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From Loss to Loneliness: The Relationship Between Bereavement and Depressive Symptoms

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Spousal bereavement can cause a rise in depressive symptoms. This study empirically evaluates 2 competing explanations concerning how this causal effect is brought about: (a) a traditional latent variable explanation, in which loss triggers depression which then leads to symptoms; and (b) a novel network explanation, in which bereavement directly affects particular depression symptoms which then activate other symptoms. We used data from the Changing Lives of Older Couples (CLOC) study and compared depressive symptomatology, assessed via the 11-item Center for Epidemiologic Studies Depression Scale (CES-D), among those who lost their partner ($N = 241$) with a still-married control group ($N = 274$). We modeled the effect of partner loss on depressive symptoms either as an indirect effect through a latent variable, or as a direct effect in a network constructed through a causal search algorithm. Compared to the control group, widow(er)s' scores were significantly higher for symptoms of loneliness, sadness, depressed mood, and appetite loss, and significantly lower for happiness and enjoyed life. The effect of partner loss on these symptoms was not mediated by a latent variable. The network model indicated that bereavement mainly affected loneliness, which in turn activated other depressive symptoms. The direct effects of spousal loss on particular symptoms are inconsistent with the predictions of latent variable models, but can be explained from a network perspective. The findings support a growing body of literature showing that specific adverse life events differentially affect depressive symptomatology, and suggest that future studies should examine interventions that directly target such symptoms.

Keywords: bereavement, depressive symptoms, latent variable model, loneliness, networks

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Major depressive disorder (MDD) is a highly prevalent disease (Kessler, Chiu, Demler, Merikangas, & Walters, 2005), and the majority of patients diagnosed with depression suffer from severely impaired functioning (Kessler et al., 2003). Experiencing an adverse life event, in turn, is a well-established predictor for developing depression (Hammen, 2005; Mazure, 1998), and de-

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pression rates are increased in individuals exposed to severe stress (Rojo-Moreno et al., 2002; Shrout et al., 1989). This has been documented in both clinical and community samples (Brown & Harris, 1989; Hammen, 2005).

A diagnosis of MDD requires the presence of at least five of the nine *DSM-5* criterion symptoms (American Psychiatric Association, 2013). These symptoms are commonly assessed via screening instruments such as the Beck Depression Inventory (BDI; Beck, Steer, & Garbin, 1988) and calculated by summing the number of symptoms one has. The idea underlying sum-scores is that depression symptoms are interchangeable indicators of the same unidimensional underlying disorder. This is called the common-cause hypothesis (Schmittmann et al., 2013), and in statistical models, reflective latent variables are used to describe this direction of causation. In such reflective models, changes in the latent variable (depression) lead to changes in the observed indicators (the symptoms). From this perspective, depression symptoms such as sadness, insomnia, or fatigue covary because they are triggered by the latent disease. Symptoms are regarded as measurements of depression, and aggregated symptoms reflect a person's position on the latent variable. The common cause for depression is often assumed to reside in the brain of individuals diagnosed with MDD (e.g., Andreasen, 2001). If depression symptoms are understood as passive consequences of an underlying brain dysfunction, then identifying and treating such a common cause is indeed the most logical procedure.

In recent years, however, a growing body of evidence has challenged the common cause model for depression. First, the *DSM-5* diagnosis for depression encompasses a large number of disparate symptoms such as sadness, insomnia, or appetite problems, and three of the symptoms consist of contrasting features (psychomotor retardation or psychomotor agitation; weight gain or weight loss; insomnia or hypersomnia). This leads to about 1,500 unique symptom profiles that all qualify for the same diagnosis (Østergaard, Jensen, & Bech, 2011), including profiles that do not share a single symptom. For example, one recent paper documented 1,030 unique symptom profiles in 3,703 patients diagnosed with depression (Fried & Nesse, 2015). Although it is possible that a disease causes various syndromes—syphilis, for instance, is often referred to as "the great imposter" for that reason—it is unlikely that it causes many symptomatic opposites. Second, individual depressive symptoms vary with respect to their risk factors (Fried, Nesse, Zivin, Guille, & Sen, 2014), and their underlying biology (Kendler, Aggen, & Neale, 2013; Myung et al., 2012). Some symptoms show greater heritability than others, with heritability factors ranging from 0.0 to 0.35 (Jang, Livesley, Taylor, Stein, & Moon, 2004). Third, the etiology of depressive symptoms is complex and multifactorial, featuring biological, psychological, and environmental influences (Kendler, 2012). Fourth, cross-sectional studies have documented that specific life events such as failing at an important goal or the death of a loved one are associated with particular depression symptom profiles (Cramer, Borsboom, Aggen, & Kendler, 2013; Keller, Neale, & Kendler, 2007; Keller & Nesse, 2005, 2006).

Novel network models offer an alternative perspective to the common cause framework. In these approaches, depressive symptoms are not understood as passive and interchangeable indicators of a latent disease, but as distinct entities with autonomous causal power that influence each other (Borsboom & Cramer, 2013; Cramer, Waldorp, van der Maas, & Borsboom, 2010). Symptoms such as insomnia or fatigue do not cluster because of a common cause—they cluster because they influence each other across time. Depression is not conceptualized as latent variable, but is understood to be constituted by the causal associations among symptoms.

Here we examine the impact of one specific adverse event—late-life spousal loss—on a variety of depressive symptoms in a prospective study of older bereaved spouses with matched control participants. Losing a loved one is a strong and well-established risk factor for the onset of depressive symptomatology (Zisook & Kendler, 2007; Zisook & Shuchter, 1991), and a large literature has documented the impact of bereavement on psychological functioning, especially among older adults (Carr, Nesse, & Wortman, 2006; Knight & Silverstein, 2014). We aim to address two main questions.

First, it is unclear how spousal bereavement affects depressive symptoms. From the perspective that depression is the common cause of its symptoms and thus explains symptom covariation, bereavement should affect a latent depression factor, which in turn should cause the symptoms (i.e., the effect of loss on symptoms is indirect and operates through the latent variable). The alternative hypothesis is that the effects are direct and propagated through a symptom network. In this case, one would expect that the life event triggers specific depressive symptoms which, in turn, activate other symptoms in a causal chain. To compare these competing hypotheses, we used data from the Changing Lives of Older Couples (CLOC) study, a prospective study of spousal loss among older adults (Carr et al., 2006). We fit both latent variable models and network models to the data and compare and discuss the results.

Second, the question of whether bereavement is conceptually distinct from MDD has been discussed for decades and remains unresolved. The bereavement exclusion (BE) introduced in the *DSM–III* (American Psychiatric Association, 1980) conceptualized grief as normal response to loss and not as a mental disorder. The *DSM–IV* (American Psychiatric Association, 2000) narrowed down the BE substantially in order to avoid false-negatives, and the BE was replaced in the *DSM-5* by a footnote that "caution[s] clinicians to differentiate between normal grieving associated with a significant loss and a diagnosis of a mental disorder" (American Psychiatric Association, 2013, p. 161). This decision was based on several systematic reviews documenting very few differences between bereavement-related depression and depression (Kendler, Myers, & Zisook, 2008; Zisook et al., 2012). Others have argued that bereavement is a normal and uncomplicated response to loss, noting that symptoms usually subside within weeks or months of the death; grief persists for a prolonged period of time among only a small minority of bereaved persons (Kersting, Brähler, Glaesmer, & Wagner, 2011). From this perspective, the removal of the BE brings the dangers of misdiagnosing normal sadness as pathological depression and medicalizing a normal condition (Bonanno et al., 2002; Friedman, 2013; Nesse & Stein, 2012; The Lancet, 2012; Wakefield, 1997). Our analysis of symptom dynamics among recently widowed individuals may offer new insights into the question of whether bereavement-related depression is a distinct condition.

Methods

Participants

Data from the CLOC study (Carr et al., 2006) were analyzed to examine the impact of bereavement on depression symptoms. A

prospective sample of 1,532 married men and women age 65 or older from the Detroit Metropolitan Area were enrolled. Participants were English-speaking, noninstitutionalized, and able to participate in a 2-hr face-to-face interview. Individuals who lost a spouse during the course of the study were invited to follow-up interviews at 6, 18, and 48 months after their partner's death.

We used data from the first follow-up interview (Wave 1) 6 months after spousal loss. Of the 335 individuals who had lost a spouse, 250 (74.6%) participated in the Wave 1 interview. Bereaved participants were matched regarding age and gender with control participants from the baseline sample who had not lost a partner. Because of the funding constraints, the number of controls at Wave 1 was small ($N = 84$). In our analysis, we thus pool control subjects from all three follow-up waves ($N = 280$).

Outcome Measures

Depressive symptoms were measured with the 11-item version of the Center for Epidemiologic Studies Depression Scale (CES-D) on each measurement occasion (Kohout, Berkman, Evans, & Cornoni-Huntley, 1993); this scale is an abbreviated version of the original 20-item CES-D (Radloff, 1977). For each item, participants indicated the frequency with which it had occurred during the past week. Response categories were "hardly ever," "some of the time," or "most of the time." The 11 CES-D items are (abbreviated names used in the remainder of this text in brackets): "I felt depressed" (depr), "I felt that everything I did was an effort" (effort), "My sleep was restless" (sleep), "I was happy" (happy), "I felt lonely" (lonely), "People were unfriendly" (unfr), "I enjoyed life" (enjoy), "My appetite was poor" (appet), "I felt sad" (sad), "I felt that people disliked me" (dislike), and "I could not get going" (getgo).

Because the behavior of skewed polytomous items in networks, such as CES-D symptoms, is not well understood, we dichotomized item-scores into an absent (0) and present (1) code. Such networks of binary variables can then be studied using the Ising model (van Borkulo et al., 2014). For the nine negative items, "hardly ever" was coded as absent symptom, whereas "some of the time" and "most of the time" were coded as present symptoms. Because the two items *enjoy* and *happy* are reverse-coded in the CES-D (where a high value indicates less frequent depressive symptoms), we dichotomized them accordingly. "Hardly ever" and "some of the time" were coded as being absent, and "most of the time" as being present. We reversed the two positive items in analyses of sum-scores.

Statistical Analysis

Because of item-specific missing data on any of the 11 CES-D items, nine participants in the widowed group and six participants in the control group were excluded. This leaves 241 bereaved and 274 nonbereaved participants in the analytic sample.

We compared the widowed and the control groups regarding their overall symptom load (the CES-D sum-score) at baseline and at follow-up, using Welch two sample *t* tests; these tests adjust the number of degrees of freedom when the variances of the compared groups are not equal to each other. Furthermore, we used multivariate analysis of variance (MANOVA)[1] to investigate whether individual symptoms differed across groups.

We then assessed two competing hypotheses that offer different explanations for the ways that bereavement affects depressive symptoms. First, the common cause perspective predicts that a latent depression variable explains symptom covariation. As such, bereavement should affect a latent depression factor, which in turn should cause the symptoms: The effect of loss on symptoms is indirect and operates via a latent variable. To test this assumption we estimated two multiple indicators multiple causes (MIMIC) models (Jöreskog & Goldberger, 1975). MIMIC models contain a reflective latent variable (depression), items that indicate the presence of the latent variable (11 depressive symptoms), and one or more variables that have an impact on the latent variable (bereavement). We set up the first model (Model 1) so that the spousal loss was only allowed to affect the latent variable. This was then compared to a nested Model 2 in which loss was allowed to directly affect symptoms (not mediated by the latent factor). If Model 2 fit the data significantly better, this means that the common cause framework (Model 1) does not describe the data well. Consistent with previous publications (Fried et al., 2014; Jones, 2006), we estimated Model 2 in an iterative process. In a first step, bereavement was allowed to have direct effects on all symptoms except for one symptom for purposes of identification. In a second step, nonsignificant paths were removed until only significant estimates remained. The weighted least squares means and variance adjusted estimator was used to fit the models, and models were compared with a χ^2 difference test. Model fit was examined using the root mean square error of approximation (RMSEA; $\leq .06$ indicating a good fit) and the comparative fix index (CFI; $\geq .95$ indicating a good fit) (Hu & Bentler, 1999).

Second, the network approach offers an alternative explanation in which the effects of loss on symptoms are propagated through a symptom network. To explore this hypothesis, we constructed a network through a causal search algorithm. In networks, each node represents a symptom, and the connections (called "edges" in the network literature) between nodes can be understood as direct influences. Consistent with the previous analysis, we integrated spousal loss into the model to examine whether it is connected to the network, and if so, to which symptoms. The network was fitted using an Ising model (van Borkulo et al., 2014) via the *R*-package IsingFit. An Ising model is a probabilistic model in which the joint distribution over K binary variables (11 items and the loss variable) is represented using threshold parameters (related to the marginal probability of endorsement of any individual item) and pairwise association parameters (related to the associations between the variables). An unconstrained Ising model for our data has 12 threshold parameters and $(12 * 11)/2 = 66$ pairwise association parameters to be estimated. Of main interest are the pairwise associations that are represented as a network. These pairwise association parameters are similar to partial correlation coefficients for continuous normally distributed variables: They are direct associations between nodes controlling for all other associations. More pairwise association parameters in the model lead to a more complex model (with possibly many spurious

[1] Instead of the MANOVA we also used a logistic regression approach to better account for the binary nature of the symptom variables. Since the results were essentially unchanged, we report the conceptually simpler MANOVA.

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connections). For this reason, the method employed here uses an estimation procedure with a penalty approach (i.e., eLasso based on the Extended Bayesian Information Criterion or EBIC; for further details, see Ravikumar, Wainwright, & Lafferty, 2010) to identify only the relevant relationships between variables. A detailed explanation of the Ising model, the estimation procedure, and its properties can be found elsewhere (van Borkulo et al., 2014).

Results of the causal search algorithm are visualized using the *R*-package qgraph (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012). The position of the nodes in the network is based on the Fruchterman–Reingold algorithm, which iteratively computes the optimal layout so that nodes with stronger and/or more connections are placed closer together (Fruchterman & Reingold, 1991).

We used Mplus v7.2 (Muthén & Muthén, 2012) to fit the latent variable models; all other tests and models were estimated in *R* v3.1.0 (R Development Core Team, 2008).

Results

Sample Characteristics

In total, 241 widows and widowers and 274 controls were included in the analytic sample ($n = 515$). Demographic characteristics of the two groups are described in Table 1. The mean age of the study participants during the enrollment phase was 73.34 years ($SD = 6.55$), and 85.4% were female. The widowed group was assessed 6 months after spousal loss, a mean of 37.3 months ($SD = 18.2$) following the baseline measurement. For the control participants who were pooled from all three waves (Wave 1: $n = 81$; Wave 2: $n = 151$; Wave 3: $n = 42$), the follow-up interview took place significantly later than in the widowed group (Table 1, row 3). We tested whether the three waves of control participants differed from each other. There were no differences regarding gender or CES-D sum-score (all comparisons $p > .05$). Participants in Wave 3, however, were significantly older than participants in Wave 1 ($p = .01$, $d = .50$, *CI* [.11, .88]) and Wave 2 ($p = .01$, $d = .43$, *CI* [.08, .77]) (no significant difference between Waves 1 and 2; Wave 1: $M = 72.94$, $SD = 6.75$; Wave 2: $M = 73.45$, $SD = 6.51$; Wave 3: $M = 76.17$, $SD = 5.92$). To test whether this age difference of Wave 3 biased the results, we repeated all analyses reported below with the control participants of Wave 3 excluded. The results were unchanged, and we thus retained all participants in the main analyses.

The five most frequent causes of spousal death were heart attacks (29.5%), cancer (25.3%), arteriosclerosis and related conditions (12.4%), strokes (8.7%), and emphysema (5%). Bereaved and nonbereaved participants did not differ significantly by gender (Table 1, row 1) or age (Table 1, row 2).

Symptom Differences at Baseline

At baseline, the widowed and control groups did not differ significantly on the CES-D sum-scores (Table 1, row 4). A MANOVA revealed no significant differences on the 11 CES-D symptoms between the groups (Table 1, row 5).

Symptom Differences After Spousal Loss

Six months after spousal loss, the mean CES-D score in the widowed group was significantly higher than in the control group (Table 1, row 6). A MANOVA documented significant overall differences between the two groups in their endorsed symptoms (Table 1, row 7). As the results in Figure 1 show, univariate post hoc tests revealed that this was due to the specific symptoms: *lonely* ($p < .001$), *sad* ($p < .001$), *happy* ($p < .001$), *enjoy* ($p < .001$), *depr* ($p = .02$), and *appet* ($p = .02$).

We identified 87 widows and widowers who endorsed at least six of the 11 CES-D symptoms. In this subsample, the most common symptoms were *lonely* and *sad* (both: $M = .98$, $SD = .15$), followed by *depr* ($M = .90$, $SD = .31$), *getgo* ($M = .80$, $SD = .40$), *sleep* ($M = .78$, $SD = .42$), and *appet* ($M = .54$, $SD = .50$).

Depression as Latent Variable

It is commonly assumed that life events such as bereavement increase the likelihood of developing depression, which in turn causes its symptoms. We tested this hypothesis by fitting two MIMIC models to the CLOC data: Model 1 represents the common cause assumption, whereas Model 2 allows bereavement to directly affect symptoms.

As the results in Table 2 reveal, the χ^2 difference test indicated that the less constrained Model 2 fit the data substantially better than Model 1 ($p < .001$). Although the RMSEA and CFI showed

Table 1
Characteristics of the Widowed Group and the Control Group

	Widowed group ($n = 241$)		Control group ($n = 274$)				
	M	*SD*	*M*	*SD*	Difference tests	Significance	Effect size, confidence interval
1. Gender (female)	86.3%	.34	84.7%	.36	$t(509.57) = .53$	$p = .60$	$d = .04$, *CI* (−.13, .22)
2. Age (years)	72.26	6.53	73.87	6.56	$t(515.69) = 1.57$	$p = .12$	$d = .12$, *CI* (−.05, .29)
3. Months since baseline	37.33	18.19	51.76	16.59	$t(489.46) = 9.39$	$p < .001$	$d = .83$, *CI* (.65, 1.02)
4. Baseline CES-D sum	3.53	2.78	3.23	2.59	$t(484.96) = 1.27$	$p = .21$	$d = .11$, *CI* (−.06, .29)
5. Baseline MANOVA					$F(1, 11) = 1.26$	$p = .24$	$\eta^2 = .03$, *CI* (.02, .05)
6. Follow-up CES-D sum	4.17	2.84	2.87	2.54	$t(497.63) = 5.45$	$p < .001$	$d = .49$, *CI* (.31, .66)
7. Follow-up MANOVA					$F(1, 11) = 17.92$	$p < .001$	$\eta^2 = .28$, *CI* (.25, .31)

Note. CES-D = Center for Epidemiologic Studies Depression Scale; MANOVA = multivariate analysis of variance.

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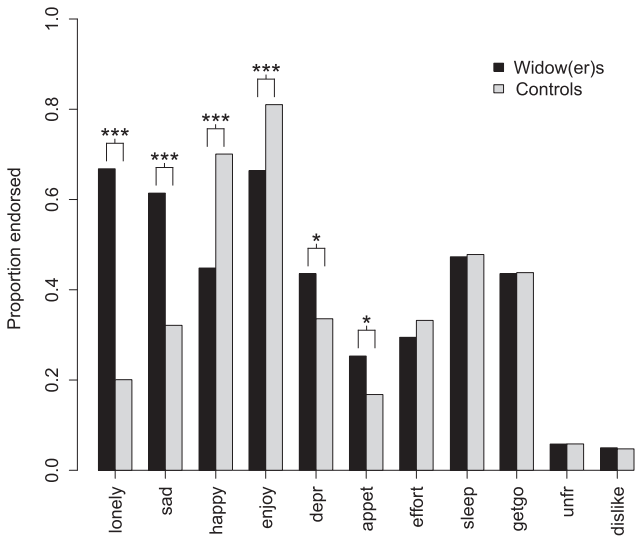

Figure 1. Means of the 11 Center for Epidemiologic Studies Depression Scale (CES-D) items in the control group and widowed group six months after spousal loss. Symptoms are ordered by the degree of severity differences between groups. Statistically significant group differences denoted as * $p < .05$, ** $p < .01$, *** $p < .001$.

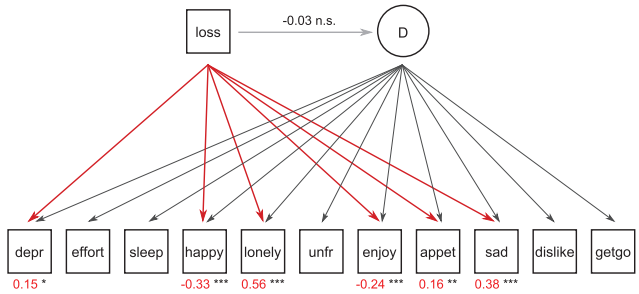

Figure 2. Schematic visualization of the Multiple Indicators Multiple Causes (MIMIC) Model 2. The red (dashed) lines depict significant direct effects of spousal loss (loss) on Center for Epidemiologic Studies Depression Scale (CES-D) items; standardized estimates of these effects are presented in red below the symptoms. All CES-D items load significantly on the latent factor (D) (black lines, estimates omitted to preserve clarity). In Model 2, the condition does not significantly affect the latent variable (gray line). depr = depressed; effort = everything is an effort; sleep = restless sleep; happy = feeling happy; lonely = feeling lonely; unfr = people are unfriendly; enjoy = enjoy life; appet = poor appetite; sad = feeling sad; dislike = people dislike me; getgo = cannot get going. Statistically significant effects denoted as * $p < .05$, ** $p < .01$, *** $p < .001$. See the online article for the color version of this figure.

that Model 2 described the data better than Model 1, they were not satisfactory in either model, and the χ^2 statistic for both models was highly significant ($p < .001$; see Table 2), rejecting both Model 1 and Model 2.

As a result of the iterative fitting process described in the Methods section, Model 2 identified six significant direct effects of bereavement on depressive symptoms after controlling for the latent variable (ordered by the strength of the standardized estimates: lonely, sad, happy, enjoy, appet, and depr) (see Figure 2). These are the exact symptoms for which the MANOVA revealed significant differences between the bereaved and control groups (see Figure 1). The effect of spousal loss on the latent variable was significant in Model 1 (.28, $p < .001$) but was no longer significant in Model 2 when bereavement was allowed to directly affect symptoms ($-.03$, $p = .67$). Overall, these results suggest that the common cause model does not account well for the impact of spousal loss on depression symptoms.

It is possible that a factor model different from the one-factor solution used in the MIMIC models above would lead to different results. More precisely, there may be a latent depression factor on which only specific CES-D items load, through which bereavement hits the symptoms increased in the bereaved group. We searched for the best factor solution using an exploratory factor analysis (EFA), estimated a confirmatory factor analysis (CFA) with two factors based on the results obtained in the EFA, and fitted two 2-factor MIMIC models based on the CFA results; the analyses are described in detail in the online supplementary materials. In sum, the results of the additional analyses were not substantially different from the ones reported above, and a different factor solution does not allow for a different general interpretation of the findings.

Table 2
Goodness-of-Fit Statistics and χ^2 Difference Test for the Two MIMIC Models

MIMIC models	χ^2	*df*	RMSEA	CFI	χ^2_{diff}	df_{diff}	*p*
Model 1[a]	288.67	54	.09	.90			
Model 2[b]	171.39	48	.07	.95	124.69	6	<.001

Note. RMSEA = root mean square error of approximation; CFI = comparative fit index; $\chi^2_{diff} = \chi^2$ statistic of the χ^2 difference test; df_{diff} = degrees of freedom of the χ^2 difference test; $p = p$ value of the χ^2 difference test; MIMIC = multiple indicators multiple causes.
[a] No direct paths from bereavement to symptoms. [b] Six direct effects from bereavement to symptoms.

Depression as Symptom Network

In a next step, we used a causal search algorithm that interprets symptoms and spousal loss as nodes in a network. Results of the model are shown in Figure 3. Overall, bereavement has a very strong impact on *lonely*, which in turn is mainly associated with *sad* (positively) and *happy* (negatively). From there, activation spreads through the network. The six symptoms in closest proximity to the spousal loss node in Figure 3 are those found to be significantly different among bereaved persons versus matched controls (lonely, sad, happy, enjoy, appet, and depr). Bereavement also exhibits weak negative associations with *happy* and *effort*. As expected, the two positively worded items *happy* and *enjoy* are strongly positively related in the network, but show pronounced negative associations with other negative affective items such as *lonely*, *depr*, and *sad*.

Discussion

We examined the impact of spousal loss on bereavement in a prospective study of widowed persons and matched controls and evaluated two competing hypotheses: The effect of partner loss

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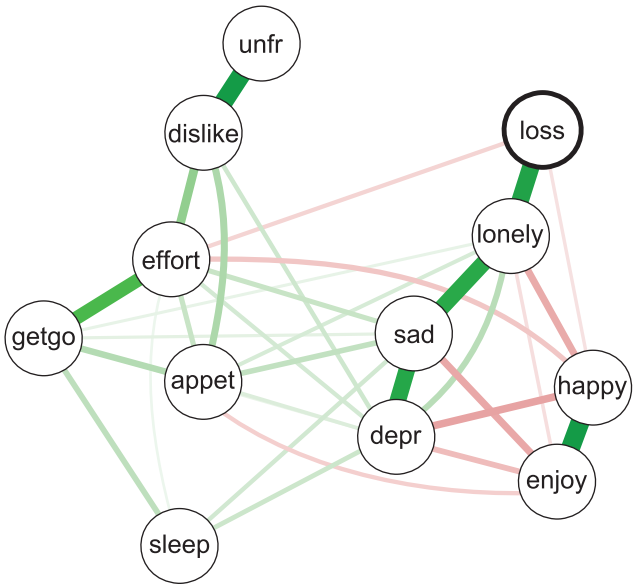

Figure 3. Network of 11 Center for Epidemiologic Studies Depression Scale (CES-D) items and spousal loss (loss). depr = depressed; effort = everything is an effort; sleep = restless sleep; happy = feeling happy; lonely = feeling lonely; unfr = people are unfriendly; enjoy = enjoy life; appet = poor appetite; sad = feeling sad; dislike = people dislike me; getgo = cannot get going. Green (solid) lines represent positive associations, red (dashed) lines negative associations, and the thickness and brightness of an edge indicate the strength of the association. The layout is based on the Fruchterman-Reingold algorithm that places nodes with stronger and/or more connections closer together. See the online article for the color version of this figure.

affects depression symptoms indirectly via a latent variable, or as a direct effect propagated through a symptom network. We found that particular symptoms such as loneliness, sadness, and loss of appetite were especially elevated in the context of bereavement, and that the effects from loss on these symptoms were not conveyed via a latent variable, but through a network. Loneliness played a key role: Bereavement mainly affected loneliness, which in turn activated other depressive symptoms.

Loneliness, however, was not only the gateway symptom that led from loss of a loved one to the development of further depressive symptoms, it was also the most pronounced negative CES-D item in both bereaved groups analyzed in this report—the 241 participants in the overall sample as well as the subgroup of 89 individuals with especially high symptom load. Our results are consistent with prior empirical research documenting that the large majority of widowed individuals describe loneliness as the biggest challenge to cope on a daily basis (Lund, 1989; see also Utz, Swenson, Caserta, Lund, & deVries, 2014). Our findings are especially relevant in the light of recent work highlighting loneliness as risk factor for compromised physical and mental health among older adults. Loneliness predicts morbidity and mortality (Luo, Hawkley, Waite, & Cacioppo, 2012) as well as reduced daytime functioning (Hawkley, Preacher, & Cacioppo, 2010), and is associated with suicidal ideation (Stroebe, Stroebe, & Abakoumkin, 2005). Moreover, lonely older adults exhibit higher levels of risky health behaviors such as smoking and physical inactivity (Shankar, McMunn, Banks, & Steptoe, 2011), and are more likely to develop Alzheimer's disease (Wilson et al., 2007) and depressive symptoms (Cacioppo, Hawkley, & Thisted, 2010). Furthermore, researchers have detected associations between loneliness and elevated systolic blood pressure, increased hypothalamic pituitary adrenocortical activity, decreased levels of sleep, and reduced immune functioning (Hawkley & Cacioppo, 2003; Masi, Chen, Hawkley, & Cacioppo, 2011).

Our results suggest that practitioners who work with older adults should recognize the distinctive needs of bereaved individuals with pronounced feelings of loneliness, and that intervention programs should directly target loneliness. Loneliness following widowhood can be considerably complicated, and there is also recent evidence that social support alone is not sufficient to remedy feeling lonely (Utz et al., 2014). Various approaches have been developed to specifically target loneliness (Peplau & Perlman, 1982); cognitive–behavioral therapy with a focus on maladaptive social cognitions have been found to be especially effective in randomized controlled trials (Masi et al., 2011). Such strategies may play a crucial role to prevent the onset and maintenance of depression and a range of other adverse outcomes following spousal loss.

Interestingly, a large community study of adults aged 50 years or older revealed that loneliness and depressive symptoms affect each other reciprocally and lead to diminished well-being (Luo et al., 2012). These results suggest that bereavement may have an especially severe impact on loneliness initially, and that the subsequent activation of other depressive symptoms initiates feedback loops that relate back to feeling lonelier than before; this, in turn, might prevent the individual taking action to reduce the loneliness. Such self-reinforcing loops may play a major role in bereavement, and their importance has been documented recently in persistent complex bereavement disorder (Robinaugh, Leblanc, Vuletich, & McNally, 2014).

Research Implications

Our findings have three important implications. First, although the idea that patients suffering from mental health problems are caught in vicious circles of problems that fuel each other is certainly not new, it is incompatible with the assumption of a common cause. Interestingly, it is consistent with the way clinicians tend to think about mental disorders (Kim & Ahn, 2002), and also with the way subjects describe their own symptoms (Frewen, Allen, Lanius, & Neufeld, 2012; Frewen, Schmittmann, Bringmann, & Borsboom, 2013). Moreover, studies using experience-sampling methods support the hypothesis of interacting symptoms (Wichers, 2014), and causal symptom chains are a well-established concept in the psychotherapy literature (Beck, Rush, Shaw, & Emery, 1979; Nolen-Hoeksema, 2000; van der Heiden, Muris, & van der Molen, 2012). Understanding depressive symptomatology as a complex system rather than as a latent variable may offer crucial insights into underlying causal mechanisms otherwise obfuscated, and we suggest that the study of such mechanisms could contribute to answering important questions. For example, mood disorders are estimated to be at least moderately heritable (Boomsma, Busjahn, & Peltonen, 2002), but identified risk alleles can explain—if anything at all—only small proportions of the variance (Hek et al., 2013; Shi et al., 2011; Wray et al., 2012). The network perspective

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suggests that large parts of the "missing heritability" (Johnson, Penke, & Spinath, 2011; Zuk, Hechter, Sunyaev, & Lander, 2012) may lie in the connections between symptoms rather than in the symptoms themselves.

Second, depressive symptomatology and underlying causal mechanisms may vary across different life events. This is consistent with cross-sectional studies documenting that particular life events are associated with specific symptoms (Cramer et al., 2013; Keller et al., 2007; Keller & Nesse, 2005, 2006). Previous reports are inconclusive regarding whether adverse events led to particular symptoms or vice versa. For example, failing to achieve an important goal may cause concentration problems, but concentration may also make it more likely to fail to achieve an important goal—our prospective analysis allows the conclusion that bereavement triggered particular depressive symptoms. Other adverse events, such as chronic stress or romantic breakups, likely lead to the development of depressive symptoms different to those elevated in the context of bereavement (Keller et al., 2007). It may thus be crucial to pay close attention to the etiology that precedes depressive symptoms to provide patients with the most effective targeted intervention strategies. To complicate things further, symptom profiles may differ among bereaved participants depending on the cause, timing, context, or perceived controllability of death of a loved one (Carr, House, Wortman, Nesse, & Kessler, 2001; Carr, 2009). The bereaved participants analyzed in this report may benefit from an intervention centered on loneliness, but this may be different for losses associated with specific events such as suicides (Mitchell, Kim, Prigerson, & Mortimer-Stephens, 2004), vehicle accidents (Lehman, Wortman, & Williams, 1987), or among widow(er)s who maintained poor-quality or distant relationships with their late spouse(Carr et al., 2000).

Third, the *DSM-5* diagnosis of MDD in its current form may not be a very useful category, seeing that depressed individuals experience very diverse problems (e.g., Fried & Nesse, 2015). From this perspective, it is not surprising that there has been little progress in identifying biomarkers for depression diagnosis and treatment response (Hek et al., 2013; Tansey et al., 2012), and that the *DSM-5* field trials revealed a questionable interrater reliability of MDD diagnosis that was much lower than the majority of other mental disorders (Regier et al., 2013). These concerns relate to the original question whether bereavement is conceptually different from MDD, and whether the BE was warranted. Because we did not compare nonbereaved depressed with bereaved depressed participants in this study, our findings cannot settle this question. Nonetheless, results of the latent variable models show that spousal loss does not increase a general depression factor, but instead affects very specific symptoms. Moreover, the network analysis revealed particular dynamics between spousal loss and MDD symptoms that are likely specific to bereavement. The results thus speak to the possibility that losing a loved one sets in motion a specific chain of symptoms, and from this perspective it is questionable to include bereaved individuals into the already highly heterogeneous disease category MDD.

Limitations

The results should be interpreted in the light of a number of limitations. First, most participants were female, and the bereaved sample was, on average, not clinically depressed. Results may thus not generalize to other bereaved samples, especially ones with high levels of depressive symptomatology. Although we did examine the prevalence of symptoms in a subsample of 84 widows and widowers who endorsed at least six MDD symptoms, this group was not sufficiently large to fit a network model. Future studies will be required to examine the validity of our findings in samples with higher levels of psychopathological load.

Second, the CLOC sample is limited to older adults who were born in the United States in the early 20th century; as such, the study findings may not be generalizable to younger persons or to other birth cohorts. Recent work suggests that older adults have lower levels of emotional reactivity than their younger counterparts, which may generate a more constrained range of responses to the CES-D items than those detected among younger or midlife adults (e.g., Charles & Carstensen, 2013).

Third, the 11-item CES-D neither assesses all *DSM-5* criterion symptoms, nor does it cover detailed information about compound symptoms such as sleep problems. Because particular subsymptoms such as poor sleep quality and difficulty in initiating and maintaining sleep—but not early morning awakening—are predictive of subsequent depression symptoms in the elderly (Jaussent et al., 2011), compounds should be more closely examined in future studies.

Fourth, although we established that the bereaved and control groups did not differ in their endorsed symptoms at baseline, and that profiles were different in widow(er)s after experiencing spousal loss, both the latent variable model and the network model were constructed with cross-sectional data. To ascertain unequivocal causality between symptoms (e.g., loneliness > sadness > appetite problems/depressed mood), a study using many time points that are very closely spaced in time would be required.

Finally, there are limitations that derive from the novelty of network models. In contrast to the latent variable models, we could not investigate the absolute goodness of fit of the Ising model, because such fit indices are not yet developed for networks; this is also why we could not examine whether the latent variable model or network model describes the data better in a direct statistical test (i.e., a χ^2 difference test). Furthermore, we dichotomized CES-D symptoms because it is currently not feasible to use highly skewed polytomous items in causal search algorithms. Although we believe that the network approach offers great opportunities, future studies will have to address these current shortcomings.

Conclusions

To our knowledge, this is the first study that empirically evaluates the common cause and the network approach to illuminate the association between spousal loss and depressive symptoms. The association pattern between symptoms is far more complex than the common cause model can explain, and the network reveals that bereavement mostly triggers loneliness, which activates further depressive symptoms. Novel statistical network models thus offer insights into underlying mechanisms obfuscated in common cause models, and differential treatment implications emerge on the basis of such new understandings.

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