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Divorce and health: good data in need of better theory David A Sbarra¹ and James A Coan²

A very large literature links the experiences of marital separation and divorce to risk for a range of poor distal health outcomes, including early death. What is far less clear, however, is the mechanistic pathways that convey this risk. Several plausible mechanisms are identified in the literature, and the central thesis of this paper is that the empirical study of divorce and health will benefit enormously from a renewed reliance on theory to dictate how these mechanisms of action may unfold over time. This review emphasizes the roles of attachment and social baseline theories in making specific mechanistic predictions and highlights the ways in which these perspectives can contribute new empirical knowledge on risk and resilience following marital dissolution.

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Research linking the experience of marital separation and divorce to distal health outcomes is unequivocal: On average, compared to married adults, people who experience a separation or divorce are at increased risk for a range of negative health outcomes, from hospital-diagnosed infectious disease [1] to cardiovascular morbidities [2], and risk for earth death [3]. Far less clear, however, are the mechanisms that might explain why people who separate from a partner in, say, their 30s or 40s might evidence increased mortality risk 30 years later. This, of course, is a prospective statement, but the cross-sectional comparison also is relevant: At any given time, why do separated and divorced adults evidence worse health than their married counterparts? At their core, questions about mechanisms are questions about theory. What perspective or theoretical account can (a) explain existing data in a useful way, and (b) be used to generate informative predictions going forward? The central thesis of this paper is that in order to more completely understand the mechanisms underpining the divorce-health association, we need timely theoretical advances and new, theory-driven research.

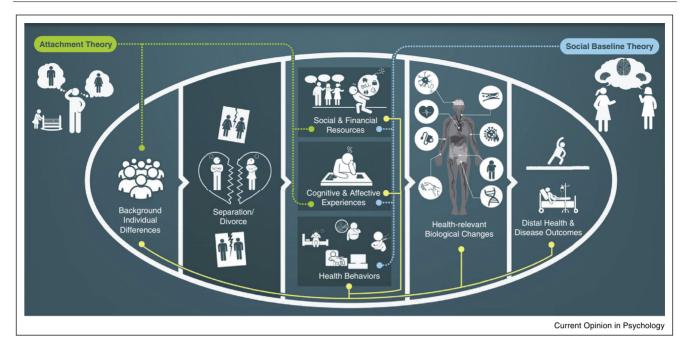
Divorce and health: what do we know?

Divorce is consistently described as one of life's most distressing events [4], and is tied to increased susceptibility to illness [5,6] and other morbidities [1,7,8**], as well as increased risk for early death [3,9°,10]. A growing consensus in the literature on divorce and health is that these aggregate risk statistics mask considerable variability in who fares well or poorly [11**]. This fact suggests that (a) risk for poor outcomes may be limited to a subset of people, and (b) any theoretical models or perspectives seeking to explain the overall patterns of risk must account for individual differences. For example, in a study linking adults' marital biographies to all-cause mortality, Sbarra and Niertart [12] found that, over a 40-year period, adults who reported being married then divorced evidenced the greatest risk for early death; however, when the marital biography was redefined as having ever experienced a divorce (e.g., being divorced, then remarried), there was no elevated risk. In this paper, the risk was limited to a specific group of people, and the authors suggested that the variables that may explain continued singlehood after divorce — for example, personality difficulties, depression, substance use — might also explain the elevated risk among these adults [12]. Similar results come from a recent study of divorce and depression [13**], which found evidence for a diagnosable depressive episode was only observed after divorce if a person had also experienced depression prior to their separation.

Mechanisms of action

In a review on the association between divorce and risk for early mortality, Sbarra et al. [10] outlined a series of mechanistic pathways that may explain how risk processes get transduced into long-term health risk [14]. Figure 1 provides a visualization of the mechanistic pathways from marital separation and divorce to long-term health outcomes; within the circle, reading from the left to the right, we illustrate the specific psychosocial behaviors that may explain the distal outcomes of interest; outside the circle (on the upper left and right) we illustrate how extant theories can inform the understanding of these mechanisms. Focusing first on the mechanisms, we highlight a few points that are important to consider as this line of research grows. First, on the far left within the circle, we emphasize and incorporate a well-known finding in this literature: individual differences predict the likelihood of divorce [13**], and many of these same variables are associated with biological changes that predict disease outcomes. For example, hostility and neuroticism predict

Figure 1



An illustration of the varied mechanistic pathways linking marital separation and divorce with distal health outcomes. Within the circle, reading from left to right, the model posits that background individual differences (e.g., hostility) predict the future likelihood of divorce, as well as each step the in the pathways toward distal outcomes, which appear on the right side of the model. Following a separation and/or divorce, the model also posits that responses in three major domains (social and financial resources; cognitive and affective experiences, and health behaviors) operate together shape health relevant biological responding, which, in turn, predict health-relevant biological changes and later health outcomes. For illustrative purposes, attachment and social baseline theories are included outside the circle with connections to specific links in the mechanistic chain of action. For example, attachment theory can be used to make specific predictions about background individual differences that may increase risk for marital dissolution, as well as the specific cognitive/affective responses people have to their breakup.

rapid declines in early marital satisfaction as well as separation and divorce 5 years later [15]; independent of divorce, these variables also predict each step in the putative change from marital separation to important health outcomes [16-18]. For these reasons, in the context of prospective correlational studies, all efforts to understand the predictors of health outcomes after a separation must account for variables that also predict the end of marriage. We illustrate this point by connecting the background individual difference variables in our figure to each step in the mechanistic chain from the left to the outcomes in the right side of the circle.

Following a separation or divorce, it is useful to think about the mechanisms that can predict poor health across three different dimensions: social and financial resources, cognitive and affective experiences, and health behaviors. A key issue in this literature is distinguishing between responses that are unique to a marital separation/divorce and those that are common to stressful life events in general. For example, in the affective domain, adults high in attachment anxiety who speak about their separation in an enmeshed, over-involved manner demonstrate a high degree of blood pressure reactivity when they are asked to

think about their former partner and separation experience [19]. On face value, this study is about attachment anxiety and blood pressure reactivity after a marital separation; upon deeper consideration, however, this type of paradigm probably speaks to the more general association between anxiety-related hyperactivation [20] and cardiovascular responding than it does to the specific mechanisms of adaptation to divorce. In this way, marital separation/divorce is an ideal model system for understanding how adults respond to and cope with interpersonal stress more generally [14].

Finally, when it comes to understanding the mechanisms linking separation/divorce to distal health, it is critical to study health-relevant biological changes or intermediaries [21], and to build transduction pathways from relationship upheavals to disease outcomes. Miller et al. [21] argue that it is important to reverse engineer disease pathways by considering the relevant pathophysiology, then working backward from a disease endpoint to life stress or a specific psychological experience. For example, cardiovascular disease morbidity and mortality is highly associated with atherosclerosis, and multiple biological changes can contribute to the development of arterial

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