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Revisiting the Effect of Marital Support on Depressive Symptoms in Mothers and Fathers: A Genetically Informed Study

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Abstract

This article uses a genetically informed design to evaluate whether (1) the well-documented association between marital support and depressive symptoms is accounted for by genetic and/or shared environmental selection, (2) gender differences are found after controlling for selection effects, and (3) parenthood moderates any nonshared environmental relation between depressive symptoms and marital support. We used a sample of 1,566 pairs of same-sexed, married twins from the Australian Twin Registry to evaluate our hypotheses that (1) the predicted effect of marital support on depressive symptoms is not fully an artifact of selection, (2) the etiological sources accounting for this effect differ between husbands and wives, and (3) parenthood status moderates the effect of marital support on depressive symptoms adjusting for selection effects. The results support the first hypotheses. However, after controlling for selection, the effect of marital support on depressive symptoms was not significantly different for husbands and wives. Parenthood moderated the effect of marital support, such that after controlling for selection, marital support is more strongly associated with depressive symptoms for full-time parents than nonfull-time parents.

Keywords

marital support; interpersonal relationships; depressive symptoms; depression; behavior genetics

Marital researchers reliably find that spouses who report unhappy marriages also report more depressive symptoms (Rehman, Gollan, & Mortimer, 2008; Whisman, 2001). A recent

meta-analysis found a mean cross-sectional correlation of .37 and a mean longitudinal correlation of .25 (Proulx, Helms, & Buehler, 2007). The association holds across several aspects of the marital relationship, including marital conflict, marital happiness, and marital satisfaction (Beach, Sandeen, & O'Leary, 1990; Whisman & Bruce, 1999).

Most investigators interpret the correlation as meaning either that poor marital quality causes depressive symptoms or vice versa. In this article, we focus primarily on the former causal interpretation, while acknowledging the plausibility of the latter (Whisman & Uebelacker, 2009). Several mechanisms have been offered to explain the effect of marital quality on depressive symptoms, including the marital discord model of depression (Beach et al., 1990) and support processes that underlie the link between marital stress and depressive symptoms (Davila, Bradbury, Cohan, & Tochluk, 1997). However, we first address an additional explanation. The association between marital quality and depressive symptoms may be an artifact of nonrandom selection.

Selection effects can be genetic or environmental or both. Genetic selection can occur when people's environmental experience is correlated with their genetic characteristics, which it might be theoretically (Scarr & McCartney, 1983) and often is empirically (D'Onofrio et al., 2005; Harden et al., 2007; Hill, Emery, Harden, Mendle, & Turkheimer, 2008). It is theoretically possible and empirically plausible that the observed association between depressive symptoms and unhappy marriage is inflated because of a gene–environment correlation. For example, people who are genetically prone to depression might “select” less desirable or responsive partners. This possibility is a reasonable concern in the present context, because marital quality (Kendler & Baker, 2007), major depression (Sullivan, Neale, & Kendler, 2000), and depressive symptomatology (McGue & Christensen, 2003) show significant heritability.

Two kinds of genetic selection effects may contribute to the observed association between marital quality and depressive symptoms. An *active* gene–environment correlation occurs when people seek out environments that match their genetic characteristics. For example, these characteristics may both increase the risk for depression and risk for seeking relatively unresponsive mates, leading to poor relationship quality. Alternatively, depressed partners may evoke negative responses from their spouses, thereby lowering marital quality. This type of genetic selection is known as an *evocative* gene–environment correlation.

Of course, environmental selection also may create the correlation between marital quality and depressive symptoms. For example, growing up and living in a poor and/or chaotic neighborhood may increase the risk of both depressive symptoms and an unhappy marriage, thus making their association an epiphenomenon of individuals' milieu.

Twin studies are a uniquely powerful method of testing and controlling for such selection effects, and as such, serve as a quasi-experimental method for determining causation. Twin studies have three essential advantages over other methods of controlling for selection. First, twin studies control for genetic selection. For example, identical (monozygotic, MZ) twins share 100% of their genes; thus any observed differences in depressive symptoms between MZs discordant for a targeted life experience (e.g., marital satisfaction) cannot be because of genetic selection. Second, twin studies control for selection because of the shared environment. Twins are reared together in the same families at the same time; thus, for example, any observed differences in depressive symptoms between MZ twins discordant for marital happiness cannot be because of poverty, neighborhood, ethnicity, parents' marital status, child rearing, and so on. Third, twin studies control for genetic and shared environmental selection whether they have been, or indeed, can be, measured. That is, twin

studies control for measured and *unmeasured* shared environmental (e.g., shared childhood experience) and genetic selection (the entire genome).

An example may help to illustrate these advantages. If in an MZ pair, Twin 1 is more happily married than Twin 2 and Twin 1 also has fewer depressive symptoms, this association *cannot* be explained by genetic or shared environmental third variables. The twins are genetically identical, and they also share a rich shared environmental history (e.g., the same family, the same parents-with the same marital status/quality-the same family values, the same schools, neighborhoods, ethnicity, and so on). Of course, despite the strengths of the twin method nonshared experiences other than the twins' marriage may account for observed correlations, such as method effects (Schmitt, 1994) and assortative mating (Vandenberg, 1972). Thus, we use the term "quasi-causal" to describe the predicted effect of marital quality on depressive symptoms after controlling for selection effects.

A small handful of behavior genetic studies have partially addressed the possibility that nonrandom selection accounts for the association between marital quality and depressive symptoms. Finding support for genetic selection, Spotts et al. (2004) argued that people who were genetically predisposed to depression were more likely to both seek out less desirable partners and to evoke marital difficulties. In a second study, Spotts et al. (2005) found similar genetic results linking marital relationships to positive mental health. In each study, they also found that nonshared environmental factors contributed to the association between marital quality and depression. Again, the implication of nonshared environmental effects is important because differences in the siblings' experience—like being married to different spouses and having more and less happy marriages—account for differences in siblings' reported depressive symptoms. However, Spotts et al. did not specifically analyze or interpret this nonshared variance, nor did they compare men and women.

South and Krueger (2008) studied the moderating role of marital quality on the genetic and environmental contributions to internalizing spectrum disorders. Although they found that essentially no unique nonshared environmental factor accounted for the observed covariation between marital quality and internalizing disorders, they demonstrated that genetic and environmental effects on internalizing disorders vary as a function of level of marital satisfaction. In keeping with our quasi-causal approach, the causal understanding of the link between marital quality and depressive symptoms remains an open question.

Overall, these three studies support the conclusion that part of the association between marital quality and depressive symptoms is genetically mediated. Questions remain as to whether nonshared environmental differences (i.e., within-family differences) in twins' marital quality contribute to differences in their depressive symptom reports. One goal of the present study was to test this possibility.

Gender Differences

Important and controversial gender differences have been observed in the link between marriage and mental health (Bernard, 1972; Waite & Gallagher, 2000). For men, marital *status* is thought to be associated with less risky behavior and better mental health whereas marital *quality* appears to be more reliably connected to women's depressive symptoms (Fincham, Beach, Harold, & Osborne, 1997; Dehle & Weiss, 1998; Davila et al., 1997). Assuming that the link is not an epiphenomenon of genetic or environmental risk factors, it is important to examine gender differences in this relationship. In fact, a twin study rules out the critical and as-of-yet untested possibility that selection effects differ by gender, thus contributing to observed differences in correlations for women and men. To date, only women have been included in behavior genetic studies of this topic.

Parenthood as a Moderator

The context in which marital quality and depressive symptoms co-occur also is important to consider. There are many possible contextual moderators of the correlation, but few have been studied (Davila, Karney, Hall, & Bradbury, 2003; Karney, 2001). Parenthood is a particularly important potential moderator. Marriages inevitably change when a couple has children (Cowan & Cowan, 2000) and marital quality declines, on average, during the transition to parenthood (Kurdek, 1999). Moreover, depressive symptoms increase during the transition to parenthood, at least under certain circumstances (Evenson & Simon, 2005). For these reasons, we test the possibility that parenthood moderates the correlation between marital quality and depressive symptoms, perhaps in a way that differs by gender. Because of traditional gender roles and potential spillover effects of marital conflict into parenting and vice versa (Katz & Gottman, 1996), mothers may be more affected by marital support processes and family dynamics, making them particularly sensitive to marital quality (Carr & Springer, 2010).

Present Study

In the present study, we used a sample of Australian twins to test three specific hypotheses. First, we hypothesized that, after statistically adjusting for genetic and shared environmental selection using the twin design, marital quality still would predict depressive symptoms, a quasi-causal effect. Second, we hypothesized that although selection mechanisms would account for part of the observed association between marital quality and depressive symptoms in men and women, and the quasi-causal effect would be stronger for women. Finally, we added parenthood as a covariate to test the 3-way interaction among marital support, parenthood, and gender. Specifically, we hypothesized that the quasi-causal effect of an unhappy marriage on depressive symptoms would be stronger for mothers than both non-mothers and fathers.

Method

Sample

The twins in this study come from the second wave of the Australian Twin Registry (ATR), a sample representative of the adult Australian population born between 1893 and 1965 (Heath & Martin, 1994). Twins volunteered to participate in three waves of data collection. The initial wave, known as the Canberra study, was conducted from 1980–1981 ($N = 8,183$ individual twins, 69% response rate; Jardine & Martin, 1984). Participating twins in the Canberra study were contacted again in 1988–1989 to participate in the Alcohol Cohort Follow-up study, which focused on alcohol use and related risk factors and outcomes ($N = 6,327$ individual twins, 83% response rate; Heath & Martin, 1994). The present analyses used twins' depressive symptoms, marital support, and parental status scores collected in this wave.

To be included in the present study, twins must have been married or in a marital-type relationship (i.e., married ($N = 2,853$), cohabiting ($N = 170$), or separated but still married ($N = 109$); $N = 3,132$ individuals or 1,595 pairs, 50.42% of the total sample) at the time of data collection. Despite differences that may exist between these marital status designations, we selected this standard under the assumption that parents in these marital situations would be involved in a daily routine with their children. However, an additional 29 pairs of twins reported neither marital support ratings nor depressive symptom ratings, thereby reducing the sample to 1,566 pairs of twins, $MZ = 1,007$ (men = 311, women = 695) and $DZ = 560$ (men = 165, women = 395). Zygosity was determined by two items that provide 95% concordance with blood-typing (Heath, Eaves, & Martin, 1998). Because of the racial

homogeneity in Australia, race status was not collected. The vast majority of the sample is White. The mean age of the sample was 41.52 ($SD = 11.50$) years. The mean age for the male twins (41.76, $SD = 12.06$) was approximately equal to the mean female age (41.41, $SD = 11.26$).

Self-Reported Depressive Symptoms

Eight self-rated items derived from the well-validated Delusions-Symptoms-States Inventory (Bedford & Foulds, 1977) were collected on the twins' *current depressive symptoms*. Sample items include: "Recently I have been so miserable that I have had difficulty with my sleep" and "Recently I have been depressed without knowing why." Twins rated each item using the 4-point Likert scale (1 = *Not at all*, 2 = *A little*, 3 = *A lot*, 4 = *Unbearably*). Of the 1,566 twin pairs, 1,441 had reports of depressive symptoms, 124 had reports for only one twin, and 1 pair had reports for neither twin. We created mean scores of the eight items for each twin, such that higher scores indicate higher levels of self-reported depressive symptoms. The reliability of the depression items using McDonald's omega (ω) coefficient methodology (McDonald, 1999) is .85.¹

Self-Reported Marital Support

We operationalized marital quality with 3 items characterizing *supportive marital interactions* often used in epidemiological research to assess relationship functioning (Schuster, Kessler, & Aseltine, 1990). These items were self-rated using a 4-point Likert scale (1 = *Not at all*, 2 = *A little*, 3 = *Quite a bit*, 4 = *A great deal*): "How much [does your spouse] listen to you if you need to talk about your worries or problems?", "How much [does your spouse] understand the way you feel and think about things?", and "How much would [your spouse] go out of [his or her] way to help you if you really needed it?" Of the 1,566 twin pairs, 1,429 had reports of marital support, 134 had reports for only one twin, and 3 pairs had reports for neither twin. Like our depressive symptom variable, we used mean scores, such that higher scores indicate higher marital support. McDonald's omega (ω) for the marital quality items was also .85.

Parenthood Status

Parenthood status is a binary variable, in which *full-time parents* were coded 1 and *nonfull-time parents* were coded -1. Because parenting is most strongly associated with depression in persons actively raising children (Evenson & Simon, 2005), we restricted the status of "parenthood" to participants with children under the age of 18 under the assumption that parents with children who are minors were still engaged in the great majority of parenting practices. All parents with children greater than 18 years of age were considered "nonfull-time" and were coded the same as nonparents (-1). Thus, for our purposes, parenthood status is operationalized as a measure of "full-time" parenting. On average, nonfull-time parents were older, married for longer, and reported slightly higher education and yearly incomes than full-time parents.

Analyses

Our analyses are of two types: descriptive statistics and multivariate twin analyses. The descriptive statistics are easily interpretable means and correlations that illustrate genetic, shared environmental, or nonshared environmental effects. In addition, we rigorously test our hypotheses using multivariate models by fitting a series of structural equation models to

¹McDonald's omega (ω) uses a factor analytic approach to partition the common variance among the items from the unique variance (McDonald, 1999). It is the ratio of the common variance to the total variance (common and unique) and may be interpreted as the square of the correlation between the total test score and the common factor.

our data. Earlier, we outlined the general logic of using twin studies for drawing quasi-causal conclusions. Figure 1 depicts the biometric model we used to statistically evaluate quasi-causal effects, gender differences, and moderation of the quasi-causal effect of marital support on depressive symptoms. First, we decomposed twins' marital support into three sources of variance: genetic (A), shared environmental (C), and nonshared environmental (E).²

However, the classical univariate ACE variance decomposition of depressive symptoms is not our primary focus. The heart of our analyses lies in a second step, the regression of depression on the phenotype and biometric components of marital quality. The regression pathways b_{AMAR} and b_{CMAR} reflect the genetic and shared environmental effects, respectively, of marital support on depressive symptoms. Holding constant these selection effects, the phenotypic regression coefficient (b_{MAR}) is the critical test by which we can evaluate our hypothesis that marital support has a quasi-causal effect on depressive symptoms. To the extent that the phenotypic effect is significant *holding constant* the genetic and shared environmental effects on depressive symptoms, we may conclude a quasi-causal effect. PAR refers to the parenthood variable and INT refers to its interaction with marital support. Finally, the residual variances for depressive symptoms were allowed to covary across zygosity and gender.

Using this approach, we tested a series of models to evaluate evidence for nonrandom selection, gender differences, and interactions (described below). Models were compared using three measures – Root Mean Squared Error of Approximation (RMSEA), Bayesian Information Criterion (BIC), and χ^2 difference tests. RMSEA values below .05 are interpreted as good model fit and values below .08 are considered acceptable (Browne & Cudeck, 1993). Lower BIC values indicate better model fit. With respect to missing data, only cases with complete data were used for our correlation analysis (R 2.11; R Development Core Team, 2010). We conducted all multivariate analyses in *Mplus* 5.2 and used Full Information Maximum Likelihood (FIML) to handle missing data (Muthén & Muthén, 2008).

Results

Descriptive Results

Table 1 presents the within and across twin correlations among marital support and depressive symptoms, by gender and zygosity. The cross-trait correlation between marital support and depressive symptoms is about equal for men and women (men = $-.29$; women = $-.30$), providing evidence of an inverse phenotypic (i.e., observed) association of approximately equal strength for men and women. A comparison of the cross-twin correlations for MZ and DZ twins provide initial insight into the etiological composition of marital support. Stronger MZ concordance than DZ concordance suggests evidence of underlying genetic sources whereas equal MZ/DZ concordance rates provide evidence of shared environmental sources. DZ males demonstrated slightly stronger concordance (.21) than MZ males (.20), suggesting neither underlying genetic nor shared environmental effects among men, contrary to our second hypothesis. However, MZ females were more concordant for marital support (.27) than DZ females (.22), suggesting a partial underlying genetic etiology.

²There are two approaches to decomposing twin covariance of a given phenotype into between-family (A and C) and within-family (E) variance components. Very often they are represented by their own latent variable (A, C, and E; see Loehlin, 1996). An equivalent approach is to estimate individual latent factors for A and C components and set the twins' residual variance of their phenotype to be equal. The residual variance replaces the latent E factor, allowing for a more intuitive understanding of the quasi-causal association between two observed variables while holding constant unobserved selection effects.

Over half of the participants report parenting children under the age of 18 (men = 56.51%; women = 55.41%). Under conditions of different parenthood status the cross-trait correlation between marital support and depressive symptoms is stronger for full-time parents (men = $-.35$, women = $-.32$) than nonfull-time parents (men = $-.22$, women = $-.29$), supporting our prediction that parenthood strengthens the observed correlation.

Multivariate Analyses

As a starting point, we regressed twins' depressive symptom reports on their marital support reports to provide an initial idea of the effect difference between men and women. All parameter estimates were constrained to be the same for twin pairs under the assumption that twin siblings are interchangeable dyads (Olsen & Kenny, 2006). As expected, marital support negatively predicted depressive symptoms for men ($b = -0.14$, 95% confidence interval [CI] = -0.18 to -0.11) and women ($b = -0.17$, 95% CI = -0.19 to -0.15).

We compared a series of structural equation models to evaluate the gender equivalence of nonrandom selection mechanisms and quasi-causal effects of marital support on depressive symptoms. The left column of Table 2 presents the full gender difference model. Our initial model (Model 1) tested for gender differences between genetic and shared environmental selection effects and the quasi-causal effect of marital support on depressive symptoms. We observed that additive genetic effects and shared environmental effects did not significantly account for any of the variance in marital support in men or women. However, the residual (nonshared environmental) variance significantly accounted for the remainder of the variance in both men [$0.245 \div (0.022 + 0.044 + 0.245) = .7878 \times 100 = 78.78\%$] and women [$0.356 \div (0.092 + 0.052 + 0.356) = .7120 \times 100 = 71.20\%$].

The nonsignificant genetic effect in women was surprising given our correlational findings. This led us to believe that including the shared environment suppressed the genetic effect (Cohen, Cohen, West, & Aiken, 2003). Indeed, past research demonstrates that shared environmental effects often do not play an etiological role in marital quality (Spotts et al., 2004). Therefore, they were excluded in Model 2, showing no loss of fit when compared with Model 1 ($\chi^2 = 92.84$, $\Delta\chi^2 = 3.49$, $\Delta df = 4$, $p = .48$). Therefore, we were comfortable proceeding without including shared environmental effects. Model 2 demonstrated findings consistent with our correlational results. The genetic variance component for marital support was significant in women ($A_{MARF} = 0.15$, 95% CI = 0.11 to 0.19 ; $R^2 = .15$) but not men. Moreover, the genetic effect of marital quality on depressive symptoms also was significant for women ($b_{AM-ARF} = -0.16$, 95% CI = -0.29 to -0.04) but not men. Lastly, we observed a significant quasi-causal effect of marital quality on depressive symptoms in women ($b_{MARF} = -0.12$; 95% CI = -0.16 to -0.08) and men ($b_{MARM} = -0.14$; 95% CI = -0.19 to -0.08).

Next, we compared Model 2 to more restricted models to evaluate gender equivalence of the quasi-causal effect of marital support and equivalent genetic confounds. Model 3 evaluated gender equivalence of the quasi-causal effect of marital support by constraining the regression estimates to be the same across gender. Again, we observed no significant reduction in model fit compared with Model 2 ($\chi^2 = 93.00$, $\Delta\chi^2 = 0.15$, $\Delta df = 1$, $p = .70$), indicating that the effect of marital support on depressive symptoms is not significantly different for men and women ($b_{MARF} = b_{MARM} = -0.13$; 95% CI = -0.16 , -0.09). Lastly, Model 4 tested the gender equivalence of the genetic effect of marital support on depressive symptoms. The chi-square difference test showed a significant reduction in model fit ($\chi^2 = 106.77$, $\Delta\chi^2 = 13.77$, $\Delta df = 2$, $p < .01$), suggesting preference for Model 3 and evidence of genetic effects in women but not men.

Biometric Moderated Analysis

Using the same logic as above, we regressed twins' depressive symptom reports on marital support, parenthood status, and the interaction term between them. We did not find a significant 3-way interaction between marital support, parenthood, and gender ($b_{males} = -0.02$, 95% CI = $-0.06 - 0.01$; $b_{females} = -0.02$, 95% CI = $-0.04 - 0.01$). However, the 2-way interaction between marital support and parenthood was significant ($b = -0.02$, 95% CI = $-0.04 - -0.001$).³

On the basis of this finding, we included parenthood and the interaction term between it and marital support into the biometric model described above. However, our third research question was modified; gender difference tests for the moderating role of parenthood status on the quasi-causal effect of marital support on depressive symptoms—holding constant genetic effects—were omitted. The 2-way interaction was significant ($b_{INTF} = b_{INTM} = -0.02$; 95% CI = $-0.03, -0.004$), as well as the simple slope for full-time parents ($b = -0.15$, $SE = 0.02$, $p < .01$) and nonfull-time parents ($b = -0.11$, $SE = 0.02$, $p < .01$). The simple slopes may be interpreted such that increases in marital support lead to greater predicted decreases in depressive symptoms for full-time parents than nonfull-time parents. That is, full-time parents' depressive symptoms are slightly more sensitive to differences in marital support than nonfull-time parents, demonstrated in Figure 2 by the slightly steeper regression line for full-time parents.

Discussion

Using a genetically informed design, we tested three hypotheses about the relation between marital support and depressive symptoms. Our results support our first hypothesis that marital support predicts depressive symptoms even after statistically adjusting for nonrandom genetic confounds. Our second hypothesis was partially supported. Based on prior research demonstrating gender differences (Dehle & Weiss, 1998; Fincham et al., 1997; Kiecolt-Glaser & Newton, 2001), we predicted that the quasi-causal effect of marital support on depressive symptoms would be significantly stronger in women than men, holding constant genetic and/or shared environmental confounds. However, our results suggest that genetic selection is evident only in women and that the quasi-causal effect of marital support on depressive symptoms was not statistically different between husbands and wives. Lastly, these results partially support our third hypothesis—that the strength of the quasi-causal effect of marital support depends on whether one is a full-time parent or nonfull-time parent. We did not observe a 3-way interaction between marital support, parenthood, and gender. However, the present results suggest that, irrespective of gender, parenthood moderated the effect of marital support on depressive symptoms, such that marital support was more strongly associated with depressive symptoms for full-time parents than for nonfull-time parents.

The study of marital relationships and depression is not unlike a game of cat's cradle: an interactive two-person game that can produce multiple outcomes, many tied up in a frustrating knot. However, behavior genetic studies dis-entangle one substantial knot—the realistic possibility that genetic and environmental selection account for part of the association between marital problems and depressive symptoms. These results corroborate

³Heterogeneity within marital status (married, remarried, co-habiting, separated but married) contributes to the interaction effect. When the *separated but married* individuals are removed from the analysis, the pattern of results holds but the effect of the interaction is no longer significant. However, separated spouses constitute the lower end of the marital support distribution of spouses in marital-type relationships, thereby reducing the total variance of marital support. Subsequent analysis showed that holding constant the effect of separation, the pattern and significance of the interaction remains although the magnitude of the effect decreases. We thank one reviewer for drawing our attention to this source of within-group variation.

prior findings (Spotts et al., 2004, 2005) and permit the strongest possible inference that low marital support predicts increases in concurrent depressive symptoms. This is because twin analyses control for measured and *unmeasured* genetic selection into having an unhappy marriage or feeling depressed.

As noted in the Introduction, an additional complication making causal interpretations of the relation between marital quality and depressive symptoms is the likelihood that they are reciprocally related, as suggested by stress generation models and erosion of support models of depression (Coyne, 1976; Hammen, 1991). Spotts et al. (2004) did not address this issue in their study of marital quality and depressive symptoms, reporting that nonshared environmental sources partly accounted for their observed association. By way of quasi-experimentation, the present study extends their work by showing that level of marital support systematically predicts level of depressive symptoms, holding constant *any* genetic influence on the association. However, like their data, ours too is cross-sectional. Even stronger conclusions about the effects of marital quality could be drawn studying it over time.

Some have argued that marital status matters more to men whereas marital quality matters more for women (Waite & Gallagher, 2000). Some findings demonstrate no gender differences (Davila et al., 2003) whereas others do (Fincham et al., 1997; Dehle & Weiss, 1998). The current findings regard marital support as equally important to both members in the marital dyad in relation to depression. Yet, the effect may be a matter of degree as well as differences in the underlying processes. This is the first evidence to date investigating the underlying genetics of marital support and depressive symptoms in men, wherein we did not find support for our hypothesis. However, these results may be unique to these data. This finding needs future replication before any firm conclusions can be drawn about the underlying pathways linking marital support to depressive symptoms in men. Other research directions may involve comparing mean changes in depressive symptoms between husbands and wives as a function of marital support to evaluate whether the effect produces equal symptomatology.

The interaction between parenthood and marital support is also noteworthy. The strains of parenthood are well documented (Evenson & Simon, 2005; Twenge, Campbell, & Foster, 2003), yet the present results support the intuitively appealing idea that a supportive marriage appears to protect parents against some of the strains of parenting, as spousal support was found to predict slightly greater decreases in depressive symptoms in full-time parents than nonfull-time parents. Indeed, these findings are supported by prior research suggesting that belonging to a partnership and having at least one child may contribute to gains in happiness (Kohler, Behrman, & Skytthe, 2005). Future exploration is needed to learn more about how the effect of marital support on depressive symptoms changes across parenthood.

These findings also have implications for intervention, especially because they indicate that the relationship between marital support and depressive symptoms is not an artifact of selection. A negative feedback loop apparently exists between an unhappy marriage and depression (Beach et al., 1990; Davila et al., 1997). Psychotherapeutic research consistently shows that couples therapy has a therapeutic impact on spouses' depressive symptoms (Beach & O'Leary, 1992). However, research also shows that cognitive-behavioral marital treatments and behavior marital treatments for depression (Butler, Chapman, Forman, & Beck, 2006) also help to reduce marital hostility, thereby supporting a bidirectional causal association between marital quality and depressive symptoms.

Other limitations are inherent in the present study. First, the data are cross-sectional, raising questions about the direction of causal effects that are better addressed by longitudinal designs. Second, the comparison between husbands and wives is not a comparison of spouses in the same marriage. Therefore, it is possible that the effect of marital support on depressive symptoms may differ between husbands and wives in the same marriage. Third, our operational definition of parenthood was broad. Important differences likely exist between parents with different aged children. Because of limited statistical power, we operationalized “full-time” parents as anyone with a child younger than the age of 18. Future research will provide the opportunity to refine our definition of parenthood and focus on a specific period of parenting, such as early childhood.

Despite these limitations, the present study makes several important contributions to the literature on marital quality and depressive symptomatology. As noted, the twin design controls for measured and unmeasured genetic and shared environmental selection. This offers the strongest realistically possible method of showing that the relationship between an unsupportive marriage and depressive symptoms is not a byproduct of genetic risk, but is, in fact, causal. The present study also shows that high marital support benefits both wives and husbands. Finally, the interaction between marital support and parenthood emphasizes the importance of studying the benefits of marriage in specific contexts.

Acknowledgments

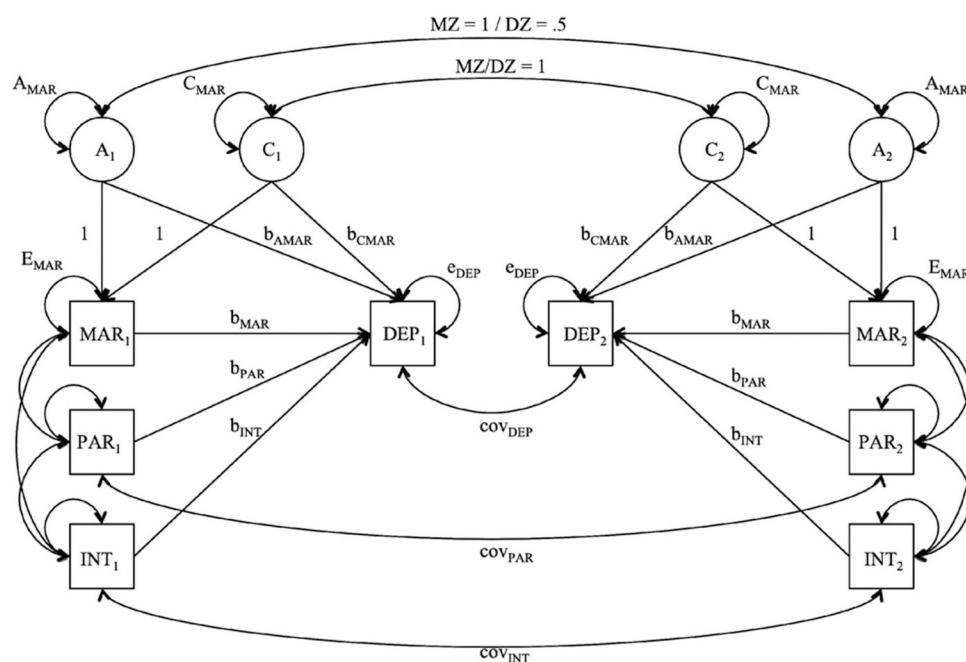
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**Figure 1.**

Full Multivariate Biometric Regression Model. A = additive genetic effect; C = shared environmental effect; E = nonshared environmental (residual) effect; MAR = Marital Support Report; DEP = Depressive Symptom Report; PAR = parenthood status; INT = Interaction between marital support and parenthood; cov = covariance; b = unstandardized regression coefficient. Subscripts 1 and 2 denote Twin 1 and Twin 2.

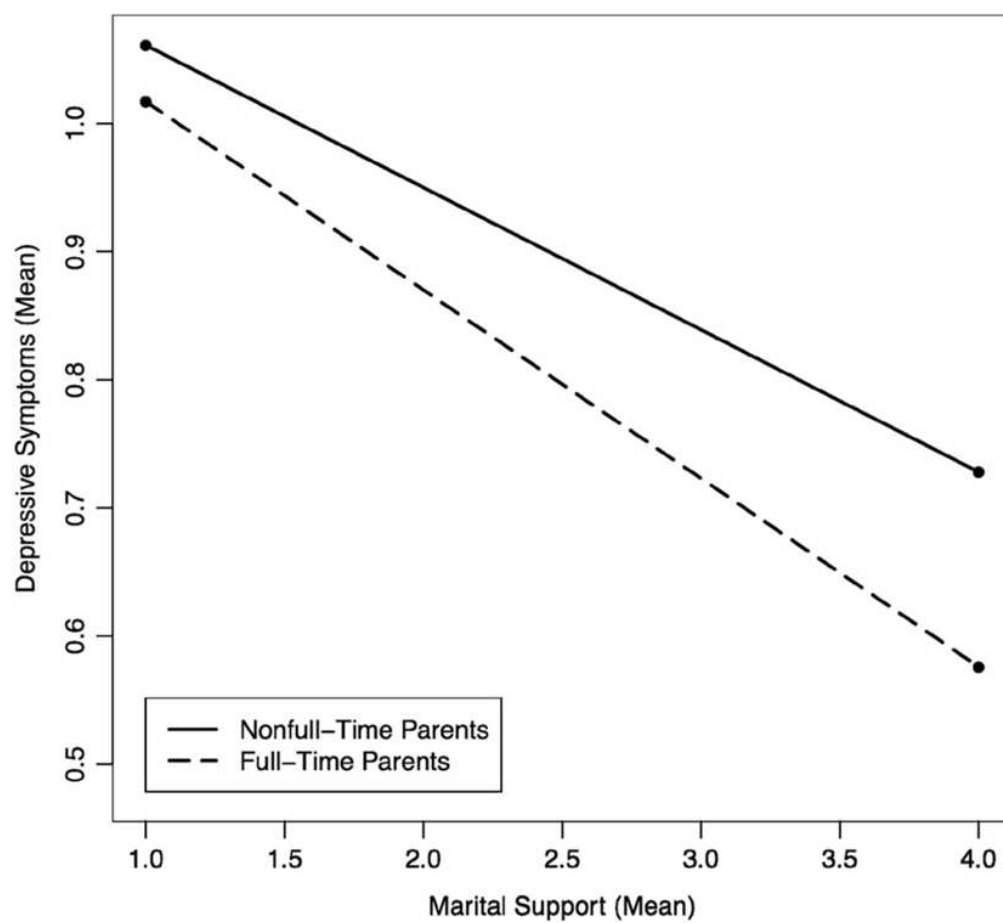


Figure 2.
Regression line plots of the effect of marital support on depressive symptoms dependent on parenthood status.

Table 1

Cross-Twin and Cross-Variable Correlations, Means, and SDs Between Marital Support and Depressive Symptoms for Male and Female MZ (Bolded) and DZ Twins

	Male		Female	
	Marital support	Depressive symptoms	Marital support	Depressive symptoms
Marital support	.20/.21	–.30	.27/.22	–.26
Depressive symptoms	–.25	.17/.20	–.36	.22/.12
Means	3.61	1.14	3.36	1.25
<i>SD</i>	0.56	0.28	0.71	0.39

Table 2

Unstandardized Parameter Estimates for the Multivariate Biometric Models Comparing Male and Female Spouses

	Model 1 full	Model 2 No C	Model 3 equal E regression	Model 4 equal A regression ^a	Model 5 interaction
Variance components					
A _{MARF}	0.09 (0.06)	0.15 (0.02)	0.15 (0.02)	0.11 (0.01)	0.15 (0.02)
A _{MARM}	0.02 (0.02)	0.07 (0.02)	0.07 (0.02)		0.06 (0.02)
C _{MARF}	0.05 (0.05)	—	—	—	—
C _{MARM}	0.04 (0.02)	—	—	—	—
E _{MARF}	0.36 (.02)	0.35 (0.02)	0.35 (0.02)	0.38 (0.02)	0.35 (0.02)
E _{MARM}	0.25 (0.02)	0.24 (0.02)	0.24 (0.02)	0.22 (0.02)	0.25 (0.02)
Regression coefficients					
b _{AMARF}	– 0.34 (0.33)	–0.16 (0.06)	–0.10 (0.06)	–0.10 (0.06)	–0.14 (0.06)
b _{AMARM}	– 1.00 (0.11)	–0.04 (0.11)	– .07 (0.08)		–0.04 (0.09)
b _{CMARF}	0.11 (0.38)	—	—	—	—
b _{CMARM}	0.38 (0.51)	—	—	—	—
b _{MARF}	– 0.12 (0.02)	–0.12 (0.02)	–0.13 (0.02)	–0.13 (0.02)	–0.13 (0.02)
b _{MARM}	– 0.12 (0.03)	–0.14 (0.03)			
b _{PARF}	—	—	—	—	–0.004 (0.01)
b _{PARM}	—	—	—	—	
b _{INTF}	—	—	—	—	–0.02 (0.01)
b _{INTM}	—	—	—	—	
Model fit					
RMSEA	.06	0.06	0.06	0.06	0.05
BIC	8,048.54	8,022.6	8,015.39	8,014.45	22,812.74
χ^2 , $df(p)$	89.36, 34 (<.001)	92.84, 38 (<.001)	93.00, 39 (<.001)	106.77, 41 (<.001)	250.49, 129 (<.001)
$\Delta\chi^2$, $\Delta df(p)$	—	3.49, 4 (.480) ^b	.15, 1 (.698) ^c	13.77, 2 (.001) ^d	—

Note. *SEs* are in parenthesis, and bolded values indicate that the parameter is significant $p < .05$. A = additive genetic effect; C = shared environmental effect; E = nonshared environmental (residual) effect; MAR = marital support; DEP = depressive symptoms; PAR = parenthood status; INT = interaction between marital support and parenthood status; b = unstandardized regression coefficient; F = female; M = male.

^aIt was necessary to constrain the genetic variances for marital quality to be equal across gender so that the equality of the unstandardized genetic regression coefficient could be tested.

- ^b Compared with Model 1.
- ^c Compared with Model 2.
- ^d Compared with Model 3.