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Depression, social support, and long-term risk for coronary heart disease in a 13-year longitudinal epidemiological study

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ABSTRACT

Depression has been linked with long-term risk for a variety of physical health ailments, including coronary heart disease (CHD). Little is known about resilience factors that may attenuate this relationship. The current study assessed whether social support moderates the long-term risk for CHD associated with this disorder. Data were drawn from the Americans' Changing Lives study, a nationally representative longitudinal survey of adults in the United States. Participants (unweighted n=1636) completed initial assessments of functional social support, body mass index, recent history of major depression, CHD, hypertension, and diabetes. Participants were again assessed for CHD at a follow-up assessment 13 years later. Social support was found to moderate the relationship between depression and the occurrence of CHD 13 years later. Specifically, among individuals with low social support, depression was prospectively associated with CHD. In contrast, depression was not prospectively associated with CHD among individuals with high social support. The results indicate that social support may function as a resilience factor against the long-term cardiovascular risk associated with depression. Clinical interventions focusing on the development of social support systems are important not only for addressing depression itself, but also for associated long-term physical health outcomes.

1. Introduction

Among psychiatric disorders, depression ranks highest in terms of its burden to society. Out of all physical and mental health conditions, depression is the second leading cause of disability in the U.S. and worldwide (Ferrari et al., 2013; US Burden of Disease Collaborators, 2013). This disorder accounts for approximately 9.6% of years lived with disability (YLDs) and 3.0% of global disability adjusted life years (DALYs; Ferrari et al., 2013). The global burden of this disorder increases substantially when considering its association with coronary heart disease (CHD). Indeed, after accounting for the approximately 4 million CHD DALYs (as well as 16 million suicide DALYs) attributed to depression, the overall burden of this disorder increases to 3.8% of global DALYs (Ferrari et al., 2013).

This link between depression and CHD has been empirically well established (Goldston and Baillie, 2008; Lett et al., 2004; Whooley and Wong, 2013). Specifically, depression is associated with a relative risk of 1.5–2.0 for developing CHD, as well as a worsening course in individuals with CHD, with an estimated relative risk of 1.5–2.0 for eventual cardiac morbidity and mortality (Lett et al., 2004). The strength of this association between depression and CHD has led the American Heart Association Science Advisory to recommend routine screening of depression in all patients with CHD (Lichtman et al., 2008).

Given that individuals with depression are at greater risk for developing CHD and tend to have a more negative prognosis once this condition develops, it remains imperative to identify resilience factors that may moderate the relation between depression and CHD, particularly ones that may exert a long-term effect, thereby potentially altering the risk trajectory of this physical health condition in these individuals. Resilience has been defined as a reduction in vulnerability to risk experiences, overcoming adversity, and the experience of a positive outcome in spite of the presence of a risk factor (Rutter, 2006). It is therefore a necessarily interactive, or moderating, construct (Rutter, 2012). Elucidating resilience factors relevant to the relation between depression and CHD has the potential to inform intervention

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efforts for at-risk individuals with depression.

One resilience factor of potential relevance is social support. Particularly influencing the concept of social support as a resilience factor relevant to physical health conditions are the observations of Cassel (1976) and Cobb (1976) that more socially integrated patients tended to exhibit a better prognosis in recovering from somatic illness and in maintaining symptom remission. Elaborating on this view, the buffering hypothesis proposes that social relationships provide resources that facilitate adaptive coping in response to a stressful experience, or illness in the case of stress-related disorders such as depression (Cohen et al., 2001). These resources essentially buffer against the detrimental effect of the stressor or illness.

There has been previous criticism about the lack of clarity with which social support has often been measured and defined (Coyne and Bolger, 1990). Although social support has been widely studied in the context of depression and CHD, past research has tended to focus on structural support (i.e., the size and frequency of contact within the individual's social network) rather than functional support (Compare et al., 2013). The concept of functional support relates to the quality, as opposed to the size, of the individual's social network (Lett et al., 2005). Specifically, it refers to the support received from the individual's social structure, and can include emotional support and support in the form of advice or appraisal. Importantly, there is some evidence that functional is more important than structural support in understanding risk for CHD (Lett et al., 2005).

Many of the studies in this area assessed for main effects in the relation between social support and CHD (Compare et al., 2013), leaving relatively unexamined the important concept of social support as a resilience factor buffering against the risk for CHD associated with depression. In fact, one review of the literature specifically identified the need for research on potential moderating relations between social support and depression on this physical health condition (Lett et al., 2005). One of the few studies in this area did not find functional support to be a resilience factor moderating the relation between depression and mortality or recurrence of acute myocardial infarction (AMI) in a sample of patients with recent AMI (Lett et al., 2007). As moderation was determined by visual inspection rather than tests of simple slopes, however, these findings must be interpreted with a degree of caution. In contrast, another study found functional support to buffer the effects of depression on one-year mortality in a sample of patients with AMI, such that when social support was high, depression was no longer associated with increased mortality risk (Frasure-Smith et al., 2000).

The current study aims to extend the existing literature in several ways in testing functional social support as a long-term resilience factor against the risk of CHD associated with depression. In contrast to prior studies that focused exclusively on AMI patient samples, we examined the interaction between functional support and depression on long-term risk for CHD in a more generalizable context, an epidemiological sample of middle-aged to older adults. To provide perhaps the most conservative evaluation of this relation, well-established risk factors for CHD were covaried, including hypertension, diabetes, body mass index (BMI), and pre-existing CHD at baseline. Additionally, whereas prior tests of this interaction have compared only minimal relative to mild depressive symptom severity (i.e., Beck Depression Inventory scores ≥10), the current investigation evaluated the relevance of this relation to clinically significant depression (i.e., major depression). To provide an especially rigorous assessment of functional support, the current study examined the long-term buffering effect of this resilience factor on CHD status over a 13-year follow-up period.

2. Methods

2.1. Participants and procedures

Data were drawn from the Americans' Changing Lives (ACL) study, a nationally representative longitudinal survey of adults in the United States conducted by the Survey Research Center of the University of Michigan. Further information about the ACL study is provided elsewhere (House et al., 1990). For the purpose of this study, we used data collected in Wave 2 and 13 years later at Wave 4, as major depression was not assessed at Wave 1. At Wave 2, participants (unweighted n =2,846; 55.90% female; $M_{age\ at\ Wave\ 2}$ =44.97, SE =0.35; $M_{years\ in\ school}$ =13.05, SE =0.08) completed assessments of depression, social support, pre-existing CHD, hypertension, diabetes, and BMI. They were assessed again for past-year CHD at Wave 4. In terms of racial composition, 86.6% of participants at Wave 2 were White, 9.0% were Black, 3.9% were American Indian and 2.1% were Asian.[1] A total of 1,642 participants (unweighted) completed the 13-year follow-up assessment, 397 (unweighted) attrited, and 807 (unweighted) were deceased. The weighted racial composition at follow-up was: 86.6% White, 9.0% were Black, 3.9% were American Indian and 2.1% were Asian (percentages exceed 100% due to rounding).

2.2. Measures

2.2.1. Major depression

At Wave 2, major depressive episodes over the past three years were determined using a diagnostic interview following DSM-IV criteria. This diagnostic interview included 10 symptom questions reflecting the nine symptoms of major depression. Respondents were asked about the presence of these symptoms during their most recent onset of depressed mood within this three-year span (for more information on the assessment of major depression in the ACL study, see Maciejewski et al., 2001).

2.2.2. Social support

Friend/relative functional social support was assessed with two questions: (1) "On the whole, how much do your friends and other relatives make you feel loved and cared for?" and (2) "How much are these friends and relatives willing to listen when you need to talk about your worries or problems?" Responses for each item were measured on a 5-point scale ranging from 1 ("not at all") to 5 ("a great deal"). The internal consistency of this measure was adequate (Cronbach's alpha =0.73; for additional information on the creation and use of this measure, see Harvey and Alexander, 2012; House and Kahn, 1985; Lynch, 1998).

2.2.3. Physical health

BMI, and past-year experiences of hypertension, diabetes, and heart attacks and other heart troubles were each assessed by self-report (for another example of epidemiological research employing self-report measures of physical health, see McWilliams and Bailey, 2010). CHD was assessed at Wave 2 and Wave 4 with the question: *"Have you had a heart attack or other heart trouble during the last 12 months?"* Self-reports of physical illnesses have been shown to be reliable and valid (Iacovino et al., 2016; Idler et al., 1999), particularly when compared to medical records (Barr et al., 2002; Colditz et al., 1986, 1987). BMI was calculated by dividing respondents' self-reported weight by height, with a BMI greater than or equal to 25 kg/m^2 defined as overweight. Supporting the validity of this self-report measure, Little to no racial or sex differences have been found between Wave 2 BMI and BMI derived by measuring height and weight in the Third National Health and Nutrition Examination Survey (NHANES III; Kuczmarski et al., 1994). Furthermore, in a systematic review of assessments of BMI, self-reports were considered to be reliable estimates of BMI in adults (Connor Gorber et al., 2007).

[1] Percentages add up to more than 100% because of rounding.

Table 1
Hierarchical logistic regression model of prospective predictors of coronary heart disease (unweighted n =1642).

Predictor	Coronary Heart Disease
	Odds Ratio (95% CI)
Step 1	
Baseline Coronary Heart Disease	4.94 (2.47–9.85)***
Hypertension	1.73 (1.07–2.80)*
Diabetes	0.96 (0.42–2.20)
BMI > 25 kg/m^2	1.36 (0.89–2.09)
Male Sex	1.03 (0.67–1.60)
Age (years)	1.03 (1.02–1.05)***
Education (years)	0.97 (0.90–1.05)
Step 2	
Baseline Coronary Heart Disease	4.86 (2.46–9.60)***
Hypertension	1.69 (1.04–2.74)*
Diabetes	0.98 (0.43–2.22)
BMI > 25 kg/m^2	1.41 (0.92–2.16)
Male Sex	1.04 (0.66–1.64)
Age (years)	1.03 (1.02–1.05)***
Education (years)	0.97 (0.90–1.05)
Major Depression	1.41 (0.79–2.49)
Functional Social Support	1.00 (0.80–1.25)
Step 3	
Baseline Coronary Heart Disease	5.01 (2.52–9.95)***
Hypertension	1.62 (0.99–2.66)
Diabetes	0.95 (0.42–2.16)
BMI > 25 kg/m^2	1.39 (0.91–2.14)
Male Sex	1.06 (0.67–1.67)
Age (years)	1.03 (1.02–1.05)***
Education (years)	0.98 (0.91–1.06)
Major Depression	1.26 (0.67–2.37)
Functional Social Support	0.55 (0.28–1.07)
Major Depression × Functional Social Support	2.07 (1.02–4.17)*

CI = confidence interval.
* $p < 0.05$.
*** $p < 0.001$.

2.3. Data analysis

To assess whether functional support moderated the relation between depression and prospective CHD, a hierarchical logistic regression analysis was conducted with Wave 4 CHD as the criterion variable. In Step 1, Wave 2 CHD,[2] hypertension, diabetes, BMI were entered to account for baseline indicators of standard risk factors for CHD on its prospective occurrence, and age, sex, and years of education were entered to covary for sociodemographic characteristics. In Step 2, depression and functional support were entered.[3] In Step 3, the interaction term for depression and functional support was entered into the model. Following recommended procedures for probing significant interactions (Aiken and West, 1991), simple slope analyses were conducted assessing the strength of the relation between Wave 2 depression and Wave 4 CHD at one *SD* above and below the mean for the functional support variable. In simple slope analyses, the covariates from Step 1 of the hierarchical logistic regression model were retained.

Sampling weights were used in all analyses to account for the complex sampling design of this nationally representative study, and thereby arrive at accurate population estimates. A four-stage stratified sampling process was achieved with an aim of arriving at a nationally representative sample on a range of characteristics, including geographical location and population at the county level, as well as county size and income. In such epidemiological surveys, it is necessary to incorporate sample weights in the analyses to account for the stratified design and to adjust for geographical and race differences in response rates, and in the case of follow-up waves, differential rates of retention. The sampling weight used in the current study was specifically created for conducting longitudinal analyses with Wave 2 and Wave 4 data combined.

3. Results

We first conducted weighted χ^2 analyses to evaluate whether participants who completed Wave 4 differed from those who did not in terms of Wave 2 depression and CHD status. No differences were observed (*ps* ≥0.407). At Wave 2, 14.47% of the participants met diagnostic criteria for major depression. In the 12 months prior to Wave 2, 16.82% had hypertension, 4.22% had CHD, and 3.88% reported having diabetes. Additionally, 54.05% had a BMI greater or equal to 25 kg/m^2 (M_{BMI} =26.14, *SE* =0.15). The participants with CHD at Wave 4 constituted 8.96% of the full sample. Of those with CHD at Wave 2, 38.32% also had it at Wave 4.

In the hierarchical logistic regression model, after account for the effects of Wave 2 CHD, hypertension, diabetes, age, sex, BMI, years of education, neither major depression nor functional support was significantly associated with CHD at 13-year follow-up. When the interaction between depression and functional support was then entered into the model, it was found to be significant (OR =2.07, 95% CI =1.02 – 4.17; see Table 1). After this interaction was decomposed, simple slope analyses revealed that when functional support was low (−1 *SD*), major depression was predictive of CHD 13 years later, even after accounting for initial CHD, hypertension, diabetes, age, sex, BMI, years of education (OR =10.12, 95% CI =2.58–39.75). In contrast, when functional support was high (+1 *SD*), major depression was no longer prospectively associated with CHD status (OR =0.79, 95% CI =0.07–8.62). This interaction is illustrated in Fig. 1.

4. Discussion

The current investigation provided the largest and longest evaluation of the long-term effect of functional social support as a resilience factor on the relation between major depression and CHD. Consistent with the buffering hypothesis, functional support moderated the relation between major depression and CHD such that depression was predictive of CHD when functional support was low, but not when functional support was high. Importantly, this relation held even after several well-known risk factors for CHD were covaried, and demonstrated the long-term beneficial influence of functional support, persisting 13 years after its initial assessment. It should also be noted that although significant main effects for depression and function support were not observed, this does not invalidate the finding of a significant interaction. A main effect is not a necessary (nor sufficient) condition to investigate potential moderators. In fact, often moderating effects have empirical or theoretical basis in the absence of a main

[2] The inclusion of Wave 2 CHD as a covariate in our analyses was based on the following considerations. Covarying Wave 2 CHD was intended to strengthen the conclusion that the interaction between depression and social support is predictive of Wave 4 CHD, over and above CHD status at Wave 2. Although there is clear appeal in restricting analyses to individuals with no prior CHD history at Wave 2, so as prospectively to predict first lifetime CHD, this was not possible in the current study as the Wave 2 measure of CHD was only for the past year. That is, it was very possible (and indeed quite likely) that some participants had a prior history of CHD but were in remission at Wave 2. Indeed, although CHD often follows a chronic, unremitting course, this is by no means always the case. As an example of high remissions rates with a specific subtype of CHD, 32–44% of new cases of angina pectoris in the Framingham Heart Study were found to achieve remission based on a rather strict criteria (no symptoms over two full years; Kannel and Sorlie, 1978). Given that we could not account for CHD prior to the 12 months immediately preceding Wave 2, excluding those with Wave 2 CHD would have made it more unclear whether or not pre-existing CHD, rather than the interaction between social support and depression, accounted for Wave 4 CHD.

[3] Although it would be possible to include changes in depression and social support between Waves 2 and 4, doing so with just two time-points would effectively reduce the study from a prospective one to a cross-sectional one, as the end-points of the independent variables and the dependent variable would have been the same. Given the focus of the current study on assessing the long-term temporal prediction of CHD by the interaction between depression and social support, the interaction between these predictor variables was assessed at Wave 2.

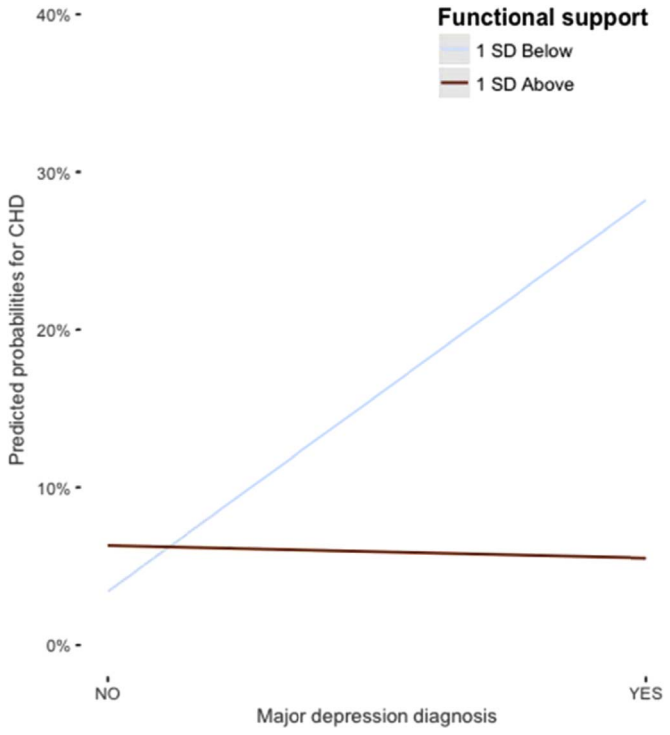

Fig. 1. Functional social support as a moderator of the relationship between major depression and coronary heart disease at follow-up.

effect (e.g., Abramson et al., 1989; Van Orden et al., 2010). Indeed, this has been asserted to be the case with resilience factors (Rutter, 2012).

An important consideration for future investigation are potential processes through which high social support may be associated with reduced risk for CHD among depressed individuals. One possibility is that a healthy social support system may promote healthy behaviors in the individual, countering the negative association between depression and physical activity (Roshanaei-Moghaddam et al., 2009). Indeed, functional support from friends and relatives has previously been linked to greater engagement in physical activity (Harvey and Alexander, 2012), which, itself, has been negatively associated with risk for CHD (Lee et al., 2012). Interestingly, recent research has demonstrated an association between loneliness and pro-inflammatory response to acute stress (Jaremka et al., 2013), which, in turn, has been associated with risk for CHD (Ridker et al., 1997). Additionally, elevated concentrations of pro-inflammatory cytokines have consistently been found in depressed individuals (Dowlati et al., 2010). Within the context of the current findings, it may be that a healthy social support system may be a resilience factor relevant to CHD through an attenuation of pro-inflammation associated with depression.

The buffering effect of social support in the association between depression and CHD is all the more relevant when considered within the context of several theories of depression positing a deleterious effect of this disorder on interpersonal relationships. According to Coyne's (1976) interpersonal theory of depression, for example, individuals susceptible to depression seek the care and interest of significant others as well as reassurance from others in an attempt to confirm their self-worth. Depression-prone individuals tend to discount the initial reassurance they receive as being insincere, causing them to solicit additional reassurance. The repetitive pattern of seeking and discounting reassurance continues until it causes irritation and frustration in others, leading to deterioration of the relationship and rejection of the depression-prone individual. Empirical support has been found for this tendency to engage in excessive reassurance-seeking to be associated with both depression (Starr and Davila, 2008) and deteriorating interpersonal relationships (Joiner et al., 1992).

Similarly detrimental to depression-prone individuals' interpersonal relationships is the tendency for such individuals to be particularly sensitive to social rejection (Downey and Feldman, 1996). This rejection sensitivity has been proposed to be deleterious to interpersonal relationships through self-fulfilling behavioral tendencies. Indeed, rejection-sensitive individuals and their romantic partners have been found to experience greater dissatisfaction in their relationship (Downey and Feldman, 1996) and are more likely to experience the dissolution of their relationship (Downey et al., 1998).

Collectively, the implication of these theoretical models of depression and associated empirical findings is that depressed individuals, who as a group are already at risk for CHD, may unknowingly increase their risk further still by diminishing the very resilience factor (i.e., social support) that could buffer them against the risk for this physical health condition. If this is indeed the case, clinical interventions that improve or maintain existing social support by targeting these dysfunctional interpersonal mechanisms would be important both for treating depression and preventing its downstream consequences in the form of CHD.

This study is not without its limitations. In particular, all health-related data were derived from self-report. Although, as previously noted, self-report data of BMI and physical health conditions have been found to be reliable and valid (Connor Gorber et al., 2007; Iacovino et al., 2016; Idler et al., 1999), future research should corroborate the current findings with data derived from medical evaluations. Furthermore, future studies would benefit from inclusion of additional detail regarding the different forms of CHD experienced by participants, medications or treatments they received for this condition, and indices of the severity of CHD. Additionally, antidepressant medication usage was not assessed, which has been associated with reduced risk for CHD-related hospitalizations among depressed individauls (Cooper et al., 2014). It would therefore be important to account for this variable in future studies. Although the aim of the current study was to evaluate the long-term prognostic value of the interaction between social support and depression, it should also be noted that these variables were only assessed at baseline, which did not allow for an evaluation of temporal changes in relation to CHD during the longitudinal phase of the study. That this interaction was associated with CHD status 13 years later in a manner consistent with the buffering hypothesis, even without accounting for changes in depression or social support over the same time period, is compelling support for its predictive power. Nonetheless, future research would benefit from assessing these variables at multiple time-points so as to allow for more temporally dynamic evaluation of their relation with CHD. Finally, although the current study documented the buffering effect of functional support on long-term risk for CHD, further research is needed to assess the generalizability of the current findings by determining whether functional support similarly serves as a resilience factor against the relation between depression and mortality specifically due to heart-related conditions.

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