Toward a Mechanistic Understanding of Links Between Close Relationships and Physical Health

Allison K. Farrell¹ and Sarah C. E. Stanton²
¹Department of Psychology, Miami University, and ²Department of Psychology, University of Edinburgh

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 toward considering relationships as a public health issue, understanding mechanistic pathways in relationships–health links is crucial for designing efficient and effective interventions. In this review, we outline criteria for establishing a construct as a relationships–health mechanism. We then discuss how best to test potential mechanisms of relationships–health links and identify some promising mechanism candidates on the basis of initial evidence (emotion, attachment, sleep, and substance abuse). We conclude by recommending key directions for future research.

Keywords
close relationships, physical health, psychological mechanisms, behavioral mechanisms

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 relationships–health research in an intervention format, we must better understand the mechanisms underlying relationships–health links.

Calls for studying mechanisms in health psychology are not new. Kemeny (2003) argued that testing pathways among psychological experiences, physiological mediators, and clinical disease outcomes is critical for psychosocial processes to gain credence as disease risk factors. Miller, Chen, and 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, 2010), 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. 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.

Corresponding Author:
Allison K. Farrell, Miami University, Department of Psychology, 90 North Patterson Ave., Oxford, OH 45056
E-mail: farrelak@miamioh.edu
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) outlined several criteria in detail when discussing the effectiveness of therapeutic interventions. Although many studies have identified mediators, the intervening variables that statistically account for the association between two other variables, fewer studies could 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 et al., 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 of but is likely not a causal mechanism underlying links between relationships and health. Separating causal mechanisms from variables that serve as significant statistical mediators—likely by being a by-product of a true mechanism—allows for the development of targeted interventions that prioritize improving truly causal factors (Onken, Carroll, Shoham, Cuthbert, & Riddle, 2014).

Kazdin (2007) proposed that in order to determine that a variable is a mechanism, there must be (a) a plausible and coherent explanation for why and how a causal pathway operates between predictor A (the independent variable), mechanism variable B, and outcome C (the dependent variable); (b) strong associations between A, B, and C; (c) consistency in these associations across studies, samples, and conditions; (d) experimental evidence for the A–B–C pathway; and (d) an established timeline showing that A changes before B, which changes before C (see Table 1). To provide this evidence, Kazdin recommended 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 tested using experimental longitudinal designs to establish experimental evidence and timeline. Critically, some studies must measure A, B, and C at multiple time points 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 goals of determining what evidence we have for establishing them as mechanisms and of determining 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 mediational pathway reveal promising results (see Farrell et al., 2018, for a full review). For example, studies have shown that greater positive affect mediates links between positive relationship functioning and outcomes such as diurnal cortisol slopes (Ditzen, Hoppmann, & Klumb, 2008) and inflammation (Tobin et al., 2015). Recently, a 20-year longitudinal study of a sample of middle-aged adults found that 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 could not determine whether 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 is through attachment orientations. Attachment orientations represent internal working models of the self and others that develop in infancy on the basis of experiences with caregivers. They are continually shaped through later relationship experiences, guiding cognition, affect, and behavior across the life span (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, Miller, Jones, & Cassidy, 2016). Unsurprisingly, individual differences in attachment anxiety (i.e., chronic worries about rejection or 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 behaviors cross-sectionally and longitudinally (Pietromonaco & Beck, 2019). However, much like emotion pathways, indirect relationships–attachment–health pathways are largely untested. A recent exception is a study by Farrell et al. (2019), which showed that attachment security in early adulthood (ages 19–23) partially mediated links between maternal sensitivity received early in life (ages 0–5) and cardiometabolic risk in adulthood (ages 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

Table 1. Criteria for Establishing Mechanistic Pathways Between Close Relationships and Physical Health

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Definition</th>
<th>Research designs required</th>
<th>Affective processes as B?</th>
<th>Attachment as B?</th>
<th>Sleep quality as B?</th>
<th>Substance abuse as B?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plausibility</td>
<td>Plausibility and coherence in the explanation of why or how the causal A → B → C mechanistic pathway operates</td>
<td>Theory-driven designs</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Strong associations</td>
<td>Robust A–B and B–C links across studies, samples, and conditions</td>
<td>Cross-sectional or longitudinal designs assessing A–B, B–C, or A–B–C links</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Consistency</td>
<td>Consistency in A–B–C links across studies, samples, and conditions</td>
<td>Multiple cross-sectional or longitudinal designs assessing A–B–C links</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Partial</td>
</tr>
<tr>
<td>Experimental manipulation</td>
<td>Experimental evidence that establishes that altering A produces changes in B and that altering B produces changes in C</td>
<td>Cross-sectional or longitudinal experimental designs or randomized controlled trials manipulating either A or B</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Timeline</td>
<td>An established timeline demonstrating that change in A precedes change in B, which in turn precedes change in C</td>
<td>Longitudinal designs assessing A, B, and C at multiple time points</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

Note: We refer to the predictor (independent 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 a dose-dependent response gradient for the mechanism’s effect on the outcome as a nonessential criterion and specificity of a single mechanism for a given intervention–outcome link, which we think is unlikely to hold in the case of relationships–health links that are probably multiply determined.
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 that 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 relationships–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, Neighbors, & Knee, 2014). Given that drug and alcohol abuse increase morbidity and mortality risk (Centers for Disease Control and Prevention, 2018; National Institute on Drug Abuse, 2017), there is likely an indirect path from relationships to health through substance abuse. 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 a timeline over which it might operate.

Unfortunately, the behavioral mechanisms of relationships–health links are studied even less than the psychological mechanisms. It is especially important to fill this gap because 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, Yu, Chen, & Miller, 2017).

**Future Directions**

When examining existing work on these potential mechanisms, one can immediately see the lack of research utilizing experimental manipulation or including assessments of constructs at multiple time points to test the mechanistic timeline. Most studies in the relationships–health domain use observational designs and assess certain constructs only at certain waves. This makes it extremely difficult to separate statistical mediators (likely proxies or by-products of mechanisms) from true mechanisms. It is therefore critical to establish causality over time to reveal mechanistic pathways that can inform interventions.

One method for identifying and clarifying these pathways is to situate the study of mechanism within relevant existing, exciting intervention work. For instance, 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 as a result of the program predicts reduced metabolic syndrome (Chen, Miller, Yu, & Brody, 2018) and inflammation (Miller, Brody, Yu, & Chen, 2014), providing causal evidence for relationships–health links. One recent study also showed that SAF reduces men's drug use, pointing to a potential behavioral mediator of biological effects (Brody, Yu, Miller, Ehrlich, & Chen, 2019). Researchers have not yet investigated potential psychological mechanisms with SAF, but this design could be very fruitful for distinguishing derivative mediators from causal mechanisms over time. However, implementing SAF does include challenges. Like many relationship interventions, SAF is very time and resource intensive, requiring 14 hr 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 useful 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, an incremental process of applying basic science to evaluate and streamline potential interventions is called for, using samples from more controlled research environments as well as communities at large. Knowledge to improve future interventions is gained at each stage in this process, including after failed interventions.

One insight that basic research may help reveal is important “active ingredients” within interventions that most 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
Perceived partner responsiveness is related to health outcomes such as diurnal cortisol profiles (Slatcher, Selcuk, & Ong, 2015) and mortality rates (Stanton, Selcuk, et al., 2019) via affective processes, even when analyses control for the effects of negative relationship functioning. Responsiveness is also associated with potential behavioral mechanisms such as 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.

Another path for future work involves providing greater depth to our understanding of when and for whom interventions will be effective. Interventions must consider changes in pathways across development at the individual and relationship levels. 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 time course for these types of interventions will be important as well. “Booster-shot” sessions of interventions may be necessary over time to ensure that 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, such as race and socioeconomic status (SES), 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, Karney, Nguyen, & Bradbury, 2019). Similarly, self-control, which typically promotes health, is negatively associated with health markers in individuals from disadvantaged backgrounds (Miller, Yu, Chen, & Brody, 2015). Given that most 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.

**Recommended Reading**

Cohen & Janicki-Deverts (2009). (See References). A review offering valid critiques (albeit 10 years old) regarding the lack of causal and mechanistic evidence for relationships–health links


Kazdin, A. E. (2007). (See References). An in-depth discussion of the requirements for demonstrating causal mechanisms and the methodological techniques that can provide this evidence.


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**ORCID iDs**

Allison K. Farrell [https://orcid.org/0000-0002-8233-4076](https://orcid.org/0000-0002-8233-4076)

Sarah C. E. Stanton [https://orcid.org/0000-0002-3562-4644](https://orcid.org/0000-0002-3562-4644)

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