

A Twin Study on Perceived Stress, Depressive Symptoms, and Marriage

Journal of Health and Social Behavior
2017, Vol. 58(1) 37–53
© American Sociological Association 2017
DOI: 10.1177/0022146516688242
jhsb.sagepub.com


Christopher R. Beam¹, Diana Dinescu², Robert Emery²,
and Eric Turkheimer²

Abstract

Marriage is associated with reductions in both perceived stress and depressive symptoms, two constructs found to be influenced by common genetic effects. A study of sibling twins was used to test whether marriage decreases the proportion of variance in depressive symptoms accounted for by genetic and environmental effects underlying perceived stress. The sample consisted of 1,612 male and female twin pairs from the University of Washington Twin Registry. The stress-buffering role of marriage was tested relative to two unmarried groups: the never married and the divorced. Multivariate twin models showed that marriage reduced genetic effects of perceived stress on depressive symptoms but did not reduce environmental effects. The findings suggest a potential marital trade-off for women: access to a spouse may decrease genetic effects of perceived stress on depressive symptoms, although marital and family demands may increase environmental effects of perceived stress on depressive symptoms.

Keywords

depressive symptoms, marital status, marriage benefit, perceived stress, stress sensitivity

An abundance of research has demonstrated the relation between stress factors and depressive symptoms (Dohrenwend et al. 1984; Kendler and Prescott 2006; Kessler et al. 1992; Segal et al. 2008), with greater stress generating greater symptomatology and vice versa (Hammen 1991). Over the past two decades, researchers have worked toward understanding the etiology of the association between stress and depressive symptoms (Caspi et al. 2003), investigating causal pathways between them (Kendler, Karkowski, and Prescott 1999; Schnittker 2010), and testing whether social support factors attenuate stress effects (Thoits 1995, 2011). Under Kessler's (1997) recommendation that "research on stress modifiers should use genetically informative designs, such as twin or adoption studies, to investigate related possibilities in more detail" (p. 209), the purpose of the following article is to report a twin study where differences in marital status were hypothesized and tested as modifiers of the genetic and environmental effects of perceived stress on depressive symptoms.

BACKGROUND

Stress-relieving Benefits of Marital Support

Married people often report fewer stress and depressive symptoms than unmarried and marital separated people (Carlson 2012; Gove, Hughes, and Style 1983; Horn et al. 2013; Johnson and Wu 2002; Pearlin and Johnson 1977). Researchers repeatedly have found that marital support reduces the association between stress and depressive symptoms for

¹University of Southern California, Los Angeles, CA, USA

²University of Virginia, Charlottesville, VA, USA

Corresponding Author:

Christopher R. Beam, Department of Psychology,
University of Southern California, 3620 South
McClintock Avenue, Los Angeles, CA, USA, and
Davis School of Gerontology, University of Southern
California, 3715 South McClintock Avenue, Los Angeles,
CA, USA.

E-mail: beamc@usc.edu

both men and women (Bierman, Fazio, and Milkie 2006; Carr and Springer 2010; Kessler and Essex 1982), despite occasional weak (Walen and Lachman 2000) or null results (Reifman, Biernat, and Lang 1991; Thoits 1984). This finding is often interpreted in light of the *stress-buffering hypothesis* (Cohen and Wills 1985; Hammen 2005), a theory positing that the presence of a spouse reduces emotional reactions to stress.

Marital support matters for preventing harmful psychological consequences of stress, but the mechanisms underlying buffering effects of marital support still remain unclear (Thoits 2011). Thoits (2011) suggests two stress-relieving social mechanisms: emotional sustenance and active coping assistance. Emotional sustenance includes the provision of care and concern during stressful times with sympathy and elevating one's sense of belongingness. Active coping assistance includes advice and instrumental aid that only close, significant others can provide during periods of high stress.

Marriage also is important for receiving invisible (i.e., given but unrecognized) support and for learning social skills that help manage stress. Unlike other forms of partner support, invisible support has been found to predict decreases in anger, anxiety, and depressive symptoms (Shrout, Herman, and Bolger 2006). Marriage also may be an essential mechanism through which people acquire skills to manage stress (Cohen, Sherrod, and Clark 1986), leading to larger social networks and better interpersonal skills (i.e., social competency and self-disclosure) with which to draw support.

Finally, genetics may be an important factor for why marriage reduces effects of stress on depressive symptoms. Research on the 5-HTTLPR gene variant—a widely recognized genetic determinant of stress-induced depressive symptoms (Caspi et al. 2003; Kendler et al. 2005)—has shown that short allele variants predict increases in depression and stress perception (Conway et al. 2011). Genetic effects underlying stress appraisals and depressive symptoms, thus, may depend on support mechanisms (e.g., invisible support and social skills) often provided in marriages.

In this article, we propose that marriage may reduce environmental effects of perceived stress on depressive symptoms as well as genetic effects that generate their association. While perceived stress may also moderate the genetic and environmental effects of marital status on depressive symptoms, we consider only whether marriage buffers effects of perceived stress on depressive symptoms. Married people, for example, have been found to have lower

heritability estimates of depressive symptoms (Heath, Eaves, and Martin 1998), possibly for the reason that attractive and wealthier people, for example, may not only have lower genetic liabilities for depressive symptoms but also are more “marriageable” than people perceived to be unattractive and impoverished. When coping resources are operationalized by marital status, however, no evidence of common genetic effects have been observed (Horn et al. 2013). Even when genetic effects have been observed to generate the association between social support and depressive symptoms (Kessler et al. 1992), perceived stress was not found to be a significant moderator of the association. For these reasons, we tested whether marriage reduces effects of perceived stress on depressive symptoms.

The moderating role of marriage may matter more for reducing effects of perceived stress on depressive symptoms in men than in women. While some research has shown little to no marital advantage for either gender (Bierman et al. 2006; Blekesaune 2008), marriage has been found to benefit the mental health of men and women in different ways, with men benefiting more often than not over women (Carr and Springer 2010; Kim and McKenry 2002; Maciejewski, Prigerson, and Mazure 2001; Waite and Gallagher 2000). For example, marriage engenders more emotional support and social connection for men than for women but more financial support for women than for men. The effects of social isolation, like lack of a spouse, have also been found to have stronger negative effects on mental health outcomes in men than in women, as found in loneliness research (Cacioppo et al. 2015). Thus, we expect marriage to have a stronger moderating role on the effects of perceived stress on depressive symptoms for the reason that men reap greater emotional support and social connectedness from marriage than women do.

Parsing Genetic Effects from Environmental Effects

Genetic effects have been found to underlie the association between stress and depressive symptoms (Bogdan and Pizzagalli 2009; Kendler et al. 1999; Schnittker 2010). *Genetic effects*, which refer to the likelihood of inheriting a disorder, occur when perceived stress and depressive symptoms are not causally related but are correlated for nonrandom genetic reasons. In contrast, *environmental* (or *nonshared environmental*) *effects*, which refer to the causal relation between perceived stress and depressive symptoms, occur when perceived stress

significantly influences depressive symptoms after appropriate controls for selection effects (e.g., genetic effects) are taken into account. Additionally, *shared environmental effects* can occur too, that is, the increased likelihood of nonrandom exposure to environments that influence the development of a disorder (e.g., parental rearing environments). Shared environmental effects, however, rarely are found in adult twin sibling samples (Bergen, Gardner, and Kendler 2007). While the term *environmental effects* could refer to shared or nonshared environmental effects, we use the term strictly to indicate a causal effect. Thus, the first aim of the present study is to replicate prior findings and parse genetic effects from environmental effects of perceived stress on depressive symptoms.

Twin sibling studies are a powerful method for testing hypothesized environmental effects between two variables, like perceived stress and depressive symptoms. Given that twins share both genetic and shared environmental experiences (e.g., including parents, child-rearing styles, and socioeconomic status [SES]), observed differences between twins, unlike differences between unrelated individuals, control for genetic and shared environmental selection. Observed differences between twins, then, are necessarily attributable to differences in the twins' cumulative nonshared experiences, like differences in social networks, educational attainment, and other social environments (Turkheimer and Harden 2014). Researchers commonly assess and control selection effects in correlational research (e.g., SES), yet inherent in the design of twin sibling studies is the control for genetic and shared environmental effects, whether measured or not (Beam et al. 2011; D'Onofrio et al. 2005). By ruling out two sweeping categories of alternative explanations, twin sibling studies allow investigators to make far stronger inferences about the causal effects between related phenomena compared with studies of unrelated individuals.

Twin sibling studies also are useful for testing potential causal hypotheses when data are cross-sectional (Heath et al. 1993). In previous studies on the stress-buffering role of marriage that used cross-sectional samples of individuals (Aneshensel and Stone 1982; Kessler and Essex 1982; Thoits 1984), genetic effects may have confounded conclusions that stress influences depressive symptomatology. In twin sibling models, once genetic effects have been taken into account, significant environmental effects mean that if identical twins with higher scores on perceived stress than their co-twins also report significantly higher depressive symptoms, the correlation can be thought to result from "within-family random

assignment" to different perceived stress conditions. With marital support included as a social modifier, twin sibling studies provide more stringent tests of the hypothesis that marriage buffers environmental effects of perceived stress on depressive symptoms.

The possibility that marriage may attenuate the genetic and shared environmental effects of perceived stress on depressive symptoms also has not been tested empirically. Consistent with the stress-buffering view, genetic factors underlying perceived stress may not account for as much variance in depressive symptoms in married people as in unmarried people. A high-strung husband might appraise daily hassles as more stressful than others, but spousal support may reduce his genetic liability for developing depressive symptoms associated with perceived stress. The second aim of the present study, thus, is to test whether marriage lowers genetic effects of perceived stress on depressive symptoms, in addition to the hypothesized environmental effects described above.

To test whether marriage has benefits for reducing effects of perceived stress on depressive symptoms, we compared the married to two unmarried groups: the never married and the divorced. Relative to the never married, marriage offers access to spousal support that may dampen perceptions of stress and consequent depressive symptoms (Cohen, Gottlieb, and Underwood 2000). Conversely, entering marriage may narrow access to broader support networks (e.g., friends) beyond married and family life, limiting coping resources that also may lower stress, particularly for women (Antonucci and Akiyama 1987). Relative to the divorced, married people tend to report fewer stress and depressive symptoms, for the reason that the emotional, social, logistical, and financial burdens prior to and following divorce may deplete people's internal and external coping resources (Lucas 2005; Sbarra et al. 2014). Thus, the stress-buffering benefits of marriage ought to be most noticeable when comparing differences between married and divorced people.

We conducted the present study with a large sample of male and female twins. On the basis of our two aims, we made three hypotheses. First, we hypothesized that genetic and environmental effects underlying perceived stress would also account for variation in depressive symptoms. Second, we hypothesized that being married would reduce the genetic and environmental effects of perceived stress on depressive symptoms compared to being divorced or never married. We expected that genetic and environmental effects of perceived stress would account for the least variation in depressive symptoms among married people and account for the

most variation in divorced people, as divorce often consists of intractable stress that married and never-married people do not undergo (Berman and Turk 1981; Emery 2011). Separate analyses were conducted in male and female twins, as marriage has been found to correlate differentially with measures of stress and depression (Carr and Springer 2010). Therefore, we made the third hypothesis that marriage would benefit men more than women, as the emotional support provided to men would lead to greater reductions in all hypothesized effects of perceived stress on depressive symptoms.

DATA AND METHODS

Sample

The University of Washington Twin Registry (UWTR) is a representative sample of citizens ages 16 years and older from the state of Washington (Afari et al. 2006). Twins were identified based on their registration for a driver's license with the Washington State Department of Licensing. All residents must have indicated on their application whether they had a twin. Once twins were identified through state licensing registration, they were sent a Health and Wellbeing (HWB) questionnaire and consent form to be returned by mail. The questionnaire consisted of items about physical health, mental health, and lifestyle habits. Although participant recruitment in the UWTR is ongoing, the HWB questionnaire was sent to all twins registered as of April 2012 and had a response rate of 76.4%. The total HWB sample included 3,587 families (6,124 individual twins). We used three criteria to select pairs of twins from the HWB subsample for the current study (remaining pairs based on each successive criteria are indicated in parentheses): (1) known zygosity status (i.e., either monozygotic [MZ] or dizygotic [DZ]; remaining pairs = 3,502), (2) DZ twins had to be of the same sex (i.e., no opposite-sex fraternal twin pairs) to eliminate within-family gender differences as a potential confound (remaining pairs = 2,755), and (3) both twins must have responded to the marital status item (i.e., reported married, never married, or divorced), with at least one twin in each family reporting being married (remaining pairs = 1,612). This sample selection process was based on the logic of proband studies and permitted the married group condition to be common across all families.

All data used for the current study were from the HWB questionnaire and were cross-sectional. The sample for this analysis consisted of 1,612 pairs

(44.94% of HWB families), with 1,606 complete pairs, 4 pairs with complete data from one twin and missing data for the co-twin, and 2 pairs with complete data from one twin and partial data for the co-twin. There were 403 MZ male pairs, 138 DZ male pairs, 768 MZ female pairs, and 303 DZ female pairs. The ethnic background of the sample was 88.83% Caucasian, 2.82% Asian, 1.49% African American, .65% Native American, .34% Pacific Islander, and 5.71% Other. The unit of analysis in the multivariate twin analyses was the twin pair.

Measures

Depressive symptoms were measured with three items from the Patient Health Questionnaire (PHQ-9), a nine-item measure used for depression diagnosis and severity (Spitzer, Kroenke, and Williams 1999). The PHQ-9 corresponds to the *Diagnostic and Statistical Manual of Mental Disorders* diagnosis of depression and assesses the severity of the nine symptom criteria in the past two weeks. Although a brief, three-item version is not as ideal as the full nine-item questionnaire, it has been shown that even a two-item version can be used as a valid and reliable assessment of clinical depression, in both diagnosis and severity (Löwe, Kroenke, and Gräfe 2005). The three-item assessment (PHQ-3) in the HWB measured anhedonia ("little interest or pleasure in doing things"), depressed mood ("feeling down, depressed, or hopeless"), and fatigue or decreased energy ("feeling tired or having little energy"). The version of the PHQ-3 in the HWB questionnaire was modified to address symptom presence over the past four weeks. Symptomatology was rated on a four-point scale ("not at all" = 0, "several days" = 1, "more than half of the days" = 2, and "nearly every day" = 3). Higher scores indicated greater symptom severity. Cronbach's alpha was used to calculate the reliability of the three items. For the male twins, $\alpha = .79$, and for the female twins, $\alpha = .81$. Participants' mean PHQ-3 score was computed and used to index depressive symptoms in the current study.

Perceived stress was measured with the 10-item version of the Perceived Stress Scale (PSS; Cohen, Kamarck, and Mermelstein 1983; Cohen and Williamson 1988). The PSS measures stress perception over the past four weeks. Examples include the following: "In the past four weeks, how often have you been upset because of something that happened unexpectedly?"; "In the past four weeks, how often have you felt nervous and 'stressed'?"; and "In the past four weeks, how often have you

felt that things were going your way?" (reverse scored). The items contained five responses ("never" = 0, "almost never" = 1, "sometimes" = 2, "fairly often" = 3, and "very often" = 4) and were scaled so that higher scores reflected greater perceived stress. The PSS has been found to consist of two facets: perceived helplessness and perceived self-efficacy (Reis, Hino, and Añez 2010; Roberti, Harrington, and Storch 2006). *Perceived helplessness* better represents the degree to which people view their lives as uncontrollable, whereas *perceived self-efficacy* better represents how overloaded by stress people view their lives (Cohen and Williamson 1988). A preliminary exploratory factor analysis of the 10 items showed that the same two-factor structure fit the male and female twin data best. Cronbach's alpha was used to calculate the reliability of the perceived helplessness items. For the male twins, $\alpha = .85$, and for the female twins, $\alpha = .86$. Cronbach's alpha of the perceived self-efficacy items was .81 for the male twins and .83 for the female twins. Participants' mean perceived helplessness and perceived self-efficacy scores were computed and used to index the two facets of perceived stress in our analyses.

Three marital status groups were considered: married, never married, and divorced. Of all male twins ($n = 1,082$), 50.55% were married, 43.44% were never married, and 6.01% were divorced. Of all female twins ($n = 2,142$), 55.14% were married, 36.18% were never married, and 8.65% were divorced. The following numbers of male twin pairs were concordant for marital status: 212 married, 197 never married, and 9 divorced. The following numbers of male twin pairs were discordant for marital status: 76 married/never married and 47 married/divorced. The following numbers of female twin pairs were concordant for marital status: 436 married, 308 never married, and 18 divorced. The following numbers of female twin pairs were discordant for marital status: 159 married/never married and 150 married/divorced.

The raw marital status variable was a nominal variable with three levels, coded as: married = 0, never married = 1, and divorced = 2. In the multivariate analyses, two dummy variables were created using Cohen's (1968) coding scheme for nominal variables to test for differences in the genetic and environmental effects of perceived stress on depressive symptoms in the three marital groups. Married twins served as the reference group. The first dummy variable was dichotomized to test the effects of perceived stress on depressive symptoms in never-married twins (married = 0,

never married = 1, divorced = 0). The second dummy variable was dichotomized to test for effects in divorced twins (married = 0, never married = 0, divorced = 1). Both dummy variables were included in all multivariate analyses and were treated as within-family difference variables so that twin pairs concordant and discordant for marital status could be included in the moderation analysis (van der Sluis, Posthuma, and Dolan 2012). Parameter estimates for each marital status group were computed in the same analysis.

Data Analysis

Data analysis consisted of descriptive analyses and multivariate twin analyses conducted separately for male and female twins. First, we present the means and standard deviations of the perceived stress facets and depressive symptoms across the different marital status groups. Second, to address our first hypothesis, MZ and DZ twin correlations are presented to provide initial evidence for whether genetic, shared environmental, and nonshared environmental factors influenced each of the three variables. To address our second and third hypotheses, phenotypic correlations between the perceived stress facets and depressive symptoms are given by marital status group and gender.

Co-twin control analyses were conducted to further examine our second hypothesis by comparing correlations between twins discordant for marital status. This analysis consisted of computing correlations between each of the perceived stress facets and depressive symptoms separately for married twins and their unmarried co-twins and then comparing differences between each set of correlations. Co-twin control analyses help to strengthen claims about causality, as differences in the correlations between twins discordant for marital status cannot be attributed to genetic and environmental factors shared between twins. Significance tests are provided for transparency but are not interpreted because the available sample sizes of discordant twin pairs were small and lacked power to discriminate differences between discordant twins.

Finally, the multivariate twin analyses included all twin pairs (concordant and discordant) to address all three hypotheses. First, twin (ACE) models were used to quantify the underlying genetic (A), shared environmental (C), and non-shared environmental (E) effects common and unique to the perceived stress facets and depressive symptoms. Genetic effects comprise the common genes twins share (MZ twins share 100% of their

segregating genes, while DZ twins share 50%, on average) that make them resemble one another, so in twin models, genetic factors are correlated 1.0 between MZ twins and .5 between DZ twins. Shared environmental effects include environmental factors that make twins from the same family resemble one another. Shared environments affect MZ and DZ twins identically, so these factors are correlated 1.0 for both zygosity types. Nonshared environmental effects include environmental factors that make twins different from one another, including measurement error, and are uncorrelated between pairs of twins. When MZ twin correlations are less than 1, this indicates that environmental influences must contribute to a trait. Standard ACE models make three additional assumptions: (1) the ACE components are uncorrelated with one another; (2) the components do not interact with one another, although they may interact with covariates, like marital status; and (3) mating between twins' parents is random.

We estimated bivariate Cholesky ACE models to quantify the proportion of variation in depressive symptoms attributed to genetic, shared environmental, and nonshared environmental effects common to the perceived helplessness and perceived self-efficacy facets. The baseline model is presented in Figure 1. The parameters of interest were the genetic (b_{0A}), shared environmental (b_{0C}), and nonshared environmental (b_{0E}) regression effects, as they represent the overlap between each biometric component underlying perceived stress and depressive symptoms (labeled PS and PHQ-3 in Figure 1, respectively). The nonshared environmental regression effect represents the strongest evidence of a causal relation in studies where random assignment is not possible for ethical and logistical reasons. The HWB data were correlational, so strict causal conclusions about the effects of perceived stress on depressive symptoms could not be drawn; other uncontrolled third variable confounds could account for the effects. Thus, b_{0E} was interpreted as the predicted effect of perceived stress on depressive symptoms while simultaneously adjusting for any genetic and shared environmental effects of perceived stress on depressive symptoms.

In the baseline model (Figure 1), all genetic and environmental effects were estimated to be the same across all marital status groups. All coefficients were constrained to be equal across the MZ and DZ twin groups. The main effects of the twins' marital status (b_1MS_{NM} and b_2MS_{DIV}) and their co-twins' marital status (b_3MS_{NM} and b_4MS_{DIV}) on twins' depressive

symptom scores were estimated to account for known biases in the estimates of gene-by-environment interaction models when all main effects pathways were not included in the models (van der Sluis et al. 2012). The correlations between twin 1's dummy-coded marital status variables and twin 2's dummy-coded marital status variables were estimated separately in the MZ and DZ twin groups to allow for differences based on their degree of consanguinity. All multivariate models adjusted for the effects of age on perceived stress and depressive symptoms.

Finally, gene-environment interaction parameters were added to the model to test whether the genetic (b_{0A}), shared environmental (b_{0C}), and nonshared environmental (b_{0E}) effects of perceived stress on depressive symptoms varied as a function of marital status. The interaction model simultaneously tested whether marital status moderates the genetic, shared environmental, and nonshared environmental effects of the perceived stress facets on depressive symptoms. All main effects of marital status were retained in the interaction models, as in the baseline main effects model.

Using the parameter estimates from the interaction models, we computed separate heritability (h^2), shared environmental (c^2), and nonshared environmental (e^2) estimates for the married, never-married, and divorced groups. Based on the dummy-coding scheme for marital status, the following equations were used to calculate the heritability estimates of depressive symptoms dependent on the perceived stress facets (the same set of equations applied for computing c^2 and e^2 in each marital group):

$$h^2_{\text{Married}} = \left[(b_{0A} + b_{1A}MS_{NM} + b_{2A}MS_{DIV})^2 * \sigma^2_{AS} \right] / \sigma^2_{\text{PHQ-3}}$$

$$\sigma^2_{\text{PHQ-3}} = \left[(b_{0A})^2 * \sigma^2_{AS} \right] / \sigma^2_{\text{PHQ-3}}$$

$$h^2_{\text{Never Married}} = \left[(b_{0A} + b_{1A}MS_{NM} + b_{2A}MS_{DIV})^2 * \sigma^2_{AS} \right] / \sigma^2_{\text{PHQ-3}}$$

$$\sigma^2_{\text{PHQ-3}} = \left[(b_{0A} + b_{1A}MS_{NM})^2 * \sigma^2_{AS} \right] / \sigma^2_{\text{PHQ-3}}$$

$$h^2_{\text{Divorced}} = \left[(b_{0A} + b_{1A}MS_{NM} + b_{2A}MS_{DIV})^2 * \sigma^2_{AS} \right] / \sigma^2_{\text{PHQ-3}}$$

$$\sigma^2_{\text{PHQ-3}} = \left[(b_{0A} + b_{2A}MS_{DIV})^2 * \sigma^2_{AS} \right] / \sigma^2_{\text{PHQ-3}}$$

The h^2 estimates are interpreted as the proportion of variance in depressive symptoms scores attributed to genetic factors underlying the perceived stress facet. They represent the chances of inheriting depressive symptoms related to stress perception for each marital status. While the h^2 , c^2 , and e^2 estimates unique to depressive symptoms are also presented in the results, they are not discussed as they do not pertain to our study hypotheses.

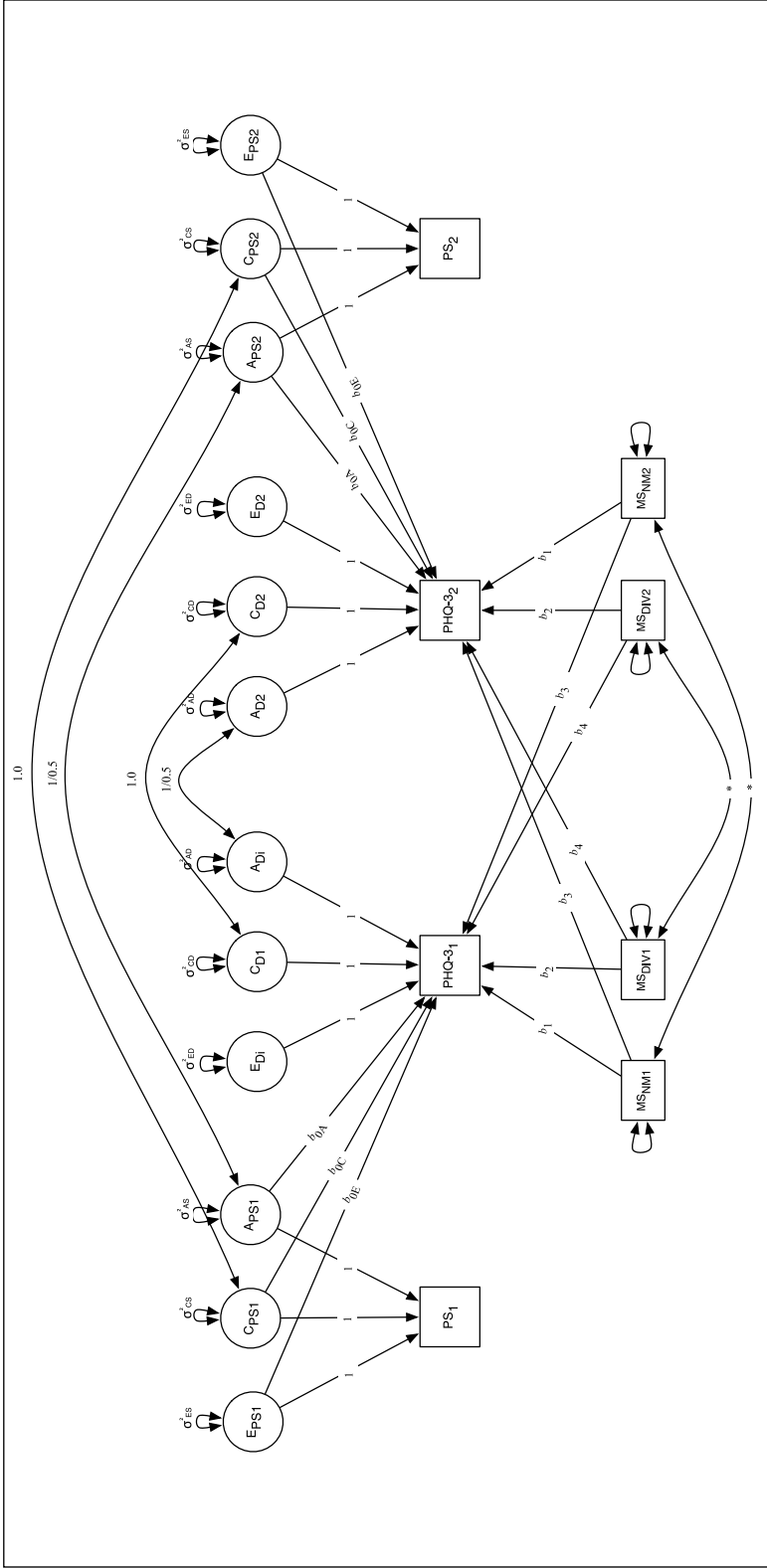


Figure 1. Bivariate Cholesky ACE Model.

Note: PS = perceived stress facet; PHQ-3 = depressive symptoms; MS = marital status; NM = never married; DIV = divorced; A = additive genetic effect; C = shared environmental effect; E = nonshared environmental effect; b_{0A} = genetic regression coefficient; b_{0C} = shared environmental regression coefficient; b_{0E} = nonshared environmental regression coefficient; b_1 and b_2 = unstandardized regression effects of twins' own marital status; b_3 and b_4 = unstandardized regression effects of co-twins' marital status; σ^2_{AS} = genetic variance common to PS and PHQ-3; σ^2_{CS} = shared environmental variance common to PS and PHQ-3; σ^2_{ES} = nonshared environmental variance common to PS and PHQ-3; σ^2_{AD} = unique genetic variance of PHQ-3; σ^2_{CD} = unique genetic variance of PHQ-3; σ^2_{ED} = unique genetic variance of PHQ-3. Asterisk (*) indicates that the covariance was estimated separately in the MZ and DZ groups. Subscripts 1 and 2 refer to twin 1 and twin 2. The interaction model modifies the b_{0A} , b_{0C} , b_{0E} pathways for when twins' never-married and divorced marital statuses equal 1 (for example, b_{0A} becomes $b_{0A} + b_{1A}MS_{NM1} + b_{2A}MS_{DIV1}$).

Table 1. Descriptive Results for Each Marital Group across All Male and Female Twins, University of Washington Twin Registry, 2012 (N = 3,224 Individuals).

Variable	Male Twins						Female Twins					
	Married (n = 547)		Never Married (n = 470)		Divorced (n = 65)		Married (n = 1,181)		Never Married (n = 775)		Divorced (n = 186)	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Perceived helplessness	1.14	.66	1.23	.80	1.19	.74	1.37	.75	1.53*	.78	1.59*	.81
Perceived self-efficacy	1.04	.69	1.31*	.78	1.04	.62	1.21	.72	1.40*	.70	1.39*	.81
Depressive symptoms (PHQ-3)	.39	.49	.52*	.60	.62	.66	.53	.58	.61*	.62	.84*	.79
Age	48.11	15.56	25.71*	8.12	52.70*	13.62	42.96	13.86	25.89*	8.36	50.84*	12.98
Education level	6.49	1.43	5.67*	1.37	6.03*	1.38	6.26	1.42	5.88*	1.32	5.89*	1.47
Number of children	1.80	1.45	.04*	.36	1.38*	1.32	1.65	1.29	.11*	.54	1.76	1.40

Note: Asterisk (*) indicates values significantly different ($p < .05$) from corresponding mean estimates in the married group based on an independent-samples *t* test. PHQ-3 = three-item Patient Health Questionnaire; M = mean; SD = standard deviation.

Model fitting was performed using the Mplus 7.3 program (Muthén and Muthén 1998–2010). In order to handle missing data and the non-normal distribution of the items, we used full-information maximum likelihood estimation with robust standard errors (MLR). As the depressive symptom and perceived stress variables were positively skewed, MLR estimation is robust to modest violations of missing-at-random and multivariate non-normality in behavioral research (Muthén and Kaplan 1985; Raykov 2005). With an MLR estimator, the Satorra-Bentler scaled chi-square difference test must be used to calculate a chi-square distributed test statistic to compare nested models (Satorra and Bentler 2001). Absolute model fit was evaluated using the root mean square error of approximation (RMSEA; Browne and Cudeck 1992). RMSEA estimates lower than .05 indicate “good” model fit to the data; estimates lower than .08 (but greater than .05) indicate “adequate” model fit to the data. The Akaike information criterion and Bayesian information criterion were used to evaluate relative model fit (Burnham and Anderson 2004). Both indexes were computed to balance model parsimony and model complexity (Kline 2005), with lower values indicating better model fit. Although for space considerations we do not provide the full covariance matrices for the male and female twin analyses, they are available upon request from the corresponding author.

RESULTS

Descriptive Analysis

Table 1 presents the means and standard deviations of perceived helplessness, perceived self-efficacy, and depressive symptoms for the married, never-married, and divorced male and female twins. Married male twins reported significantly lower mean perceived self-efficacy and depressive symptom scores than never-married male twins. There were no significant differences on any of the variables between married and divorced male twins. On average, married male twins were significantly older than never-married male twins but significantly younger than divorced male twins. Married male twins reported significantly higher educational attainment and more children than never-married and divorced male twins.

Married female twins reported significantly lower mean perceived helplessness, perceived self-efficacy, and depressive symptom scores than never-married and divorced female twins. Married female twins were significantly older, on average, than their never-married counterparts but significantly younger than divorced female twins. Married female twins reported significantly higher educational attainment and significantly more children than never-married female twins, whereas they reported only significantly higher educational attainment compared with divorced female twins.

Table 2. Monozygotic (MZ) and Dizygotic (DZ) Twin Correlations, University of Washington Twin Registry, 2012.

Variable	Male Twins				Female Twins			
	MZ	DZ	z	p	MZ	DZ	z	p
Perceived helplessness	.32	.18	1.47	.140	.35	.13	3.44	.001
Perceived self-efficacy	.32	.09	2.48	.010	.38	.13	4.03	.001
Depressive symptoms (PHQ-3)	.30	.21	.95	.340	.30	.23	1.07	.280

Note: Sample size of male twins: $n_{MZ} = 413$ pairs and $n_{DZ} = 138$ pairs; sample size of female twins: $n_{MZ} = 768$ pairs and $n_{DZ} = 303$ pairs. PHQ-3 = three-item Patient Health Questionnaire; z = z score.

Next, we estimated MZ and DZ twin correlations to investigate the first hypothesis that both genetic and environmental effects account for variation in the perceived stress facets and depressive symptom scores. In the male twins, MZ twin correlation was significantly greater than the DZ twin correlation for perceived self-efficacy but not perceived helplessness or depressive symptoms (Table 2). Genetic effects, but not shared environmental effects, partially accounted for a significant portion of the variation in perceived self-efficacy, as indicated by an MZ correlation more than twice as great as the DZ correlation. The small to moderate-sized MZ twin correlations suggest underlying nonshared environmental effects for all three variables.

In the female twins, the MZ twin correlations were significantly greater than the DZ twin correlations for perceived helplessness and perceived self-efficacy but not depressive symptoms (Table 2). Again, only genetic effects partially accounted for variation in both perceived stress facets, as indicated by MZ twin correlations more than twice as great as DZ twin correlations. Nonshared environmental effects accounted for the greatest proportion of variance in all three variables.

In sum, genetic and nonshared environmental effects appeared to influence perceived helplessness (for women only) and perceived self-efficacy (for both men and women) scores, whereas only nonshared environmental effects influenced depressive symptom scores. Significant genetic and nonshared environmental influences underlying both perceived stress facets are the basis for testing whether they also account for variation in depressive symptoms.

Next, the phenotypic correlations between the perceived stress facets and depressive symptoms are presented for each gender and then subdivided by marital status to begin addressing our second hypotheses—that is, the magnitudes of the correlations between the perceived stress facets and depressive

symptoms would be lower for married twins compared to unmarried twins. Across all MZ and DZ male twins, the correlation between perceived helplessness and depressive symptoms was .56, and the correlation between perceived self-efficacy and depressive symptoms was .40. Across all female twins, the correlation between perceived helplessness and depressive symptoms was .61, whereas the correlation between perceived self-efficacy and depressive symptoms was .49.

There were no appreciable differences in the observed correlations across marital status groups. For all male twins, the correlations between perceived helplessness and depressive symptoms for the married, never-married, and divorced groups were .59, .58, and .59, respectively. The correlations between perceived self-efficacy and depressive symptoms for the married, never-married, and divorced groups were .46, .42, and .45, respectively. For all female twins, the correlations between perceived helplessness and depressive symptoms for the married, never-married, and divorced groups were .61, .60, and .60, respectively. The correlations between perceived self-efficacy and depressive symptoms for the married, never-married, and divorced groups were .48, .48, and .49, respectively.

Co-twin control analyses are presented next to further address the second hypothesis. The results in Table 3 suggest larger contrast between married and unmarried groups in the correlations between the perceived stress facets and depressive symptoms than found in the phenotypic correlations reported in the previous paragraph. In the male twins, although the correlation between perceived helplessness and depressive symptoms was stronger for married twins than for their never-married co-twins, the same correlation was cut in half for married twins when compared to their divorced co-twins. This latter result is consistent with the second hypothesis. In the female twins, the results were inconsistent with our

Table 3. Co-twin Control Analysis of the Correlations between the Two Perceived Stress Facets and PHQ-3 Scores, University of Washington Twin Registry, 2012 ($N = 432$ Pairs).

Group	<i>n</i>	Correlation between Perceived Helplessness and Depressive Symptoms				Correlation between Perceived Self-efficacy and Depressive Symptoms			
		<i>r</i> _{Married Twin}	<i>r</i> _{Unmarried Twin}	<i>z</i>	<i>p</i>	<i>r</i> _{Married Twin}	<i>r</i> _{Unmarried Twin}	<i>z</i>	<i>p</i>
<i>Male Twins</i>									
Married–never married	76	.63	.48	1.33	.180	.49	.51	.16	.870
Married–divorced	47	.24	.48	1.32	.190	.14	.44	1.51	.130
<i>Female Twins</i>									
Married–never married	159	.60	.50	1.32	.190	.43	.48	.59	.550
Married–divorced	150	.60	.64	.58	.560	.54	.62	.93	.350

Note: PHQ-3 = three-item Patient Health Questionnaire; $z = z$ score.

hypothesized predictions about the stress-reducing benefits of marriage. The correlation between perceived helplessness and depressive symptoms was stronger for married twins compared to their never-married co-twins and nearly equal in comparison between married twins and their divorced co-twins.

Differences in the correlations between perceived self-efficacy and depressive symptoms for twins discordant for marital status, however, more clearly support the second hypothesis. Differences in correlations were small between married twins and their never-married co-twins for both male and female twins (Table 3). The correlation was reduced by more than two thirds for married male twins compared to their divorced male co-twins and slightly reduced for married female twins compared to their divorced co-twins.

Multivariate Analysis

Finally, multivariate twin analyses were first performed to formally test our first hypothesis and estimate the proportions of variation in depressive symptoms attributed to genetic and environmental factors underlying the perceived stress facets. They were then used to formally test our second hypothesis, that marriage reduces both the genetic and environmental effects of perceived stress on depressive symptoms, as well as our third hypothesis, that men would benefit more from marriage than women. The baseline bivariate Cholesky ACE (“main effects”) models suggest that both genetic and nonshared environmental factors significantly accounted for the observed association between the perceived stress facets and depressive symptoms. All main effects models fit the data adequately, with RMSEA estimates ranging from .045 to .056.

Table 4 presents the heritability and environmental estimates of stress-induced depressive symptoms. The estimates in Table 4 are adjusted for the main effects of marital status on depressive symptoms. Genetic factors common to both facets significantly accounted for variation in depressive symptoms (range: 15% to 26% of the total variance in PHQ-3; see column labeled h^2). In other words, genetic factors generated, in part, the correlation between perceived stress and depressive symptoms. The e^2 estimates suggest that environmental effects of underlying both perceived stress facets accounted for significant proportions of variation in depressive symptoms (range: 5% to 18% of the total variance in PHQ-3; see column labeled e^2).

To sum, these results support our first hypothesis that genetic and nonshared environmental factors partially account for the observed correlation between perceived stress and depressive symptoms. There was no evidence that shared environmental effects mediated the correlations between the perceived stress facets and depressive symptoms; these effects were deleted from further analyses.

Next, we fit moderated bivariate Cholesky ACE (“interaction effects”) models to the male and female twin data sets and compared the fit of these models to the main effects models (Table 5). In the male twins, marital status was not observed to be a significant moderator, although the interaction effects model was nearly significant for perceived self-efficacy compared to the main effects model ($p < .06$). In the female twins, marital status significantly moderated the genetic and environmental effects of both perceived stress facets on depressive symptoms.

We computed heritability and environmental estimates for both male and female twins based on the parameter estimates obtained in Model 2 of each

Table 4. Proportion of Total Variance in PHQ-3 Attributed to Common and Unique Genetic and Environmental Effects across All Male Twins and All Female Twins, University of Washington Twin Registry, 2012 (N = 1,612 pairs).

Variable	h^2	SE	e^2	SE	h^2_{dep}	SE	c^2_{dep}	SE	e^2_{dep}	SE
<i>Perceived Helplessness</i>										
Male twins	.26	.06	.11	.03	.01	.17	.05	.15	.58	.04
Female twins	.20	.04	.18	.03	–	–	.11	.02	.51	.03
<i>Perceived Self-efficacy</i>										
Male twins	.16	.06	.05	.02	.06	.22	.08	.20	.65	.05
Female twins	.15	.03	.10	.02	–	–	.15	.03	.59	.03

Note: n_{male} = 541 pairs and n_{female} = 1,071 pairs. PHQ-3 = three-item Patient Health Questionnaire; h^2 = heritability of stress-induced depressive symptoms; SE = standard error; e^2 = environmental liability of stress-induced depressive symptoms; h^2_{dep} = heritability unique to PHQ-3; c^2_{dep} = variance accounted for by shared environmental effects unique to PHQ-3; e^2_{dep} = variance accounted for by nonshared environmental effects unique to PHQ-3. h^2_{dep} not given due to negative variance estimates.

Table 5. Model Fit Results for the Bivariate Cholesky ACE Models, University of Washington Twin Registry, 2012 (N = 1,612 pairs).

Model	Description	–2LL	df	$\Delta S-B\chi^2$	Δdf	p	RMSEA	AIC	BIC
<i>Male Twins: Perceived Helplessness</i>									
1	Main effects model	–4466.29	73	—	—	—	.045	9002.58	9152.85
2	Interaction effects model	–4461.92	69	5.20	4	.268	—	9001.85	9169.29
<i>Male Twins: Perceived Self-efficacy</i>									
1	Main effects model	–4579.14	73	—	—	—	.052	9228.28	9378.55
2	Interaction effects model	–4571.13	69	9.30	4	.054	—	9220.26	9387.70
<i>Female Twins: Perceived Helplessness</i>									
1	Main effects model	–9461.32	73	—	—	—	.054	18992.64	19166.81
2	Interaction effects model	–9441.05	69	29.16	4	.000	—	18960.10	19154.18
<i>Female Twins: Perceived Self-efficacy</i>									
1	Main effects model	–9508.77	73	—	—	—	.056	19087.54	19261.71
2	Interaction effects model	–9487.94	69	27.87	4	.000	—	19053.88	19247.95

Note: n_{male} = 541 pairs and n_{female} = 1,071 pairs. –2LL = –2 log-likelihood; $\Delta S-B\chi^2$ = Satorra-Bentler likelihood ratio difference for nested model comparison; RMSEA = root mean square error of approximation; AIC = Akaike information criterion; BIC = Bayesian information criterion.

analysis. These estimates and their standard errors are given in Table 6, with the significant effects found in the female twins presented here. Genetic factors underlying perceived helplessness and perceived self-efficacy did not account for as much variance in depressive symptoms in married women compared to never-married and divorced women. Thus, heritability of stress-induced depressive symptoms was less among those with access to marital support. Consistent with predictions in our second hypothesis, h^2 was lowest in the married group and greatest in the divorced group. Inconsistent with our third hypothesis, however, marriage did not appear to benefit men more than women.

Marriage did not attenuate the environmental effects of either perceived facet on depressive symptoms in women. In contrast to predictions made in the second hypothesis, the environmental effect of perceived helplessness on depressive symptoms is .17 units lower (95% CI: [–.28, –.06]) in never-married female twins compared to married female twins and .26 units lower (95% CI: [–.39, –.13]) for the effect of perceived self-efficacy. The environmental effects found here also mean that the environmental estimates for stress-induced depressive symptoms increased for married women (Table 6). No significant environmental effects were found between married and divorced female twins.

Table 6. Proportion of Total Variance in PHQ-3 Attributed to Common and Unique Genetic and Environmental Effects in Married, Never-married, and Divorced Groups, University of Washington Twin Registry, 2012 (N = 1,612 pairs).

	h^2	SE	e^2	SE	h^2_{dep}	SE	c^2_{dep}	SE	e^2_{dep}	SE
<i>Male Twins: Perceived Helplessness</i>										
Married	.22	.06	.13	.04	—	—	.09	.14	.56	.11
Never married	.36	.11	.06	.03	—	—	.08	.13	.50	.10
Divorced	.51	.10	.03	.05	—	—	.06	.11	.40	.09
<i>Female Twins: Perceived Helplessness</i>										
Married	.14	.03	.23	.04	—	—	.14	.07	.49	.04
Never married	.31	.07	.09	.03	—	—	.14	.06	.47	.06
Divorced	.52	.07	.09	.04	—	—	.09	.05	.31	.04
<i>Male Twins: Perceived Self-efficacy</i>										
Married	.08	.05	.07	.04	—	—	.14	.20	.71	.17
Never married	.32	.12	.02	.11	—	—	.11	.16	.55	.12
Divorced	.45	.15	.03	.05	—	—	.09	.13	.43	.10
<i>Female Twins: Perceived Self-efficacy</i>										
Married	.08	.03	.16	.03	—	—	.20	.08	.57	.05
Never married	.29	.06	.02	.01	—	—	.18	.07	.51	.06
Divorced	.42	.08	.05	.04	—	—	.14	.06	.39	.05

Note: $n_{male} = 541$ pairs and $n_{female} = 1,071$ pairs. PHQ-3 = three-item Patient Health Questionnaire; h^2 = heritability of stress-induced depressive symptoms; SE = standard error; e^2 = environmental liability of stress-induced depressive symptoms; h^2_{dep} = heritability unique to PHQ-3; c^2_{dep} = variance accounted for by shared environmental effects unique to PHQ-3; e^2_{dep} = variance accounted for by nonshared environmental effects unique to PHQ-3. h^2_{dep} not given due to negative variance estimates.

Overall, while married male twins neither profited nor lost in terms of fewer stress-induced depressive symptoms, we observed costs of marriage for female twins that never-married female twins did not incur.

DISCUSSION

The purpose of this study was to use a genetically informative design to evaluate the relation between perceived stress and depressive symptoms and to test whether marital status modified the association, as recommended by Kessler (1997). We made three hypotheses. First, both genetic and nonshared environmental factors underlying perceived stress would account for variation in depressive symptoms. Second, the genetic and environmental effects underlying perceived stress would account for less variance in depressive symptoms in married men and women, compared to their unmarried counterparts. Third, marriage would be a stronger buffering mechanism of the genetic and environmental effects of perceived stress on depressive symptoms in men than in women. We found support for our first hypothesis and replicated prior twin sibling studies

on stress and depressive symptomatology in the UWTR twin sample (Bogdan and Pizzagalli 2009; Schnittker 2010). We found partial support for our second and third hypotheses.

The main contribution of the present study is that marriage significantly reduced the genetic effects of both perceived stress facets on depressive symptoms in female twins in support of our second hypothesis. The findings suggest that the heritability of stress-induced depressive symptoms was lowest among married women and highest among divorced women. Marriage, thus, may lower the heritability of depressive symptoms related to perceived stress. Divorced women, on the other hand, may be at increased risk for depressive symptoms that are attributable to genes shared between depressive symptoms and perceived stress. The significant difference between the married and divorced may be associated with the social, health, and financial upheaval woman may experience following marital dissolution (Emery 2011).

Contrary to our second hypothesis, marriage did not reduce environmental effects of perceived stress on depressive symptoms in female twins. Indeed, the depressogenic effects of perceived stress were

strongest for married women, consistent with Bernard's (1982) argument that marrying leads to worse mental health outcomes for women than not marrying at all. One possibility is that even if marriage were to lower women's natural, biologic vulnerabilities to stress perception, they must negotiate stressful marital and family situations, which may, in turn, raise depressive symptoms that never-married women do not have to negotiate.

At least for women, there may be a trade-off between partner access and quality of partner support. Simply having an intimate relationship (marital or nonmarital) may lower heritability of stress-induced depressive symptoms but may introduce daily stressors in the form of partner expectations and familial obligations. Lapses in good-quality partner support may trigger increases in stress perception, which may lead to upticks in depressive symptoms. Conversely, never-married women may have higher genetic vulnerabilities for stress-induced depressive symptoms, but available support from extended social networks may be protective. Never-married women do not have to cope with fluctuations in the quality of partner support, which do predict mood symptoms (Bolger, Zuckerman, and Kessler 2000). The descriptive results presented in Table 1 support this interpretation, as married women reported having significantly more children than never-married women, a life condition that may engender increased daily stress. For married women, consistent high-quality partner support (e.g., emotional sustenance, coping assistance, and invisible support) may be critical for maintaining good mental health.

Divorced women appeared to have the greatest heritability estimates of stress-induced depressive symptoms, up to 5.5 times that of married women. This finding is consistent with previous longitudinal findings that marital dissolution has greater negative effects on psychological well-being than never marrying (Hope, Rodgers, and Power 1999). Divorced people's increased genetic vulnerability to stress-induced depressive symptoms also is consistent with research demonstrating that divorce often engenders stressful transitions around financial loss, changes in family structure and social networks, and practical problems around establishing new living arrangements (Berman and Turk 1981).

Implications for Gender Differences in Stress Protection

We also observed three notable gender differences in the current study. First, marriage significantly lowered genetic effects of perceived stress on depressive symptoms in women but not men. Contrary to past

research (Waite and Gallagher 2000), these findings suggest that marriage may benefit women more than men. Whereas marriage may independently lower mean depressive symptom scores for both men and women (Aneshensel and Stone 1982; Thoits 1984), only married women had significantly lower heritability estimates of stress-induced depressive symptoms. Weaker, but statistically null, effects were observed in male twins. The significant difference between perceived self-efficacy and depressive symptoms scores between married and never-married male twins in the descriptive results suggests that the multivariate analyses were underpowered to reject the null hypothesis.

The significant moderating role of marriage observed in the female twins could mean that the benefits married women receive in terms of decreased heritability of stress-induced depressive symptoms may be offset by increased environmental effects of perceived stress on mean depressive symptom scores. Quality of extended social networks may be one reason for the trade-off. Women, in contrast to men, have been found to surrender extended social networks as the demands of married and family life unfold over time (Antonucci and Akiyama 1987). Married women have smaller social networks outside of the family because of marital- and family-related obligations, which may mean they have fewer social outlets to process stressful life experiences. These stressful life experiences, in turn, may cause increases in their stress perception of daily situations that subsequently cause increases in their depressive symptomatology. Thus, diminished access to social networks beyond marriage and family that previously were venues for processing daily stress may actually cause increases in depressive symptoms.

Finally, gender differences were also observed with respect to both facets of perceived stress. The division of the PSS into two distinct facets is useful for clarifying the ways in which people might appraise life as stressful (Cohen and Williamson 1988). Consistent with prior research on the psychometric properties of the PSS, the correlations between depressive symptomatology and the facets of perceived helplessness (an index of lack of control) and perceived self-efficacy (an index of feeling overloaded by stress) were moderate across both the male and female twin samples. The results clearly indicate significantly lower heritability estimates of depressive symptoms related to both perceived helplessness and perceived self-efficacy for women but not men. Unlike the men in the HWB, women with high genetic propensities for experiencing depressive symptom spillover from both forms of perceived stress may benefit the most from marital support.

Limitations

While the main strength of the current twin study is a more nuanced representation of the genetic and environmental pathways through which marital—and more generally, relationship—support might protect against stress-induced depressive symptomatology, one limitation of the current study is that the HWB twin data are cross-sectional. While we tested whether perceived stress influences depressive symptoms, the reverse may also be true (e.g., see Hammen's [1991] stress generation model of depression). More endogenous forms of depression, like dysthymia, may influence stress perception. Similarly, depressive people may have lower thresholds for stress reactivity, which implies that increases in depressive symptoms ought to predict increases in stress perception, particularly around daily life hassles (Harkness 2008).

In addition to the reasons provided in the introduction, we did not conduct tests of reverse causality for three reasons. First, prior research typically has focused on the effects of stress factors on depression to understand stress reactivity (Aneshensel and Stone 1982; Hammen 2005), which was also the aim in this report. Second, perceived stress measures are good predictors of affective and clinical outcomes (Hewitt, Flett, and Mosher 1992; Pbert, Doerfler, and DeCosimo 1992). In keeping with past research, the current findings make a stronger contribution to the existing literature by elucidating the genetic and environmental pathways through which perceived stress influences depressive symptoms. Third, the buffering role of marriage fits within the social support theory framework (Lakey and Cronin 2008), which posits that people's depressive reactions to stress depend on people's support resources.

Several other limitations in the current study are worthy of note and should be kept in mind when interpreting these findings. First, we considered only two unmarried groups—the never married and the divorced—whereas other groups, like the marital separated and the widowed, also might be expected to differ from married populations. We possessed too few twins in these other unmarried groups to include them as separate groups in the current study. When separated and widowed twins were combined with divorced twins in post hoc analyses, the results were unchanged. Any advantages of marriage, thus, are limited to the comparison with divorced and never-married people.

Second, the self-report nature of the perceived stress and depressive symptom items may have introduced interpretation bias into the variables, as the meaning of the items may have differed across

marital groups (Hammen 2005; Kessler 1997). Married people, for example, may have a different threshold for appraising stress and depressive symptoms than unmarried people.

Third, although the generalizability of twin populations to nontwin populations always remains a concern, as twin samples increase in size, they tend to be more generalizable to the general population (Robinson et al. 1992). The generalizability of the HWB sample is also bolstered by the similarity of the correlations among the perceived facets and depressive symptoms measure to other nontwin samples (Bogdan and Pizzagalli 2009; Hewitt et al. 1992; Pbert et al. 1992).

Finally, educational status was not included in our multivariate analyses and may be confounded with the effects of marital status, as many never-married people may be too young to have finished their formal education. Post hoc analyses suggest that inclusion of the main effects of educational status on perceived stress and depressive symptoms does not alter the results.

Future Research

The current study raises an important issue for further research on how social relationships should be operationalized to better understand how social support reduces negative consequences of perceived stress. There is increasing evidence that simply having support does not reliably reduce effects of stress on depressive symptomatology (Thoits 2011). Social relationships, however, matter for protecting against mental health threats of acute and chronic stress. Measures of the quality of support transactions ought to be integrated into future twin studies on social support, as differences in the quality of transactions—like emotional, practical, and invisible support—contribute to people's stress reactions.

FUNDING

This work was supported by the National Institute On Aging 1F31AG044047-01A1, T32AG020500, and T32 AG000037-38 and the National Institute of Child Health & Human Development 1R01HD056354-01.

REFERENCES

- Afari, Nilofar, Carolyn Noonan, Jack Goldberg, Karen Edwards, Kiran Gadepalli, Bethany Osterman, Cynthia Evanoff, and Dedra Buchwald. 2006. "University of Washington Twin Registry: Construction and Characteristics of a Community-based Twin Registry." *Twin Research and Human Genetics* 9(6):1023–29.

- Aneshensel, Carol S., and Jeffrey D. Stone. 1982. "Stress and Depression: A Test of the Buffering Model of Social Support." *Archives of General Psychiatry* 39(12):1392–96.
- Antonucci, Toni C., and Hiroko Akiyama. 1987. "An Examination of Sex Differences in Social Support among Older Men and Women." *Sex Roles* 17(11–12):737–49.
- Beam, Christopher R., Erin E. Horn, Stacy Karagis Hunt, Robert E. Emery, Eric Turkheimer, and Nick Martin. 2011. "Revisiting the Effect of Marital Support on Depressive Symptoms in Mothers and Fathers: A Genetically Informed Study." *Journal of Family Psychology* 25(3):336–44.
- Bergen, Sarah E., Charles O. Gardner, and Kenneth S. Kendler. 2007. "Age-related Changes in the Heritability of Behavioral Phenotypes over Adolescence and Young Adulthood: A Meta-analysis." *Twin Research and Human Genetics* 10(3):423–33.
- Berman, William H., and Dennis C. Turk. 1981. "Adaptation to Divorce: Problems and Coping Strategies." *Journal of Marriage and Family* 43(1):179–89.
- Bernard, Jessie. 1982. *The Future of Marriage*. 2nd ed. New Haven, CT: Yale University Press.
- Bierman, Alex, Elena M. Fazio, and Melissa A. Milkie. 2006. "A Multifaceted Approach to the Mental Health Advantage of the Married: Assessing How Explanations Vary by Outcome Measure and Unmarried Group." *Journal of Family Issues* 27(4):554–82.
- Blekesaune, Morten. 2008. "Partnership Transitions and Mental Distress: Investigating Temporal Order." *Journal of Marriage and Family* 70(4):879–90.
- Bogdan, Ryan, and Diego A. Pizzagalli. 2009. "The Heritability of Hedonic Capacity and Perceived Stress: A Twin Study Evaluation of Candidate Depressive Phenotypes." *Psychological Medicine* 39(2):211–18.
- Bolger, Niall, Adam Zuckerman, and Ronald C. Kessler. 2000. "Invisible Support and Adjustment to Stress." *Journal of Personality and Social Psychology* 79(6):953–61.
- Browne, Michael W., and Robert Cudeck. 1992. "Alternative Ways of Assessing Model Fit." *Sociological Methods & Research* 21(2):230–58.
- Burnham, Kenneth P., and David R. Anderson. 2004. "Multimodel Inference: Understanding AIC and BIC in Model Selection." *Sociological Methods & Research* 33(2):261–304.
- Cacioppo, Stephanie, Angela J. Grippo, Sarah London, Luc Goossens, and John T. Cacioppo. 2015. "Loneliness: Clinical Import and Interventions." *Perspectives on Psychological Science* 10(2):238–49.
- Carlson, Daniel L. 2012. "Deviations from Desired Age at Marriage: Mental Health Differences across Marital Status." *Journal of Marriage and Family* 74(4):743–58.
- Carr, Deborah, and Kristen W. Springer. 2010. "Advances in Families and Health Research in the 21st Century." *Journal of Marriage and Family* 72(3):743–61.
- Caspi, Avshalom, Karen Sugden, Terrie E. Moffitt, Alan Taylor, Ian W. Craig, Hona Lee Harrington, Joseph McClay, Jonathan Mill, Judy Martin, Antony Braithwaite, and Richie Poulton. 2003. "Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene." *Science* 301(5631):386–89.
- Cohen, Jacob. 1968. "Multiple Regression as a General Data-analytic System." *Psychological Bulletin* 70(6):426–43.
- Cohen, Sheldon, Benjamin H. Gottlieb, and Lynn G. Underwood. 2000. "Social Relationships and Health." Pp. 3–25 in *Social Support Measurement and Interventions: A Guide for Health and Social Scientists*, edited by S. Cohen, B. H. Gottlieb, and L. G. Underwood. New York: Oxford University Press.
- Cohen, Sheldon, Tom Kamarck, and Robin Mermelstein. 1983. "A Global Measure of Perceived Stress." *Journal of Health and Social Behavior* 24(4):385–96.
- Cohen, Sheldon, Drury R. Sherrod, and Margaret S. Clark. 1986. "Social Skills and the Stress-protective Role of Social Support." *Journal of Personality and Social Psychology* 50(5):963–73.
- Cohen, Sheldon, and Gail M. Williamson. 1988. "Perceived Stress in a Probability Sample of the United States." Pp. 31–67 in *The Social Psychology of Health: Claremont Symposium on Applied Social Psychology*, edited by S. Spacapan and S. Oskamp. Newbury Park, CA: Sage.
- Cohen, Sheldon, and Thomas A. Wills. 1985. "Stress, Social Support, and the Buffering Hypothesis." *Psychological Bulletin* 98(2):310–57.
- Conway, Christopher C., Costance Hammen, Emmanuel P. Espejo, Naomi R. Wray, Jake M. Najman, and Patricia A. Brennan. 2011. "Appraisals of Stressful Life Events as a Genetically-linked Mechanism in the Stress–Depression Relationship." *Cognitive Therapy and Research* 36(4):338–47.
- D'Onofrio, Brian M., Eric Turkheimer, Robert E. Emery, Wendy S. Slutske, Andrew C. Heath, Pamela A. Madden, and Nicholas G. Martin. 2005. "A Genetically Informed Study of Marital Instability and Its Association with Offspring Psychopathology." *Journal of Abnormal Psychology* 114(4):570–86.
- Dohrenwend, Barbara S., Bruce P. Dohrenwend, Margaret Dodson, and Patrick E. Shrout. 1984. "Symptoms, Hassles, Social Supports, and Life Events: Problem of Confounded Measures." *Journal of Abnormal Psychology* 93(2):222–30.
- Emery, Robert E. 2011. *Renegotiating Family Relationships: Divorce, Child Custody, and Mediation*. 2nd ed. New York: Guilford Press.
- Gove, Walter R., Michael Hughes, and Carolyn B. Style. 1983. "Does Marriage Have Positive Effects on the Psychological Well-being of the Individual?" *Journal of Health and Social Behavior* 24(2):122–31.

- Hammen, Constance L. 1991. "Generation of Stress in the Course of Unipolar Depression." *Journal of Abnormal Psychology* 100(4):555–61.
- Hammen, Constance L. 2005. "Stress and Depression." *Annual Review of Clinical Psychology* 1:293–319.
- Harkness, Kate L. 2008. "Life Events and Hassles." Pp. 317–41 in *Risk Factors in Depression*, edited by K. S. Dobson and D. J. A. Dozois. San Diego, CA: Academic Press.
- Heath, Andrew C., Lindon J. Eaves, and Nicholas G. Martin. 1998. "Interaction of Marital Status and Genetic Risk for Symptoms of Depression." *Twin Research* 1(3):119–22.
- Heath, Andrew C., Ronald C. Kessler, Michael C. Neale, John K. Hewitt, Lindon J. Eaves, and Kenneth S. Kendler. 1993. "Testing Hypotheses about Direction of Causation Using Cross-sectional Family Data." *Behavior Genetics* 23(1):29–50.
- Hewitt, Paul L., Gordon L. Flett, and Shawn W. Mosher. 1992. "The Perceived Stress Scale: Factor Structure and Relation to Depression Symptoms in a Psychiatric Sample." *Journal of Psychopathology and Behavioral Assessment* 14(3):247–57.
- Hope, Steven, Brian Rodgers, and Chris Power. 1999. "Marital Status Transitions and Psychological Distress: Longitudinal Evidence from a National Population Sample." *Psychological Medicine* 29(2):381–89.
- Horn, Erin E., Yishan Xu, Christopher R. Beam, Eric Turkheimer, and Robert E. Emery. 2013. "Accounting for the Physical and Mental Health Benefits of Entry into Marriage: A Genetically Informed Study of Selection and Causation." *Journal of Family Psychology* 27(1):30–41.
- Johnson, David R., and Jian Wu. 2002. "An Empirical Test of Crisis, Social Selection, and Role Explanations of the Relationship between Marital Disruption and Psychological Distress: A Pooled Time-series Analysis of Four-wave Panel Data." *Journal of Marriage and Family* 64(1):211–24.
- Kendler, Kenneth S., L. M. Karkowski, and Carol A. Prescott. 1999. "Causal Relationship between Stressful Life Events and the Onset of Major Depression." *American Journal of Psychiatry* 156(6):837–41.
- Kendler, Kenneth S., Jonathan W. Kuhn, Jen Vittum, Carol A. Prescott, and Brien Riley. 2005. "The Interaction of Stressful Life Events and a Serotonin Transporter Polymorphism in the Prediction of Episodes of Major Depression: A Replication." *Archives of General Psychiatry* 62(5):529–35.
- Kendler, Kenneth S., and Carol A. Prescott. 2006. *Genes, Environment and Psychopathology: Understanding the Causes of Psychiatric and Substance Use Disorders*. Boston: Guilford Press.
- Kessler, Ronald C. 1997. "The Effects of Stressful Life Events on Depression." *Annual Review of Psychology* 48:191–214.
- Kessler, Ronald C., and Marilyn Essex. 1982. "Marital Status and Depression: The Importance of Coping Resources." *Social Forces* 61(2):484–507.
- Kessler, Ronald C., Kenneth S. Kendler, Andrew C. Heath, Michael C. Neale, and Lindon J. Eaves. 1992. "Social Support, Depressed Mood, and Adjustment to Stress: A Genetic Epidemiologic Investigation." *Journal of Personality and Social Psychology* 62(2):257–72.
- Kim, Hyoun K., and Patrick C. McKenry. 2002. "The Relationship between Marriage and Psychological Well-Being: A Longitudinal Analysis." *Journal of Family Issues* 23(8):885–911.
- Kline, Rex B. 2005. *Principles and Practice of Structural Equation Modeling*. 2nd ed. New York: Guilford Press.
- Lakey, Brian, and Arika Cronin. 2008. "Low Social Support and Major Depression: Research, Theory and Methodological Issues." Pp. 385–408 in *Risk Factors in Depression*, edited by K. S. Dobson and D. J. A. Dozois. San Diego, CA: Academic Press.
- Löwe, Bernd, Kurt Kroenke, and Kerstin Gräfe. 2005. "Detecting and Monitoring Depression with a Two-item Questionnaire (PHQ-2)." *Journal of Psychosomatic Research* 58(2):163–71.
- Lucas, Richard E. 2005. "Time Does Not Heal All Wounds." *Psychological Science* 16(12):945–50.
- Maciejewski, Paul K., Holly G. Prigerson, and Carolyn M. Mazure. 2001. "Sex Differences in Event-Related Risk for Major Depression." *Psychological Medicine* 31(4):593–604.
- Muthén, Bengt O., and David Kaplan. 1992. "A Comparison of Some Methodologies for the Factor Analysis of Non-normal Likert Variables: A Note on the Size of the Model." *British Journal of Mathematical and Statistical Psychology* 45(1):19–30.
- Muthén, Linda K., and Bengt O. Muthén. 1998–2010. *Mplus User's Guide*. 6th ed. Los Angeles: Muthén & Muthén.
- Pbert, Lori, Leonard A. Doerfler, and Diana DeCosimo. 1992. "An Evaluation of the Perceived Stress Scale in Two Clinical Populations." *Journal of Psychopathology and Behavioral Assessment* 14(4): 363–75.
- Pearlin, Leonard I., and Joyce S. Johnson. 1977. "Marital Status, Life-strains and Depression." *American Sociological Review* 42(5):704–15.
- Raykov, Tenko. 2005. "Analysis of Longitudinal Studies with Missing Data Using Covariance Structure Modeling with Full-information Maximum Likelihood." *Structural Equation Modeling: A Multidisciplinary Journal* 12(3):493–505.
- Reifman, Alan, Monica Biernat, and Eric L. Lang. 1991. "Stress, Social Support, and Health in Married Professional Women with Small Children." *Psychology of Women Quarterly* 15(3):431–45.
- Reis, Rodrigo Siqueira, Adriano Akira Ferreira Hino, and Ciro Romélio Rodriguez Añez. 2010. "Perceived

- Stress Scale: Reliability and Validity Study in Brazil." *Journal of Health Psychology* 15(1):107–14.
- Roberti, Jonathan W., Lisa N. Harrington, and Eric A. Storch. 2006. "Further Psychometric Support for the 10-item Version of the Perceived Stress Scale." *Journal of College Counseling* 9(2):135–47.
- Robinson, JoAnn L., Jerome Kagan, J. Steven Reznick, and Robin Corley. 1992. "The Heritability of Inhibited and Uninhibited Behavior: A Twin Study." *Developmental Psychology* 28(6):1030–37.
- Satorra, Albert, and Peter M. Bentler. 2001. "A Scaled Difference Chi-square Test Statistic for Moment Structure Analysis." *Psychometrika* 66(4):507–19.
- Sbarra, David A., Robert E. Emery, Christopher R. Beam, and Bailey L. Ocker. 2014. "Marital Dissolution and Major Depression in Midlife: A Propensity Score Analysis." *Clinical Psychological Science* 2(3):249–257.
- Schnittker, Jason. 2010. "Gene–Environment Correlations in the Stress–Depression Relationship." *Journal of Health and Social Behavior* 51(3):229–43.
- Segal, Daniel L., Frederick L. Coolidge, Brian S. Cahill, and Alisa A O'Riley. 2008. "Behavior Modification of the Beck Depression Inventory-II (BDI-II) among Community-dwelling Older Adults." *Behavior Modification* 32(1):3–20.
- Shrout, Patrick E., Craig M. Herman, and Niall Bolger. 2006. "The Costs and Benefits of Practical and Emotional Support on Adjustment: A Daily Diary Study of Couples Experiencing Acute Stress." *Personal Relationships* 13(1):115–34.
- van der Sluis, Sophie, Danielle Posthuma, and Conor V. Dolan. 2012. "A Note on False Positives and Power in $G \times E$ Modelling of Twin Data." *Behavior Genetics* 42(1):170–86.
- Spitzer, Robert L., Kurt Kroenke, and Janet B. W. Williams. 1999. "Validation and Utility of a Self-report Version of PRIME-MD." *Journal of the American Medical Association* 282(18):1737–44.
- Tohits, Peggy A. 1984. "Explaining Distributions of Psychological Vulnerability: Lack of Social Support in the Face of Life Stress." *Social Forces* 63(2):453–81.
- Tohits, Peggy A. 1995. "Stress, Coping, and Social Support Processes: Where Are We? What Next?" *Journal of Health and Social Behavior* 35(Extra Issue):53–79.
- Tohits, Peggy A. 2011. "Mechanisms Linking Social Ties and Support to Physical and Mental Health." *Journal of Health and Social Behavior* 52(2):145–61.
- Turkheimer, Eric, and K. Paige Harden. 2014. "Behavior Genetic Research Methods: Testing Quasi-causal Hypotheses Using Multivariate Twin Data." Pp. 159–87 in *Handbook of Research Methods in Personality and Social Psychology*, edited by H. T. Reis and C. M. Judd. New York: Cambridge University Press.
- Waite, Linda J., and Maggie Gallagher. 2000. *The Case for Marriage: Why Married People Are Happier, Healthier, and Better off Financially*. New York: Doubleday.
- Walen, Heather R., and Margie E. Lachman. 2000. "Social Support and Strain from Partner, Family, and Friends: Costs and Benefits for Men and Women in Adulthood." *Journal of Social and Personal Relationships* 17(1):5–30.

AUTHOR BIOGRAPHIES

Christopher R. Beam is a postdoctoral research fellow in clinical geropsychology in the Davis School of Gerontology and a lecturer in the Department of Psychology at the University of Southern California. His research focuses on sex differences in the genetic and environmental mechanisms underlying depression, dementia, and mortality risk in older adults.

Diana Dinescu is a PhD candidate in clinical psychology at the University of Virginia (Charlottesville, VA). Her research investigates the effect of marriage and family relationships on health outcomes using genetically informed methodology. She is currently completing a clinical internship at the Kennedy Krieger Institute/Johns Hopkins School of Medicine.

Robert Emery, PhD, is a professor of psychology and director of the Center for Children, Families, and the Law at the University of Virginia. He has authored over 150 scientific publications and several books. His research focuses on family relationships and children's mental health, including parental conflict, divorce, child custody, family violence, and genetically informed studies of all these topics as well as associated legal and policy issues.

Eric Turkheimer is the Hugh Scott Hamilton Professor of Psychology at the University of Virginia. He is a clinical psychologist whose research focuses on the genetics of complex human behavior. Current research involves the effect of poverty on genetic variability of intelligence.