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Good theories in need of better data

Good Theories in Need of Better Data: Combining Clinical and Social Psychological

Approaches to Study the Mechanisms Linking Relationships and Health

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Abstract

The study of intimate relationships and health is a fast-growing discipline with numerous well-developed theories, many of which outline specific interpersonal behaviors and psychological pathways that may give rise to good or poor health. The central argument of this review paper is that the study of relationships and health can move toward interrogating these mechanisms with greater precision and detail, but doing so will require a shift in the nature of commonly used research methods in this area. Accordingly, our review draws heavily on recent work on the science of behavior change and discusses six key methodologies that may galvanize the mechanistic study of relationships and health: dismantling studies, factorial studies, experimental therapeutics, experimental mediation research, multiple assessments, and recursive modelling. We provide empirical examples for each strategy and outline new ways in which a given approach may be used to study the mechanisms linking intimate relationships and health. The paper concludes with a discussion of the key challenges and limitations for using these research strategies as well as novel ideas about how to integrate this work into existing paradigms within the field.

Keywords: intimate relationships, health, mechanisms, mediation, indirect effects, social psychology, clinical psychology, intervention science

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Approaches to Study the Mechanisms Linking Relationships and Health

The quantity and quality of our intimate relationships are associated with a broad range of health outcomes, including immunological functioning (Kiecolt-Glaser, 2018), cardiovascular disease (Kiecolt-Glaser, Gouin, & Hantsoo, 2010) , cancer (Kroenke, Kubzansky, Schernhammer, Holmes, & Kawachi, 2006; Trudel-Fitzgerald et al., 2019), and even mortality risk (Holt-Lunstad, Smith, & Layton, 2010; Holt-Lunstad, Smith, Baker, Harris, & Stephenson, 2015; Stanton, Selcuk, Farrell, Slatcher, & Ong, 2019). Indeed, intimate relationships are increasingly recognized as a public health priority akin in magnitude and scope to other social determinants of health (Holt-Lunstad, Robles, & Sbarra, 2017). As researchers interrogate the pathways between relationship quality[1] and health, a common finding is that relationship processes do not often predict health *directly*; rather, elements of relationships are most frequently associated with health *indirectly* through a series of intermediate mechanisms. These putative mechanisms include, for example, health behaviors, chronic stress, and access to social resources (Burman & Margolin, 1992; Farrell, Imami, Stanton, & Slatcher, 2018; Kiecolt-Glaser & Newton, 2001; Pietromonaco & Collins, 2017; Robles, Slatcher, Trombello, & McGinn, 2014).

Understanding the pathways linking intimate relationships and distal health outcomes, as well as the mechanisms underlying these pathways, is vital to designing

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[1] We recognize that there are many different ways to characterize and measure marital and relationship quality, and debates about the best way to do so date back over 30 years (Fincham & Bradbury, 1987). In many ways, differences in measurement hinder the study of mechanisms. With no agreed-upon, "gold standard" measurement of relationship quality, the critical predictor variable discussed throughout this paper often varies between the different studies we describe. In general, we are agnostic here about best practices for measuring marital and relationship quality, but we recognize from the outset that advances in measurement and assessment in this area will be critical to advancing the mechanistic study of relationships and health.

effective interventions. Thus, theorizing and testing potential mechanisms has become a key interest for many social, health, and clinical psychologists. Yet, when it comes to studying these potential mechanisms, we feel similarly to Mark Twain who, in paraphrasing his colleague's thoughts on the weather in New England, remarked how often it was discussed but that "no one seemed to do anything about it" (Johnson, 1923, p. 322). Changing how we study psychosocial mechanisms is hardly as immutable as changing the weather in New England, but perhaps one reason we are better at theorizing about the mechanisms than we are about studying said mechanisms is that key methodological advances (which allow us to do so) emerge in the literature quite slowly. We have a surplus of theoretical models that propose mechanistic pathways between relationships and health, but very little data that adequately fulfil all the criteria necessary to truly warrant identification of a specific behavior or psychosocial experience as a *mechanism of action*. To be sure, the field is ripe with experimental studies showing that variation in the ways people think about and behave in relationships are associated with health-relevant outcomes (Bourassa, Ruiz, & Sbarra, 2019; Smith, Ruiz, & Uchino, 2004). This said, experimental effects are necessary but not sufficient to identify health-relevant mechanisms of action. The goals of this review are to discuss commonalities across the existing theoretical accounts of the mechanisms explaining associations between intimate relationships and health outcomes; highlight the evidence that is needed for a deeper understanding of these potential mechanisms; and illustrate how distinct methodologies in the fields of social and clinical psychology can inform each other to propel this body of knowledge forward and help design targeted interventions.

We begin by providing a review of theoretical models that specify potential mechanistic pathways between intimate relationships and health outcomes. In this

analysis, we distill common themes among extant theoretical models. The focus of our analysis is on adult intimate relationships (i.e., romantic pair bonds; marriage or marriage-like relationships), including relationship functioning (e.g., high vs. low relationship quality) and status (e.g., whether people are married, separated/divorced, or widowed). Intimate relationships represent a subset of all close relationships, but may be particularly potent in the health domain. Worldwide, nearly all people form intimate relationships at some point in adulthood (Copen et al., 2012). Despite the ubiquity of intimate relationships, however, there is quite a range in the extent to which people maintain stable pair bonds. Nearly 40% of all first marriages end in divorce (Smock & Schwartz, 2020) and, based on taxometric analyses, approximately 3 out of 10 marriages can be described as highly discordant—so much so that this discordance represents a true taxon, one that is different in kind rather than quantity (Whisman, Beach, & Snyder, 2008). Although we limit our analysis to intimate relationships, we draw on relevant neighboring literatures to make critical points, including the study of loneliness/isolation, the social support literature, and both parent-child and caregiving literatures. We also describe and reference work that may provide a good illustration of the ways the relationships and health literature can import novel advances that are happening in other parts of the field. In this sense, although this paper is ultimately aimed at advancing the study of intimate relationships and health, we are hopeful our review can be informative to other areas within psychological science that undoubtedly struggle with many of the same challenges.

After discussing relevant theories, we turn to the types of evidence necessary for distinguishing true mechanisms from derivative mediators, and we describe the current state of empirical evidence for some of the mechanisms proposed to underlie relationship-health pathways. The literature defining mechanisms of action and

distinguishing mechanisms from mediators, indirect effects, and proxy variables is well developed (Kraemer et al., 2001), and we are not making novel claims about how biopsychosocial mechanisms operate to connect relationships and health. Our contribution rests in understanding how the basic elements of this literature can be used to galvanize the empirical study of intimate relationships and health. To do so, the bulk of the paper discusses a variety of robust methodologies used in social and clinical psychology, and we evaluate the strengths and weaknesses of each for providing evidence for causal mechanisms and explaining how these methods can (and likely should) be combined to more completely understand mechanistic puzzles in the area of relationships and health. Throughout, we discuss the implications of robust mechanistic research for intervention development.

Review of Extant Theory on Mechanisms Linking Relationships and Health

As our title implies, the key proposition of this paper is that when it comes to the mechanistic study of intimate relationships and health, we have more theories than data. Although the existing theories are distinct, there exists quite a degree of overlap as well. In many ways, the review of these theories is a critical set-up for exploring new empirical ways of testing mechanisms in the relationships-health domain. To begin with this end in mind, our assessment of the literature in this area is relatively straightforward: The study of intimate relationships and health will benefit most from a deeper empirical focus on mechanisms of action. Theoretical advances are certainly important, and we are *not* calling for a moratorium on theory but instead a reinvigorated empirical focus on the ways in which the putative mechanistic effects are conveyed. In other words, we argue that, with the advent of rigorous methods and interdisciplinary initiatives, researchers are now in an exciting position to empirically

test the various theoretical models specifying links between intimate relationships and health.

The existing theoretical writing on relationships and health maps directly onto the sub-topics within the field. We have unique—but definitively interrelated—mechanistic models of the ways in which marriage and relationship quality (Burman & Margolin, 1992; Kiecolt-Glaser & Newton, 2001), social integration, isolation, and loneliness (Cacioppo & Patrick, 2008), social support and stress buffering (Uchino, 2009), partner responsiveness (Stanton, Slatcher, & Reis, 2019), social networks (Smith & Christakis, 2008), social evaluative threat (Dickerson, Gruenewald, & Kemeny, 2004), social ambivalence (Holt-Lunstad & Uchino, 2019), and both divorce (Sbarra, Law, & Portley, 2011) and bereavement (Shor et al., 2012) may shape health-relevant psychology, behavior, and physiology, all of which are related to disease incidence, progression, and endpoint outcomes (also see Farrell & Simpson, 2017; Slatcher & Selcuk, 2017).

One broad question in this literature is whether intimate relationships exert health-*protective* effects (via positive relationship processes such as support and physical intimacy), or if outcomes are driven largely by the health-*damaging* effects of lacking intimate relationships or experiencing low-quality relationships (via negative relationship processes such as conflict or hostility). Evidence exists for both pathways (Farrell & Simpson, 2017). For example, perceived social support, partner responsiveness, and social networks of close others may buffer against stress in adulthood (Cohen & Janicki-Deverts, 2009; Manvelian & Sbarra, 2020; Slatcher, Selcuk, & Ong, 2015), which is clearly health-protective. At the same time, marital separation increases risk for smoking behavior (Bourassa, Ruiz, & Sbarra, 2019), which is clearly health-damaging, and loneliness is associated with increased pro-inflammatory gene

expression motifs (Slavich & Cole, 2013). As these examples illustrate, there are distinct ways in which relationship resources may contribute positively to health whereas social stress, hostility, and relationship discord may contribute negatively. Furthermore, changes in relationship functioning in either direction may also make unique contributions to health; for example, Stanton and colleagues (2019) found that decreases in partner responsiveness over a 10-year period predicted mortality rates above and beyond mean levels of responsiveness. In the remainder of this section, we review three cross-cutting psychosocial and behavioral pathways that are believed to link intimate relationship quality/status with distal health: health behaviors, affect, and cognition. Nearly all the existing theories in this area point to the critical roles of these constructs as potential mechanistic engines linking intimate relationships and health.

Health Behaviors as Mechanisms

At the broadest level, any behavior that alters health-relevant physiology is a *health* behavior. Behaviors overtly related to health enhancement (e.g., exercise, diet) and health impairment (e.g., drug and alcohol use) are often the primary behavioral mediators in models linking relationships and health. Behavior plays a critical role—if not *the* critical role—in shaping disease incidence and outcomes, with empirical estimates suggesting that roughly 40% of all deaths in the United States are attributable to *modifiable* health behaviors (McGinnis, Williams-Russo, & Knickman, 2002). Within a mediational framework, the central questions of interest for this paper are largely about the ways in which intimate relationship quality or status may organize, shape, constrain, or drive health-relevant behaviors (Skoyen, Blank, Corkery, & Butler, 2013; Umberson, Crosnoe, & Reczek, 2010; Umberson, Williams, Powers, Liu, & Needham, 2006). For example, in a large sample from the Study of Women's Health Across the Nation (SWAN) cohort, marital happiness is associated with fewer sleep disturbances

(Troxel, Buysse, Hall, & Matthews, 2009), and work using the Midlife in the United States (MIDUS) dataset shows perceived partner responsiveness is associated with better sleep (Selcuk et al., 2017). Similarly, poor sleep in one member of a couple is associated with a lower ratio of positive to negative affect in a laboratory conflict task (Gordon & Chen, 2014). In other domains, marital separation and divorce appear to increase risk for smoking, especially relapse among prior smokers (Bourassa et al., 2019). Perceived support from one's partner for exercise and healthy eating is associated with lower weight, and spousal interactions involving influence, regulation, and constraint of health practices that encourage engagement in a healthy lifestyle are associated with better health practices and more health-enhancing behaviors (Skoyen, Kogan, Novak, & Butler, 2013).

There are multiple ways in which close relationships may affect health behaviors. First, relationship partners shape the way we think and feel about different health behaviors. The social control of health behaviors refers to efforts by one person to directly regulate the behavior of another person (by telling, reminding, or threatening another person in order to effect a health behavior change) or to indirectly influence that person through feeling of obligation and responsibility to others (Tucker, 2002; Umberson, 1992). In an extensive review of this topic, Umberson et al. (2010) discussed a lifecourse perspective on health behaviors and outlined a model in which many health habits are established in childhood, largely from parental influences. These developed health behaviors are then heavily affected by peers and social norms in adolescence, organized by relationship partners within a marriage, and finally change once again for older adults, who are more likely to lose these powerful social forces in later life (Tucker, Klein, & Elliott, 2004).

Second, low-quality relationships can deplete the self-regulatory resources needed to engage in more taxing health-enhancing behaviors and avoid pleasurable but harmful behaviors. Committing to goals for changing health behaviors, creating plans for achieving those goals, and avoiding distractions and obstacles all require ample self-regulatory resources (Mann, De Ridder, & Fujita, 2013), and low-quality relationships characterized by high levels of conflict and hostility use up self-regulatory resources (Smith et al., 2011). However, higher-quality relationships can allow for greater resources to be shared across partners to achieve health behaviour-related goals (Fitzsimons, Finkel, & Vandellen, 2015; vanDellen, Beam, & Fitzsimons, 2018).

It is beyond the scope of this review to cover all conceivable ways in which intimate relationship quality and status are associated with health behaviors; nevertheless, even our brief review highlights the breadth of this literature—health behaviors unfold in a social context and are strongly associated with relationship quality and status.

Affective Processes as Mechanisms

It is now widely recognized that affect—the valenced experience of whether something is good or bad and the general term used for a variety of emotion-relevant concepts—plays a direct role in shaping critical health outcomes (DeSteno, Gross, & Kubzansky, 2013; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). The experience of psychological stress itself, including concomitant physiological changes involving the autonomic and neuroendocrine systems, is believed to be an affective process (DeSteno et al., 2013). Recently, Farrell et al. (2018) reviewed the literature suggesting emotions experienced and expressed, emotion regulation strategy use, and affective reactivity to stress all serve as mediators linking intimate relationship functioning and health. Similarly, Sbarra and Coan (2018) posited that what appear to be direct effects from

relationships to health and health-relevant physiology may be better understood as effects that occur *via* affective responding; in this way, intimate relationships provide a social context that organizes many of our affective experiences.

Psychological distress, for example, is unambiguously linked to a range of negative health outcomes (Segerstrom & Miller, 2004), and in many instances relational conflict contributes directly to more exaggerated cardiovascular reactivity. For instance, greater hostility during marital interactions is associated with higher blood pressure and heart rate (Smith, Glazer, Ruiz, & Gallo, 2004; Smith, Ruiz, & Uchino, 2004). Moreover, vigilance to threat is a negatively-valenced affective state of high arousal, and evidence suggests that vigilance for threat is associated with exaggerated cardiovascular responses in social situations (Ruiz et al., 2017). Social vigilance is proposed to be a key mediator of links between attachment anxiety and health—it is believed that anxiously attached individuals are constantly monitoring their partners' emotions and reactions, and this high state of arousal is one that contributes negatively to health (Jaremka et al., 2013; Stanton & Campbell, 2014); we return to this topic later in the paper when discussing experimental mediation approaches for identify mechanisms of action. In other domains, exaggerated emotional responding is also associated with health outcomes. For example, using data from the national MIDUS study, Stanton et al. (2019) recently reported that negative affect reactivity to daily stressors mediated the association between change in perceived partner responsiveness in marriage or marriage-like relationships and mortality across 20 years.

Affect and emotion regulation—that is, what people do to manage their emotional experiences—are centrally related to health as well (DeSteno, Gross, & Kubzansky, 2013). Perhaps the best example from the intimate relationships literature

centers on attachment insecurity. Fundamentally, individual differences in attachment anxiety and avoidance involve relative predispositions to engage in specific emotion regulatory strategies (Mikulincer & Shaver, 2019). People high in attachment anxiety tend to favour other-oriented *hyperactivating* strategies, defined as exaggerated social responses to relational threat that often involves repetitive efforts to engage the threatening content (e.g., romantic pursuit in the context of jealousy). In contrast, people high in attachment avoidance engage in self-reliant *deactivating* strategies in which they minimize or suppress their emotional experiences. A growing literature now links both of these processes to health-relevant outcomes (Ehrlich, 2019; Stanton & Campbell, 2014), and Pietromonaco and Beck (2019) recently outlined a dyadic model of attachment and health in which each person's reactions and responses relate to and drive those of their partner. This idea is consistent with prior theorizing in the marriage and health literature—one person's disposition is their partner's social context (Ruiz, Hamann, Coyne, & Compare, 2006).

Cognitive Processes as Mechanisms

In reality, it is often difficult to separate affective and cognitive processes. For example, situational appraisals (a cognitive process) may drive psychological stress (an affective experience), but the experience of stress itself shapes cognitive evaluation of the social world (Neff & Karney, 2004). Furthermore, relative to affective responses and health behaviors, it is often difficult to determine whether cognitive processes are themselves the key predictors of outcomes or the intervening variables that explain the outcome. Put in the language of statistical mediation, are cognitive processes the X variables (e.g., perceived social support, perceived partner responsiveness, marital attributions, or loneliness), or the mediating M variables that follow from specific relational circumstances (e.g., depression or anxiety symptoms)? This distinction

ultimately matters for the study of mechanistic effects. As we discuss later, if an experiment or intervention intends to target a specific mechanism (to determine if altering that variable alters a health-relevant outcome), we need a very clear idea about which mechanisms to target. In other words, which variables may ultimately exert a direct effect on key outcomes?

Some prospective longitudinal studies point to the role of cognitive factors as mediators of pathways between relationship experiences and health outcomes. Farrell and colleagues (2019) tested the mediating role of two different aspects of the Adult Attachment Interview in explaining links between observations of maternal sensitivity in the first few years of life and cardiometabolic risk in middle adulthood: *Secure base script knowledge*, which assesses the extent to which individuals seek and expect effective support from attachment figures during stressful situations, and *coherence of mind*, which reflects the ability to produce a consistent, open, and detailed narrative and is believed to reflect attentional strategies implemented during distress. They found that secure base script knowledge, but not coherence of mind, partially mediated paths between maternal sensitivity in infancy and cardiometabolic risk in adulthood. This suggests that the awareness and engagement of cognitive scripts for successful support seeking may be one way in which previous relationship experiences translate into long-term health. Cognition is the least-studied category of mediating variables in the relationships and health literature, but findings such as these suggest a cognitive approach may be a fruitful direction for future research.

All Mechanisms can be Moderated

Within the broad relationship-health literature, many of the existing mechanistic models and theories focus not only on the intervening processes, but also on the ways in which these processes may be moderated by sociodemographics or individual

differences across a range of psychological domains. For instance, in their model linking marital quality to health, Robles and colleagues (2014) are clear in noting that gender and individual difference variables may differentially impact the processes of interest. Based on a thorough meta-analysis, however, these authors also noted that empirical evidence for their proposed moderators (i.e., gender and age) is quite limited. In the study of marital status and health, Sbarra and colleagues (2015) suggest the association between divorce and health is likely moderated by individual differences that contribute to the likelihood of becoming over-involved in one's psychological experiences (cf. Kross & Ayduk, 2011). For example, self-reported attachment anxiety, conceptualized at the trait-like tendency to engage in maladaptive emotion regulation is associated with a stronger association between linguistic markers of emotional overinvolvement and blood pressure reactivity following a marital separation (Lee, Sbarra, Mason, & Law, 2011). This study highlights how a specific mediational process (hypothesized to be associated with poor health outcomes following marital separation) may be moderated by individual differences.

Beyond gender or individual difference variables, there is increasing awareness that stressful environmental contexts may also impact relationship dynamics, especially the stress conferred by lower socioeconomic standing (Neff & Karney, 2017; Randall & Bodenmann, 2009). The normative developmental course of most marriages is one of relational decline, but considerable evidence indicates that this decline accelerates among people who are economically disadvantaged (Neff & Karney, 2017). Economic disadvantage is a broad term that can encompass multiple stressors, include unemployment, neighborhood risks, inconsistent transportation, and limited network support, which presumably affects reserve capacity to respond to these stressors (Gallo, de los Monteros, & Shivpuri, 2009). Neff and Karney (2017) have suggest two routes

through which these external stressors may impact relationship satisfaction—via the creation of additional marital problems (e.g., conflict over escalating debt) and as hindrances to constructive problem solving (e.g., multiple job requirements make solving childcare problems increasingly difficult). As far as we know, no studies to date have used this framework within the relationship-health literature, but it is clear that external stressors alter relationship processes in a manner than may have a direct impact on the mediational processes discussed above (Lavner & Bradbury, 2017). Central to this issue is the need to collect diverse and representative samples in all corners of the discipline (Rad, Martingano, & Ginges, 2018). As with other sub-fields (e.g., developmental psychology—see Nielsen, Haun, Kärtner, & Legare, 2017), there is little doubt that the study of intimate relationships and health suffers from considerable sampling bias; all endeavors to study mechanisms should be built on efforts to collect more diverse and representative samples across the spectrum of relationships types, gender representation, socioeconomic status, ethnicity, and race.

Biological Intermediaries Link Mechanistic Variables to Health

Many of the broad, "pathway models" in the relationships-health literature focus on the biological mechanisms that may give rise to distal physical health outcomes (e.g., Robles et al., 2014; Slatcher & Selcuk, 2017). The basic idea underpinning these models is that some relational circumstance or experience alters how people think, feel, and/or behave, and these changes have physiological correlates, including changes in endocrine, autonomic, and immune system functioning (Kiecolt-Glaser, 2018). Sustained dysregulation of multiple physical systems can result in allostatic load (McEwen, 1998), believed to reflect sustained wear-and-tear on the body, and the basic conception here is that maintained physiological dysregulation of these systems poses a direct risk for long-term health outcomes. This pathway perspective is consistent with

classic models in health psychology (Miller, Chen, & Cole, 2009), which argue for the need to study *biologically plausible intermediaries* that ultimately link psychosocial experiences with endpoint physical health.

There are two important corollaries related to this point. First, risks accumulate over time (see Kuh et al., 2003) and the accumulation of health-damaging effects may take decades to alter disease incidence. Thus, it is critical that any study of these intermediaries at least attempts to speak to long-term health risks in a manner that is consistent with the slow accumulation of effects. Second, most studies on relationships and health represent only a snapshot into this larger window of accumulation. The intensity and the timing of the risk exposure or stress buffering are hypothesized to represent a process that would be health-damaging or health-protective if maintained over time. Consider, for example, month-long daily study of stressful interactions, ratings of perceived partner responsiveness, and ambulatory blood pressure; this study observes that greater perceptions of responsiveness across daily stressful interactions are associated with reduced ambulatory blood pressure reactivity. Are these effects health-relevant? Many papers in the literature gloss over the fact that a study like this provides only snapshot from a lifetime of social interactions. As our mechanistic studies advance, these points must become front-and-center in the literature. We encourage scientists working in this area to make explicit the assumptions in their methodology; for example, for responsiveness to be considered health-protective, we may need to assume that couples who report greater responsiveness over the course of many years also experience less chronic stress and, by extension, potentially less blood pressure reactivity.

As we consider the relevance of relationship processes to clinical endpoints, it is also important to reverse engineer the pathways that may link disease incidence,

progression, and morbidity to social risk factors *via changes* in basic disease pathogenesis, gene expression and inflammation, and autonomic nervous system (ANS) and neuroendocrine activity. In the relationships and health domain, we have a growing literature on ANS, neuroendocrine, and immune outcomes, but it will be increasingly important to connect functioning in these systems to clinical endpoints. It is no longer enough to demonstrate, say, that marital quality is associated with blood pressure reactivity during emotionally sensitive conversations; rather, the field needs to show that this reactivity does, in fact, explain the association between marital quality and a more distal health mediator (e.g., intermedial thickness of the coronary artery) *en route* toward risk for clinical dysfunction (e.g., coronary heart disease diagnosis).

Studying health intermediaries that have a causal connection with distal health (i.e., distinguishing between risk markers and causal risk factors) is equally important to advance in this area. A classic example in the biomedical literature is the study of C-reactive protein (CRP), a marker of systemic inflammation that was long-believed to play a unique causal role in the development of atherosclerosis. However, recent studies have established CRP as a risk marker rather than as a causal agent in the development of cardiovascular disease (see Pingault et al., 2018). To the extent that the relationship and health literature has relied on CRP as a critical health intermediary, some of the putative pathways toward disease endpoints may need deeper consideration.

The Mechanisms of Action are Reciprocal

A final point about the broad theoretical literature on relationships and health is that the putative mechanisms of action are likely reciprocal and highly interdependent (Smith & Weihs, 2019). We see recursive cycles between behaviors like sleep and hostility between partners, with poor sleep quality predicting more negative partner interactions the next day, which, in turn, predict worse sleep the following night (Hasler

& Troxel, 2010). Psychological and behavioral processes also affect one another. Emotional processes, for instance, are linked to eating behavior in several different ways, including eating to cope with negative affect or suppressing food intake after intense emotional experiences (Macht, 2008). However, for those who develop disordered eating problems, negative affect tends to increase further after binging episodes (Haedt-Matt & Keel, 2011). There are even recursive cycles between psychological and health-relevant biological processes; for example, depression and stress promote pathogenic gut bacteria survival and replication, and these bacteria can also affect vagus nerve and neurotransmitter responsivity to influence stress reactivity and mood (Madison & Kiecolt-Glaser, 2019).

These illustrations are broadly consistent with Butler's (2011) model of Temporal Interpersonal Emotional Systems (TIES) in which the time-based organization of emotional experience in one person is directly connected to the emotional experience of another person, and the interpersonal nature of these emotional processes can be understood as a dynamic system (Sbarra & Coan, 2018). As we discuss later, the conceptual promise of understanding relationships and health in terms of reciprocal or recursive systems awaits realization, perhaps largely, we contend, because the field has not yet embraced statistical methods that are well suited to capturing these processes (Butler & Barnard, 2019). Moreover, to the extent that these approaches are computationally demanding, the field will benefit enormously through enhanced multidisciplinary collaborations with computer scientists, engineers, biologists, and ecologists.

Summary of Extant Theories

Existing theoretical models highlight that the pathways between intimate relationships and physical health are most often explained by a relatively common set of

underlying mechanisms. Although individual theoretical models make unique contributions to our understanding of relationships and health, our review of the literature has distilled several common themes and arguments across different models. First, many theoretical models propose that the psychosocial mechanisms underlying relationship-health associations fall into the broad categories of health behaviors (e.g., eating patterns), affective processes (e.g., emotion regulation), and cognitive processes (e.g., mental representations of relationships). Second, the vast majority of theoretical models of relationships and health include at least one of the following tenets: (a) Any given mechanism explaining a relationship-health link can be moderated by person- and situation-level variables (e.g., gender, individual differences, socioeconomic status); (b) Psychosocial mechanisms themselves are linked to health outcomes via biological intermediaries (e.g., cortisol, immunological markers); and (c) The pathways between predictor, mechanism, and outcome variables are reciprocal and interdependent.

Searching for Mechanisms: The State of the Science

Given the many models outlining the mechanistic processes linking intimate relationships to physical health outcomes, we might expect many of the key tenets to be supported by data as well. In reviewing the existing literature, we see several constructs frequently arising in empirical work as mediators, particularly affective processes like emotional expression and regulation (Farrell et al., 2018; Sbarra & Coan, 2018), attachment orientations, sleep, and substance use (Farrell & Stanton, 2019). However, a mediating variable and an underlying mechanism are not necessarily the same, and the terms should not be used interchangeably. Is the evidence strong enough to consider mediating variables like affective processes and sleep fully established mechanisms?

Establishing a causal mechanism can be a challenging task. According to Kazdin (2007, 2014), there are five[2] criteria that must be fulfilled for a mediating variable to be a true mechanism: Plausibility, strong associations, consistency, experimental manipulation, and timeline (see Table 1). In a recent review, Farrell and Stanton (2019) applied these criteria to four established mediators (affective processes, attachment orientations, sleep quality, and substance use) in the relationships-health literature. The results were humbling. Although all four potential mechanisms met Kazdin's plausibility and strong associations criteria, only a few met the consistency criterion (affective processes, attachment orientations, and sleep quality), and none met the experimental manipulation and timeline criteria. In this sense, given that we currently lack fully convincing evidence for the variables that should be targeted as mechanisms, it may be premature to design relationship-level interventions to improve health outcomes (cf. Cacioppo, Grippo, London, Goossens, & Cacioppo, 2015; Cohen & Janicki-Deverts, 2009).

One illustration of the ways in which well-intentioned interventions may fall short comes from the Enhancing Recovery in Coronary Heart Disease (ENRICHD) study, which was a large-scale (N = 2,481) randomized control trial designed to test the effectiveness of cognitive-behavioral treatment on reducing early mortality after a myocardial infraction (MI) via two common comorbid issues, high depression and low social support (ENRICHD Investigators, 2001). Drawing upon literature showing low social support was associated with morbidity and mortality in heart disease patients, the researchers included modules designed to improve behavioral skills related to building social connection and seeking support, reduce cognitive biases that contributed

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[2] Kazdin (2007, 2014) also proposed dose-dependent response gradient for the mechanism's effect on the outcome as a non-essential criterion, and specificity of a single mechanism for a given intervention-outcome link which we think is unlikely to hold in the case of relationship-health pathways that are probably multiply determined.

to the perception and maintenance of unsatisfying social support, and encouraging network development through social outreach (ENRICHD Investigators, 2001).

Unfortunately, the results of this randomized control trial were underwhelming. Although the intervention did modestly increase perceptions of social support and reduce depression symptoms, there was no difference in event-free survival rates after 6 months between intervention and control participants. The lack of results may have been in part due to notable improvements in depression and social support in patients in the control group who received no therapy (ENRICHD Investigators, 2003). Although the authors could not conclude why their intervention was only slightly more effective than the control, many of their suggested explanations dealt with mechanistic pathways, including medications for depression or heart disease also targeting the biological pathways that link depression and social support to cardiovascular disease; the intervention not having a large enough effect on social support to lead to biological changes; and the duration and timing of the intervention not including key causal windows. A larger empirical base that includes evidence for all five mechanistic criteria may have allowed the ENRICHD researchers to design a more effective intervention, or at least a more effective critical test. The field needs to move from relying on broad-based epidemiological findings to inform interventions (e.g., low social support is associated with post-MI complications; therefore, treatments should improve social support) to considering which *mechanistic levers of change* have the greatest likelihood of exerting a causal effect on target outcome. The work of the ENRICHD trial is almost 30 years old at this point; our illustration is not intended to slight the efforts of this important work, but instead emphasize that the field should learn from past efforts and aspire to build future intervention studies from more firmly established mechanistic work. Much of the work required to do so, however, remains to be completed.

Combining Social and Clinical Psychological Methods to Study Mechanisms of

Action

Determining how to gather the evidence needed to establish causal mechanisms and to separate them from derivative mediators is complicated. Many different applied fields wrestle with the issues of studying mechanisms. For example, the mechanisms underlying the effectiveness of psychotherapy in improving mental health are notoriously difficult to identify. By the late 20th and early 21st centuries, there was ample evidence that behavioral interventions for several clinical disorders were highly effective (Kazdin, 2007). However, there were also several concerns stemming from researchers and therapists not understanding *why* these interventions were effective. First, there were major gaps in effectiveness and efficiency. Some therapies were shown to be highly effective in controlled research settings, but when disseminated to community practitioners, proved too complicated to be carried out with fidelity (Onken, Carroll, Shoham, Cuthbert, & Riddle, 2014). Without convincing insights into the mechanisms and key ingredients of these complicated interventions, it was difficult to streamline them effectively (Kazdin, 2001). Second, for each disorder, there were a plethora of different interventions that were difficult to group and organize. Identifying *common mechanistic pathways* would allow for the grouping and organization of these interventions, making it easier to draw comparisons and broad conclusions. Some potential mechanisms were proposed, including change in cognitive tendencies following cognitive therapy and a strong therapeutic alliance between patient and therapist, but, as Kazdin (2007) discusses, the evidence for these mechanisms was weak and the theoretical basis for these and other commonly proposed mechanisms was lacking.

In wrestling with these issues, clinical research and intervention science have developed several methodological tools for identifying mechanisms of action, many of which are now organized under the National Institute of Health's (NIH) initiative on the Science of Behavior Change (SOBC; Haedt-Matt & Keel, 2011). The goals of the SOBC initiative (see: https://scienceofbehaviorchange.org/) center on identifying the "active ingredients"—the how and why—of successful behavior change. In the context of intimate relationships and health, we know that relationship *quality* (vs. *quantity*) is a key correlate of health outcomes (Holt-Lunstad et al., 2010; Kiecolt-Glaser & Newton, 2001; Robles et al., 2014). Nonetheless, experimentally manipulating "relationship quality" as a global construct would be quite arduous, and must therefore relies on a series of more basic SOBC-style questions: Which element(s) of relationship quality should be enhanced, and which should be diminished? Are some elements of relationship quality more influential than others? How can intervention design be optimized for downstream beneficial effects on multiple distinct elements of relationship quality, given that there is unlikely to be a "one-size-fits-all" intervention method? Investigating and attempting to manipulate specific relationship-level active ingredients known to play a role in the health domain (e.g., perceived responsiveness, Stanton et al., 2019; hostile communication patterns, Kiecolt-Glaser et al., 2005) is more feasible, which will advance our understanding of potential mechanisms more rapidly and allow researchers to design more targeted and effective interventions.

In the following sections, we describe six methodological techniques drawn from clinical/intervention science and social psychology that can best fill the gaps in our mechanistic understanding of pathways between intimate relationships and physical health. These techniques fall into three major categories. First, dismantling studies and full factorial designs take existing broad interventions and break them down to

determine the most important components for affecting the outcome(s) of interest. Second, targeted experimental techniques, such as experimental therapeutics and experimental mediation, develop and test relatively simpler, theory-based interventions experimentally to study mechanism. Third, intensive longitudinal approaches, such as using multiple assessments and recursive modelling techniques, allow researchers to better study mechanistic processes with observational studies. We selected these six techniques to require differing levels of investment (in terms of both time and resources) and to cover different portions of the existing mechanistic gaps in the relationships-health literature. For each, we describe the technique, identify which of the five types of mechanistic evidence it supports, provide examples of previous studies that have successfully used this technique to study mechanism (in various domains), and discuss the unique strengths and challenges of each type of design for studying pathways between close relationships and physical health. Our discussion is summarized in Table 2.

<div align="center">**Factorial Designs**</div>

Single Factorial Designs: Dismantling Studies to Identify "Active Ingredients"

In clinical psychology, there is increasing attention and concern paid to the idea that although the field has a number of empirically-supported treatments that work to alleviate emotional distress, often there is little sense of precisely why these treatments work (Kazdin, 2007). Dismantling or component experimental designs seek to distill the most essential elements of any given intervention into their constituent parts, then test the efficacy of the specific elements alone or in combination (Papa & Follette, 2015). In a multi-session manualized intervention, dismantling studies allow researchers to identify the key "active ingredients" that may drive overall change. The broad approach to distilling active ingredients is consistent with efforts toward therapeutic

optimization: How can we deliver the most potent, trimmed-down behavioral intervention programs in a scalable way?

A classic example of dismantling work is Jacobson and colleagues' (1996) efforts to distill the activity ingredients in Cognitive Behavioral Therapy (CBT) for depression. The first empirical study on this topic separated the behavioral activation and automatic thought components from the core schema change and full CBT treatment package to treat 150 outpatients with major depression, and found no evidence that that the full treatment package improved outcomes over the combination of behavioral activation and automatic thought restructuring. In a follow-up randomized trial with 241 people suffering from depression, Dimidjian and colleagues (2006) compared behavioral activation alone to cognitive therapy and antidepressant medication and found that behavioral activation alone was comparable to antidepressant medication and outperformed cognitive therapy, especially among people with more severe mood disturbances. In a more recent example, Lindsay and colleagues (2018) dismantled a mindfulness intervention for stress management into the components of (a) monitoring and (b) accepting present moment experiences. Across two studies, this work showed that the inclusion of acceptance skills (toward present-moment experiences) is critical for improving daily positive emotional experiences.

The examples above provide a few illustrations of the ways in which dismantling designs can inform mechanisms of action. There are a variety of ways we can apply this approach to understanding intimate relationships and health. Clearly, couple-based interventions have established efficacy for treating depressive illness (Whisman et al., 2012) and a recent review suggests that intimate relationship functioning is a *causal risk factor* contributing to depressive illness (Whisman, Sbarra, & Beach, 2021). To the extent that major depression may shape health behaviors, cognitive/affective processes,

or plausible biological intermediaries that ultimately impact distal health outcomes (DeSteno, Gross, & Kubzansky, 2013), targeting mood disorders in the context of couple interventions may be useful for altering health intermediaries or outcomes. When thinking about dismantling these interventions it will be critical for investigators to consider the basic relationship processes that may improve the outcomes in question (see Barbato & D'Avanzo, 2020). For example, we might seek to separate classic ideas about behavioral exchange, problem-solving, and constructive communication from those that involve promoting relational closeness and emotional intimacy, including, conceivably, perceived partner responsiveness (Greenman, Johnson, & Wiebe, 2019; Stanton, Slatcher, & Reis, 2019). Ultimately, though, the question is not whether specific elements of these interventions can improve relationship functioning and emotional/mental health outcomes, but whether changes in the specific relational targets yield positive experimental effects on health-relevant intermediaries (e.g., blood pressure, inflammation, heart rate variability, sleep disturbances, and/or subjective symptoms). There is a growing experimental literature examining couple interventions for chronic health conditions (e.g., Badr & Krebs, 2013; Berry, Davies, & Dempster, 2017;Matire et al., 2010; Shields et al., 2012) and beginning to dismantle some of the effective interventions in this area may help pinpoint mechanisms of action.

Full Factorial Designs

Another way of breaking down complex interventions is using full factorial designs. Interventions should be as efficient as possible, with no inactive components taking up resources. However, the effectiveness of components may depend on one another: For example, a social support training intervention may not be effective unless partners improve their responsiveness as well (Selcuk & Ong, 2013). In order to assess what component(s) are most critical to a treatment's efficacy, factorial designs break

down a large intervention into component parts and create versions that test different combinations (e.g., only A, only B, only C; A and B, B and C, A and C; A, B, and C) while considering the level of investment required for each (Collins et al., 2016; Onken et al., 2014). Factorial designs are conceptually similar to dismantling studies, but instead of trying to disentangle two competing elements, these designs compare and contrast the merits of different combinations of possible mechanisms within a broader treatment. Then, researchers reassess the remaining components to develop new factorial designs to further break down the new and improved intervention, until it is as streamlined and effective as possible.

As with dismantling studies, this approach requires there to be multifaceted existing interventions that can be broken down into different components, and these are somewhat lacking in the relationships and health literature thus far. There are well-validated interventions for parenting that appear to impact health outcomes (e.g., Dozier, Peloso, Lewis, Laurenceau, & Levine, 2008; Miller, Brody, Yu, & Chen, 2014), but the experimental literature for couples and health is scant. We are aware of one small study (with 20 couples) that randomly assigned participants and their partners to communication training or an assessment-only control (Ewart et al., 1984); couples in the communication training condition showed less blood pressure reactivity at post-test. In another study of 68 couples, a support intervention (vs. control) involving "warm touch" enhancement reduced alpha amylase and systolic blood pressure (Holt-Lunstad et al., 2008). With the exception of this work and the couple therapy and depression treatment literature, we are unaware of intervention and experimental studies showing that altering relationship functioning shows a concomitant change in a health-relevant biomarker or physiological intermediary. Furthermore, these types of designs require fairly large samples in order to have reasonable representation in each

cell, which can be difficult when recruiting couples or families. However, combining groups to make comparisons can make required sample sizes more feasible (e.g., all conditions containing B versus all conditions without B).

As one example of the full factorial design comes from Yousafzai et al. (2014), who studied an intervention for child health and well-being consisting of two parts: an enhanced nutrition component (that included nutrition education and supplying participants with micronutrient powders) and a responsive stimulation component (that presented mothers with a variety of play and communication activities and taught them to use play and communication to strengthen responsiveness to child cues). The researchers recruited 1,302 four-year-old children and their mothers in Pakistan and randomized them into four groups: a control group (no intervention), an enhanced nutrition component only group, a responsive stimulation component only group, and an enhanced nutrition *and* responsive stimulation components group. Generally, the responsive stimulation component had positive effects on child outcomes regardless of the presence of the enhanced nutrition component, suggesting this is the more critically important component of this intervention. However, individuals who received both components showed especially high levels of pro-social behavior, suggesting that in some cases, addressing both features together is especially beneficial (Yousafzai et al., 2014). Although this study not focused on intimate relationships and health, it is a compelling example of how specific relationship processes—derived either from the theoretical or empirical literature—can be studied in combination with other intervention components. From our perspective, a study like this holds great value for the future investigation of the mechanisms linking intimate relationships and health.

Targeted Experimental Techniques

Experimental Therapeutics/Medicine

As noted above, a key element of NIH's SOBC program is a call to action for increased research identifying and quantifying specific mechanisms of action that drive lasting behavior change (Nielsen et al., 2018; Sumner, Beauchaine, & Nielsen, 2018). In the growing field of experimental medicine, *targets of change* represent variables that maintain poor health and, when altered, can initiate and/or support positive behavior change. The basic approach of experimental medicine has two elements: *target engagement* and *target validation*. Target engagement provides evidence that the intervention of interest alters the putative target mechanism of action, and target validation shows that change in the target is causally related to changes in the outcomes. Thus, experimental medicine takes a highly focused approach to target specific putative mechanisms directly. This is in contrast to randomized controlled trials (RCTs) of behavior change programs, which historically focus on the relatively efficacy of different treatment packages or a given treatment package relative to a control condition. As explained in the section on dismantling and factorial designs, these treatment packages can be bloated and may not be optimized to target mechanisms directly.

Following the basic principles of the SOBC movement, we argue that interpersonal and social processes are an expansive class of potential and generally understudied targets of behavior change, and the existing literature provides some relatively straightforward, theoretically based "wise interventions" (Walton, 2014; Walton & Wilson, 2018) that could be especially useful in an experimental medicine framework. Clearly, we cannot randomly assign people to stressful or hostile relational situations, and we certainly cannot randomly assign people to divorce or to experience greater loneliness in the context of marriage. The way around this obstacle is to

prioritize interventions or experiments that have the potential to improve functioning in these different spheres, rather than increase negative aspects of relationships.

Although not explicitly framed as an experimental medicine study, Finkel and colleagues' (2013) "marriage hack" prevention program illustrates many of the basic principles involved in the direct targeting of mechanisms. Drawing from literature indicating that negative marital attributions contribute to the normative decline in marital satisfaction over time (Bradbury, Beach, Fincham, & Nelson, 1996), Finkel et al. reasoned that an intervention designed to help adults *reappraise* interactions with their partner in a more benign light—as a third-party observer might see the interaction— and to maintain this perspective when they interacted with their partner would forestall the decline in relationship quality over a two year period. This is exactly what they found, and the unique promise of this preventative intervention is that it is relatively brief and potentially, if replicated, quite scalable. Adding health measures to designs like this would provide convincing evidence for the causal role of mechanisms linking relationships and health. For example, among the couples that engage in the mechanistically-focused reappraisal prevention program, does this maintain perceived partner responsiveness, which, in turn, explains a distal health-relevant outcome (e.g., resting blood pressure, actigraphy-derived measures of sleep quality)?

When it comes to targeting the mechanisms that shape the link between intimate relationships and health, we must return to a point we raised earlier: Ultimately, for experimental medicine studies to be successful, they must be designed with some clear insights into the timescale of how the causal mechanism unfolds, and this hinges on both the timing of assessments as well as the outcomes in question. If assessments are too narrowly focused or too widely spaced, effects will be missed and the insights these studies could provide will be limited (Farrell & Stanton, 2019). Moreover, clinical

disease endpoints take decades to emerge (Kuh et al., 2003), but a focus on, say, health

behavior intermediaries (e.g., smoking or sleep quality) or health-relevant biomarkers

(resting blood pressure or heart rate variability, or glucocorticoid resistance) can be

studied from weeks to months after the experimental interventions.

Experimental Mediation

Ideally, the field of intimate relationships and health will pursue rigorous

longitudinal, experimental studies designed to target putative mechanisms of action, but

in many instances the resources needed for these studies exceed what is available to

most scientists. Basic, cross-sectional research studies can also be invaluable in

providing the groundwork for the predictors and mechanisms that *should be targeted* in

subsequent large-scale longitudinal investigations (cf. Farrell & Stanton, 2019; Onken et

al., 2014). Scholars in the social psychology field (e.g., Cook & Groom, 2004; Spencer,

Zanna, & Fong, 2005) have argued that establishing a causal chain can be accomplished

by manipulating the constructs of interest in a series of experiments or across time

points, where each experiment or time point addresses a different path in the causal

chain. This approach, often termed *experimental mediation* or *experimental causal-chain*

design, is more robust than simply using mediation analysis in a single study.

Researchers typically manipulate X (the predictor variable) and observe its effects on M

(the mediator) in one study or at one time point. They then manipulate M and observe

its effects on Y (the outcome variable) in a separate study or later time point. Some

researchers follow up by testing their X–M and M–Y effects in a different sample or

using different methods to establish consistency, and others conclude their

investigation with a final study that manipulates X and observe its effects on Y via M

(i.e., establishing mediation in a more traditional analytic manner). This approach is

similar to experimental therapeutics/medicine in that both approaches are concerned

with establishing causal links between the predictor, mediator, and outcome.

Experimental mediation, however, is focused primarily on effecting short-term change

and is arguably less intensive than experimental therapeutics/medicine; for instance,

the causal paths might be established through brief experimental sessions in the lab and

measure immediate changes in relationship perceptions, behavior, and physiology.

Experimental mediation approaches have been used to test questions that fall

within the domains of social (e.g., Callan et al., 2011; O'Mara & Gaertner, 2017; Singh et

al., 2017) and health psychology (e.g., Jimenez et al., in press). However, to our

knowledge, there is not yet research testing the associations between intimate

relationships and health using an experimental causal-chain design. Nevertheless, we

can draw on an illustrative example from the intimate relationships literature: Cortes et

al. (2018) demonstrated that people who were satisfied with their current relationship

(X) placed more importance on positive past relational events and less importance on

negative past relational events (Y). This association was mediated by a tendency to feel

subjectively closer in time to positive events and subjectively distant from negative

events (M). In Study 1, participants reported their relationship satisfaction and wrote a

brief paragraph recalling a positive or negative relational event (X), after which they

reported how close or far in time the memory felt (M). Study 2 used a similar procedure

where positive versus negative memory recall (X) was manipulated, and its effects were

observed on subjective time distance (M) and perceived event importance (Y). In Study

3, the researchers manipulated both memory recall (X) and subjective time distance (M)

and observed their effects on perceived event importance (Y) by testing mediation

models with positive and negative memories tested separately. Establishing potential

causal links between X, M, and Y variables across separate studies or short timeframes

allows researchers to identify potentially important mechanistic pathways that can inform other types of designs.

In the intimate relationships and health domain, a promising experimental manipulation that might lend itself to this type of design involves attachment security priming (Gillath, Selcuk, & Shaver, 2008). Priming attachment security involves experimentally activating cognitive representations of feeling secure, comfortable, and close with a person, and has frequently been shown to enhance positive views of the self and others (Bryant & Chan, 2017; Pan, Zhang, Liu, Ran, & Teng, 2017; Rowe & Carnelley, 2003). There is also some evidence for attachment security priming being particularly helpful for reducing symptoms of individuals struggling with depression (Carnelley, Bejinaru, Otway, Baldwin, & Rowe, 2018; McGuire, Gillath, Jackson, & Ingram, 2018). Researchers interested in understanding how attachment anxiety and avoidance are linked to later health and disease outcomes may be able to use attachment security priming to observe its influences on, for instance, participants' sense of vigilance in the laboratory. Psychological vigilance may be a key mediator linking the cognitive-emotional sense of security with health intermediaries (Ruiz et al., 2017). In turn, and consistent with the steps of experimental mediation, vigilance itself can be manipulated to assess its causal role in shaping cardiovascular reactivity. These manipulations need not be limited to in-lab activities. With an effective mobile attachment security priming technique, it would be reasonable to assess vigilance via ecological momentary assessments in daily life over the course of a week or more (this would be a manipulation of a putative X variable; "engaging the target," according to the language of the SOBC initiative). Similarly, with an effective manipulation of vigilance (the key X variable) in the laboratory, it would be reasonable to assess potential sleep disturbances (the key mediatory) that are set in motion as a function of this heightened

arousal state (a putative outcome). Ultimately, the X-M and the M-Y associations would need to be organized together in a single study using established methods for evaluating X-M-Y mediation, but the ideas here illustrate the ways in which specific relational processes can be targeted and manipulated experimentally to pinpoint potential mechanisms of action in a theoretically and empirically coherent manner.

Intensive Longitudinal Approaches

Multiple Assessments

One major requirement for demonstrating mechanism is clearly establishing a causal timeline and temporal precedence to show that changes in the predictor precede changes in the mechanism, which in turn precede changes in the outcome (Kazdin 2007, 2014). Scientifically, if we wish to study a window into a causal process, we must understand the temporal resolution under which it unfolds (e.g., hours, days, months, years), which can often be a wildly difficult undertaking (Cole & Maxwell, 2003). Some studies do not begin measuring the mechanism or outcome of interest until after the manipulation, which makes it impossible to establish change from baseline. It can also be easy to miss a true effect because of the sampling window: If a follow-up is too early, change in the outcome or even the mechanism may not have occurred yet. If a follow-up is too late, changes in both the mechanism and the outcome may have occurred between assessments, leaving the researcher unable to establish the order of changes, or the effect may have occurred and dissipated before they attempted to measure it. To increase the likelihood of being able to identify the temporal order of change, we recommend that study designs include multiple assessments before, during, and after the intervention. Including multiple assessments on the shorter end of where the causal window(s) are expected and sampling beyond the expected timeline increases the likelihood of being able to capture the order of changes occurring in the mechanistic

pathway of interest. Materially, once we identify the correct window of temporal causal processes, we need the resources to study people over the entire window. We could easily envision a timescale of several months for a study on whether sleep quality mediates links between relational distress and blood pressure, and this would require a considerable longitudinal study with significant financial support.

There are some examples of multiple assessment designs in literature on relationships and health that illustrate their utility, though most are not focused on intimate relationships specifically. For example, Dekovic et al. (2012) investigated the temporal order of mechanisms linking multisystemic therapy (MST) to improved parenting and reduced externalizing problems. They followed families of adolescents with antisocial behavior issues receiving MST versus a control therapy over six months. By including 5 monthly assessments during therapy, as well as a pre-test and 6-month post-therapy follow-up of all variables, Dekovic and colleagues were able to determine that MST produced improvements in parental perceptions of competence, which then predicted greater use of positive discipline practices, which in turn predicted reductions in adolescent externalizing problems. These findings not only rule out alternative pathways (e.g., use of positive discipline promoting greater perceptions of parenting competence) and provide evidence for two variables in a causal chain and not just an association, but also highlight promoting competence as a key first step in this pipeline that this and other parenting interventions should be sure to maintain.

Combining multiple assessment designs with experimental research illustrates ways in which the recursive processes implicit in many of the existing theoretical accounts linking relationships and health may be studied. For example, Kok et al. (2013) showed that increased positive emotions, increased social connections, and vagal tone build upon one another to create an upward spiral towards improved well-being by

using a 9-week daily diary design. Unlike the previous example, these pathways were shown to be bidirectional, suggesting that changing either positive emotions, social connections, *or* vagal tone may lead to changes in the other interconnected outcomes. Studies with multiple assessments—either observation or experimental in nature— over time are ideal for establishing the timeline of a mechanistic process and consistency of an effect over time.

Studying Reciprocal Mechanisms and Dynamic Change

As alluded to above, we believe that a major rate-limiter on the speed of understanding mechanisms in this area of study is the statistical methods employed for most of our research questions. On the one hand, relationship researchers are certainly leaders in adopting newer methods to answer questions of process (e.g., contributing to new models on the longitudinal modeling of couple-related dynamics; Bolger & Laurenceau, 2013; Sakaluk et al., in press). On the other hand, we do not yet see many of these models capturing reciprocal and recursive mechanisms in the study of relationships and health. Here, we point to two statistical modeling approaches that may prove promising in this regard and note that many different models may ultimately be used to represent interdependent change in relationship and health-relevant variables (Estrada, Sbarra, & Ferrer, in press). First, latent change score (LCS) models (McArdle, 2009; McArdle & Hamagami, 2001) are conceptually similar to bivariate latent growth curves, but are better suited to capture dynamic associations between two parallel processes; this is achieved by specifying an unmeasured, latent difference score that compounds over time. Ultimately, this allows for an examination of cross-lagged or coupling effects on the change process itself. These latent scores, then, represent the accumulation of first-order difference scores and effectively create a non-linear system of interdependence. A concrete example here is illustrative. Sbarra and

Allen (2009) studied the interdependence of mood and sleep disturbances over six occasions and found that mood and sleep symptoms operate as two forces acting on each other depending on their specific levels; when sleep problems are relatively low, any sleep problems that occur have large effects increasing negative mood, but the opposite is true when sleep problems are relatively high: fluctuations in sleep problems have a much smaller effect on mood. In this case, the movement of the system (i.e., the rate of change of each variable) depends on the level of the other variable.

In our opinion, the LCS specification has tremendous promise for studying relationships and health. Proulx and Snyder-Rivas (2013) applied LCS models to the study of marital happiness and self-rated health over a 20-year period (including six major assessment timepoints). In this study, marital happiness predicted greater changes in self-rated health, but not the other way around (although the model tested a coupling parameter from self-rated health to participants' reports of marital happiness). Although this analysis is limited in its use of self-rated health, it is instructive that the health-relevant outcome follows from higher levels of happiness and not vice-versa. In the future, it will be ideal for models of this nature to include data from couples in which each trajectory represents a variable or construct from one partner within the relationship. Admittedly, however, repeated longitudinal assessments of health-relevant constructs in dyads are hard to come by. This consideration raises a larger point when it comes to studying dynamic mechanisms over time: The ability to do so with any degree of satisfaction depends on the available data. As longitudinal dyadic studies begin to come to fruition, the application of LCS models will continue to grow.

Another means of studying mechanisms that operate at an interpersonal level is through the use of a newly developed R package, *rties* (Butler & Barnard, 2019; see https://github.com/ebmtnprof/qid). A growing body of work suggests that

interpersonal emotional systems play an important role in a variety of health variables (e.g., Reed, Barnard, & Butler, 2015), and the *rties* package formalizes two models researchers can test to evaluate the degree of interdependence in emotional dynamics between two people. First, the inertia-coordination model represents the interdependence in two variables assessed over time and is conceptually similar to the "stability-influence" model (Thorson, West, & Mendes, 2018). These can be two physiological variables or two psychological variables, or even two ratings of different constructs over time. The key parameters in this model capture the interdependence in the variables over time as a function of auto-regression (within a person) and cross-regression (between people in a dyad) parameters. Second, a coupled-oscillator model characterizes the interdependent frequency of *oscillation* of two variables (e.g., emotional dampening or amplification). This latter model has the potential to be highly useful in testing theories about homeostatic set-points within relationships. For example, the coupled-oscillator model can evaluate the hypothesis that ability for couples to create and maintain homeostatic set points around their emotional functioning is a mechanism driving the potential health benefits of intimate relationships. The coupled-oscillatory model can characterize this process at the level of the couple, and this parameter estimate can then be associated with markers of health or well-being. This approach would be quite useful in explaining what it is that changes over, for example, the course of couple therapy and how these changes may yield health benefits.

Moving Forward

In research on the mechanisms linking intimate relationships and health, we are calling for a shift from the theoretical to the empirical, and from the conceptual to the methodological. Of course, theory is invaluable for specifying *potential* mechanisms of

action, and we are not calling for an end to theoretical developments in this area of study. Rather, we are encouraging the field to enhance its commitment to testing the hypothesized mechanisms of action that have already made their way into the literature and form the basis of most theoretical models in the field. What is the best way to make the next set of empirical advances? We have detailed six methodological strategies that have the potential to galvanize research in this field and to provide us with a better causal understanding of mechanism and clearer timeframes for identifying the emergence of effects. Similarly, these approaches will help us identify precise active ingredients within broad constructs like relationship quality and affective processes that are most impactful and most malleable.

Although each of these methods would help fill gaps in the existing literature, none of them alone can address all of Kazdin's (2007, 2014) criteria for establishing mechanism of action. Thus, these methods are most effective when used in conjunction within a program of research or teams of collaborators, with investigators outlining and studying short-term illustrations of meaningful changes complementing and informing the work of those running intensive longitudinal studies. For example, if researchers used an experimental medicine intervention to alter perceived partner responsiveness in couples, then studied ambulatory blood pressure across two weeks, it could be quite plausible to see illustrative causal effects that, when scaled up, would have a large impact on health.

Capitalizing on these methodologies is no small feat, but the rewards are well worth the effort. Researchers can supplement their own empirical endeavors—particularly smaller-scale lab studies—with data from large *N*, publicly-available datasets. Studies such as MIDUS, the National Social Life, Health, and Aging Project [NSHAP], the English Longitudinal Study of Aging [ELSA], and the Health and

Retirement Study [HRS] include several measures of relationship functioning. These projects may allow researchers to pursue questions about relationships-health mechanisms beyond the typical resources and timeframe provided by a standard grant. Furthermore, researchers will be able to gain novel, important understanding of the mechanistic pathways linking intimate relationships and health by investing in multidisciplinary initiatives and cross-research-group collaborations. We recommend that scientists who have samples with relationship and health outcomes consider submitting the datasets to the Love Consortium data science initiative (https://www.theloveconsortium.org/).

Another key consideration will be the diversity of samples recruited to test mechanisms linking intimate relationships and health. As discussed in the section on moderators of mediation above, mediators may not be consistent across demographic groups (e.g., culture, race, socioeconomic status). Furthermore, the overreliance on homogenous convenience samples for developing and testing mechanistic theory may lead us to ignore or miss important mechanisms. As individuals who are not White, well-educated, and upper-class are often the target of health interventions, studying mechanistic processes in these groups is critical to ensure interventions are optimized for the populations they are intended for. Thus, recruiting more diverse samples for testing the generalizability of basic mediational effects, as well as moderated mediation models, will be a critical task for the work in this field going forward.

The available evidence suggests that intimate relationships are one of the most potent social determinants of health (Holt-Lunstad, Robles, & Sbarra, 2017). To initiate the next generation of advances in this field, we need to better understand precisely how these effects unfold over time; ultimately, we can harness this knowledge to help people live longer, healthier, and happier lives.

Table 1

Kazdin's (2007, 2014) Five Criteria for Establishing a Mechanism of Change Applied to the Study of Close Relationships and Health

Criterion	Definition
Plausibility	Plausible and coherent explanation (e.g., a theoretical account) for why the causal path from X→M→Y should exist and how it should operate
Strong Associations	Evidence demonstrating robust associations between X and M, M and Y, or all three
Consistency	Evidence demonstrating consistency in the associations between X, M, and Y across different samples and paradigms (i.e., replicability)
Experimental Manipulation	Causal evidence demonstrating that altering X changes M and altering M changes Y
Timeline	Temporal evidence demonstrating that change in X precedes change in M, which then precedes change in Y

Note. We refer to the predictor/independent relationship variable as X, the mechanism of change as M, and the outcome/dependent physical health variable as Y. Criteria and definitions are adapted from Kazdin (2007, 2014).

Table 2

Summary of Six Methodological Techniques Suited to Investigating and Establishing Mechanisms Underlying Pathways between Close Relationships and Health

	Factorial Designs		Targeted Experimental Techniques		Intensive Longitudinal Approaches	
	Single Factorial Designs (Dismantling Studies)	Full Factorial Designs	Experimental Therapeutics (Experimental Medicine)	Experimental Mediation	Multiple Assessments	Recursive Modelling
Top-Down or Bottom-Up	Top-Down	Top-Down	Bottom-Up	Bottom-Up	Bottom-Up	Bottom-Up or Top-Down
Mechanism Criteria Fulfilled	Plausibility, Strong Associations, Consistency	Plausibility, Strong Associations, Consistency	Plausibility, Strong Associations, Experimental Manipulation	Plausibility, Strong Associations, Experimental Manipulation	Plausibility, Strong Associations, Consistency, Timeline	Plausibility, Strong Associations, Consistency, Timeline
Time Investment (Low, Moderate, High)	Moderate	High	High	Low	High	Moderate
Resource Investment (Low, Moderate, High)	High	High	Moderate	Low	Moderate	Moderate

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