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Psychological Pathways Linking Social Support to Health Outcomes: A Visit with the "Ghosts" of Research Past, Present, and Future

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Abstract

Contemporary models postulate the importance of psychological mechanisms linking perceived and received social support to physical health outcomes. In this review, we examine studies that directly tested the potential psychological mechanisms responsible for links between social support and health-relevant physiological processes (1980s to 2010). Inconsistent with existing theoretical models, no evidence was found that psychological mechanisms such as depression, perceived stress, and other affective processes are directly responsible for links between support and health. We discuss the importance of considering statistical/design issues, emerging conceptual perspectives, and limitations of our existing models for future research aimed at elucidating the psychological mechanisms responsible for links between social support and physical health outcomes.

Keywords

Social support; psychological mechanisms; physiology; review

Charles Dickens' classic tale "A Christmas Carol," tells the story of Ebenezer Scrooge who is visited by the ghosts of Christmas past, present, and yet to come. Through these encounters, Scrooge is transformed from a cynical individual into someone who appreciates the past, celebrates the present, and looks optimistically into the future. In this paper, we would like to tell a similar tale, one that is rooted in the search for psychological mechanisms linking social support to disease.

In this paper, we first briefly review evidence linking social support to health and the theoretical models that postulate the importance of psychological mechanisms in such links. We refer to such mechanisms broadly as those involving cognitive and affective states (e.g., appraisals, depression) at a psychological level of analysis (Cacioppo & Berntson, 1992). These psychological mechanisms are typically examined in the context of perceived or received social support. Perceived support refers to the perception that support would be available if needed, whereas received support refers to the exchange of support resources (Dunkel-Schetter & Bennett, 1990; Uchino, 2009). Although these aspects of support can be

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further differentiated by the specific type of support (e.g., emotional, instrumental; Cohen & Wills, 1985), most of the health literature has focused more broadly on this distinction.

We next critically examine the actual empirical evidence for psychological mechanisms linking these aspects of social support to biological processes as this literature is the most well-developed. Potential studies were identified using PsycINFO and Medline and needed to include data on perceived/received support, psychological mechanisms, and health-relevant physiological outcomes. We thus review the literature examining psychological mediation of links between social support and cardiovascular, neuroendocrine, and immune function as these physiological pathways have important implications for physical health (Chida & Steptoe, 2010; Dickerson & Kemeny, 2004; Kiecolt-Glaser et al., 2002). Finally, we discuss salient issues when examining psychological mechanisms that might be responsible for links between social support and health. Overall, we believe that there are important lessons to be learned based on past and present approaches which can provide much needed guidance to future work.

Psychological Mechanisms Potentially Linking Social Support to Health

Perceived social support is one of the most well-documented psychosocial factors influencing physical health outcomes (see reviews by Berkman et al., 2000; Cohen, 1988; Holt-Lunstad et al., 2010; House et al., 1988; Pinquart & Duberstein, 2010; Uchino, 2004). Epidemiological studies indicate that individuals with low levels of social support have higher mortality rates; especially from cardiovascular disease (Barth et al., 2010; Berkman et al., 1992; Orth-Gomer et al., 1993). Although more research is needed, there is also evidence linking support to lower cancer and infectious disease mortality (Ell et al., 1992; Lee & Rotheram-Borus, 2001; Pinquart & Duberstein, 2010). In perhaps the most compelling evidence to date on the health effects of social support, Holt-Lunstad and colleagues (2010) conducted a meta-analysis of the existing literature and found that perceived support was related to significantly lower risk for all-cause mortality (Holt-Lunstad et al. 2010). Many of these studies statistically controlled for other variables such as basic demographics as well as additional risk factors (e.g., health behaviors). Indeed effect sizes from their meta-analysis appeared as large, if not larger, than standard medical factors such as exercise and obesity.

Importantly, major models of social support and health have postulated psychological mechanisms such as perceived stress, depression, and positive affect as important pathways (Berkman et al., 2000; Cohen, 1988; Uchino, 2004). Directly elucidating the psychological mechanisms linking social support to physical health outcomes is generally considered a primary research agenda for both theoretical and applied reasons (Reis et al., 2000). For instance, knowing the psychological mechanisms by which social support influences health highlights different approaches (e.g., stress-buffering) or arenas of intervention (e.g., positive affect).

As a starting point, Cohen and Wills (1985) classic review on the buffering hypothesis predicts that support may prevent or reduce stress appraisals with corresponding influences on emotion-linked physiological responses. They further suggest that “direct effects” of social support on health may be mediated by positive affect, predictability, and a sense of self-worth (also see Cohen, 1988). In a recent paper, Cohen (2004) argued the following:

> In this view, the belief that others will provide necessary resources may bolster one’s perceived ability to cope with demands, thus changing the appraisal of the situation and lowering its effective stress … (p. 677)

> Belief that support is at hand may also dampen the emotional and physiological responses to the event or alter maladaptive behavioral responses … (p. 677, see Table 1).

In their theoretical analysis, Berkman and colleagues (2000) also hypothesized that psychological mechanisms such as self-efficacy, self-esteem, depression, distress, and sense of well-being, represent important "downstream" pathways linking support to health. For instance, they noted that:

> … social networks via any number of pathways influence cognitive and emotional states such as self-esteem, social competence, self-efficacy, depression and affect (p. 850).

> Social support may additionally operate through its influence on emotion, mood, and perceived well-being (p. 851, see Figure 1).

Uchino (2004) argued for similar psychological mechanisms based on an integration of prior models. In fact, we are not aware of a major theoretical model linking social support to health that does not postulate psychological mechanisms as potential pathways. However, it should be noted that many of these authors also caution about making strong inferences about these psychological mechanisms given the lack of direct available evidence (Berkman et al., 2000; Cohen, 2004; Uchino, 2004).

It is important to note that many of these frameworks were informed by and hence overlap with models linking social support to mental health outcomes (e.g., Gore, 1981; Lin, 1986). Although not a large literature, research in the mental health domain has been able to directly model relevant psychological mediators including self-esteem and perceived control (Atienza et al., 2001; Krause, 1987; Symister & Friend, 2003). So what is the current evidence for these psychological mechanisms in the physical health domain? There is certainly strong evidence linking social support to these psychological factors when they are treated as outcomes in their own right. Social support has been linked to appraisal patterns (Dunkel-Schetter et al., 1987), as well as greater feelings of control, self-efficacy, and self-esteem (Atienza et al., 2001; Shaw et al., 2004; Symister & Friend, 2003). High levels of social support are also related to lower perceptions of stress, less stress exposure, and lower depression (Sarason et al., 1990; Russell & Cutrona, 1991). Of course, these data are necessary but not sufficient to demonstrate that these factors are direct mechanisms responsible for associations between social support and physical health. We now turn to a discussion of direct evidence for psychological mechanisms based on a representative review of research linking social support to health-relevant biological outcomes.

The Ghosts of Research Past and Present: Testing Psychological Processes as Confounds or Mechanisms

A starting point for examining psychological mechanisms linking social support to health occurred in the 1980s to mid-1990s. Many of the early health models which postulated psychological mechanisms were meant to guide future work (Cohen, 1988; Cohen & Wills, 1985). However, as is typical with emerging potential risk factors, there was a concern that social support might be confounded with other factors, especially those related to mental health. For instance, it was shown that depression might bias perceptions of support (e.g., Bolger & Eckenrode, 1991) which raised concerns about whether social support was the primary factor responsible for links to health. In these studies, researchers would statistically control for such mental health variables (primarily depression or perceived stress) to demonstrate that social support links to health-relevant physiological processes were still significant. In these cases, perceived social support maintained its association to cardiovascular (Uchino et al., 1992) and immune function (Baron et al., 1990; Kiecolt-

Glaser et al., 1991) when considering these psychological processes. Of course, these studies also provided some evidence against psychological mediation as statistically controlling for them should have partially or completely eliminated the links between social support and physiological outcomes (Baron & Kenney, 1986; MacKinnon, 2008).

There were a number of social support studies during this time period in which it was less clear if these psychological factors were viewed as confounds or mechanisms (Kirschbaum et al., 1995; Unden et al., 1991; Linden et al., 1993; Thomas et al., 1985). Nevertheless, none of these studies reported evidence that would be consistent with strong psychological mediation. For instance, Thomas et al. (1985) examined the links between social support and proliferative responses to mitogens. They found that women high in social support were characterized by a stronger proliferative response (indicative of a better immune response) than women low in social support even when statistically controlling for reported psychological distress.

Of particular interest are the laboratory reactivity studies emerging during this period that examined whether received support during acute stress was linked to reduced cardiovascular reactivity (Thorsteinsson & James, 1999). These laboratory models became popular following the work by Kamarck and colleagues (1990) who hypothesized explicit links between the reactivity hypothesis of disease and the buffering model of support (e.g., Krantz & Manuck, 1984). In general, such reactivity studies in the laboratory are thought to model how receiving support over time in the real world might impact the long-term development or exacerbation of clinically diagnosed cardiovascular disease (Uchino et al., 2011). In their important study, the mere presence of a friend was associated with reduced cardiovascular reactivity during acute psychological stress (Kamarck et al., 1990).

These laboratory studies should in theory provide a more powerful test of psychological mechanisms due to the controlled context. In many of these laboratory studies, measures of affective reactions (e.g., anxiety, distress), and in some cases, appraisals (e.g., threat) were measured under carefully controlled conditions of receiving support from a stranger or friend. For instance, Gerin and colleagues (1992) had women engage in a stressful group event discussion. Participants' views regarding a controversial topic were attacked by two other confederates while a third confederate either provided support or sat quietly. Results revealed that social support was uniformly associated with lower blood pressure reactivity to the discussion task. However, participants in the support condition did not report that they felt significantly less stressed, anxious, or angry during the discussion task. This pattern of findings in which physiological processes, but not psychological processes, differentiated support links is the norm in this literature (e.g., Cosley et al., 2010; Glynn et al., 1999; Uchino & Garvey, 1997). It is important to note that most laboratory studies are based on the receipt of support which is separable from perceived support (Uchino, 2009). As will be discussed later, received support is more sensitive to the context in which it is provided (e.g., responsiveness), so different psychological mechanisms that have thus far received little attention may be at work in these laboratory reactivity studies.

Starting around the late 1990's to the present, researchers became more explicit about testing the possible psychological mechanisms linking social support to physiological outcomes. For instance, one study examined age-related differences in resting blood pressure as a function of perceived social support (Uchino et al., 1999). These authors found that social support was associated with lower resting blood pressure in older adults; an effect that was not statistically mediated by perceived stress, depression, or satisfaction with life. Stone and colleagues (1998) similarly reported that links between perceived social support and lower prostate-specific antigen were not due to perceived stress. Lutgendorf and colleagues (2000) also directly tested the possibility that mood profiles might be responsible

for links between receiving social support and IL-6 in cancer patients. They found no evidence that alterations in mood were responsible for such links. In general, the available recent literature provides no evidence that the influence of perceived or received social support on cardiovascular, neuroendocrine, and/or immunity is statistically mediated by anxiety, life stress, subjective distress, or depression (Clark, 2003; Cosley et al., 2010; Hilmert et al., 2002; Lutgendorf et al., 2000, Lutgendorf et al., 2005; Marsland et al., 2007; Taylor et al., 2010). As noted earlier, this is not a recent trend (Baron et al., 1990; Kiecolt-Glaser, et al., 1991; Thomas, et al., 1985).

The Ghost of Research Yet to Come: Learning from the Past for a more Optimistic Future?

The tale thus far does not inspire confidence in finding answers to this important theoretical question. Although social support has proven to be a robust predictor of future morbidity and mortality (Holt-Lunstad et al., 2010), decades of research have consistently been unable to demonstrate the psychological mechanisms directly responsible for such links. We believe we are at a point where staying the present course is unlikely to lead to answers to this question and indeed may sidetrack us from the major issues involved. We now turn our attention to potential explanations for the current state of the literature starting from the most obvious to the most exploratory or provocative (see Table 1). In doing so, we strongly advocate systematically pursuing all of these possibilities in future work.

Potential Explanation 1: Statistical and design issues are obscuring our search for psychological mediation

There are several salient statistical and design issues based on the prior literature that might explain the present set of findings. First, as true of any inferential test, minimizing measurement error allows for more sensitive tests of potential associations. One obvious possibility then is that heightened measurement error may be limiting our tests of psychological mediation. However, there are two lines of evidence that argue against this being a primary problem. First, many of the studies used well-validated measures (e.g., profile of mood states, perceived stress scale, Lutgendorf et al., 2000; Uchino et al., 1999). Although it is possible that measurement error on the social support - physiological outcome link may operate, whatever error exists has not been sufficient to mask any such associations.

A second argument against measurement error is the consistent pattern of null findings for laboratory studies which usually result in larger effect sizes and less measurement error (Thorsteinsson & James, 1999). In these studies, receiving support from a friend, confederate, or experimenter has been associated with reductions in cardiovascular reactivity during acute psychological stress. However, the majority of these studies found no evidence that receiving support altered psychological mechanisms such as perceived distress, affect, or anxiety; or that these psychological mechanisms were directly responsible for links between social support and physiological outcomes (e.g., Cosley et al., 2010; Gerin et al., 1992; Glynn et al., 1999). Thus, although researchers must always be aware of measurement error, we do not find sufficient evidence that this is a primary reason why past research has been unable to uncover the psychological mechanisms linking social support to health outcomes.

A second salient statistical issue is that researchers may need to use more sensitive statistical tests of mediation. It is certainly the case that most studies testing for psychological mechanisms have utilized the Baron and Kenny (1986) approach. As noted by MacKinnon (2008), not all the criteria laid out by Baron and Kenny (1986) are necessary for mediation

and overall this approach is quite conservative and thus more likely to result in such null findings. More recently, there is consensus that a test of a nonzero a*b path is what is necessary to determine at least partial mediation (Preacher & Hayes, 2004). A common way of testing for this product term is the Sobel test which assumes normality of the coefficients. However, this assumption is not typically met so a more widely accepted method of testing for mediation is bootstrapping (Preacher & Hayes, 2004). Bootstrapping involves treating your sample as population-level data and sampling with replacement to create confidence limits for mediated effects (Preacher & Hayes, 2004). None of the prior studies that we are aware of has tested for mediation using bootstrapping so this remains an issue that needs to be examined in future tests. It should be noted, however, that evidence for mediation in this literature has been virtually nonexistent using the Baron and Kenny (1986) approach. It is thus unclear whether or not the gain in precision using bootstrapping would be able to detect relevant psychological mechanisms in prior work.

A third, design issue, is that researchers may need to model changes over time in social support, psychological mechanisms, and health-relevant outcomes. Most of the prior work examining links between social support and health-relevant physiological outcomes has utilized cross-sectional designs. In such studies it is assumed that the health-related variance in the psychological “state” is adequately captured at that point in time. However, these psychological mechanisms are multiply-determined and at any point in time may reflect other sources of variance such as time of day or exposure to more transient events (Ahadi & Diener, 1989). Modeling these processes over time may thus provide a more sensitive test of psychological mediation as systematic changes in social support and psychological processes that are health-relevant can be directly modeled. Of course, such studies are expensive and time intensive but important given the prospective hypotheses linking social support to disease (e.g., development of cardiovascular disease). Nevertheless, even studies using shorter repeated sampling over time (e.g., daily experience sampling linked to biological processes; Kamarck et al., 2005) may provide relevant mechanistic data on change.

Finally, a common approach in the studies reviewed above is to examine one potential psychological mechanism at a time. However, it has been argued that one is unlikely to find evidence for a single dominant psychological mechanism given the complexity of links between social support and physical health that unfold over time (Uchino, 2004; also see Ahadi & Diener, 1989). There are other techniques that we believe are underutilized that will allow for an examination of multiple pathways which may be a more accurate way of modeling psychological mechanisms. For instance, the multiple mediator approach based on additive models has had some success in documenting mechanisms responsible for links between SES and health outcomes (Evans & Kim, 2010). In addition, covariance structural modeling (CSM) allows for a test of multiple mechanisms via overall model fit as well as individual pathways. Although CSM models are more reliably estimated based on larger sample sizes, recent developments may allow for such modeling using considerable smaller samples (i.e., Bayesian approach, Lee & Song, 2004). Optimally, these models would take into account the sequence of events that lead to physical health changes so longitudinal designs would also be a particularly powerful complement to this approach.

Potential Explanation 2: Researchers need to focus on emerging and underutilized conceptual approaches of relevance to psychological mediation

There are several salient conceptual issues based on the broader social support and mental health literature that have relevance for this discussion. First is the conceptualization of social support as perceived or received (Dunkel-Schetter & Bennett, 1990). Importantly, these two dimensions are not interchangeable as the separability of perceived and received support is well-documented (Haber et al., 2007). This is important because there is

significant variability in links between received support and health compared to perceived support (Holt-Lunstad et al., 2010; Uchino, 2009). One reason for this is that unsolicited received support that is not responsive to the recipient may negatively influence psychological mechanisms (e.g., threats to independence, decreases in esteem, Bolger & Amarel, 2007; Maisel & Gable, 2009). These reactions may cancel out any positive influences of actually receiving support. The implication of these findings is that researchers examining received support may need to consider an expanded set of psychological mechanisms related to these potential deleterious influences. Although more work is needed, recent laboratory reactivity studies have not been able to consistently identify the direct psychological mechanisms responsible for such adverse effects when they do occur (Hilmert et al., 2002; Taylor et al., 2010).

Recent research is also highlighting the importance of greater attention to the support context. In many cases, social support is typically measured using global assessments that tap into a person's perception of support across contexts (Holt-Lunstad et al., 2010). Similarly, assessments of psychological mechanisms often focus on more global processes such as perceived stress or depression (Uchino, 2004). There is some recent theoretical work highlighting the context of support which has implications for providing a more sensitive test of mediation. Thoits (2011) has argued for a distinction between primary and secondary groups in understanding links between social support and health. Primary group members are typically long-lasting, informal, and consist of close or "significant others." Secondary group members can be short or long-lasting, but are characterized by more formal roles, are less personal, and members can exit or enter such groups on their own discretion (also see Granovetter, 1973). Thoits (2011) predicts that emotional support is more effective coming from primary group members given the closeness characterizing these relationships, whereas informational support is less likely to be effective given such group members' lives are also disrupted by the stressor (which may create impatience, anger, resentment etc.). In addition, such primary social ties are less likely to have direct experience with the stressor (i.e., experiential dissimilarity) and hence their attempts at providing informational support may be off-track, ingenuous, or ineffective. Consistent with this possibility, spouses of chronic disease patients appear to lack insight into their partners' level of pain, fatigue, and physical limitations which can lead to problematic support (Lehman et al., 2011). Secondary group members, in comparison, may be better sources of informational support given their past experience (typically in context, e.g., asking a coworker about a job problem). This perspective suggests that a more sensitive test of psychological mechanisms would need to take into account the support context (e.g., the type of group member) and more specific psychological perceptions of such processes (e.g., experiential similarity).

A second contextual issue that may shed greater light on mechanisms is reflected by work on relational regulation theory (Lakey & Orehek, 2011). These authors have argued that support perceptions develop primarily in the context of daily, mundane interactions (e.g., talking about sports, music, gossip). These interactions help to regulate individuals on a daily basis and serve as the initial basis for expectations about support from particular relationships. This perspective suggests that greater attention to such daily life interactions (as opposed to stressful contexts) may provide a better understanding of the interpersonal and psychological mechanisms associated with support which may then have implications for health. This context also suggests more specific types of psychological mechanisms that unfold in daily life (e.g., general comfort interacting with person) that might be responsible for links between social support and health.

A focus on the more specific context of support emphasizes psychological mechanisms that have been largely unexamined in prior health work. However, researchers may need to expand their consideration of psychological mechanisms more generally. The psychological

mechanisms most examined and postulated in work on social support and health include distress, anxiety, and depression. However, there are other factors that might be health-relevant. Thoits (2011) has argued that "mattering" appears to be an important but unexamined mechanism in prior work. Mattering reflects one's view that one is important and meaningful to others, which also provides purpose to life (Thoits, 2011). Accordingly, this sense of mattering is hypothesized to protect individuals from mental health problems, including "existential despair." This perspective is rooted in social identity perspectives (Thoits, 1983) and suggests additional psychological mechanisms that have been largely untested in work on social support and physical health such as self-esteem and control (Thoits, 2011).

There are also salient psychological mechanisms from a relationship science perspective that may be health-relevant. For instance, researchers Maisel & Gable and Reis and colleagues (Maisel & Gable, 2009; Reis et al., 2004) have presented a more integrated view of the close relationship literature by arguing for the importance of perceived responsiveness. Perceived responsiveness reflects an individual's perception that another is aware of and sensitive to his or her needs (Reis et al., 2004). This construct is related to social support and appears to moderate the influence of received support on adjustment (Maisel & Gable, 2009). That is, receiving support indeed appears beneficial to adjustment if it is perceived as responsive to the recipient (Maisel & Gable, 2009). More generally, there are a number of relevant psychological mechanisms based on the relationship science literature that remains largely untested in social support and health work (e.g., perceptions of intimacy, trust, acceptance, companionship, reciprocity; Reis, 2007). In fact, most of the prior work testing mechanisms has focused on affective or emotional processes so more attention is needed on important cognitive processes based on this larger literature. The emphasis in relationship science is also on how individuals interact and influence each other in specific relational contexts (Berscheid, 1999). As a result, a contextual focus on dyads and the larger family unit may provide a more sensitive test of potential psychological mechanisms.

Finally, social support can be separated into specific functional components such as emotional, informational, and tangible support (Cohen, 1988). Most studies examining links between social support and health examine general support, perhaps with the assumption that "more is better" especially in explaining links to long-term health endpoints. However, not all support dimensions are equivalent and their influence may be moderated by the contextual processes highlighted above (Thoits, 2011). For example, emotional support is seen as a friendly and less controlling form of support compared to informational support (Trobst, 2000). Cohen (1988) has also argued for relatively distinct psychological mechanisms depending on the functional support component. For instance, emotional support should be related to increased self-esteem which facilitates coping with stress (Cohen, 1988). Linking these more specific support components to distinct psychological mechanisms remain largely untested in the physical health literature.

Potential Explanation 3: Models postulating direct psychological mediation are wrong (or at least need important modifications)

As noted earlier in the review, contemporary models of social support and health highlight the importance of psychological mechanisms (Berkman et al., 2000; Cohen, 1988; Uchino, 2004). Is it possible that these models are wrong? This is perhaps the most provocative and potentially controversial explanation but is plausible based on prior work. For instance, consider the following statement from House and colleagues (1988) over 22 years ago:

> We must further understand the psychological and biological processes or mechanisms linking social relationships to health, either as extensions of the social processes just discussed … or as independent mechanisms. In the latter regard,

> psychological and sociobiological theories suggest that the mere presence of, or sense of relatedness with, another organism may have relatively direct motivational, emotional, or neuroendocrinal effects that promote health either directly or in the face of stress or other health hazards but that operate independently of cognitive appraisal or behavioral coping and adaptation (House et al., 1988, pp. 543–544).

The possibility that there is no psychological mediation of links between social relationships on health was also discussed in 1986 when the National Heart, Lung, and Blood Institute and the University of California at Irvine co-sponsored a workshop on social support and cardiovascular disease (Davidson & Shumaker, 1987). This conference brought together leading experts working broadly in the area who noted three distinct ways that social support might be related to cardiovascular health:

> "Three mechanisms that related social support to cardiovascular outcomes suggested in the conference were: 1) support impacts directly on emotions, which, in turn, produce neuroendocrine and hemodynamic responses; 2) support affects behavior which influences physiological responses; and 3) support directly alters cardiovascular risk indicators." (Davidson & Shumaker, 1987, p. 102).

Such a "direct link" explanation is reminiscent of early work in psychophysiology that showed weak associations between psychological reports of affect/arousal and peripheral physiological responses (Skelton & Pennebaker, 1990). This explanation is a salient one because of the consistent lack of findings across different paradigms (e.g., laboratory received support, cross-sectional studies on perceived support). Phrased in terms of the present analysis, if the mediator (e.g., affect) does not predict the outcome (e.g., physiology) it cannot function as a statistical mediator of links between social support and health. From this literature, it has been argued that self-report measures of such affective processes are probably not derived from internal bodily sensations (Skelton & Pennebaker, 1990) as self-reported psychological states can be influenced by other situational cues and individual differences (e.g., neuroticism). Reasons for such discrepancies over the years have varied, however, with some focusing on methodological explanations (e.g., MacKay, 1980), while others suggesting that physiological measures represent a different source of information about person-environment interactions (Cacioppo & Petty, 1986). The later point was often seen as a strength because many early and contemporary social psychophysiological studies on relationships had been aimed at tapping into processes that individuals were either unwilling or unable to report (see Berntson & Cacioppo, 2000).

This issue was recently re-visited by Denson and colleagues (2009) who argued that prior work demonstrating weak links between affect and physiology were flawed because they did not consider more specific appraisals and emotional states. Results of their analyses lead them to conclude that although global affect was not related to these physiological processes, more specific dimensions of appraisals and affect were suggesting stronger links between affect and biology. However, as noted by Miller (2009), their findings could have been due to chance given the number of tests that were performed. In addition, with the exception of links between threat/social evaluation and cortisol responses, few of these findings produced interpretable links that were replicated across different measures. Miller (2009) concluded "So where does this leave us? I'm afraid that the answer is back where we started, with lots of affection for the concept of integrated specificity but little in the way of definitive evidence to refute or accept it."

One intriguing reason why there might be a "direct" effect of relationships on physiological outcomes is due to the possibility that they reflect automatic processes associated with familiarity (Spitzer et al., 1992). Several studies have reported that ambulatory blood

pressure during daily life is lowest when with a family member and highest when with a stranger (e.g., Spitzer et al., 1992). Holt-Lunstad and colleagues (2003) similarly found that social interactions with familial ties were associated with lower ambulatory blood pressure compared to non-familial ties; an effect that was not statistically mediated by self-reported positive affect, negative affect, self-disclosure, intimacy, or influence. Similar to Spritzer and colleagues (1992), these researchers argued that the familiarity associated with family ties has a calming effect on the cardiovascular system which is automatically activated and perhaps reflective of a conditioned response shaped by many years of contact. Consistent with this possibility, Rusbult & Van Lange (2008) in their review of interdependence theory argue that close relationships are often characterized by a habit of responding that requires little or no conscious thought.

If indeed these associations reflect more automatic processes, then paradigms that examine these less conscious processes may represent alternative approaches to testing psychological mechanisms. This possibility is consistent with the general view that people construct working models of their relationships with others as an automatic organizational guide in navigating complex and important social worlds (Baldwin, 1992; Ogilvie & Ashmore, 1991) and that simply calling to mind feelings of connectedness and support can attenuate physiological reactivity to a stressor task (Smith et al., 2004). For instance, Smith and colleagues (2004) used a supraliminal prime by having participants write about a supportive tie or casual acquaintance (e.g., what you appreciate about this person) and then exposed them to acute psychological stressors. Results revealed that writing about a supportive tie was associated with lower cardiovascular reactivity to a subsequent speech stressor compared to writing about a casual acquaintance. In addition, the links between supportive ties and lower cardiovascular reactivity were statistically mediated by decreases in reported state anxiety levels using this paradigm which highlights the increased sensitivity that may be obtained using such protocols.

These automatic processes can also be examined by focusing on brain regions that might coordinate both psychological and physiological response systems as a function of social support. For instance, it is clear that self-report measures of psychological mechanisms reflect complex information processing pathways that may rely on separable neural substrates (Eisenberger, Gable et al., 2007; Lieberman, 2007). Some of these neural substrates are more closely linked to health-relevant physiological alterations than others (Gianaros & O'Connor, 2011). The most commonly activated structures in stress protocols include the dorsal anterior cingulate cortex (sometimes extending into the dorsomedial prefrontal cortex), anterior insula, and periaqueductal gray and the most commonly deactivated structures include the ventral medial prefrontal cortex (Gianaros & O'Connor, 2011; Wager et al., 2009a,b). These structures are particularly important as there are links between these regions and subcorticol brain structures important for autonomic nervous system responses (Gianaros & O'Connor, 2011). These data are thus relevant for stress-related models of support and may provide a more direct test of links between support-related appraisals, emotions, and peripheral physiology (Coan et al., 2006; Eisenberger et al., 2007), as well as other psychological states that individuals are unable or unwilling to report.

How Will This Story End?

Contemporary models clearly postulate psychological mechanisms as theoretically important pathways linking social support to health (Berkman et al., 2000; Cohen, 1988; Cohen, 2004; Thoits, 2011; Uchino, 2004). However, decades of research have failed to directly document these mechanisms. While none of us have self-identified as "Scrooges," the current state of the literature is at best discouraging in terms of direct answers to this

crucial question. It is always difficult to forecast the future of any research domain. However, we believe that continuing to test for psychological mechanisms as we are currently doing is unlikely to lead to answers to this question. As a result, we argue that systematic attention to each of the explanations presented may significantly enhance our understanding of potential psychological mechanisms. In fact, consideration of these issues in epidemiological research should receive priority. This literature has been focused on documenting important links between social support and morbidity/mortality and is now poised to ask important "2nd generation" questions regarding mechanisms.

One interesting question is why this issue has not received sufficient attention over the years. One potential reason is due to the fact that documenting links between social support and physiological outcomes continues to be a primary research agenda because of its direct implications for health. Such studies are playing a key role in testing the biological plausibility of social support as a potential risk factor. As a result, it is relatively easier to ignore these null findings for psychological mechanisms in the context of this larger research question. However, the weight of evidence regarding what we know about social support and health versus its psychological mechanisms is so unbalanced as to hinder attempts at theoretical modeling or the design of well-informed interventions. Of course, this means that we need to be doing both - preferably in studies that also examine health outcomes so that direct evidence (or lack thereof) for psychological mechanisms can be obtained. Based on the current set of issues and recommendations, even some null findings for such tests (e.g., Potential Explanation 3) can be informative so we would encourage researchers to consider performing such analyses in their future work.

To facilitate the search for psychological mechanisms, we have thus discussed a number of potential explanations. Our suggestions range from more straight-forward statistical and design issues, like minimizing measurement error, to more radical suggestions that our models might simply be wrong. In truth, we believe systematic attention to each of these potential explanations can foster our modeling of psychological mechanisms and many of these can be examined in combination. For instance, future work can examine multiple psychological mechanisms simultaneously (Potential Explanation 1) that can also be informed by advances in the conceptualization of support, a consideration of the support context, and related work in relationship science (Potential Explanation 2).

We would also like to highlight particularly noteworthy "ghosts of the future" that we believe to be especially promising areas of inquiry. First, there has consistently been a call for more longitudinal research linking social support to various outcomes (Potential Explanation 1), especially because cross-sectional links between social support and psychological processes may represent support-seeking for more stressful events (Barrera, 2000). Given the longstanding nature of this issue and the lack of such studies in the health domain it is time to again call attention to the importance of this issue. Of course such longitudinal designs are time consuming and expensive. Thus, research using daily experience sampling may complement such studies by providing important information on short-term processes that tend to be stable over time (Kamarck et al., 2005).

Second, we view the integration of relationship science with health-relevant social support and health work to be critical to the goal of "updating" models postulating psychological mechanisms. Relationship science has examined the contextual processes related to relationship functioning, including social support (Berscheid, 1999; Gottlieb, 1985; Reis et al., 2000; Reis, 2007). However, very little work has directly modeled these relationship-specific processes as mechanisms possibly linking social support to health-relevant outcomes (e.g., responsiveness: Reis, 2007). In addition, a focus on dyads and families from a relationship science perspective can provide a more sensitive context for examining such

mechanisms. Although progress in integrating these areas are being made, we recommend a stronger relationship science approach to understanding psychological mechanisms linking social support to health.

Finally, if model testing is hindered because some of these psychological mechanisms reflect processes that individuals are unable or unwilling to report (Potential Explanation 3, Davidson & Shumaker, 1987; House et al., 1988), testing our models may require alternative paradigms (e.g., neural mechanisms, less conscious protocols) that are then used to infer psychological significance. Examining these, as well as other explanations, may then provide stronger inferences over the long-term for the provocative possibility that our models might be wrong or are at least in need of important modifications. For instance, implicit measures of relationship processes appear uniquely related to outcomes and hence need consideration in future work (Lee et al., 2010).

In conclusion, social support is one of the most well-documented psychosocial factors linked to physical health (Holt-Lunstad et al., 2010). However, over 25 years of research has failed to directly uncover the psychological mechanisms responsible for such links. We view this paper as simply the first step in raising an issue that is in need of much greater systematic attention. So unlike the story that serves as the analogy for the present review, our ending is incomplete and not nearly as satisfying. Future research will ultimately complete this story for us at perhaps an accelerated pace by paying greater attention to the messages of these “ghosts.”

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Highlights

- Reviews almost 30 years of research that directly examined the evidence for models that propose psychological pathways linking social support and health.
- No evidence was found that psychological factors were mediators of links between social support and biological outcomes.
- Future research avenues are suggested to test these models (e.g., expanded set of mediators).
- Raises the possibility that our existing theoretical models are wrong or in need of important modifications.

Table 1

Potential explanations for lack of psychological mediation for associations between social support and health outcomes.

Potential Explanation	Specific Issues
Statistical and Design	Measurement error
	Less precise statistical tests of mediation
	Lack of longitudinal designs examining change
	Single mediator modeling
Emerging or Underutilized Conceptual Considerations	Difference between perceived and received support
	Lack of attention to the support context
	Limited testing of potential mediators
	Specific functional support components
Models Wrong or Need modification	Lack of correspondence between self-reported affect and biology
	Alternative approaches
	- Brain imaging
	- Less conscious protocols