor, T. G. (1998). Children's adjustment and prosocial behaviour in step-, single-, and non-stepfamily settings: Findings from a community study. *Journal of Child Psychology and Psychiatry, 39*, 1083–1095.

Eaves, L. J., Heath, A. C., Martin, N. G., Neale, M. C., Meyer, J. M., Silberg, J. L., Corey, L. A., Truett, K., & Walters, E. (1999). Biological and cultural inheritance of stature and attitudes. In C. Robert Cloninger (Ed.), *Personality and psychopathology*. American Psychopathological Association, Washington, DC.

Epstein, N. B., Baldwin, L. M., & Bishop, D. S. (1983). The McMaster Family Assessment Device. *Journal of Marital and Family Therapy, 9,* 171–180.

Formoso, D., Gonzales, N. A., & Aiken, L. S. (2000). Family conflict and children's internalizing and externalizing behavior: protective factors. *American Journal of Community Psychology, 28*, 175–199.

Harold, G. T. H., Fincham, F. D., Osborne, L. N., & Conger, R. D. (1997). Mom and dad are at it again: adolescent perceptions of marital conflict and adolescent psychological distress. *Developmental Psychology, 33*, 333–350.

Herndon, R. W., McGue, M., Krueger, R. F., & Iacono, W. G. (2005). Genetic and environmental influences on adolescents' perceptions of current family environment. *Behavior Genetics, 35*, 373–380.

Hughes, A. A., Hedtke, K. A., & Kendall, P. C. (2008). Family functioning in families of children with anxiety disorders. *Journal of Family Psychology, 22*, 325–328.

Jaffee, S. R., Moffitt, T. E., Caspi, A., & Taylor, A. (2003). Life with (or without) father: the benefits of living with two biological parents depend on the father's antisocial behavior. *Child Development, 74*, 109–126.

Jaffee, S. R. & Price, T. S. (2007). Gene-environment correlations: A review of the evidence and implications for prevention of mental illness. *Molecular Psychiatry, 12*, 432–442.

Jacobson, K. C. & Rowe, D. C. (1999). Genetic and environmental influences on the relationships between family connectedness, school connectedness, and adolescent depressed mood: sex differences. *Developmental Psychology, 35*, 926–939.

Jinks, J. L. & Fulker, D. W. (1970). Comparison of the biometrical genetical, MAVA, and classical approaches to the analysis of human behavior. *Psychological Bulletin, 73*, 311–349.

Jardine, R., Martin, N. G., & Henderson, A. S. (1984). Genetic covariation between neuroticism and the symptoms of anxiety and depression. *Genetic Epidemiology, 1*, 89–107.

Kendler, K. S. & Baker, J. H. (2007). Genetic influences on measures of the environment: A systematic review. *Psychological Medicine, 37*, 615–626.

Kendler, K. S., & Eaves, L. J. (1986). Models for the joint effect of genotype and environment on liability to psychiatric illness. *The American Journal of Psychiatry, 143*, 279–289.

Kendler, K. S., Gardner, C. O., Neale, M. C., & Prescott, C. A. (2001). Genetic risk factors for major depression in men and women: Similar of different heritabilities and same or partly distinct genes? *Psychological Medicine, 31*, 605–616.

Kurdek, L. A., & Fine, M. A. (1994). Family acceptance and family control as predictors of adjustment in young adolescents — linear, curvilinear, or interactive effects. *Child Development, 65*, 1137–1146.

Leary, A. & Katz, L. F. (2004). Coparenting, family-level processes, and peer outcomes: The moderating role of vagal tone. *Development and Psychopathology, 16*, 593–608.

Leeuwen van, M., Berg van den, S. M., & Boomsma, D. I. (2007). A twin-family study of general IQ. *Learning and Individual Differences, 18*, 76–88.

McHale, J. P., & Rasmussen, J. L. (1998). Coparental and family group-level dynamics during infancy: early family precursors of child and family functioning during preschool. *Development and Psychopathology, 10*, 39–59.

McFarlane, A. H., Bellissimo, A., & Norman, G. R. (1995). Family structure, family functioning and adolescent well-being: The transcendent influence of parental style. *Journal of Child Psychology and Psychiatry, 36*, 847–864.

Millikam, E., Wamboldt, M. Z., & Bihun, J. T. (2002). Perceptions of the family, personality characteristics, and adolescent internalizing symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry, 41*, 1486–1494.

Moos, R. H. (1974). *Family Environment Scale: Preliminary manual*. Palo Alto, CA: Consulting Psychologists Press.

Neale, M. C., Boker, S. M., Xie, G., & Maes, H. H. (2006). *Mx: Statistical modeling (7th ed)*. Department of Psychiatry, Richmond, VA.

Neiderhiser, J. M., Pike, A., Hetherington, E. M., & Reiss, D. (1998). Adolescent perceptions as mediators of parenting: genetic and environmental contributions. *Developmental Psychology, 34*, 1459–1469.

Nes, R. B., Røysamb, E., Tambs, K., Harris, J. R., & Reichborn-Kjennerud, T. (2006). Subjective well-being: Genetic and environmental contributions to stability and change. *Psychological Medicine, 36*, 1033–1042.

O'Connor, T. G., Dunn, J., Jenkins, J. M., Pickering, K., & Rabash, J. (2001). Family settings and children's adjustment: Differential adjustment within and across families. *British Journal of Psychiatry, 179*, 110–115.

Pike, A., McGuire, S., Hetherington, E. M., Reiss, D., & Plomin, R. (1996). Family environment and adolescent depressive symptoms and antisocial behavior: A multivariate genetic analysis. *Developmental Psychology, 32*, 574–589.

Plomin, R. & Bergeman, C. S. (1991). The nature of nurture: Genetic influences on 'environmental' measures. *Behavioral and Brain Sciences, 14*, 373–427.

Price, T. S. & Jaffee, S. R. (2008). Effects of the family environment: gene-environment interaction and passive gene-environment correlation. *Developmental Psychology, 44*, 305–315.

Proctor, C. L., Linley, P. A., & Maltby, J. (2009). Youth life satisfaction: A review of the literature. *Journal of Happiness Studies, 10*, 583–630.

Purcell, S. (2002). Variance components models for gene-environment interaction in twin analysis. *Twin Research, 5*, 554–571.

Richmond, M. K. & Stocker, C. M. (2006). Associations between family cohesion and adolescent siblings' externalizing behavior. *Journal of Family Psychology, 20*, 663–669.

Rietveld, M. J. H., van der Valk, J. C., Bongers, I. L., Stroet, T. M., Slagboom, P. E., & Boomsma, D. I. (2000). Zygosity diagnosis in young twins by parental report. *Twin Research, 3*, 134–141.

Robbers, S. C. C., Bartels, M., Van Beijsterveldt, C. E. M., Verhulst, F. C., Huizink, A. C., & Boomsma, D. I. (under review). Pre-divorce problems in 3-year-olds: A prospective study in boys and girls. *Social Psychiatry and Psychiatric Epidemiology*.

Røysamb, E., Harris, J. R., Magnus, P., Vittersø, J., & Tambs K. (2002). Subjective well-being. Sex specific effects of genetic and environmental factors. *Personality and Individual Differences, 32*, 211–223.

Størksen, I., Røysamb, E., Holmen, T. L., & Tambs, K. (2006). Adolescent adjustment and well-being: Effects of parental divorce and distress. *Scandinavian Journal of Psychology, 47*, 75–84.

Vandewater, E. A. & Lansford, J. E. (2005). A family process model of problem behaviors in adolescents. *Journal of Marriage and the Family, 67*, 100–109.

Wenniger, W. F. M. D. B., Hageman, W. J. J. M., & Arrindell, W. A. (1993). Cross-national validity of dimensions of family functioning: first experiences with the Dutch version of the McMaster Family Assesment Device (FAD). *Personality and Individual Differences, 14*, 769–781.