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Intimate Relationships and Depression: Searching for Causation in the Sea of Association

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Abstract

This article provides a critical review of existing research on intimate (marriage or marriage-like) relationship distress and risk for depression. Using the meta-framework of research triangulation, we seek to synthesize research evidence across several different methodologies and study designs and to draw the most reliable conclusion regarding a potential causal association between relationship distress and depression. Focusing on existing correlational (i.e., observational), genetically informed, and intervention (i.e., experimental) research on the association between relationship distress and depression, we conclude that the existing body of research evidence supports the claim that relationship distress is a causal risk factor for depression. A secondary aim of the article is to highlight a variety of effective methods that, when viewed from the perspective of triangulation, enhance the pursuit of causal inference, including propensity score matching, target trial emulation, directed acyclic graph approach, and Mendelian randomization.

Contents

1. INTRODUCTION

In *Darkness Visible*, William Styron (1992, p. 62) shares one of the most gripping and experiential accounts of what it is like to be depressed:

> The pain is unrelenting, and what makes the condition intolerable is the foreknowledge that no remedy will come—not in a day, an hour, a month, or a minute. If there is mild relief, one knows that it is only temporary; more pain will follow. It is hopelessness even more than pain that crushes the soul. So the decision-making of daily life involves not, as in normal affairs, shifting from one annoying situation to another less annoying—or from discomfort to relative comfort, or from boredom to activity—but moving from pain to pain. One does not abandon, even briefly, one's bed of nails, but is attached to it wherever one goes.

Styron's memoir on the depths of his emotional despair is consistent with the scientific evidence base on this common and serious psychiatric disorder. The global point prevalence of major depressive disorder (MDD) (i.e., the proportion of people with MDD at a particular point in time) is 4.7%, and the annual incidence (i.e., the proportion of people who develop MDD during a year) is 3.0% (Ferrari et al. 2013b). In the United States, the lifetime prevalence of MDD is 16.2% (Kessler et al. 2003), which is equivalent to nearly 38 million individuals ≥18 years of age in the United States with lifetime MDD, and many more with subthreshold but clinically significant levels of depressive symptoms (Yu et al. 2020). Depression not only is associated with considerable psychic pain and suffering but also is a predictor of physical morbidity and mortality. For example, both clinical depression and depressive symptoms are associated with the development of coronary heart disease in initially healthy participants (Rugulies 2002). Indeed, depressive disorders are highly associated with years living with disability, second only to cardiovascular disease burden (Ferrari et al. 2013a). Depression is a complex, multifactorial disorder with many interrelated

etiological variables (Kendler & Gardner 2014). Identifying potentially modifiable causal risk factors for depression is a major priority for researchers, and the results from this line of research have far-reaching public health implications for the prevention and treatment of this pernicious disorder.

In the search for potentially modifiable causal risk factors for depression, there is much to be gained by studying intimate relationships. Humans are social animals, and the drive to form and maintain strong, stable interpersonal relationships is a fundamental human motivation (Baumeister & Leary 1995). Many people seek connection with another person through forming and maintaining a long-term intimate (marriage or marriage-like) relationship. For example, the worldwide percentage of women aged 45–49 who have ever married is estimated to be 95.7% (UN Women 2020). Furthermore, there is great variability in the extent to which strong, stable interpersonal relationships are maintained. For example, in the United States, approximately 40–50% of first marriages end in separation or divorce (Smock & Schwartz 2020), and approximately one out of every three married individuals is in a discordant relationship (defined in terms of a nonarbitrary cut point based on the results of a taxometric analysis) (Whisman et al. 2008). Likewise, there is evidence of a broad secular trend toward increased instability in long-term relationships around the world (Härkönen 2014). Given the importance ascribed to interpersonal relationships, the near universal experience of marriage or marriage-like relationships, and the high prevalence of negative relationship outcomes, the reciprocal impact of relationship functioning and mental health is an obvious target for investigation. Intimate relationship distress[1] is strongly associated with a range of clinically significant mental health outcomes (for reviews, see Bodenmann & Randall 2013, Braithwaite & Holt-Lunstad 2017, Whisman & Baucom 2012). However, identifying patterns of causality remains difficult. Despite well-established associations, much of the work linking relationship distress with mental health is correlational, and the literature lacks a synthetic examination of causation across multiple methods. Testing causation between relationship distress and depression, and explicating and testing potential causal mechanisms when causal connections are established, is important not only for theory development and evaluation but also for developing more powerful interventions.

On the surface, reviewing available literature to address questions about direction of causality might seem simple, but it is hardly easy; the literature on intimate relationships and depression is vast, and drawing an integrative conclusion about causality requires a keen awareness of the biases inherent in different research methodologies (Lawlor et al. 2016). To address whether relationship distress plays a causal role in the emergence of depression, this review relies on the metaframework of triangulation (Gage et al. 2016, Lawlor et al. 2016), which involves synthesizing research evidence across different methodologies and study designs, each with a different form of confounding bias, to ascertain the most reliable conclusions and, ideally, increase the theoretical precision of those conclusions. Although there are potentially many different ways to triangulate across methodologies, this review is limited to three different methods, with each method

[1]Although much of the research on relationships and depression has focused on married individuals, we use the term intimate relationship (or simply relationship) to also include nonmarital relationships, such as cohabiting relationships. Furthermore, various measures have been used in prior research, including measures of satisfaction and quality, which assess intrapersonal aspects of relationship functioning (e.g., subjective evaluations), and measures of adjustment, which assess interpersonal aspects (e.g., communication) and typically also include subjective relationship evaluation (Fincham & Rogge 2010). Because the focus of this review is on poor relationship functioning as a potential risk factor for depression, we use the term relationship distress to refer to low levels of relationship satisfaction or adjustment, and we reframe the results of reviewed studies as necessary so that higher scores on the original measures indicate greater relationship distress.

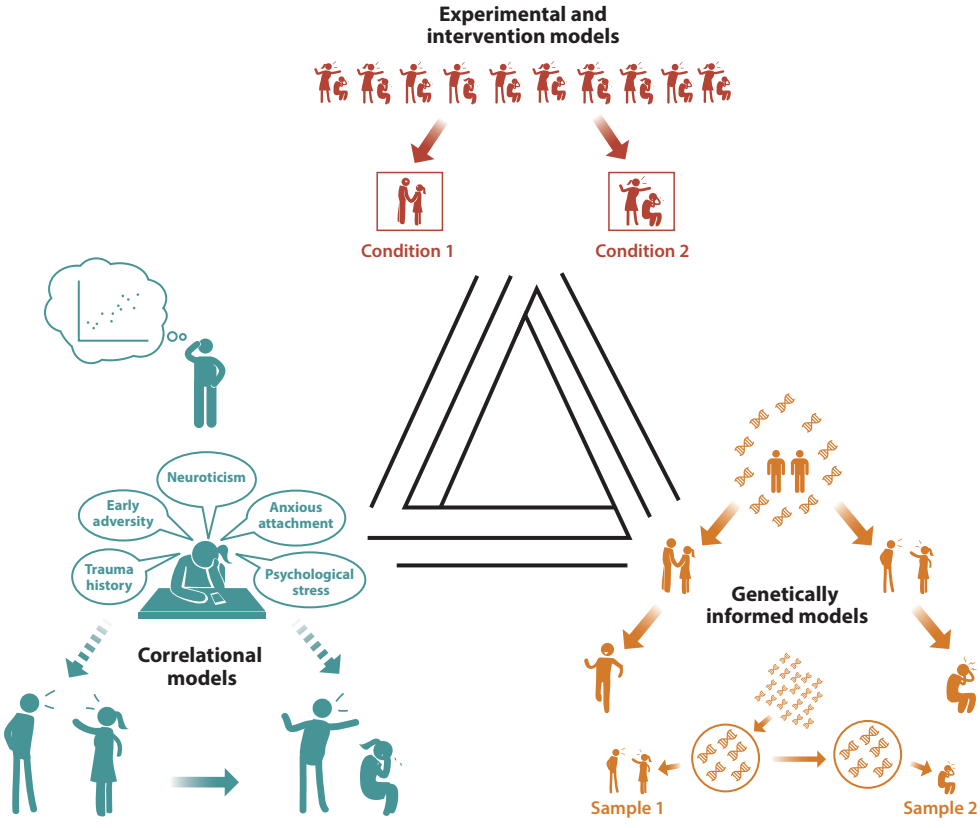

Figure 1

Illustration of the meta-framework of research triangulation that guides this review. This article focuses first on evidence for the association between relationship distress and depressive symptoms from correlational (i.e., observational) studies (*lower left corner*); the analysis then considers findings from studies using genetically informed samples (*lower right corner*); and, finally, the article reviews evidence from intervention (i.e., experimental) studies (*top of triangle*). Necessarily, a triangulation analysis reviews the limits and biases inherent in each methodological approach and integrates findings across the different methods in the service of making stronger statements about causation than can be provided by any one method alone.

powerful in its own way: correlational (i.e., observational) methods, genetically informed methods, and intervention (i.e., experimental) methods, as illustrated in **Figure 1**. The organization of the review follows the triangle depicted in **Figure 1**, and each section of the review discusses the existing empirical literature as well as new approaches that may hold promise for ascertaining answers to the question of causation in this area of study. The review begins with an overview of core theoretical models in this area and then shifts to a review of the available evidence and a discussion of emerging methodologies for each of the three major topics illustrated in **Figure 1**. The review concludes with new directions for future research.

The goals for this review are twofold. First, we seek to draw conclusions regarding the potential causal association between intimate relationship distress and depression and the mechanisms that may account for any causal linkage. Although theoretical models and empirical findings suggest that relationship distress and depression may influence one another in a bidirectional fashion consistent with reciprocal causation, the focus of this review is on the pathway leading from

relationship distress to depression and whether relationship distress is a causal risk factor for depression. Second, we use the meta-framework of triangulation to illustrate several methodologies—many emerging from epidemiology—that can be used in future research to increase the strength of causal inferences regarding the role of relationship distress not only with respect to depression but also with respect to other areas of mental health, physical health, and well-being. Although a comprehensive review of all such methodologies is beyond the scope of this article, we highlight several approaches that may be particularly beneficial for strengthening causal inferences regarding this and related topics (for examples of more detailed discussions of these topics, see Grosz et al. 2020, Hernán & Robins 2020, Kendler 2017).

2. INTIMATE RELATIONSHIPS AND DEPRESSION: RELEVANT THEORY

Causation, in the context of this review, focuses on the following counterfactual question (Pearl 2000): Other things being equal, if a person were not exposed to relationship distress, or a particular facet of relationship distress, would their level of depression be different? If so, it is worth considering how relationship distress may give rise to the signs and symptoms of depression (i.e., mechanisms of causation). At its heart, this question is a theoretical one, and several models may help to explain how functioning in intimate relationships such as marriage may be associated with the onset, severity, and course of depression.

Although they were not specifically developed to advance an intimate relationship model of depression, several theoretical models may, nonetheless, help to explain the association between relationship distress and depression. For example, Coyne (1976a,b) proposed an interpersonal model of depression in which mildly depressed (i.e., depression-prone) individuals seek reassurance of approval and acceptance from others as a function of their doubts of their own self-worth and of whether other people truly care about them. Although others often initially respond with reassurance, the depression-prone person is not convinced by this reassurance, and the pattern is repeated. Because the pattern is repetitive and resistant to change, this seeking of reassurance results, over time, in negative interpersonal reactions (e.g., frustration, feeling misunderstood) from other people, which in turn result in withdrawal and ultimately rejection, which then exacerbates symptoms of depression. A second model that may help to explain the association between relationship distress and depression is the stress generation model of depression (Hammen 1991). This perspective proposes that compared with nondepressed individuals, depression-prone individuals experience more stressful life events that are due, at least in part, to their depression or to maladaptive, enduring characteristics (e.g., cognitions, traits, behaviors) that are associated with low mood. Thus, this perspective makes a distinction between independent life events, which are events whose occurrence is outside the individual's control, and dependent life events, such as relationship problems, whose occurrence is influenced by characteristics of an individual. The experience of dependent life events, in turn, is likely to result in continuing or recurrent symptoms, further increasing the propensity for future stress-generating behavior. This creates a vicious cycle in which symptoms and stress-inducing behavior may become mutually sustaining, thereby contributing to the chronic course of depression.

Beach et al. (1990) developed a marital discord model of depression in which they hypothesized that poor relationship functioning may increase risk for depression by increasing each partner's experience of social stress and/or decreasing their experience of social support. The marital discord model allows for the possibility that marital discord is discontinuous (Gottman et al. 2002), a theme that subsequently was elaborated and tested using taxometric analyses. Taxometric analyses examine the covariation among indicators (e.g., test scores) to identify patterns that indicate

either latent categories (i.e., taxa) on the one hand or dimensions on the other hand (Waller & Meehl 1998). Results from two studies (Beach et al. 2005, Whisman et al. 2008) suggested that relationship distress is taxonic, such that there is a nonarbitrary latent category whose members (i.e., discordant individuals or couples) differ qualitatively from nonmembers (i.e., nondiscordant individuals or couples). Furthermore, preliminary findings suggested a particularly strong association between relationship processes and depressive symptoms at the point of transition to the taxon (i.e., a step function in the association between relationship distress and depression), insofar as taxon membership accounted for unique variance in depressive symptoms for women over and above the variance accounted for by a continuous measure of relationship distress (Whisman et al. 2015). The original marital discord model was expanded by integrating it with stress generation theory (Beach et al. 2014). This expansion indicates that depression will tend to increase the experience of dependent interpersonal stressors over time and thus help maintain chronic depressive episodes. Likewise, the model has incorporated negative reassurance seeking and other interpersonal strategies associated with depression that appear to erode social support (Joiner et al. 1999) and thereby increase vulnerability to stressors external to the relationship. Finally, the model's applicability to different types of intimate relationships (Beach 2014) has been expanded to make it more relevant to a diverse range of families and circumstances. The resulting heuristic framework is inclusive of both individual and contextual processes; posits modifiable, causal, bidirectional effects between relationship distress and depression; and can easily incorporate additional potential mechanisms as they are suggested by new theoretical developments in the field.

Of particular interest for future expansion of the general marital discord model are insights from the targeted rejection and behavioral models of depression. The targeted rejection model (Slavich et al. 2009) highlights the potential importance of distinguishing between negative relationship processes that meet the definition of being targeted and those that do not. Specifically, targeted social rejections are those directed at, and meant to affect, an individual and that involve an active, intentional severing of relational ties with that person. It is likely that the experience of the recipient rather than the intent of the person initiating the rejection is more proximal to its depressogenic effect; some situations are likely to evoke broad consensus about whether they were truly intended as targeted rejections, and other situations are more ambiguous, allowing for individual difference variables to amplify their impact. Because many, but not all, negative events in distressed relationships have the potential to be experienced as targeted rejection, identifying the subset most likely to be viewed as targeted rejection may substantially increase the magnitude of observed associations between relationship distress and depression. Relative to other, more chronic sources of relationship distress, targeted rejections may trigger reactions such as humiliation, self-blame, and uncertainty (i.e., hopelessness) about the future, which may in turn be more likely to increase risk for depression. Supporting the application of the targeted rejection model to the marital context, Cano & O'Leary (2000) found that people who had experienced a severe relationship stressor such as a partner's infidelity or threats of marital dissolution, which often are perceived as targeted rejection, were more likely to be diagnosed with a major depressive episode (MDE) and reported higher levels of nonspecific depression (and anxiety) compared with people with similar levels of relationship distress who had not experienced severe relationship stressors. Similarly, Brown et al. (1995) reported that women who experienced severe stressors that created feelings of humiliation and entrapment were also more likely to become depressed.

The behavioral model of depression, which posits that a low rate of response-contingent positive reinforcement leads to and maintains depression (Dimidjian et al. 2011), suggests another avenue for enhancing the marital discord model. Because couples with high levels of relationship distress often avoid engaging in positive activities together, relationship distress commonly results in a substantial decline in joint pleasant activities, as well as interruption of shared positive

activities with friends and others. Assessment of changes in shared and individual rewarding activities in response to relationship distress would allow researchers to evaluate the degree to which decline in positive activities accounts for the effect of relationship distress on depression.

3. CORRELATIONAL METHODS

Identification of criteria that are necessary to infer causation based on correlational evidence has a long history (Hill 1965). In his book *Correlation and Causality*, Kenny (1979, p. 4) advanced three criteria that are necessary to conclude that *X* causes *Y*: (*a*) *X* and *Y* must be associated with one another (i.e., relationship), (*b*) *X* must precede *Y* in time (i.e., time precedence), and (*c*) "there must not be a *Z* that causes both *X* and *Y* such that the relationship between *X* and *Y* vanishes once *Z* is controlled" (i.e., nonspuriousness or what has been discussed as the third variable or rival explanation problem by other theorists). Regarding tests of time precedence, Granger (1969) proposed that in regression analyses of longitudinal data, the past (i.e., baseline) value of *Y* needs to be included in the analysis predicting the future (i.e., follow-up) value of *Y*; if the baseline value of *X* significantly adds to the equation, then there is evidence of what is referred as Granger causality. Also with respect to time precedence, Kraemer and colleagues (2001) argued that a distinction should be made between a risk factor, which they defined as a correlate that precedes the outcome, and a causal risk factor, which they defined as a risk factor that, when changed, is shown to change the outcome.

3.1. Do Correlational Studies Indicate a Causal Relationship? Reviewing the Evidence

There is a large literature examining the association between relationship distress and depression. In this section, we review evidence for the covariation, time precedence, and nonspurious nature of this association.

3.1.1. Covariation. The cross-sectional association between relationship distress and depressive symptoms is well established. A meta-analysis of the association between these constructs yielded a weighted effect size (*r*) of 0.42 for women and 0.37 for men (Whisman 2001). Similarly, a meta-analysis of the association between relationship distress and well-being, measured in terms of depressive symptoms, anxiety symptoms, self-esteem, and physical health, yielded a weighted *r* of 0.37 (Proulx et al. 2007). Researchers have found associations of similar magnitude between relationship distress and depressive symptoms in more targeted samples. For example, meta-analytic studies have found that relationship distress is significantly associated with postpartum depression, with weighted *r* values ranging from 0.29 to 0.39 (Beck 1996, 2001). In addition, an emerging literature supports the cross-national generalizability of this association, as researchers have replicated the association between relationship distress and depressive symptoms in collectivist societies such as China (Miller et al. 2013) and in several European countries, including Belgium (Heene et al. 2003), France (Dayan et al. 2010), Germany and Austria (Atkins et al. 2011), Sweden (Bergström 2013), and Switzerland (Falconier et al. 2015).

Research on the association between relationship distress and depressive symptoms has also been advanced by using population-based probability sampling. The advantage of this type of sampling is that it yields "an unbiased sample that is representative of the target population" (Bornstein et al. 2013, p. 359) and, therefore, findings from such studies generalize to the target population. Researchers have replicated cross-sectional associations between relationship distress and depressive symptoms in probability samples from Australia (Leach et al. 2013), Canada (St John &

Montgomery 2009), England (Whisman & Uebelacker 2009), Ireland (Whisman et al. 2018b), Norway (Gustavson et al. 2012), Singapore (Sandberg et al. 2012), and the United States (Bookwala & Jacobs 2004; Choi & Ha 2011; Nolen-Hoeksema & Ahrens 2002; Whisman et al. 2004, 2006). For example, one study found relationship distress covaried with depressive symptoms in a probability sample of over 4,700 couples from 11 European countries (Salinger et al. 2020).

Providing an alternative window on the assessment of depression, researchers have evaluated the cross-sectional association between relationship distress and the prevalence of diagnosis-based depressive disorders. Early research found that relationship distress was higher on average among people with depressive disorders in treatment compared with nonpsychiatric controls (e.g., Bauserman et al. 1995, Rodríguez Vega et al. 1993). However, epidemiological studies in the United States have suggested that only 57% of people with 12-month MDD received some type of treatment in the prior 12 months (Kessler et al. 2003). Creating greater confidence that the early observations were not merely an artifact of treatment seeking, studies based on probability samples from Canada and the United States replicated associations between relationship distress and major and minor depressive disorder and dysthymia (Goldfarb et al. 2019; McLeod & Eckberg 1993; Weissman 1987; Whisman 1999, 2007; Whisman et al. 2000; Zlotnick et al. 2000). Furthermore, results from probability samples have provided evidence for cross-racial and cross-ethnic consistency in the association between relationship distress and depressive disorders in the United States (McShall & Johnson 2015, Uebelacker & Whisman 2006). The association between relationship distress and MDD has also been found in probability samples of specific populations, including active-duty soldiers in the US Army (Whisman et al. 2020).

3.1.2. Temporality. Much of the longitudinal research on temporal precedence of relationship distress and depression has used two-wave panel designs, in which researchers use multiple regression analyses or structural equation modeling to evaluate the degree to which residual changes in depressive symptoms from baseline to follow-up are predicted by relationship distress at baseline. Two-wave panel studies have found that relationship distress predicts increases in depressive symptoms in community samples in the United States (e.g., Beach & O'Leary 1993, Beach et al. 2003, Peterson-Post et al. 2014) and Brazil (Hollist et al. 2007), including community samples of understudied groups of individuals, such as racial and ethnic minorities (e.g., Thomas et al. 2019) and sexual minorities (e.g., Gilmour et al. 2019). A meta-analysis of the longitudinal association between relationship distress and well-being yielded a weighted r of 0.25 (Proulx et al. 2007). Statistically significant longitudinal associations between relationship distress and depressive symptoms have been found in probability samples from England (Stafford et al. 2011, Whisman & Uebelacker 2009), Ireland (Whisman et al. 2018b), Norway (Gustavson et al. 2012), and the United States (Miller et al. 2004, Thomas 2016); note that most of these studies were published after Proulx and colleagues' (2007) meta-analysis.

The potential bidirectional association between relationship distress and depressive symptoms also is examined in probability samples, and results indicate not only that there is evidence for a bidirectional association but also that the strength of the association of the path leading from relationship distress to depressive symptoms does not significantly differ from the strength of the association of the path leading from depressive symptoms to relationship distress (Gustavson et al. 2012, Whisman & Uebelacker 2009). The two-wave panel design was expanded upon in a study that used autoregressive cross-lagged models to test the longitudinal association of these constructs across 4 years in a probability sample of German couples (Morgan et al. 2018). Results from annual assessments indicated that for these couples, (*a*) each partner's relationship distress predicted their own depressive symptoms 1 year later, (*b*) each partner's depressive symptoms

predicted their own and their partner's relationship distress 1 year later, and (*c*) the path leading from depressive symptoms to relationship distress was significantly greater than the opposite direction. Taken together, these results indicate that relationship distress and depressive symptoms are associated in a bidirectional fashion over time, consistent with the perspective of reciprocal causation.

Providing an alternative window on assessment of depression, two-wave panel studies also have shown that baseline relationship distress predicts incidence of depressive disorders at follow-up. For example, having a poor relationship with one's spouse at baseline was associated with incidence of depressive disorder at 5-year follow-up in a community sample of men in Finland (Kivelä et al. 1996). Furthermore, baseline relationship distress was associated with annual incidence of MDE in a US probability sample, and this association remained statistically significant after adjustment for prior history of MDE (Whisman & Bruce 1999). In a separate US probability sample, baseline relationship distress predicted annual incidence of MDE assessed 10 years later (Teo et al. 2013). Similarly, baseline relationship distress was associated with 2-year total incidence of MDD (i.e., all onsets of MDD) and new case incidence of MDD (i.e., first onsets of MDD) assessed at 1-year and 3-year follow-up in a probability sample from the Netherlands (Overbeek et al. 2006), and a significant association between baseline relationship distress and major or minor depression at 12-month follow-up was observed in a probability sample from Quebec, Canada, whereas baseline depression did not predict relationship distress at follow-up (Goldfarb et al. 2019). The longitudinal association between relationship distress and incidence of 30-day MDD at 5-year follow-up was also observed in a probability sample of active-duty soldiers in the US Army (Whisman et al. 2021). Finally, in a sample of female twins from a US population-based registry, baseline relationship distress predicted 13-month onset of major depression 5 years later, baseline major depression predicted higher levels of relationship distress 5 years later, and path coefficients for these reciprocal associations were identical in magnitude (Wade & Kendler 2000).

Researchers also study within-subject longitudinal associations between relationship distress and depression; in these studies, researchers assess both variables on multiple occasions and examine the degree to which within-subject changes in one variable are associated with within-subject changes in the other, controlling for the trajectory of each variable. This design evaluates the extent to which change in one variable accounts for change in a second variable, above and beyond the way in which each variable is already changing. Consistent with the perspective that relationship distress causes depression, increases in relationship distress were associated with increases in depressive symptoms over time in (*a*) a pooled sample of women who were newlyweds or in distressed relationships, who were assessed daily (Smith et al. 2012); (*b*) cohabiting or married women, who were assessed weekly (Whitton et al. 2008); (*c*) pregnant women who were at high risk due to a history of depression, who were assessed monthly (Whisman et al. 2011); (*d*) newlywed couples, who were assessed every 3 months (Vento & Cobb 2011) or 6 months (Davila et al. 2003, Karney 2001); and (*e*) couples in established relationships with at least one child, who were assessed every year (Kouros et al. 2008). These results indicated that when someone's relationship distress was higher than usual, their depressive symptoms tended to be higher. Furthermore, consistent with the perspective of reciprocal causation, within-subject analyses indicated that increases in depressive symptoms were associated with increases in relationship distress over time (Davila et al. 2003, Kouros et al. 2008, Vento & Cobb 2011): When someone's depressive symptoms were higher than usual, their relationship distress tended to be higher. The analysis of data collected on multiple occasions also allows researchers to examine the degree to which variability (i.e., fluctuation) in relationship distress over time, which may be experienced as stressful and may reduce emotional security and one's confidence in the relationship, is associated with depression (Whitton & Whisman 2010). In a sample of married and cohabiting women, within-person variability in

relationship distress over 12 weekly assessments accounted for additional variance in depressive symptoms beyond that predicted by mean level of relationship distress; women whose relationship distress fluctuated more widely tended to report higher levels of depressive symptoms.

3.1.3. Nonspuriousness. In testing nonspuriousness of the association between relationship distress and depression, researchers have adopted several approaches. The within-subject associations between relationship distress and depressive symptoms reviewed above support a causal hypothesis because this approach excludes genetic confounding (i.e., a person's genotype does not change over time) (De Moor et al. 2008). In addition, a study conducted to test nonspuriousness examined the longitudinal association between relationship distress and depressive symptoms in a sample of married women who, at baseline, were nondepressed and in nonconflicted marital relationships (Monroe et al. 1986). The sample was restricted in this way to avoid confounds between constructs for people who already were acutely or chronically distressed at the beginning of the study. Consistent with a causal effect, baseline relationship distress was significantly associated with residual change in depressive symptoms 1 year later. Another study that was conducted to examine nonspuriousness focused on dyadic relationship quality, which was operationalized as a latent factor based on both partners' reports of relationship distress; covariation between partners in their level of relationship distress reflected partners' synchronized view of their relationship (Gustavson et al. 2012). In a 2-year longitudinal study involving a probability sample of couples from Norway, dyadic relationship quality and depressive symptoms each predicted changes in the other. The authors concluded that the longitudinal associations between relationship distress and depressive symptoms were "not due to reporter bias or confounded by other individual factors," such as negative thinking and mood bias (Gustavson et al. 2012, p. 776).

The most common approach to study nonspuriousness is to statistically adjust (i.e., control) for potential confounders, which are defined as characteristics that are associated with both the risk factor and the outcome and therefore may account for the association between the two. One important point to consider in testing for confounders is that variables that are a part of the causal chain leading from the risk factor to the outcome are not confounders but rather are mechanisms (i.e., mediators) of the association between the risk factor and the outcome; researchers should not statistically adjust for such variables because doing so may underestimate the influence of the risk factor (Ross & Mirowsky 2013). Researchers have found that the association between relationship distress and depression remains even after they have statistically adjusted for several possible confounders. For example, relationship distress was significantly associated with residual change in depressive symptoms at follow-up after adjusting for upsetting life events (Monroe et al. 1986), health (Miller et al. 2004), and religion and health (Morgan et al. 2018). The cross-sectional (Whisman et al. 2006) and longitudinal (Gustavson et al. 2012) association between relationship distress and depression is obtained when adjusting for personality traits, which is important because personality is associated both with relationship distress (Malouff et al. 2010) and with depression (Kotov et al. 2010). Other research has found that the longitudinal association between relationship well-being and depressive symptoms remains significant after controlling not only for neuroticism but also for self-esteem and stressful life events (Cao et al. 2017). Furthermore, to rule out the possibility that the association between relationship distress and depression may be due to a general tendency to perceive problems in, and be dissatisfied with, close relationships in general, researchers have statistically adjusted for participants' perceived quality of other close relationships (i.e., relationships with relatives and friends). Researchers have found that, after statistical adjustment for perceived quality of other close relationships, there remained a statistically significant (*a*) cross-sectional association between relationship distress and 6-month prevalence of MDD in a Canadian probability sample (Whisman et al. 2000); (*b*) cross-sectional association between

relationship distress and depressive symptoms in a US probability sample with additional adjustments for health status, frequency of physical activity, and religious service attendance (Choi & Ha 2011); (*c*) longitudinal association between relationship distress and change in depressive symptoms in probability samples from England (Stafford et al. 2011), Ireland (Whisman et al. 2018b), and the United States (Thomas 2016); and (*d*) longitudinal association between relationship distress and MDE at 10-year follow-up in a US probability sample with adjustments for several other measures of mental and physical health (Teo et al. 2013). Finally, results from within-person analyses indicated that increases in relationship distress are associated with increases in depressive symptoms over time, adjusting for neuroticism (Vento & Cobb 2011) and within-person fluctuations in mood states (Whitton et al. 2008).

3.2. Correlational Methods: Underused and Novel Approaches

Regression-based correlational studies—both cross-sectional and longitudinal—dominate the research landscape regarding intimate relationships and depression. In this subsection, we introduce three approaches that have the potential to advance work in this area. Fundamentally, these approaches share the biases of all correlational studies, but their strength rests in their ability to help rule out confounding explanations and isolate the effects of interest.

3.2.1. Propensity score matching. As discussed above, the most common regression-based approach to nonspuriousness is the statistical control of confounding variables. In many cases, however, statistical control can introduce its own set of biases (Miller & Chapman 2001). Propensity score matching (PSM) (Thoemmes & Kim 2011) is increasingly used in nonexperimental settings to equate groups of people who cannot be randomized. This methodology is appropriate when trying to make comparisons between an exposure group (e.g., people who experience relationship distress) and a comparison group (e.g., people who do not experience relationship distress). The goal of matching is to identify a subset of people in the comparison group whose scores on relevant control variables are similar to those in the exposure group. Membership in the exposure group is predicted from a set of characteristics, and then people from the exposure group are matched with people in the comparison group on these characteristics (e.g., they are matched on their propensity to be in a distressed relationship). After this matching, differences in outcome (e.g., depression) between the exposure group and comparison groups can be attributed more confidently to group membership (e.g., relationship distress) than to the other characteristics being controlled through propensity matching. One illustration of the ways in which PSM may yield insights into a potentially causal association is a study by Sbarra et al. (2014), in which PSM was used to assess the probability of a depressive episode following the end of marriage. When only statistical controls were used (i.e., a standard regression analysis), the effect of divorce on depression emerged as significant; in the PSM analyses, for people who were equally matched on their probability of becoming divorced, the experience of divorce was associated with future depression only among people who had been depressed in the past. To our knowledge, no studies have used this approach for investigating relationship distress and depression.

3.2.2. Target trial emulation. There are many situations in which a target clinical trial would be unethical, and this is certainly the case for random assignment to relationship distress. Recently, epidemiologists have proposed methods to emulate target trials in observational data sets (Hernán & Robins 2016). Target trial emulation outlines the steps that can be taken in an observational study to make it comparable in terms of eligibility and sampling, timing of the treatment exposure, follow-up period, and outcome assessments observed in an ideal clinical trial (Labrecque

& Swanson 2017). Researchers can emulate target trials to study the outcomes associated with the worsening and improving of relationship distress in large, probability samples. For example, a series of eligibility rules could be created to identify people experiencing a certain level of relationship distress, and then those who evidenced clinically meaningful improvements in relationship distress could be identified; this group would be considered exposed to the emulated trial that improved relationship functioning. The researchers could ask whether, compared with people not exposed to improvements in their relationships, the exposed group demonstrated concomitant improvements in or decreased risk for depression. The target trial methodology allows researchers to explore questions that would clearly be unethical in a clinical trial—for instance, compared with people exposed to increased relationship distress (over, say, two occasions in an observational study), do we observe concomitant increases in depression? Moreover, this method may avoid many of the selection biases typically associated with choosing to participate in a treatment trial, allowing broader generalization of observed effects. At face value, this approach may resemble a standard rank-order, correlational assessment of change, but the target trial emulation methodology includes several steps to improve unbiased estimation, such as the use of techniques like propensity scores to ensure that the "treatment"-exposed and unexposed adults are matched on variables that predict greater likelihood of group membership in an observational trial.

3.2.3. Directed acyclic graph approach. Graphical representations of causal effects are frequently used in epidemiology as a means of identifying sources of confounding bias, and directed acyclic graphs (DAGs) provide a formalized system for recognizing and specifying different sources of bias in causal modeling (Shrier & Platt 2008). Although researchers often use process models to illustrate the nomological constellation of variables that may be associated with an outcome of interest (for an illustration of one such model in relation to divorce and health, see Sbarra & Coan 2017), the specific roles of the variables in the models and their confounding influences are rarely explicated in a formal way. DAGs provide an approach for formalizing these interdependencies; their primary functions are identifying which variables to control and making the model's assumptions explicit (Shrier & Platt 2008). Thinking through the pathways associated with DAGs will be familiar to people who use structural equation modeling; the major contribution of DAGs in psychological science is to help investigators formalize conceptual models that are, in turn, represented as mathematical or statistical models (Pearl 2000). DAGs that link relationship distress to depression through multiple hypothesized causal pathways unfolding over time can be quite complex, but their formal explication may ultimately help identify alternative causal mechanisms that may be operative for some individuals but not for others. Likewise, this exercise is likely to better explicate indirect and buffering pathways from relationship distress to depression and vice versa. Finally, DAGs provide an opportunity to explicate sources of bias that may be specific to particular pathways or a concern for more or all pathways in the observational study of this association.

4. GENETICALLY INFORMED METHODS

Turning to the second vertex in the triangulation analysis, this section focuses on genetically informed research. Although the methods described below are nonexperimental, they are useful tools for evaluating confounding biases that cannot be addressed in correlational studies alone. Twin studies provide a powerful tool for identifying whether evidence supports a potential causal influence of a life event exposure (e.g., relationship distress) on a health outcome of interest (McGue et al. 2010). For example, does relationship distress predict increased depression, or is this association due to shared genetic or environmental factors common to both relationship distress and depression that confound the phenotypic association commonly reported in the literature?

This is an important question because there are genetic influences on both relationship distress (Spotts et al. 2004b) and depression (Sullivan et al. 2000). First, differences between monozygotic twins, who share 100% of their genes, and dizygotic twins, who share only 50% of their segregating genes on average, are used to decompose the variance in a trait or phenotype into additive genetic effects, shared environmental effects (environmental influences shared between twins in a family), and unique or nonshared environmental influences (environmental influences unique to each twin). This decomposition can be used to derive the heritability of a phenotype as well as the genetic, shared environmental, and nonshared environmental influences on the covariation between phenotypes.

Twin studies can also be used to examine whether twins who are discordant for phenotypic exposure (e.g., relationship distress) demonstrate differences in an outcome (e.g., depression) after shared genetic and environmental influences are taken into account (Osler et al. 2008). The twin biometric model allows researchers to examine whether an intrapair difference in a predictor, such as relationship distress, is associated with an intrapair difference in an outcome, such as depression. If the phenotypic association remains statistically significant after accounting for genetic and shared environmental confounds, then the findings are consistent with a causal association, and the estimated effect size of the association will be less biased because two major sources of confounding are removed; the residualized effect (from the exposure to the outcome after accounting for these confounds) is referred to as a quasi-causal effect (Turkheimer & Harden 2014).

4.1. Do Genetically Informed Studies Indicate a Causal Relationship? Reviewing the Evidence

Several studies have used twin samples to evaluate the association between relationship distress and depression. A study of Swedish female twins found shared genetic influences on the covariation between twins' (i.e., wives') self-reported relationship distress, social support, and depressive symptoms (Spotts et al. 2004a). Importantly, there also was evidence for nonshared environmental influences on the covariation between twins' depressive symptoms and both twins' and their partners' (i.e., husbands') reporting of relationship distress, which may reflect the influence of partners' characteristics (i.e., twins being married to different partners). In an independent sample of Swedish male and female twins that focused exclusively on relationship distress and depressive symptoms (i.e., that did not include social support in the analyses), there was evidence of nonshared environmental influences on the covariation between both twins' and partners' reporting of relationship distress and twins' depressive symptoms (Whisman et al. 2018a). The inclusion of partners' reports of relationship distress in these two studies suggests that the association between relationship distress and depression is not simply the result of having the same person evaluate both their relationship and their depression (i.e., single-reporter bias).

A third twin study (Beam et al. 2011), involving a sample of Australian male and female twins, used the Turkheimer & Harden (2014) model to test quasi-causal effects. Results indicated that the association between relationship distress and depressive symptoms was statistically significant after adjusting for genetic effects of relationship distress on depressive symptoms. These findings suggest that the phenotypic association between relationship distress and depressive symptoms is not an artifact of selection (i.e., people genetically prone to depression selecting less desirable partners, leading to greater relationship distress).

Genetically informative designs can also be helpful in testing potential causal associations between variables. For example, provided that several assumptions are satisfied, cross-sectional twin data can be used to distinguish causal hypotheses from correlated genetic or environmental influences; the power to test for causation between variables increases when the two variables have

rather different modes of inheritance (e.g., one variable is largely determined by genetic factors, whereas the other is largely determined by environmental factors) (Heath et al. 1993).

Finally, reverse causation and bidirectional effects can be examined using longitudinal genetically informed samples. One such study examining the longitudinal association between relationship distress (i.e., partner support, partner problems) and depression was conducted on a sample of female twins from a US population-based registry who were assessed at baseline and then at follow-up an average of 5 years later (Wade & Kendler 2000). Results from the longitudinal analyses for partner support contained one cross-variable cross-time path: Major depression at baseline was associated with reduced level of partner support at follow-up. Furthermore, there was evidence of two cross-variable cross-time paths for partner problems: Higher levels of partner problems at baseline predicted an increased risk for major depression at follow-up, and major depression at baseline predicted higher levels of partner problems at follow-up. A lifetime history of major depression in one twin was associated with significantly lower levels of partner support but was unrelated to partner problems in the cotwin; this finding suggests that shared genetic factors may partly contribute to the association between partner support and depression but not to the association between partner problems and depression. The pattern of results suggests that relationship distress as operationalized in terms of level of partner problems (e.g., too many demands, criticism, disagreements) is causally associated with major depression (and that major depression is causally associated with relationship distress).

4.2. Genetically Informed Methods: Underused and Novel Approaches

Notwithstanding its ability to rule out shared environmental and genetic factors as confounders of the association between relationship distress and depression, the cotwin control design cannot rule out confounding by environmental factors that are unique to each twin within a pair. The random assortment of alleles during gamete formation creates a natural experiment in which genetic variants are typically independent of the factors that confound the association between a risk exposure and disease outcome (Smith & Ebrahim 2004). That is, genetic variants for a given phenotype are typically independent of the genetic variants of common behavioral and environmental confounds. In Mendelian randomization (MR), which is a special type of instrumental variable analysis, gene variants associated with a risk exposure (e.g., relationship distress) in a very large data set—typically identified in one sample with many thousands of participants (Lawlor et al. 2008)—are then used to predict an outcome of interest (e.g., depression) in another sample. The two-sample MR is useful not only for deconfounding third-variable influences but also for studying reverse causation via bidirectional MR. To the extent that the gene variants associated with a risk exposure identified in one sample are associated with the outcome of interest in another sample, confidence in a causal association between the variables can be high (Davey Smith & Hemani 2014). Although there are no MR reports that specifically assess relationship distress and depression, a recent two-sample MR study using data from the UK Biobank found that, among a large list of potentially modifiable risk factors for depression, self-reported frequency of confiding in others was associated with incident depression in a manner consistent with a causal effect (Choi et al. 2020). Of course, the "frequency of confiding in others" variable examined in this study does not map perfectly onto the assessment of relationship distress; however, we would expect that, relative to people in high-quality relationships, those in distressed relationships would be less likely to report confiding in others, and this perception may be a key causal ingredient linking intimate relationship distress to depression. Other MR studies on social well-being and cardiometabolic health may be instructive regarding the use of this methodology to evaluate causal risk factors for depression (Abdellaoui et al. 2019, Day et al. 2018). At the level of specific genotypes, the

BEYOND RELATIONSHIP DISTRESS: OTHER RELATIONAL CONTRIBUTORS TO DEPRESSION

Although relationship distress is generally viewed as the "final common pathway" connecting intimate relationships to outcomes (Jacobson 1985, p. 327), it may underestimate the full effect of relationship functioning on depression. Other factors, including intimate partner violence (Spencer et al. 2019), poor communication marked by high rates of negative behavior and low rates of positive behavior (Rehman et al. 2008), and severe marital stressors, such as discovery of a partner's affair or having a partner threaten to end the relationship (Cano & O'Leary 2000, Cano et al. 2004, Whisman 2016), are associated with depression and may have an impact on depression over and above their shared association with relationship distress. Reactions to relationship distress may also vary depending on whether partners feel trapped, humiliated, and/or rejected (Brown et al. 1995, Slavich et al. 2009) and depending on the presence of genetic, personality, and other characteristics that confer greater vulnerability for depression in response to relationship distress (Goldfarb & Trudel 2019, Uebelacker & Whisman 2006, Whisman & South 2017). Because various indices of relationship functioning are associated with depression, our focus on relationship distress likely provides a conservative estimate of the impact of relationship functioning on depression, particularly among vulnerable individuals.

vasopressin receptor 1a gene (*AVPR1A*) (Walum et al. 2008) and the oxytocin receptor gene (*OXTR*) (Schneiderman et al. 2014, Walum et al. 2012) may be promising starting points for future research using an MR approach, given their association with pair-bonding and social behavior in prior research (Whisman & South 2017) (see the sidebar titled Beyond Relationship Distress: Other Relational Contributors to Depression).

5. EXPERIMENTAL AND INTERVENTION METHODS

The experimental and intervention vertex is the third and final focus of this review. The randomized controlled trial (RCT), generally considered the gold standard for treatment evaluation, provides some of the strongest evidence for causal inferences regarding risk factors. In an RCT, participants are randomly assigned to either an experimental (i.e., treatment) group or a control group, and their outcomes are compared. Because participants are randomly assigned, the groups should not differ on known and unknown confounding variables; they should differ only on manipulation of the purported risk factor. The treatment group and control group are compared on changes in both the risk factor and the outcome variable. If the treatment group has greater changes in the risk factor and the outcome variable than the control group does, then the results support a causal role for the risk factor.

Several approaches to couple-based interventions have been evaluated as treatments for depression, and the efficacy of these treatments has been the subject of narrative (Whisman et al. 2012) and meta-analytic reviews (Barbato & D'Avanzo 2020), both of which concluded that couple-based interventions for depression are efficacious in producing reductions in depressive symptoms. The focus of the current review is on results from these intervention studies that are most relevant to causal inferences regarding the association of relationship distress and depression. Beach & O'Leary (1992) conducted an RCT in which they compared cognitive behavioral couple therapy (CBCT) for depression with a waitlist control condition. CBCT for depression is a structured, short-term treatment that seeks to eliminate major stressors, reestablish positive activities, and improve communication and problem solving (Whisman & Beach 2012). Results indicated that relative to the waitlist control condition, CBCT resulted in greater reductions in both depressive symptoms and relationship distress.

Researchers have also examined longitudinal associations between relationship distress and depression for people in couple-based treatments for depression. One study found that pre- to posttreatment change in relationship distress was significantly associated with pre- to posttreatment change in depressive symptoms for couples receiving CBCT for depression (Jacobson et al. 1991). Moreover, the RCT that found that CBCT for depression resulted in changes in depressive symptoms and relationship distress relative to a waitlist control group also found that pre- to posttreatment change in depressive symptoms was correlated with pre- to posttreatment change in relationship distress, and the difference between the CBCT and control groups in change in depressive symptoms was no longer statistically significant when adjusting for change in relationship distress (Beach & O'Leary 1992). Whereas these two studies were RCTs within university settings (i.e., efficacy studies), Baucom et al. (2018) examined the degree to which change in relationship distress was associated with change in depression in couples who received CBCT for depression in routine clinical settings (i.e., an effectiveness study). A decrease in level of relationship distress from pre- to posttreatment was associated with a decrease in level of depressive symptoms at posttreatment.

Providing a complementary view of the impact of couple-based interventions on depressive symptoms, researchers have also examined changes in relationship distress and depression for people in treatment for relationship distress. One RCT examined the efficacy of an Internet-based intervention that was based on integrative behavioral couple therapy (IBCT), which promotes emotional acceptance and resulting behavior change (Doss et al. 2016). Compared with couples assigned to a waitlist control condition, those assigned to the intervention condition demonstrated significantly greater improvement in relationship distress and greater reduction in depressive symptoms. In another study, married couples with serious and stable relationship distress were randomly assigned to one of two types of behaviorally oriented couple therapy (CBCT or IBCT); results showed that improvements in relationship distress were significantly associated with decreases in depressive symptoms (Atkins et al. 2009). Therefore, similar to the findings from couple-based treatment for one partner's depression, couples in treatment for relationship distress show reductions in depressive symptoms, and change in relationship distress is associated with change in depressive symptoms.

Finally, one study examined changes in relationship distress and depressive symptoms in couples who participated in a Marriage Checkup (MC) program, a two-session assessment and feedback intervention based on motivational interviewing and IBCT techniques and intended to be equivalent to an annual physical or dental checkup (Gray et al. 2020). MC is designed to attract a range of couples, including those in the at-risk category, who might not otherwise seek relationship treatments. In a sample of couples assigned to MC or a waitlist control group, MC resulted in significant declines in depressive symptoms, changes in relationship distress were associated with changes in depressive symptoms, and three-quarters of the treatment effect on depression was mediated by changes in relationship distress.

6. FUTURE DIRECTIONS AND REMAINING QUESTIONS

6.1. Mechanisms for the Association Between Relationship Distress and Depression

Although the focus of this review is on relationship distress as a potential causal risk factor for depression, there is also a need for research on the mechanisms that may lead from relationship distress to depression. As noted above, there are many potential causal mechanisms that may play this role, but few have received adequate empirical attention. Cross-sectional studies reveal indirect paths linking relationship distress and depression for several constructs, including communication

styles, self-silencing (i.e., the tendency to suppress one's thoughts and feelings to avoid conflict), social support behaviors and perceived social support, relationship attributions, self-esteem, mastery, and adult attachment; however, results often differ by gender and population (e.g., community sample, clinical sample), and most constructs have not been examined in more than one study (for a review, see Goldfarb & Trudel 2019). Moreover, although establishing a nonzero indirect path provides evidence for statistical mediation, it does not, by itself, support the case for a causal mechanism (Kazdin 2007), and making this case involves a return to the basic elements for establishing causation, which requires the use of longitudinal data (Kraemer et al. 2001). Although testing and providing evidence for hypothesized indirect paths in cross-sectional research does not establish mediation, it is an important first step in identifying potential mediators that could be evaluated as mechanisms of action in future longitudinal studies.

In comparison to the cross-sectional data, there is little longitudinal research examining characteristics that could account for the prospective association between relationship distress and depression. One longitudinal study involving behavioral observation of couples engaging in conflict resolution tasks found that constructive, angry, and depressive conflict resolution styles mediated the longitudinal association between relationship distress and depressive symptoms (Du Rocher Schudlich et al. 2011), consistent with the marital discord model of depression (Beach et al. 1990). Further such research is needed to advance understanding of the mechanisms for the association between relationship distress and depression.

6.2. Relationship Distress as a Moderator of the Association Between Stress and Depression

Although our discussion of causality has focused on methods to more confidently detect direct (i.e., main) effects that lead from relationship distress to depression and to refine the understanding of causal mechanisms for these effects, a substantial theoretical literature suggests that relationship processes such as support provision and relationship security may play an important role in moderating (i.e., buffering) the impact that stressors that are external to the relationship have on depression. Because external stressors may have less impact when individuals are embedded in relationships perceived to be supportive (Beach et al. 2019, Hostinar 2015), it is important also to examine the moderating effects of relationship distress on other causal factors involved in depression. As sources of external threat proliferate, the buffering impact of protective relationships—and the negative consequences of losing that buffering because of unsupportive relationships—may increase (Cole 2014). This perspective suggests the potential importance of what may be labeled causal buffering effects, whereby experimentally induced changes in relationship adjustment exert an effect on depression by buffering the potentially causal association between life stress (or other contributing causes) and depression. Direct and indirect causal moderation of the effects of stress on depression is described by Howe (2019) and empirically examined in a study by Lei & Beach (2020). In the context of relationship intervention programs, similar analyses could be used to examine the degree to which improving particular facets of relationship functioning and decreasing relationship distress can result in significant decreases in the strength of the association between external stressors and depressive symptoms. Through moderating (i.e., weakening) the association between stress and depression (i.e., through causal buffering effects), relationship distress may explain additional variance in depression over and above its direct effect. Therefore, a full understanding of the magnitude and scope of the association between relationship distress and depression should consider not only the direct effect of relationship distress but also the moderating effect that relationship distress (or more accurately, relationship adjustment) may have in buffering the impact of other risk factors (e.g., stress) on depression.

7. SUMMARY AND CONCLUSIONS

The aims of this review were twofold. First, we introduced the idea of triangulation (borrowed from epidemiology) and called for an increased application of this meta-framework when researchers seek to evaluate causal inferences. In calling for this work—in any field of study—we illustrated a plurality of methods (each with its own biases and limitations) that, when viewed from the perspective of triangulation, can ultimately enhance the pursuit of causal inference. Triangulation can include established methods of inferring causation, such as correlational, genetically informed, and intervention methods, as well as newer and less frequently used methods, such as propensity score matching, target trial emulation, directed acyclic graph approach, and Mendelian randomization.

Our second aim was to apply the triangulation perspective in reviewing the correlational, genetically informed, and intervention research on relationship distress and depression and to draw conclusions about causation for the association between relationship distress and depression. With respect to correlational studies, the first vertex of the triangulation analysis, "we can never be certain about causal processes. We can only seek for increased confidence that causal effects are likely present" (Kendler 2017, p. 562). With this caveat in mind, a large body of research involving both convenience and probability samples that differ in key demographic characteristics and geographical location provides evidence for Kenny's (1979) three criteria for inferring causation based on correlational data, although additional research on time precedence and nonspuriousness would help increase confidence in this conclusion. That is, correlational data indicate that relationship distress may be considered a risk factor for depression. Furthermore, because regression analyses have shown that relationship distress at baseline predicts depressive symptoms and depression status at follow-up, adjusting for depressive symptoms and depression status at baseline, there is evidence of Granger (1969) causality for the association between relationship distress and depression. With respect to future research on time precedence, there is a need to better specify optimal time lags for evaluating the longitudinal association between relationship distress and depression (i.e., the time interval between assessments needs to be long enough for prospective effects to occur, but not so long that they have already faded) (Selig & Preacher 2009), a need to better examine potential variation in this longitudinal association due to context and characteristics of people, their partners, and their relationships, and a likely need for additional design improvements. Regarding the second vertex of the triangulation analysis, results from genetically informed methods (i.e., twin studies) build on the evidence from the correlational studies to suggest that covariation between relationship distress and depression is not secondary to shared genetic influences or selection effects. Finally, regarding the third vertex of the triangulation analysis, results from intervention studies of couples seeking treatment for depression and/or relationship distress indicate that experimentally reducing relationship distress results in improvement in depression. The findings from intervention studies are particularly important insofar as they address Kraemer and colleagues' (2001) distinction between a risk factor, defined as a correlate that precedes the outcome, and a causal risk factor, defined as a risk factor that, when changed, is shown to change the outcome. Because couple-based interventions are effective in reducing relationship distress and depression for couples in which one partner is depressed (Barbato & D'Avanzo 2020), and because changes in relationship distress are shown to change depression, relationship distress meets the criteria for a causal risk factor. Therefore, summating across the evidence obtained from research using correlational, genetically informed, and intervention methods, we conclude that relationship distress is a causal risk factor for depression.

In summary, depression is a common and serious psychiatric disorder with substantial levels of distress and impairment (Kessler et al. 2003). Identifying modifiable causal risk factors for

depression is a major public health priority, and this review indicates that relationship distress is one such causal risk factor. Therefore, the use of couple-based interventions for preventing and reducing relationship distress has important public health implications for the prevention and treatment of depression.

SUMMARY POINTS

1. Relationship distress, a key component of intimate relationship functioning, is consistently associated with depression, but there are many ways in which the association between these constructs may be confounded.
2. Using the meta-framework of research triangulation, this review synthesizes evidence across several different methodologies and study designs to draw a firm conclusion on whether relationship distress may be considered a causal risk factor for depression.
3. The field abounds with cross-sectional and longitudinal correlational studies linking relationship distress and depression, and we review this evidence as well as introduce methodologies and study designs that are relatively underused in psychological science but that can strengthen causal inferences from correlational studies.
4. Studies using genetically informed samples also help inform causal conclusions, and we review evidence about the association between relationship distress and depression from twin studies as well as highlight the merits of Mendelian randomization for future application in this area.
5. Intervention (i.e., experimental) research and randomized controlled trials represent the gold standard for establishing causation, and we review the experimental evidence showing that the successful amelioration of relationship distress reduces depression.
6. This review affirms that relationship distress plays a causal role in risk for depression, and we outline a number of areas for future studies, including the need to investigate the putative mechanisms that account for this association as well as the ways in which relationship functioning may buffer the impact of external stressors on depression.
7. Although this review focuses on drawing causal inferences regarding the association between relationship distress and depression, research triangulation is a meta-framework that can be applied fruitfully to many areas of study in psychological science, especially in cases where potential confounding or reverse causation may be a concern.

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LITERATURE CITED

Abdellaoui A, Sanchez-Roige S, Sealock J, Treur JL, Dennis J, et al. 2019. Phenome-wide investigation of health outcomes associated with genetic predisposition to loneliness. *Hum. Mol. Genet.* 28:3853–65

Atkins DC, Bortnik KE, Hahlweg K, Klann N. 2011. The association between marital discord and depression in a community-based sample of couples seeking treatment. *J. Couple Relationsh. Ther.* 10:20–33

Atkins DC, Dimidjian S, Bedics JD, Christensen A. 2009. Couple discord and depression in couples during couple therapy and in depressed individuals during depression treatment. *J. Consult. Clin. Psychol.* 77:1089–99

Barbato A, D'Avanzo B. 2020. The findings of a Cochrane meta-analysis of couple therapy in adult depression: implications for research and clinical practice. *Fam. Process* 59:361–75

Baucom DH, Fischer MS, Worrell M, Corrie S, Belus JM, et al. 2018. Couple-based intervention for depression: an effectiveness study in the National Health Service in England. *Fam. Process* 57:275–92

Baumeister RF, Leary MR. 1995. The need to belong: desire for interpersonal attachments as a fundamental human motivation. *Psychol. Bull.* 117:497–529

Bauserman SAK, Arias I, Craighead WE. 1995. Marital attributions in spouses of depressed patients. *J. Psychopathol. Behav. Assess.* 17:231–49

Beach SRH. 2014. The couple and family discord model of depression: updates and future directions. In *Interpersonal Relationships and Health: Social and Clinical Psychological Mechanisms*, ed. CR Agnew, SC South, pp. 133–55. New York: Oxford Univ. Press

Beach SRH, Fincham FD, Amir N, Leonard KE. 2005. The taxometrics of marriage: Is marital discord categorical? *J. Fam. Psychol.* 19:276–85

Beach SRH, Katz J, Kim S, Brody GH. 2003. Prospective effects of marital satisfaction on depressive symptoms in established marriages: a dyadic model. *J. Soc. Pers. Relationsh.* 20:355–71

Beach SRH, Lei MK, Simons RL, Barr AB, Simons LG, et al. 2019. Perceived relationship support moderates the association of contextual stress with inflammation among African Americans. *J. Fam. Psychol.* 33:338–48

The first clinical trial to demonstrate that reducing relationship distress ameliorates depression.

Beach SRH, O'Leary KD. 1992. Treating depression in the context of marital discord: outcome and predictors of response of marital therapy versus cognitive therapy. *Behav. Ther.* 23:507–28

Beach SRH, O'Leary KD. 1993. Marital discord and dysphoria: For whom does the marital relationship predict depressive symptomatology? *J. Soc. Pers. Relationsh.* 10:405–20

Introduced the marital discord model of depression and is essential reading on the topic.

Beach SRH, Sandeen EE, O'Leary KD. 1990. *Depression in Marriage: A Model for Etiology and Treatment*. New York: Guilford

Beach SRH, Whisman MA, Bodenmann G. 2014. Couple, parenting, and interpersonal therapies for depression in adults: toward common clinical guidelines within a stress-generation framework. In *Handbook of Depression*, ed. IH Gotlib, CL Hammen, pp. 552–70. New York: Guilford

Beam CR, Horn EE, Hunt SK, Emery RE, Turkheimer E, Martin N. 2011. Revisiting the effect of marital support on depressive symptoms in mothers and fathers: a genetically informed study. *J. Fam. Psychol.* 25:336–44

Beck CT. 1996. A meta-analysis of predictors of postpartum depression. *Nurs. Res.* 45:297–303

Beck CT. 2001. Predictors of postpartum depression: an update. *Nurs. Res.* 50:275–85

Bergström M. 2013. Depressive symptoms in new first-time fathers: associations with age, sociodemographic characteristics, and antenatal psychological well-being. *Birth* 40:32–38

Bodenmann G, Randall AK. 2013. Close relationships in psychiatric disorders. *Curr. Opin. Psychiatry* 26:464–67

Bookwala J, Jacobs J. 2004. Age, marital processes, and depressed affect. *Gerontologist* 44:328–38

Bornstein MH, Jager J, Putnick DL. 2013. Sampling in developmental science: situations, shortcomings, solutions, and standards. *Dev. Rev.* 33:357–70

Braithwaite S, Holt-Lunstad J. 2017. Romantic relationships and mental health. *Curr. Opin. Psychol.* 13:120–25

Brown GW, Harris TO, Hepworth C. 1995. Loss, humiliation and entrapment among women developing depression: a patient and nonpatient comparison. *Psychol. Med.* 25:7–21

Cano A, O'Leary KD. 2000. Infidelity and separations precipitate major depressive episodes and symptoms of nonspecific depression and anxiety. *J. Consult. Clin. Psychol.* 68:774–81

Cano A, O'Leary KD, Heinz W. 2004. Short-term consequences of severe marital stressors. *J. Soc. Pers. Relationsh.* 21:419–30
Cao H, Zhou N, Fang X, Fine M. 2017. Marital well-being and depression in Chinese marriage: going beyond satisfaction and ruling out critical confounders. *J. Fam. Psychol.* 31:775–84
Choi KW, Stein MB, Nishimi KM, Ge T, Coleman JRI, et al. 2020. An exposure-wide and Mendelian randomization approach to identifying modifiable factors for the prevention of depression. *Am. J. Psychiatry* 177:944–54
Choi NG, Ha JH. 2011. Relationship between spouse/partner support and depressive symptoms in older adults: gender difference. *Aging Ment. Health* 15:307–17
Cole SW. 2014. Human social genomics. *PLOS Genet.* 10:e1004601
Coyne JC. 1976a. Depression and the response of others. *J. Abnorm. Psychol.* 85:186–93
Coyne JC. 1976b. Toward an interactional description of depression. *Psychiatry* 39:28–40
Davey Smith G, Hemani G. 2014. Mendelian randomization: genetic anchors for causal inference in epidemiological studies. *Hum. Mol. Genet.* 23:R89–98
Davila J, Karney BR, Hall TW, Bradbury TN. 2003. Depressive symptoms and marital satisfaction: within-subject associations and the moderating effects of gender and neuroticism. *J. Fam. Psychol.* 17:557–70
Day FR, Ong KK, Perry JRB. 2018. Elucidating the genetic basis of social interaction and isolation. *Nat. Commun.* 9:2457
Dayan J, Creveuil C, Dreyfus M, Herlicoviez M, Baleyte JM, O'Keane V. 2010. Developmental model of depression applied to prenatal depression: role of present and past life events, past emotional disorders and pregnancy stress. *PLOS ONE* 5:e12942
De Moor MHM, Boomsma DI, Stubbe JH, Willemsen G, de Geus EJC. 2008. Testing causality in the association between regular exercise and symptoms of anxiety and depression. *Arch. Gen. Psychiatry* 65:897–905
Dimidjian S, Barerra MB Jr., Martell C, Muñoz RF, Lewinsohn PM. 2011. The origins and current status of behavioral activation treatments for depression. *Annu. Rev. Clin. Psychol.* 7:1–38
Doss BD, Cicila LN, Georgia EJ, Roddy MK, Nowlan KM, et al. 2016. A randomized controlled trial of the web-based OurRelationship program: effects on relationship and individual functioning. *J. Consult. Clin. Psychol.* 84:285–96
Du Rocher Schudlich TD, Papp LM, Cummings EM. 2011. Relations between spouses' depressive symptoms and marital conflict: a longitudinal investigation of the role of conflict resolution styles. *J. Fam. Psychol.* 25:531–40
Falconier MK, Nussbeck F, Bodenmann G, Schneider H, Bradbury T. 2015. Stress from daily hassles in couples: its effects on intradyadic stress, relationship satisfaction, and physical and psychological well-being. *J. Marital Fam. Ther.* 41:221–35
Ferrari AJ, Charlson FJ, Norman RE, Patten SB, Freedman G, et al. 2013a. Burden of depressive disorders by country, sex, age, and year: findings from the Global Burden of Disease Study 2010. *PLOS Med.* 10:e1001547
Ferrari AJ, Somerville AJ, Baxter AJ, Norman R, Patten SB, et al. 2013b. Global variation in the prevalence and incidence of major depressive disorder: a systematic review of the epidemiological literature. *Psychol. Med.* 43:471–81
Fincham FD, Rogge R. 2010. Understanding relationship quality: theoretical challenges and new tools for assessment. *J. Fam. Theory Rev.* 2:227–42
Gage SH, Munafò MR, Davey Smith G. 2016. Causal inference in developmental origins of health and disease (DOHaD) research. *Annu. Rev. Psychol.* 67:567–85
Gilmour AL, Whisman MA, Randall AK. 2019. Relationship discord and depressive symptoms in sexual minority individuals over time. *J. Fam. Psychol.* 33:994–99
Goldfarb MR, Trudel G. 2019. Marital quality and depression: a review. *Marriage Fam. Rev.* 55:737–63
Goldfarb MR, Trudel G, Boyer R, Preville M. 2019. Till depression do us part? Examining the prospective associations between depression and marital adjustment in older adults. *Rev. Eur. Psychol. Appl.* 69:41–49
Gottman JM, Swanson C, Swanson KA. 2002. A general systems theory of marriage: nonlinear difference equation modeling of marital interaction. *Pers. Soc. Psychol. Rev.* 6:326–40
Granger CW. 1969. Investigating causal relations by econometric models and cross-spectral methods. *Econometrica* 37:424–38

Gray TD, Hawrilenko M, Cordova JV. 2020. Randomized controlled trial of the Marriage Checkup: depression outcomes. *J. Marital Fam. Ther.* 46:507–22

Grosz MP, Rohrer JM, Thoemmes F. 2020. The taboo against explicit causal inference in nonexperimental psychology. *Perspect. Psychol. Sci.* 15:1243–55

Gustavson K, Røysamb E, Soest TV, Helland MJ, Karevold E, Mathiesen KS. 2012. Reciprocal longitudinal associations between depressive symptoms and romantic partners' synchronized view of relationship quality. *J. Soc. Pers. Relationsh.* 29:776–94

Hammen C. 1991. Generation of stress in the course of unipolar depression. *J. Abnorm. Psychol.* 100:555–61

Härkönen J. 2014. Divorce: trends, patterns, causes, consequences. In *The Wiley-Blackwell Companion to the Sociology of Families*, ed. J Treas, J Scott, M Richards, pp. 303–22. Oxford, UK: Wiley-Blackwell

Heath AC, Kessler RC, Neale MC, Hewitt JK, Eaves LJ, Kendler KS. 1993. Testing hypotheses about direction of causation using cross-sectional family data. *Behav. Genet.* 23:29–50

Heene E, Buysse A, Van Oost P. 2003. A categorical and dimensional perspective on depression within a nonclinical sample of couples. *Fam. Process* 42:133–49

Hernán MA, Robins JM. 2016. Using big data to emulate a target trial when a randomized trial is not available. *Am. J. Epidemiol.* 183:758–64

Hernán MA, Robins JM. 2020. *Causal Inference: What If*. Boca Raton, FL: Chapman & Hall/CRC

Hill AB. 1965. The environment and disease: association or causation? *Proc. R. Soc. Med.* 58:295–300

Hollist CS, Miller RB, Falceto OG, Fernandes CL. 2007. Marital satisfaction and depression: a replication of the marital discord model in a Latino sample. *Fam. Process* 46:485–98

Hostinar CE. 2015. Recent developments in the study of social relationships, stress responses, and physical health. *Curr. Opin. Psychol.* 5:90–95

Howe GW. 2019. Preventive effect heterogeneity: causal inference in personalized prevention. *Prev. Sci.* 20:21–29

Jacobson NS. 1985. Uses versus abuses of observational measures. *Behav. Assess.* 7:323–30

Jacobson NS, Dobson K, Fruzzetti AE, Schmaling KB, Salusky S. 1991. Marital therapy as a treatment for depression. *J. Consult. Clin. Psychol.* 59:547–57

Joiner TE, Metalsky GI, Katz J, Beach SRH. 1999. Depression and excessive reassurance-seeking. *Psychol. Inq.* 10:269–78

Karney BR. 2001. Depressive symptoms and marital satisfaction in the early years of marriage: narrowing the gap between theory and research. In *Marital and Family Processes in Depression: A Scientific Foundation for Clinical Practice*, ed. SRH Beach, pp. 45–68. Washington, DC: Am. Psychol. Assoc.

Kazdin AE. 2007. Mediators and mechanisms of change in psychotherapy research. *Annu. Rev. Clin. Psychol.* 3:1–27

Kendler KS. 2017. Causal inference in psychiatric epidemiology. *JAMA Psychiatry* 74:561–62

Kendler KS, Gardner CO. 2014. Sex differences in the pathways to major depression: a study of opposite-sex twin pairs. *Am. J. Psychiatry* 17:426–35

Provides a classic discussion of causal inference from correlational analysis in psychological science.

Kenny DA. 1979. *Correlation and Causality*. New York: Wiley

Kessler RC, Berglund P, Demler O, Jin R, Koretz D, et al. 2003. The epidemiology of major depressive disorder: results from the National Comorbidity Survey Replication (NCS-R). *JAMA* 289:3095–105

Kivelä SL, Köngäs-Saviaro P, Laippala P, Pahkala K, Kesti E. 1996. Social and psychosocial factors predicting depression in old age: a longitudinal study. *Int. Psychogeriatr.* 8:635–44

Kotov R, Gamez W, Schmidt F, Watson D. 2010. Linking "big" personality traits to anxiety, depressive, and substance use disorders: a meta-analysis. *Psychol. Bull.* 136:768–821

Kouros CD, Papp LM, Cummings EM. 2008. Interrelations and moderators of longitudinal links between marital satisfaction and depressive symptoms among couples in established relationships. *J. Fam. Psychol.* 22:667–77

Kraemer HC, Stice E, Kazdin A, Offord D, Kupfer D. 2001. How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *Am. J. Psychiatry* 158:848–56

Labrecque JA, Swanson SA. 2017. Target trial emulation: teaching epidemiology and beyond. *Eur. J. Epidemiol.* 32:473–75

Lawlor DA, Harbord RM, Sterne JAC, Timpson N, Davey Smith G. 2008. Mendelian randomization: using genes as instruments for making causal inferences in epidemiology. *Stat. Med.* 27:1133–63

Lawlor DA, Tilling K, Davey Smith G. 2016. Triangulation in aetiological epidemiology. *Int. J. Epidemiol.* 45:1866–86

Leach LS, Butterworth P, Olesen SC, Mackinnon A. 2013. Relationship quality and levels of depression and anxiety in a large population-based survey. *Soc. Psychiatry Psychiatr. Epidemiol.* 48:417–25

Lei MK, Beach SRH. 2020. Can we uncouple neighborhood disadvantage and delinquent behaviors? An experimental test of family resilience guided by the social disorganization theory of delinquent behaviors. *Fam. Process* 59:1801–17

Malouff JM, Thorsteinsson EB, Schutte NS, Bhullar N, Rooke SE. 2010. The Five-Factor Model of personality and relationship satisfaction of intimate partners: a meta-analysis. *J. Res. Pers.* 44:124–27

McGue M, Osler M, Christensen K. 2010. Causal inference and observational research: the utility of twins. *Perspect. Psychol. Sci.* 5:546–56

McLeod JD, Eckberg DA. 1993. Concordance for depressive disorders and marital quality. *J. Marriage Fam.* 55:733–46

McShall JR, Johnson MD. 2015. The association between relationship distress and psychopathology is consistent across racial and ethnic groups. *J. Abnorm. Psychol.* 124:226–31

Miller B, Townsend AL, Ishler KJ. 2004. Change in marital dissatisfaction, health, and depression in older married couples. *J. Ment. Health Aging* 10:65–77

Miller GA, Chapman JP. 2001. Misunderstanding analysis of covariance. *J. Abnorm. Psychol.* 110:40–48

Miller RB, Mason TM, Canlas JM, Wang D, Nelson DA, Hart CH. 2013. Marital satisfaction and depressive symptoms in China. *J. Fam. Psychol.* 27:677–82

Monroe SM, Bromet EJ, Connell MM, Steiner SC. 1986. Social support, life events, and depressive symptoms: a 1-year prospective study. *J. Consult. Clin. Psychol.* 54:424–31

Morgan P, Love HA, Durtschi J, May J. 2018. Dyadic causal sequencing of depressive symptoms and relationship satisfaction in romantic partners across four years. *Am. J. Fam. Ther.* 46:486–504

Nolen-Hoeksema S, Ahrens C. 2002. Age differences and similarities in the correlates of depressive symptoms. *Psychol. Aging* 17:116–24

Osler M, McGue M, Lund R, Christensen K. 2008. Marital status and twins' health and behavior: an analysis of middle-aged Danish twins. *Psychosomat. Med.* 70:482–87

Overbeek G, Vollebergh W, de Graaf R, Scholte R, de Kemp R, Engels R. 2006. Longitudinal associations of marital quality and marital dissolution with the incidence of DSM-III-R disorders. *J. Fam. Psychol.* 20:284–91

Pearl J. 2000. *Causality: Models, Reasoning and Inference*. Cambridge, UK: Univ. Cambridge Press

Peterson-Post KM, Rhoades GK, Stanley SM, Markman HJ. 2014. Perceived criticism and marital adjustment predict depressive symptoms in a community sample. *Behav. Ther.* 45:564–75

Proulx CM, Helms HM, Buehler C. 2007. Marital quality and personal well-being: a meta-analysis. *J. Marriage Fam.* 69:576–93

Rehman US, Gollan J, Mortimer AR. 2008. The marital context of depression: research, limitations, and new directions. *Clin. Psychol. Rev.* 28:179–98

Rodríguez Vega B, Bayón C, Franco B, Cañas F, Graell M, Salvador M. 1993. Parental rearing and intimate relations in women's depression. *Acta Psychiatr. Scand.* 88:193–97

Ross CE, Mirowsky J. 2013. Theory and modeling in the study of intimate relationships and health. *J. Health Soc. Behav.* 54:67–71

Rugulies R. 2002. Depression as a predictor for coronary heart disease: a review and meta-analysis. *Am. J. Prev. Med.* 23:51–61

Salinger JM, Whisman MA, Randall AK, Hilpert P. 2020. Associations between marital discord and depressive symptoms: a cross-cultural analysis. *Fam. Process*. In press. **https://doi.org/10.1111/famp.12563**

Sandberg JG, Yorgason JB, Miller RB, Hill EJ. 2012. Family-to-work spillover in Singapore: marital distress, physical and mental health, and work satisfaction. *Fam. Relat.* 61:1–15

Sbarra DA, Coan JA. 2017. Divorce and health: good data in need of better theory. *Curr. Opin. Psychol.* 13:91–95

Sbarra DA, Emery RE, Beam CR, Ocker BL. 2014. Marital dissolution and major depression in midlife: a propensity score analysis. *Clin. Psychol. Sci.* 2:249–57

Describes the meta-framework of triangulation and its utility in causal inference.

Review paper on the cotwin control or discordant-twin-pairs design and its applications in the study of causal inference.

Meta-analysis providing summary effect sizes on the association between relationship distress and well-being, including depression.

Example of propensity score analysis, examining the association between marital dissolution and depression.

Schneiderman I, Kanat-Maymon Y, Ebstein RP, Feldman R. 2014. Cumulative risk on the oxytocin receptor gene (OXTR) underpins empathic communication difficulties at the first stages of romantic love. *Soc. Cogn. Affect. Neurosci.* 9:1524–29

Selig JP, Preacher KJ. 2009. Mediation models for longitudinal data in developmental research. *Res. Hum. Dev.* 6:144–64

Shrier I, Platt RW. 2008. Reducing bias through directed acyclic graphs. *BMC Med. Res. Methodol.* 8:70

Slavich GM, Thornton T, Torres LD, Monroe SM, Gotlib IH. 2009. Targeted rejection predicts hastened onset of major depression. *J. Soc. Clin. Psychol.* 28:223–43

Smith DA, Breiding MJ, Papp LM. 2012. Depressive moods and marital happiness: within-person synchrony, moderators, and meaning. *J. Fam. Psychol.* 26:338–47

Describes the basic rationale of Mendelian randomization and its utility in causal inference.

Smith GD, Ebrahim S. 2004. Mendelian randomization: prospects, potentials, and limitations. *Int. J. Epidemiol.* 33:30–42

Smock PJ, Schwartz CR. 2020. The demography of families: a review of patterns and change. *J. Marriage Fam.* 82:9–34

Spencer C, Mallory AB, Cafferky BM, Kimmes JG, Beck AR, Stith SM. 2019. Mental health factors and intimate partner violence perpetration and victimization: a meta-analysis. *Psychol. Violence* 9(1):1–17

Spotts EL, Neiderhiser JM, Ganiban J, Reiss D, Lichtenstein P, et al. 2004a. Accounting for depressive symptoms in women: a twin study of associations with interpersonal relationships. *J. Affect. Disord.* 82:101–11

Spotts EL, Neiderhiser JM, Towers H, Hansson K, Lichtenstein P, et al. 2004b. Genetic and environmental influences on marital relationships. *J. Fam. Psychol.* 18:107–19

St John PD, Montgomery PR. 2009. Marital status, partner satisfaction, and depressive symptoms in older men and women. *Can. J. Psychiatry* 54:487–92

Stafford M, McMunn A, Zaninotto P, Nazroo J. 2011. Positive and negative exchanges in social relationships as predictors of depression: evidence from the English Longitudinal Study of Aging. *J. Aging Health* 23:607–28

Styron W. 1992. *Darkness Visible: A Memoir of Madness*. New York: Vintage

Sullivan PF, Neale MC, Kendler KS. 2000. Genetic epidemiology of major depression: review and meta-analysis. *Am. J. Psychiatry* 157:1552–62

Teo AR, Choi H, Valenstein M. 2013. Social relationships and depression: ten-year follow-up from a nationally representative study. *PLOS ONE* 8:e62396

Introduction to propensity score methods to match "exposed" and "unexposed" samples on potential confounds.

Thoemmes FJ, Kim ES. 2011. A systematic review of propensity score methods in the social sciences. *Multivar. Behav. Res.* 46:90–118

Thomas AL, Caughy MO, Anderson LA, Owen MT. 2019. Longitudinal associations between relationship quality and maternal depression among low-income African American and Hispanic mothers. *J. Fam. Psychol.* 33:722–29

Thomas PA. 2016. The impact of relationship-specific support and strain on depressive symptoms across the life course. *J. Aging Health* 28:363–82

Turkheimer E, Harden KP. 2014. Behavior genetic research methods: testing quasi-causal hypotheses using multivariate twin data. In *Handbook of Research Methods in Social and Personality Psychology*, ed. HT Reis, CM Judd, pp. 159–87. New York: Cambridge Univ. Press

Uebelacker LA, Whisman MA. 2006. Moderators of the association between relationship discord and major depression in a national population-based sample. *J. Fam. Psychol.* 20:40–46

UN Women. 2020. *Progress of the world's women 2019–2020: families in a changing world*. Rep., United Nations, New York. **https://www.unwomen.org/en/digital-library/progress-of-the-worlds-women**

Vento PWP-D, Cobb RJ. 2011. Chronic stress as a moderator of the association between depressive symptoms and marital satisfaction. *J. Soc. Clin. Psychol.* 30:905–36

Wade TD, Kendler KS. 2000. The relationship between social support and major depression: cross-sectional, longitudinal, and genetic perspectives. *J. Nerv. Ment. Dis.* 188:251–58

Waller NG, Meehl PE. 1998. *Multivariate Taxometric Procedures: Distinguishing Types from Continua*. Thousand Oaks, CA: Sage

Walum H, Lichtenstein P, Neiderhiser JM, Reiss D, Ganiban JM, et al. 2012. Variation in the oxytocin receptor gene is associated with pair-bonding and social behavior. *Biol. Psychiatry* 71:419–26

Walum H, Westberg L, Henningsson S, Neiderhiser JM, Reiss D, et al. 2008. Genetic variation in the vasopressin receptor 1a gene (*AVPR1A*) associates with pair-bonding behavior in humans. *PNAS* 105:14153–56

Weissman MM. 1987. Advances in psychiatric epidemiology: rates and risks for major depression. *Am. J. Public Health* 77:445–51

Whisman MA. 1999. Marital dissatisfaction and psychiatric disorders: results from the National Comorbidity Survey. *J. Abnorm. Psychol.* 108:701–6

Whisman MA. 2001. The association between depression and marital dissatisfaction. In *Marital and Family Processes in Depression: A Scientific Foundation for Clinical Practice*, ed. SRH Beach, pp. 3–24. Washington, DC: Am. Psychol. Assoc.

Whisman MA. 2007. Marital distress and DSM-IV psychiatric disorders in a population-based national survey. *J. Abnorm. Psychol.* 116:638–43

Whisman MA. 2016. Discovery of a partner affair and major depressive episode in a probability sample of married or cohabiting adults. *Fam. Process* 55:713–23

Whisman MA, Baucom DH. 2012. Intimate relationships and psychopathology. *Clin. Child Fam. Psychol. Rev.* 15:4–13

Whisman MA, Beach SRH. 2012. Couple therapy for depression. *J. Clin. Psychol.* 68:526–35

Whisman MA, Beach SRH, Snyder DK. 2008. Is marital discord taxonic and can taxonic status be assessed reliably? Results from a national, representative sample of married couples. *J. Consult. Clin. Psychol.* 76:745–55

Whisman MA, Bruce ML. 1999. Marital dissatisfaction and incidence of major depressive episode in a community sample. *J. Abnorm. Psychol.* 108:674–78

Whisman MA, Davila J, Goodman SH. 2011. Relationship adjustment, depression, and anxiety during pregnancy and the postpartum period. *J. Fam. Psychol.* 25:375–83

Whisman MA, du Pont A, Rhee SH, Spotts EL, Lichtenstein P, et al. 2018a. A genetically informative analysis of the association between dyadic adjustment, depressive symptoms, and anxiety symptoms. *J. Affect. Disord.* 237:18–26

Whisman MA, Johnson DP, BE D, Li A. 2012. Couple-based interventions for depression. *Couple Fam. Psychol. Res. Pract.* 1:185–98

Whisman MA, Robustelli BL, Beach SR, Snyder DK, Harper JM. 2015. Marital discord and depression in middle-aged and older couples: Is taxon status associated with depression? *J. Soc. Pers. Relationsh.* 32:967–73

Whisman MA, Robustelli BL, Labrecque LT. 2018b. Specificity of the association between marital discord and longitudinal changes in symptoms of depression and generalized anxiety disorder in the Irish Longitudinal Study on Ageing. *Fam. Process* 57:649–61

Whisman MA, Salinger JM, Gilmour AL, Steele BA, Snyder DK. 2021. Love and war: prospective associations between relationship distress and incidence of psychiatric disorders in active-duty Army personnel. *J. Abnorm. Psychol.* 130(1):3–8

Whisman MA, Salinger JM, Labrecque LT, Gilmour AL, Snyder DK. 2020b. Couples in arms: marital distress, psychopathology, and suicidal ideation in active-duty Army personnel. *J. Abnorm. Psychol.* 129:248–55

Whisman MA, Sheldon CT, Goering P. 2000. Psychiatric disorders and dissatisfaction with social relationships: Does type of relationship matter? *J. Abnorm. Psychol.* 109:803–8

Whisman MA, South SC. 2017. Gene–environment interplay in the context of romantic relationships. *Curr. Opin. Psychol.* 13:136–41

Whisman MA, Uebelacker LA. 2009. Prospective associations between marital discord and depressive symptoms in middle-aged and older adults. *Psychol. Aging* 24:184–89

Whisman MA, Uebelacker LA, Tolejko N, Chatav Y, McKelvie M. 2006. Marital discord and well-being in older adults: Is the association confounded by personality? *Psychol. Aging* 21:626–31

Whisman MA, Uebelacker LA, Weinstock LM. 2004. Psychopathology and marital satisfaction: the importance of evaluating both partners. *J. Consult. Clin. Psychol.* 72:830–38

Whitton SW, Stanley SM, Markman HJ, Baucom BR. 2008. Women's weekly relationship functioning and depressive symptoms. *Pers. Relationsh.* 15:533–50

Evidence for the prospective association between relationship distress and major depression in a probability sample.

Whitton SW, Whisman MA. 2010. Relationship satisfaction instability and depression. *J. Fam. Psychol.* 24:791–94

Yu B, Zhang X, Wang C, Sun M, Jin L, Liu X. 2020. Trends in depression among adults in the United States, NHANES 2005–2016. *J. Affect. Disord.* 263:609–20

Zlotnick C, Kohn R, Keitner G, Della Grotta SA. 2000. The relationship between quality of interpersonal relationships and major depressive disorder: findings from the National Comorbidity Survey. *J. Affect. Disord.* 59:205–15