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**Towards a Mechanistic Understanding of Links between Close Relationships and Physical  
Health**

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### **Abstract**

Although researchers have made great strides in identifying links between close relationship processes and physical health, we know less about the psychological and behavioral mechanisms underlying these links. As we move towards considering relationships as a public health issue, understanding mechanistic pathways in relationship-health links is crucial for designing efficient and effective interventions. In this review, we outline criteria for establishing a construct as a relationship-health mechanism. We then discuss how best to test potential mechanisms of relationship-health links and identify some promising mechanism candidates based on initial evidence (emotion, attachment, sleep, substance abuse). We conclude by describing some of the unique challenges for studying mechanisms in the relationship-health domain and recommend key directions for future research.

*Keywords:* Close relationships, physical health, psychological mechanisms, behavioral mechanisms

## **Towards a Mechanistic Understanding of Links between Close Relationships and Physical Health**

Over the past 30 years, links between close relationships and physical health have been thoroughly researched. Individuals' quantity and quality of social ties are associated with a wide range of health outcomes, including cardiovascular disease, cancer, and mortality (Farrell & Simpson, 2017; Holt-Lunstad, 2018). This research has roused calls for treating relationships as a public health issue and encouraging intervention work to improve relationships as a pathway to better health (e.g., Holt-Lunstad, Robles, & Sbarra, 2017). However, to apply relationship-health research in an intervention format, we must better understand the mechanisms underlying relationship-health links.

Calls for studying mechanisms in health psychology are not new. Kemeny (2003) argued that testing pathways between psychological experiences, physiological mediators, and clinical disease outcomes is critical for psychosocial processes to gain credence as disease risk factors. Miller, Chen, & Cole (2009) emphasized a reverse-engineering approach, in which psychologists identify biological precursors to disease and then measure the impact of psychosocial processes on those pathways. This focus on biological mechanisms is heavily reflected in existing relationships-health literature. For example, researchers have established that close relationship functioning prospectively predicts endocrine (Hostinar, Sullivan, & Gunnar, 2014), immunological (Kiecolt-Glaser, Gouin, & Hantsoo, 2009), metabolic (Kiecolt-Glaser & Wilson, 2017), and genomic (Robles et al., 2018) responses known to lead to health outcomes.

Focusing only on biological mechanisms, however, neglects other key pathways. Simply having supportive or contentious interactions with a relationship partner may not have direct biological effects. Instead, the ways in which people *interpret* and *internalize* these interactions

likely yield psychological and behavioral changes that affect biological functioning. We argue that, in addition to studying biological mechanisms, understanding psychological and behavioral mechanisms will help researchers and practitioners identify promising targets for interventions and make interventions more effective.

### **Establishing Mechanisms**

Ten years ago, scholars highlighted findings from correlational studies that point to promising mechanisms in the relationships-health domain (e.g., emotion regulation, health behaviors), but lamented that a lack of intervention-based evidence meant that the literature could not confirm these variables as causal mechanisms (Cohen & Janicki-Deverts, 2009; Thoits, 2011). Unfortunately, this research pattern persists today. Studies have continued testing either associations between relationship functioning and potential mechanisms, or associations between potential mechanisms and health; very few studies test mediation models linking all three cross-sectionally or longitudinally, and practically none use experimental, intervention-based designs. Thus, we still require direct evidence of causal mechanistic pathways underlying relationships and health.

If the existing body of evidence does not make a convincing case for mechanisms in the relationships-health domain, what would? Kazdin (2007) outlines several criteria in detail when discussing the effectiveness of therapeutic interventions. Although many studies identify *mediators*, the intervening variables that statistically account for the association between two other variables, fewer studies can clearly establish that the mediating variable is a *mechanism* that is causally responsible for the association. This distinction is critically important. For example, c-reactive protein (CRP), an inflammatory protein, is strongly associated with both psychosocial distress and heart disease (Kiecolt-Glaser, Gouin, & Hantsoo, 2010). However,

when researchers tested associations between genotypes that raised CRP and heart disease prevalence (controlling for other risk factors), they found no causal links between CRP and heart disease (C-Reactive Protein Coronary Heart Disease Genetics Collaboration, 2011). Thus, CRP may be a mediator, but likely not a causal mechanism, underlying relationships and health. Separating causal mechanisms from variables that serve as significant statistical mediators—likely by being a byproduct of a true mechanism—allows for the development of targeted interventions that prioritize improving truly causal factors (Onken et al., 2014).

Kazdin (2007) proposes that in order to determine that a variable is a mechanism, there must be (1) **a plausible and coherent explanation** for why and how a causal pathway operates between predictor/independent variable A, mechanism variable B, and outcome/dependent variable C; (2) **strong associations** between A, B, and C; (3) **consistency in these associations** across studies, samples, and conditions; (4) **experimental evidence** for the A-B-C pathway; and (5) an **established timeline** showing A changes before B, which changes before C (see Table 1). To provide this evidence, Kazdin recommends designing programs of research that build up to establishing robust mechanisms. For example, cross-sectional and longitudinal correlational designs can show plausibility, strong associations, and consistency of mechanistic pathways. Potential mechanisms emerging from this work can then be extended using experimental longitudinal designs to establish experimental evidence and timeline. Critically, some studies must measure A, B, and C at multiple timepoints before, during, and after the intervention to test alternative pathways (e.g., that C changes before B). However, researchers should be careful to avoid statistically unsound assumptions when building a body of evidence; for instance, a significant direct A-C path is not required for an indirect path through a mediator to be

significant (Zhao, Lynch, & Chen, 2010), and indirect effects that exist longitudinally may not emerge cross-sectionally (Maxwell & Cole, 2007).

In the following sections, we use these criteria to evaluate the most promising potential psychological and behavioral mechanisms emerging from recent relationships-health research, with the goal of determining what evidence we have for establishing them as mechanisms and what is still needed. This is summarized in Table 1 and described in greater detail below.

### **Potential Psychological Mechanisms**

Affective processes are among the most promising psychological mechanisms linking relationships and health. Social relationships shape the ways in which we appraise stressors, perceive our resources for dealing with stressors, and interact with one another, all of which have implications for emotion and health (Farrell, Imami, Stanton, & Slatcher, 2018; Sbarra & Coan, 2018). The few studies testing the full relationships-emotion-health mediation pathway reveal promising results (see Farrell et al., 2018 for a full review). For example, studies have shown greater positive affect mediates links between positive relationship functioning and outcomes like diurnal cortisol slopes (Ditzen et al., 2008) and inflammation (Tobin et al., 2015). Recently, a longitudinal study found, in a sample of middle-aged adults followed over 20 years, negative changes in perceived partner responsiveness over 10 years were linked to higher negative affect reactivity to daily stressors at the 10-year follow-up, which then prospectively predicted higher mortality rates another decade later (Stanton, Selcuk, Farrell, Slatcher, & Ong, 2019). This study did not test measures of affective reactivity to stressors before the first 10-year follow-up, however, so it cannot determine if changes in partner responsiveness preceded changes in negative affect reactivity. Furthermore, there are currently no interventions designed to alter

perceived partner responsiveness or negative affect reactivity, so testing these links experimentally is not yet feasible.

Another promising psychological pathway involves attachment orientations. Attachment orientations represent internal working models of the self and others that develop in infancy based on experiences with caregivers, which are continually shaped through later relationship experiences, guiding cognition, affect, and behavior across the lifespan (Mikulincer & Shaver, 2016). In turn, these working models influence how individuals assess and cope with stressors and how effectively they seek support in later relationships (Ehrlich et al., 2016). Unsurprisingly, individual differences in attachment anxiety (i.e., chronic worries about rejection/abandonment and desire for extreme closeness) and attachment avoidance (i.e., discomfort with intimacy and desire for independence) are associated with health-relevant biological processes and health behavior cross-sectionally and longitudinally (Pietromonaco & Beck, 2019). However, much like emotion, relationships-attachment-health indirect paths are largely untested. A recent exception is Farrell et al. (2019), who showed that attachment security in early adulthood (age 19-23) partially mediated links between maternal sensitivity received early in life (age 0-5) and cardiometabolic risk in adulthood (age 37-39). This research suggests that relationship functioning in early life may have enduring effects on health; however, without measuring these constructs repeatedly over time or manipulating them directly, we cannot be confident that attachment orientations are mechanisms. Nevertheless, with growing interest in how individuals can help insecure romantic partners become more secure over time (Arriaga, Kumashiro, Simpson, & Overall, 2018), the field is poised to better test the mechanistic role of attachment orientations.

### **Potential Behavioral Mechanisms**



In addition to psychological pathways, there is preliminary evidence suggesting close relationships predict health via health behaviors. Among the most researched behavioral mechanisms linking relationships to health is sleep. In adulthood, sleep is typically a shared activity between romantic partners (Hasler & Troxel, 2010). Spousal support (Jakubiak & Feeney, 2016) and self-disclosure (Kane, Slatcher, Reynolds, Repetti, & Robles, 2014) are associated with better sleep. Sleep quality, in turn, is negatively associated with morbidity and mortality (e.g., Dew et al., 2003). The literature thus far contains robust evidence for relationship-sleep and sleep-health links separately; however, to the best of our knowledge, no studies have yet tested the indirect path between these variables or attempted to manipulate relationship or sleep variables.

Relationship functioning is also associated with substance abuse. Receiving responsive parenting is associated with reduced drug use in adolescence (Baumrind, 1991). In adulthood, individuals may drink to cope with relationship difficulties (Rodriguez, Knee, & Neighbors, 2014). Given that drug and alcohol abuse increase morbidity and mortality risk (Center for Disease Control, 2018; National Institute of Drug Abuse, 2017), there is likely a relationships-substance abuse-health indirect path. Furthermore, given the body of work on interventions to treat substance abuse, a logical next step for research is to manipulate this potential mechanism and study its impact on relationships and health to establish timeline.

Unfortunately, the behavioral mechanisms of relationship-health links are studied even less than the psychological mechanisms. This gap is especially important to fill as research in this area may uncover effects of relationships on diseases that are less linked to biological stress systems, but include large behavioral components, such as Type 2 diabetes (e.g., Brody et al., 2019).

### **Unique Challenges and Future Directions**

When examining existing work on these potential mechanisms, the lack of research utilizing experimental manipulation or including assessments of constructs at multiple timepoints to test the mechanistic timeline is immediately apparent. Most studies in the relationship-health domain use observational designs and assess certain constructs only at certain waves. This makes it extremely difficult to separate statistical mediators (likely proxies/byproducts of mechanisms) from true mechanisms. We now discuss what research is needed to better identify mechanism, inform current interventions, and develop new interventions to improve relationships and health.

The Strong African-American Families (SAAF) Program is an exemplar of intervention work on relationships and health: It has been shown to improve parenting, and change in parenting from the program predicts reduced metabolic syndrome (Chen et al., 2018), and inflammation (Miller et al., 2014), providing causal evidence for relationship-health links. One recent study also shows that SAAF reduces men's drug use, pointing to a potential behavioral mediator of biological effects (Brody et al., 2019). However, researchers have not yet investigated potential psychological mechanisms with SAAF. Furthermore, like many relationship interventions, SAAF is very time- and resource-intensive, requiring 14 hours of in-person training including parents, children, and facilitators that covers a wide range of topics.

Ideally, we will see more longitudinal, experimental designs with many assessment points that can provide both kinds of evidence in the future, but major contributions to this field can still be made using less intensive approaches. As outlined in the Stage Model (Onken et al., 2014), basic research is critical for developing and refining interventions until they are maximally effective and efficient when implemented in their target populations. Rather than aiming for fast, widespread implementation, it calls for an incremental process of applying basic

science to evaluate and streamline potential interventions, using samples from more controlled research environments as well as communities at large. Knowledge is gained at each stage in this process, including after failed interventions.

For example, basic research may help reveal important “active ingredients” within interventions, which mostly strongly predict mechanisms, which could allow for effective streamlining. One potential active ingredient is *perceived partner responsiveness*, the extent to which individuals believe their partners understand them, validate their thoughts and feelings, and show caring and affection (Stanton, Slatcher, & Reis, in press). Perceived partner responsiveness is related to health outcomes such as diurnal cortisol profiles (Slatcher, Selcuk, & Ong, 2015) and mortality rates (Stanton et al., 2019) via affective processes. Responsiveness is also associated with potential behavioral mechanisms like sleep (Selcuk, Stanton, Slatcher, & Ong, 2017). Testing methods to increase perceived partner responsiveness would provide better insights into its causal effects, which could potentially inform future interventions to improve health. Alternately, experimental work in this domain could demonstrate that aspects of existing interventions that improve responsiveness are particularly indispensable.

Interventions must also consider changes in pathways across development at the individual and relationship level. For example, different types of relationships (e.g., parent-child, romantic) are most impactful at different life stages, and some diseases of aging (e.g., cardiovascular disease) are unlikely to emerge until later life regardless of relationship functioning (which requires conducting longitudinal studies to detect these effects). Determining the most effective timecourse for these types of interventions will be important as well. “Booster-shot” sessions of interventions may be necessary over time to ensure effects endure. Identifying when different interventions are most effective, how long they remain effective, and

when their effects emerge in biological functioning and health will provide valuable information for tailoring and evaluating interventions.

It is also important to note that individuals are nested within broader social contexts, like race and socioeconomic status, which affect potential mechanistic pathways (Holt-Lunstad, 2018). For example, withdrawal during conflict is associated with reduced relationship satisfaction for more affluent couples but increased satisfaction for lower SES couples (Ross et al., in press). Similarly, self-control, which typically promotes health, is negatively associated with health markers in individuals from disadvantaged backgrounds (Miller et al., 2015). Given that most of psychological research, particularly in relationship science, is conducted on white, heterosexual, higher-SES individuals, there is still much to know regarding how generalizable these patterns are across different populations (particularly those most likely to be targeted by health-promoting interventions).

As close relationships gain attention as a public health issue, it is critically important to understand the mechanisms linking relationship functioning to health. This will require programs of research that use longitudinal and experimental designs to test promising psychological and behavioral mechanistic targets before presenting them as causal mechanisms, and that then build on this basic science to design effective interventions that can be efficiently employed with people in need (Onken et al., 2014). This call for mechanistic research has been made previously (Cohen & Janicki-Deverts, 2009; Thoits, 2011) but has gone largely unheeded. We hope that the growing desire to apply the impressive body of work linking relationships and health to the real world will provide an impetus to tackle these challenges head-on. Understanding mechanisms can encourage the development of more streamlined, efficient, and effective interventions, and will be well worth the effort.

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Table 1

*Criteria for Establishing Mechanistic Pathways between Close Relationships and Physical Health*

<b>Criterion</b>	<b>Definition</b>	<b>Research Designs Required</b>	<b>Affective Processes as B?</b>	<b>Attachment as B?</b>	<b>Sleep Quality as B?</b>	<b>Substance Abuse as B?</b>
Plausibility	Plausibility and coherence in the explanation of why or how the causal $A \rightarrow B \rightarrow C$ mechanistic pathway operates	Theory-driven designs	✓	✓	✓	✓
Strong Associations	Robust A–B and B–C links	Cross-sectional or longitudinal designs assessing A–B, B–C, or A–B–C links	✓	✓	✓	✓
Consistency	Consistency in A–B–C links across studies, samples, and conditions	Multiple cross-sectional or longitudinal designs assessing A–B–C links	✓	✓	✓	Partial
Experimental Manipulation	Experimental evidence that establishes that altering A produces changes in B, and that altering B produces changes in C	Cross-sectional or longitudinal experimental designs/randomized control trials manipulating either A or B				
Timeline	An established timeline demonstrating that change in A precedes change in B, which in turn precedes change in C	Longitudinal designs assessing A, B, and C at multiple timepoints				

*Note.* We refer to the predictor/independent relationship variable as A, the mechanism of change as B, and the outcome/dependent physical health variable as C. Criteria and definitions are adapted from Kazdin (2007). Kazdin also proposed dose-dependent response gradient for the mechanism's effect on the outcome as a non-essential criteria, 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 links that are probably multiply determined.