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Deconstructing and Reconstructing Resilience: A Dynamic Network Approach

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Abstract

Resilience is still often viewed as a unitary personality construct that, as a kind of antinosological entity, protects individuals against stress-related mental problems. However, increasing evidence indicates that maintaining mental health in the face of adversity results from complex and dynamic processes of adaptation to stressors that involve the activation of several separable protective factors. Such resilience factors can reside at biological, psychological, and social levels and may include stable predispositions (such as genotype or personality traits) and malleable properties, skills, capacities, or external circumstances (such as gene-expression patterns, emotion-regulation abilities, appraisal styles, or social support). We abandon the notion of resilience as an entity here. Starting from a conceptualization of psychiatric disorders as dynamic networks of interacting symptoms that may be driven by stressors into stable maladaptive states of disease, we deconstruct the maintenance of mental health during stressor exposure into time-variant dampening influences of resilience factors onto these symptom networks. Resilience factors are separate additional network nodes that weaken symptom–symptom interconnections or symptom autoconnections, thereby preventing maladaptive system transitions. We argue that these hybrid symptom-and-resilience-factor networks provide a promising new way of unraveling the complex dynamics of mental health.

Keywords

resilience, stress, mental health, dynamic system, symptom network, emotion regulation

Mental illness, such as in the form of depressive or fear- and anxiety-related disorders, is currently an important cause of disability worldwide. Mental illness not only affects the life of the inflicted person but also can have a profound and long-lasting impact on a patient's social environment and on society. Stressful life experiences (e.g., trauma, difficult life circumstances, challenging life transitions, or physical illness) are important risk factors for the development of mental disorders (Green et al., 2010). For instance, adverse experiences in childhood account for an estimated 44% of childhood-onset mental-health disorders and about 30% of adult-onset mental-health disorders (Green et al., 2010; Kessler et al., 2010). However, not all individuals exposed to adversity develop lasting mental problems. So, whereas about 90% of people in Western countries experience at least one potentially traumatizing event in their lives,

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the lifetime prevalence of posttraumatic stress disorder (PTSD) is estimated to be only 8% (de Vries & Olff, 2009; Kilpatrick et al., 2013), and summed lifetime prevalences of any mental disorder lie between 20% and 50% (Kessler et al., 2007). In line with these observations, many prospective studies have shown that approximately two thirds of adults confronted with significant stressors show either no psychological or functional impairment or recover quickly (Bonanno, Westphal, & Mancini, 2011; Werner, 1992). These data suggest that the study of resilient individuals and of the mechanisms that protect them from lasting dysfunction is one crucial strategy for discovering new ways of battling stress-related disease (Kalisch et al., 2017).

Resilience refers to the maintenance and/or quick recovery of mental health during and after times of adversity (Kalisch et al., 2017). It is becoming increasingly clear that resilience—as opposed to lasting stress-induced mental and functional impairments—is the result of a dynamic process of successfully adapting to stressors (Bonanno, Romero, & Klein, 2015; Kalisch et al., 2017; Kent, Davis, & Reich, 2014; Rutter, 2012; Sapienza & Masten, 2011). Indeed, there is now ample evidence that individuals change while they cope with stressors. Such changes can take the form of psychological growth or maturation processes (Johnson & Boals, 2015; Joseph & Linley, 2006; Tedeschi & Calhoun, 2004); emergence of new skills and competences (Luthar, Cicchetti, & Becker, 2000); stress inoculation—that is, an immunization against the deleterious effects of future stressors (Seery, Holman, & Silver, 2010; Seery, Leo, Lupien, Kondrak, & Almonte, 2013); or modification in epigenetic marks and gene expression (Boks et al., 2015; Breen et al., 2015). Furthermore, neurobiological studies of animals have identified changes in central-nervous-system functions that are causal for the preservation of normal adaptive behavior (e.g., Friedman et al., 2014; Krishnan et al., 2007; Maier, 2015; Wang, Perova, Arenkiel, & Li, 2014). Hence, resilience is not simply insensitivity or unresponsiveness to stressors or only a passive response to adversity (Russo, Murrough, Han, Charney, & Nestler, 2012). In the same vein, resilience should no longer be understood simply as a stable, fixed trait or predisposition (the "resilient-personality" or "resilience-gene" misconception) that guarantees long-term mental health whatever stressor the organism is exposed to. Rather, resilience research must attempt to understand the complex, interactive, and time-varying processes that lead to a positive outcome in the face of adversity (Kalisch et al., 2017; Rutter, 2012).

One of the most interesting recent developments in clinical psychology and psychiatry is the discovery that mental disorders can be understood as networks of symptoms that interact (i.e., causally affect one another; Borsboom, 2017; Cramer, Waldorp, van der Maas, & Borsboom, 2010; Hofmann, Curtiss, & McNally, 2016; Kendler, Zachar, & Craver, 2011). This approach is different from traditional psychiatric thinking, which views mental disorders as nosological entities and is concerned with finding the single common underlying cause for the various distinct symptoms that define a given mental disorder. The traditional approach essentially copies a disease model successfully used in somatic disease research in which, for instance, a lung tumor may be found to cause chest pain, shortness of breath, and the coughing up of blood (Borsboom, 2017). Then, if the single cause (i.e., the tumor) is removed early enough, this also removes the symptoms (Borsboom, 2017). However, such simple and unique pathogenic pathways have never been found to be the case in depression, pathological anxiety, PTSD, or other complex stress-related diseases. The network approach, by contrast, abandons the idea of one underlying cause—or a few underlying causes—and instead assumes that disease symptoms cause each other; that is, each of a number of symptoms may be a starting point for the activation of other symptoms. A psychiatric disorder is thus a network of interrelated symptoms. Here, we start by briefly explaining the idea of modeling mental disorders as symptom networks and then explore to what extent this idea might also be helpful for understanding resilience as resulting from a dynamic process of adaptation.

Network Modeling of Mental Disorders

The key tenet of network modeling of mental disorders is that psychiatric symptoms interact such that the activation of one symptom (or network "node") promotes the activation of another symptom or symptoms. Such interactions may occur via biological, psychological, and social mechanisms (Borsboom, 2017; Cramer et al., 2010; Fried & Cramer, 2017; Kendler et al., 2011). As an example for a possible biological mechanism of interaction, frequent hypervigilance, worrying, and anxiety (schematically depicted as symptom node S1 in Fig. 1) may lead to high levels of stress hormones; these may impair prefrontal function, including executive control, which in turn may lead to problems in social functioning, another type of typical stress-related symptoms (S2 in Fig. 1). Via a psychological mechanism of interaction, the frequent negative social feedback coming with social dysfunctions may lead to a generalized negative appraisal of one's abilities and competences, which may induce feelings of despair or hopelessness (the depressive symptom node S3). Via a social mechanism, social dysfunction may also lead to a lack of social

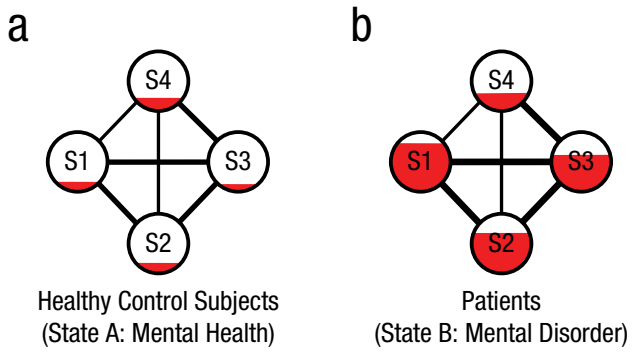

Fig. 1. Mental disorders modeled as networks of symptoms. Graphs show a symptom network consisting of nodes S1 through S4, which represent symptoms 1 through 4. For illustrative purposes only, S1 in the text always refers to a symptom cluster of anxiety, worry, and fear; S2 always refers to social dysfunctions; S3 always refers to depressive symptoms; and S4 always refers to somatic symptoms. The strength of connections (edges) between symptoms is shown by the thickness of the connecting lines. So, symptoms S1, S2, and S3 are highly interconnected (strongly statistically associated). The network in (a) represents a group of healthy subjects in which symptom activation—shown by the red filling of the nodes (arbitrary values)—is low. The network in (b) represents a patient group with several symptoms showing high, correlated activation. Symptoms are measured at one time point, and the corresponding cross-sectional (contemporaneous) network models do not contain information on the directionality of connections (is S1 caused by S2, or vice versa, or both?). For the sake of simplicity, here and in subsequent figures, we show only positive symptom connections. Note, however, that psychiatric symptoms may occasionally also inhibit each other (interact negatively).

support from others, which in turn can further enhance anxiety (S1). In the network model shown in Figure 1, S4 represents somatic symptoms of tension, exhaustion, pain, or irritability, another typical class of stress-related symptoms. In the remainder of the article, we refer to these broad symptom classes (S1–S4) that are found in most stress-related disorders to facilitate the discussion.

Network models formulate the tendency of a symptom to activate another symptom by giving the edge that connects the corresponding network nodes a strong positive weight (expressed in Fig. 1 by the strong thick line between, e.g., S1 and S2). Such connections express the purely statistical relation (usually a partial correlation) between two variables (symptom nodes) as estimated from empirical data in a cohort of subjects. As such, they do not establish causality. Further, they do not inform the directionality of the causal interactions that presumably underlie the observed statistical relation. Underlying causal interactions may be unidirectional or reciprocal. It may be that anxiety and worry (S1) generate somatic stress symptoms (S4) or that somatic stress symptoms (S4) generate anxiety or worry (S1), or both. In the simplest form of a cross-sectional network model as shown in Figure 1 (i.e., a model that incorporates data from only one measurement time point), this cannot be resolved. The same limitation applies when data from several measurement time points are analyzed in isolation (i.e., when modeling does not include time-lagged correlations between symptoms).

However, the more interesting case of dynamic time-series models, as shown in Figure 2, permits connections between symptom x at time point t and symptom y at time point $t + 1$ (time-lagged symptom–symptom interconnections) as well as between symptom x and itself at subsequent time points (time-lagged symptom autoconnections, expressing the extent to which a symptom tends to maintain its own activation; Fried & Cramer, 2017). In principle, at least, this allows one to test whether changes in symptom x systematically precede changes in symptom y or in itself and thereby to establish temporal directionality (consistent with Granger causality; Epskamp et al., 2018). It further allows one to also observe possible vicious cycles that unfold over time through reciprocal connections or also through positive feedback loops involving more than two nodes. For instance, socially induced anxiety (S1) may increase stress hormone levels and thus reduce

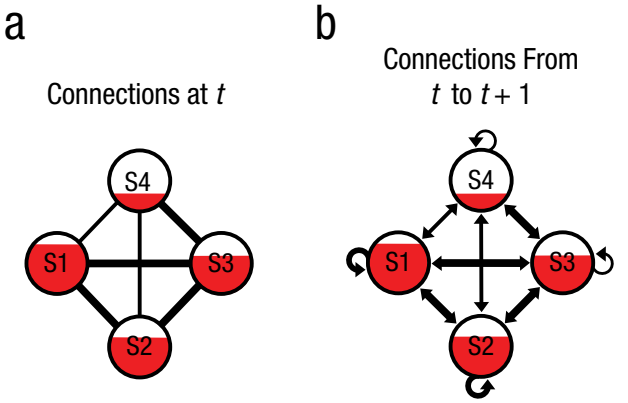

Fig. 2. Dynamic network models. When symptoms (S) are measured at several time points t, this allows for estimating both cross-sectional (contemporaneous) symptom connections (at the same time point t; a) and longitudinal (time-lagged or temporal) symptom connections (from t to $t + 1$; b), including autoconnections. Temporal connections are directional, as indicated by the arrowheads. For simplicity, here and in subsequent figures, all temporal connections between a given pair of nodes are depicted as reciprocal and equivalent. Note, however, that temporal symptom–symptom connections may well also be unidirectional and that reciprocal connections may be of different strengths. In this and subsequent figures it is assumed that symptom measurements will occur at more or less equidistant time points t in most studies (e.g., every 3 months ± 1–2 weeks). However, the network-modeling approach is not principally limited to studies with discrete and equidistant measurement time points. Further, despite measurements usually being available from only relatively few discrete time points, it could in principle also represent continuous time processes. The strength of connections (edges) between symptoms is shown by the thickness of the connecting lines.

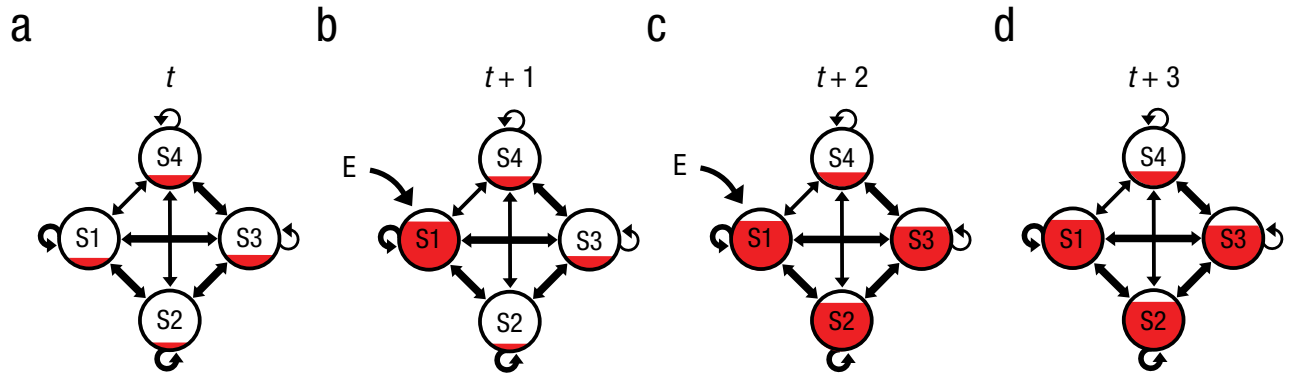

Fig. 3. Evolution of disease states in dynamic network models. Only temporal connections (connections from one time point t to the next time point $t + 1$) are shown; cross-sectional connections at any given time point are omitted for simplicity. In (a), no symptom (S) is strongly active: There are no stressors. The system is in a stable state of mental health. In (b), an external factor or stressor (E) drives the activation of S1. In (c), S2 and S3 are now also highly active, and the system has reached a new state of mental disorder characterized by the coactivation of the highly interconnected S1, S2, and S3. This state can be stable (self-sustaining) in that it persists even if the stressor has ceased (d). The strength of connections (edges) between symptoms is shown by the thickness of the connecting lines.

prefrontal executive function and social functioning (S2) and increase despair (S3) and anxiety of social failure and isolation (S1).

It is crucial for our discussion to state that networks that exhibit strong connectivity—that is, networks in which it is relatively easy for one symptom to activate another because they are strongly associated—are vulnerable. This means that, at a certain level of activation, these networks tend to develop self-sustaining states of general high activation of several strongly interconnected symptoms, which would then be called a mental disorder (Cramer et al., 2016; Fig. 3). Symptom activation may initially be caused by factors that are external to the network, such as a stressor in the environment or in one's own body (e.g., a physical disease or pain; E in Fig. 3b; Borsboom, 2017; Cramer et al., 2016). But once activation reverberates within the network, external activation is no longer necessary to maintain network activation (Fig. 3c and 3d).

The abrupt transitioning of the system into a new stable state of disease can be compared to a chemical reaction that occurs only when the system has crossed an energy barrier (Kramers, 1940; see Fig. 4a). Like a temporary rise in temperature of the surrounding medium can induce a relatively rapid state transition in chemistry, stressors may provide the activation energy

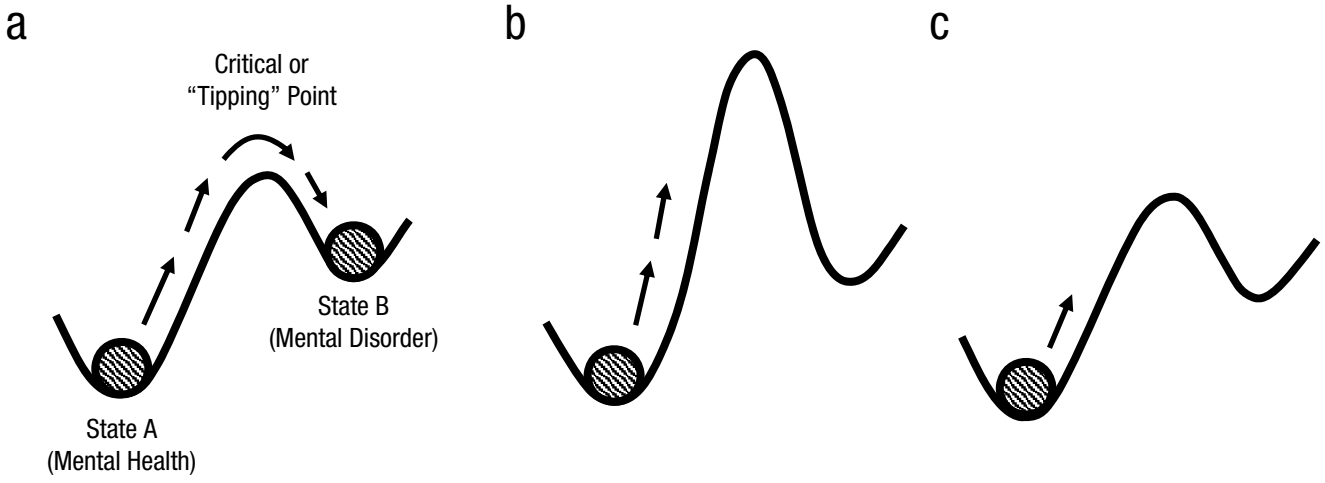

Fig. 4. Network-state transitions. Illustration of the double-well potential model (Kramers, 1940) applied to psychiatry. The diagram in (a) illustrates the transition of a network from a stable state of mental health (State A) over an energy barrier into a new stable state of mental disorder (State B). The curve expresses the overall level of symptom activation, or activation energy, of the system; the wells are energy minima corresponding to stable system states. Activation changes may, for instance, occur as a result of stressors (E in Fig. 3) acting on the system. The maintenance of mental health despite the action of such external system-activating factors (i.e., resilience) is achieved by raising the energy barrier to state transitions (b) or dampening system activation (c).

sufficient to induce a mental disorder (State B in Fig. 4a) that even persists after the stressor has ceased (i.e., hysteresis; Cramer et al., 2016). This network property of bistability reflects the clinical observation that individuals often continue to function relatively well despite a high burden of individual symptoms until a certain overall level of symptom burden or "tipping point" is reached that then leads to a sudden and lasting breakdown. Next to having great conceptual elegance and ecological validity, this new network conceptualization of mental disorders has the advantage that the same mathematical tools that have been used to analyze complex dynamic system behavior in physics, chemistry, or biology can now be applied in psychiatry (Borsboom, 2017).

Can the Network Approach to Mental Disorders Inform Resilience Research?

If resilience is not a trait or predisposition or any other fixed characteristic of an individual, then—like mental disorders—it should not be conceptualized as any kind of entity, be it antinosological or not. Resilience is no single common cause of good mental health, as would be suggested by the widely occurring naive use of resilience questionnaires as predictors of mental health under adversity (Kalisch et al., 2017). Rather, resilience is the maintenance or quick recovery of mental health during and after adversity. In the language of network modeling then, resilience is a symptom network that does not transit into disease State B in Figure 4a, despite its being driven by substantial activating factors.

What could resilient symptom networks look like? On an abstract level, a resilient network could be conceived as a network with a high energy barrier between health and the disease state (Fig. 4b; Hofmann et al., 2016; Scheffer et al., 2018) or with only dampened overall network activation (Fig. 4c). If the energy barrier is high (Fig. 4b), a network may well exhibit states of comparatively high symptom coactivation, but these states remain temporary; that is, they do not stabilize. In Figure 4, this corresponds to the system reaching an activation level comparable to that of State B but falling back into weaker activation levels once a stressor (E) has subsided. If the overall network activation is dampened (Fig. 4c), a network does not even reach activation levels comparable to those of State B, even if stressor input E is high. In other words, both abstract network properties have the effect that the bistable system is farther away from its tipping point.

Transferring these ideas into a network graph, one could attribute the raised energy barrier of Figure 4b to the network having only weak symptom autoconnections, as in Figure 5a. In such a network with weak symptom autoconnections, one or several symptoms may well be highly active at a given moment, but activation is not as easily maintained over time. The dampened overall network activation of Figure 4c in turn could be attributed to weaker symptom–symptom connections, as in Figure 5b. Here, although a single activated symptom may well maintain its activation level over time, for example, because of its high autoconnection, activation does not easily spread to other symptom nodes and, consequently, overall network activation remains low. In both cases, the transition into a new stable State B is less likely. In support of this general idea, simulation studies have already provided evidence that weakly connected networks do not necessarily exhibit bistability (Cramer et al., 2016). In such resilient networks, stressor-induced transitions into disease states may still occur but in a smoother, continuous fashion; that is, they take more time and may be observable only with massive chronic or repetitive stressor exposure.

Resilience Factors as Additional Network Nodes: Hybrid Symptom-and-Resilience-Factor Models

If these global formulations of resilient networks are viable, then one must, as a next step, try to identify the factors that confer the desirable network properties of weak self-connectedness (Fig. 5a) and/or weak interconnectedness (Fig. 5b). In the past few decades, resilience research has identified a range of individual biological or psychological properties as well as social factors that are supposed to enhance the chances that someone will overcome adversity in good mental health (Bonanno et al., 2015; Fritz, de Graaff, Caisley, van Harmelen, & Wilkinson, 2018; Hunter, Gray, & McEwen, 2018; Kalisch et al., 2017; Russo et al., 2012; Sapienza & Masten, 2011; Southwick & Charney, 2012). Predictors of resilience in quantitative prospective-longitudinal studies include good reactivity of the hypothalamus–pituitary–adrenal gland axis to stressors, a heterozygous catechol-*o*-methyl transferase (*COMT*) genotype, appraisal styles such as a tendency to positively reappraise and to not catastrophize about stressors, or a tendency for self-enhancement, the ability to recall specific positive memories, and strong familial and/or social support (Askelund, Schweizer, Goodyer, & van Harmelen, 2019; Kalisch et al., 2017). Each one of these predisposing factors may have only a very small effect on resilient outcomes, and it has been noted that replications of significant findings in independent samples are largely absent (Bonanno et al., 2015; Kalisch et al., 2017). Nevertheless, some of the predictors identified in quantitative studies are theoretically highly plausible, which is

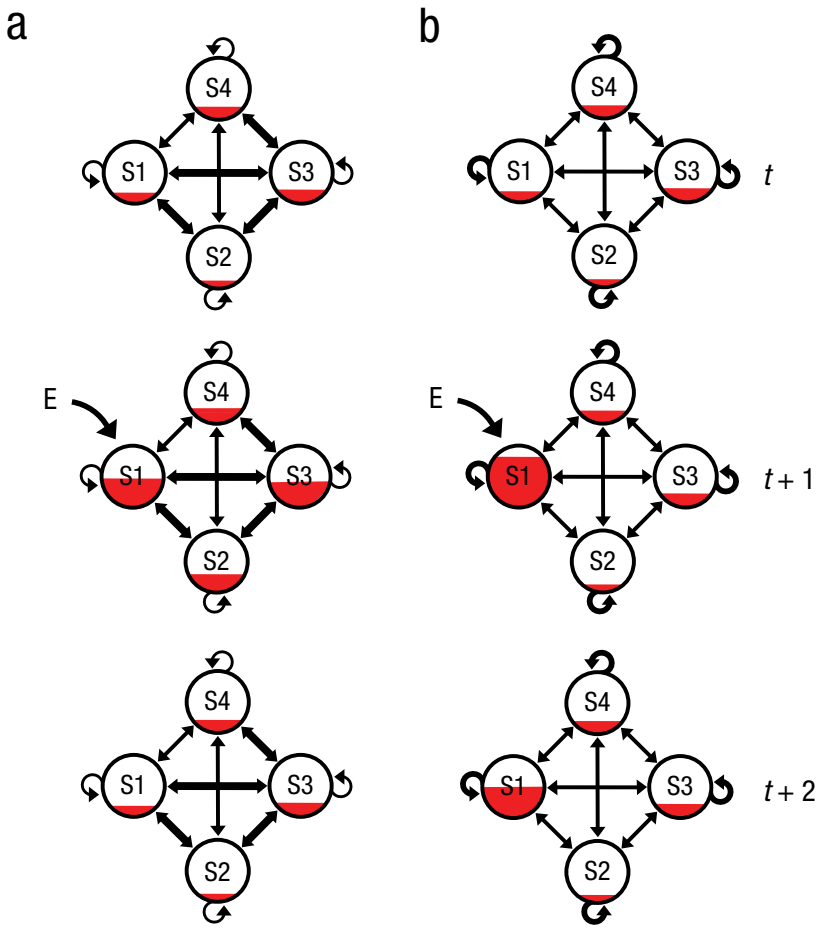

Fig. 5. Resilient symptom networks. Networks with (a) weak symptom (S) autoconnections or (b) weak symptom interconnections are more likely to maintain a stable state of mental health during stressor exposure (E; time point $t + 1$) and once stressor exposure has ended ($t + 2$). The strength of connections (edges) between symptoms is shown by the thickness of the connecting lines.

why they are currently the best starting points for a search for resilience factors that act by keeping symptom networks away from their tipping point.

Take the example of an individual's ability and tendency to volitionally regulate emotions away from negative and toward more positive emotional states using verbal strategies of reappraising the meaning of, or reframing, potentially threatening situations ("positive reappraisal"; Gross, 1998; Lazarus & Folkman, 1984). The habitual use of positive reappraisal, measured with a self-report questionnaire, is generally linked with good mental health (Gross & John, 2003) and has been shown in two prospective studies to moderate the effect of exposure to life stressors (intense media coverage of a local terror attack, problems adjusting to college) on posttraumatic and internalizing symptoms, respectively, in emerging adults (Jenness et al., 2016; Zahniser & Conley, 2018). There is good theoretical reason to believe that volitional reappraisal can protect mental health under stressor exposure (Kalisch, Müller, & Tüscher, 2015; Troy & Mauss, 2011), mainly based on a large range of laboratory studies showing that the application of instructed or self-selected reappraisal strategies can effectively reduce acute aversive or stress reactions (Webb, Miles, & Sheeran, 2012). Hence, reappraisal may reduce symptoms of anxiety, fear, sadness, or anger during and after stressor exposure, thereby also reducing the expense of resources (time, energy, cognitive capacity, financial or social capital) and the ensuing allostatic load such reactions usually come with (Kalisch et al., 2015).

On this basis, it is conceivable that volitional reappraisal—in the sense of a cognitive skill and habit—is a resilience factor and might dampen symptom network activation by acting, for instance, on anxiety and worry symptoms (S1 in the figures). It might therefore make

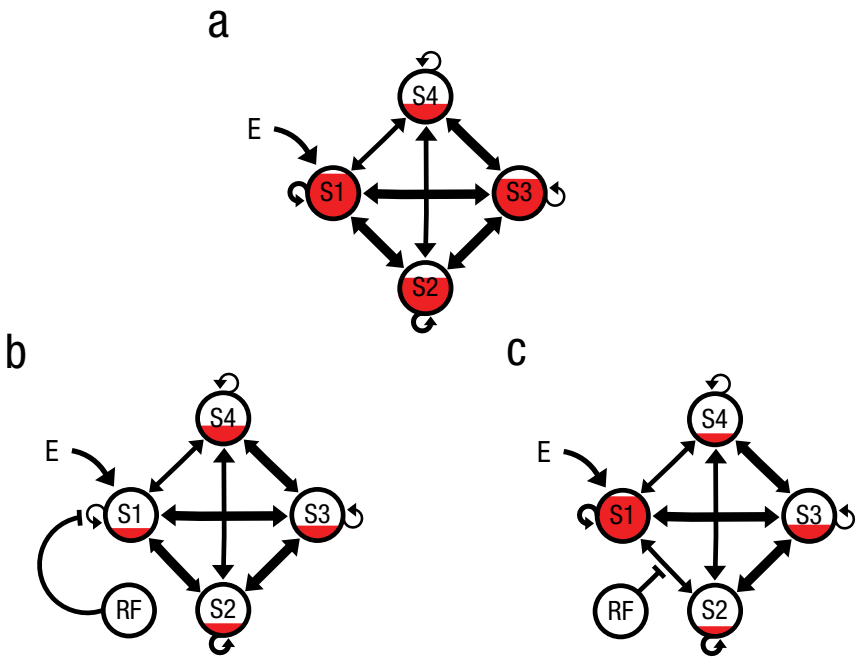

Fig. 6. Hybrid symptom-and-resilience-factor networks. The introduction into a symptom network (a) of a resilience factor (RF) allows for formalizing dampened symptom autoconnections (b; of S1) or dampened symptom interconnections (c; between S1 and S2). The strength of connections (edges) between symptoms is shown by the thickness of the connecting lines.

sense to incorporate a new node into the symptom network (shown in Figure 6a) and to allow this new node (RF, for resilience factor; see Fig. 6b and 6c) to either dampen a symptom autoconnection (Fig. 6b) or a symptom–symptom interconnection (Fig. 6c). Such an extended network could be called a *hybrid symptom-and-resilience-factor* (HSR) network. Through the immediate inhibitory influences shown in Figures 6b and 6c the HSR network would be less likely to transition into to a new stable state of correlated high overall symptom activation (i.e., the mental-disorder State B in Fig. 4a). Resilience factors thus take the role of what are called moderators in the causality literature.

On a mechanistic level, the type of network dampening illustrated in Figure 6b might correspond to someone who frequently uses his or her own reappraisal ability to not appraise a state of anxiety (or other stress reactions; S1) as a threat in itself (as in "fear of fear") but as a normal reaction to real existing problems, thereby avoiding catastrophizing-like vicious cycles of self-reinforcing anxiety. As a result, the likelihood of strong anxiety reactions would be decreased. The other type of network dampening, illustrated in Figure 6c, might correspond to someone specifically reinterpreting social dysfunctions (S2), such as reduced interest in complying with work-related demands or in acquiescing in conflicts with family or friends, again not as a threat but as a sign that it is time to try something new and to change one's way of life. At the network level, this would weaken the social dysfunction–anxiety (S2–S1) interconnection, which ultimately would also reduce anxiety (S1 activation).

Although such mechanistic explanations for the way in which a resilience factor might interact with symptom interconnections or autoconnections are plausible, it is important to note that they are not necessary to formulate the extended network model in Figure 6 and that the inhibitory links departing from the RF node do not imply any particular mechanism of action of that resilience factor. Interactions between the factor and the rest of the network might also occur through other effects. For instance, it might be that someone sees her social dysfunctions as truly problematic and realizes that they cost her social support but then reappraises the vanishing social support as something that she can still easily deal with and can be compensated for by other available coping resources, that is, not as a threat. The network model would be mathematically formulated in the same way as in Figure 6c.

Symptom networks are generally limited in that they model only the degree of activation and connectedness of symptoms but remain agnostic as to how, mechanistically, a symptom is generated at a biological or psychological level and as to what the specific biological,

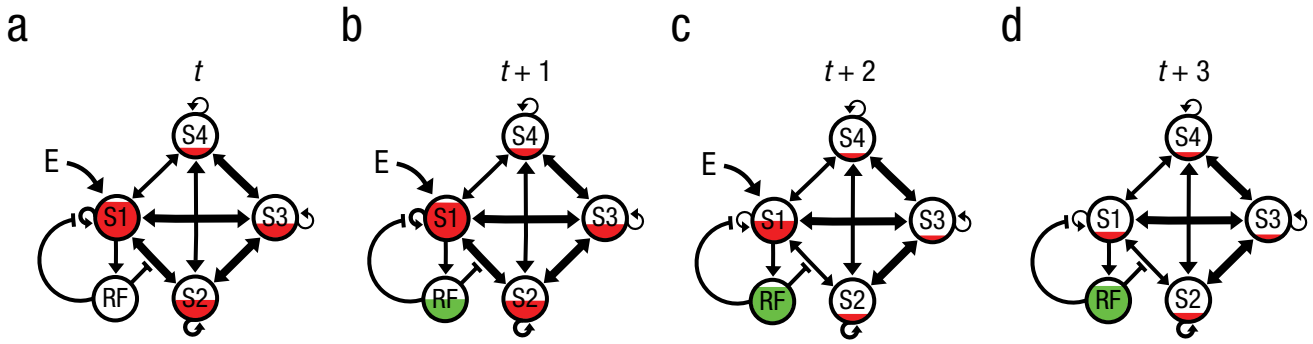

Fig. 7. Resilience factors as dynamic nodes in hybrid symptom-and-resilience networks. The time-variant degree of efficiency/effectiveness of a resilience factor (RF) over time points *t* to *t* + 3 (a through d) is indicated by the green filling of the RF node. As the RF becomes stronger, its dampening effects on a given symptom autoconnection (here, S1) or symptom–symptom interconnection (here, S1-S2) also increase.

psychological, or social mechanisms are that effectively link one symptom to another or to itself. That is, they do not take into account the nature of symptoms and symptom connections (Fried & Cramer, 2017). We propose to deal with resilience factors as a new type of network node in the same way: That is, to *not* to try to define the exact mechanism(s) through which a resilience factor affects symptom interconnections or autoconnections. Provided dedicated hypotheses, such mechanisms might be tested using other statistical and empirical methods. The goal of adding resilience factors as a new type of distinct node into symptom network models and allowing them a variety of potential links to symptom interconnections and autoconnections is to (a) obtain a formal means to test the influence of hypothesized resilience factors on symptoms and (b) improve the explanation of variance in symptoms that is commonly observed between and within individuals relative to pure symptom networks.

Dynamic Resilience Factors: The Processes of Stressor Adaptation

So far, we have treated symptom interconnections, symptom autoconnections, and resilience factors as time-invariant and stable. Changes in the system consisted exclusively in changes in symptom activation conferred exclusively by the influence of stressors and the spreading of the evoked activation through the network via its fixed connections. This assumption is, however, unrealistic. For example, the negative social feedback that may contribute to turning social dysfunction into depression symptoms (hypothetical S2-S3 connection) may well fluctuate; in addition, prefrontal functional impairment from stress-hormone release, contributing to social dysfunction, presumably depends on many other biological factors as well, making the S1-S2 connection time-variant. The same may apply to the ill-understood mechanisms governing symptom autoconnections, such as an individual's tendency to become and remain despaired when feeling incompetent. Given the scope of this article, we focus in the remainder on a discussion of the likely time variance of resilience factors.

To illustrate this point, we again turn to the example of volitional reappraisal. Although individuals differ in how well they can positively reappraise threats and in how much they tend to use such type of emotion regulation, reappraisal is not a completely stable, fixed trait that is entirely determined by genetics or early childhood experience. Rather, a reappraisal skill can be learned and trained and is likely to vary in efficacy and efficiency on both short and long time scales (within hours, within weeks or months, within a lifetime). For instance, it is conceivable that states of unpleasant negative emotion that last over days or weeks, such as anxiety or worry (S1 in Fig. 7a) provide a motivation to reappraise a stressful life situation as also having its benefits or positive aspects or to probably have a positive outcome. Alternatively, a sustained negative emotional state may motivate the use of a more self-focused reappraisal strategy of distancing or detachment that reduces the self-relevance attributed to the stressor. If a person were successful in dampening anxiety and worry with the help of such a reappraisal strategy, this would most likely further reinforce the use of the strategy. This in turn would effectively exert a training effect, enhancing the likelihood that he or she will continue to use the strategy during the current challenging life situation and also enhancing reappraisal performance and success rates. In the longer run, it would make it more likely that the person will rely on such a proven emotion-regulation strategy the next time he or she is exposed to stressful situations. In short,

through stress one might eventually become a better (more efficient and effective) and more habitual reappraiser.

To formalize this link between a symptom (here, anxiety) and a resilience factor (here, reappraisal), we introduce a positive connection between S1 and RF in Figure 7. Through this connection, a resilience factor that may otherwise be only a dormant potential (Fig. 7a) can be activated in stressful situations and trained over time (Fig. 7b). The green filling of the RF node, increasing from time point *t* (Fig. 7b) to time point *t* + 2 (Fig. 7c), indicates the time-varying efficiency/effectiveness of the resilience factor. This could be measured repeatedly over the course of and after a period of adversity with a laboratory reappraisal test or inferred indirectly from a self-report questionnaire of reappraisal-use tendency. If a subject's reappraisal efficiency/effectiveness increases lastingly (Figs. 7c and 7d), it might prevent further increases of anxiety and/or limit the spread of activation from S1 to other symptoms despite continued stressor exposure (Fig. 7c), and it might help the system recover if stressor exposure is over (Fig. 7d).

This logic of dynamic resilience factors that evolve over time under the influence of other network nodes can be applied to hypothesize any other pathway of interaction involving symptoms and resilience factors. So, one could theorize that improved reappraisal ability also enhances positive social support (another potential resilience factor; van Harmelen et al., 2016, 2017) because it facilitates interactions with well-meaning others. This would be a case of a resilience factor being activated by another resilience factor (an RF–RF interaction), an ultimate effect of which might be reduced depressive symptoms. Hence, depending on the hypothesized nature and pathways of action of a resilience factor, different constellations of interactions with the other network elements may have to be formulated.

In sum, HSR networks describe dynamic processes of adaptation to stressors that do not exclude temporary states of overall high network activation but make it less likely that a network will pass over a tipping point into a maladaptive new energy minimum in which symptoms do not recover. HSR networks model resilience—the maintenance or quick recovery of mental health during and after stressor exposure—as emerging from time-varying external influences and internal network interactions. Resilience factors in these networks can be protective skills, abilities, appraisal patterns, coping styles, social resources, molecular feedback mechanisms, gene-expression patterns, or other symptom-dampening factors. This approach of modeling resilience is entirely different from treating resilience as an entity or as a temporally fixed construct. It first deconstructs resilience into its constituent parts (i.e., resilience factors) and then reconstructs it as a complex set of dynamic processes of interaction between these resilience factors and the symptom nodes and connections in a hybrid network.

Interindividual Differences in Resilience Processes

For further terminological clarification, we now refer to specific subsets of interacting symptom and resilience-factor nodes and their connections (i.e., subparts of the network such as the interacting S1, S2, and RF nodes in Figs. 6 and 7) and to their typical ways of evolving over time (such as anxiety increasing reappraisal ability and reappraisal ability in turn decreasing anxiety) as *resilience processes*. Thus, resilience processes are typical constellations of symptom and resilience-factor nodes and their self- and interconnections with time-variant nodes and connection strengths. We assume that, although resilience processes differ between individuals in their dynamics (i.e., in the extent to which they are recruited at different time points), a limited set of generic node/connection constellations is shared by all or most individuals.

By measuring only the efficiency/effectiveness or "strength" of a given resilience factor in isolation from the other elements with which it typically interacts, it would not be possible to identify these central mental-health-protection processes. Hence, dynamic HSR network modeling—combined with high-frequency measurements of stressors, symptoms, and resilience factors—in theory offers a tool to identify and quantify critical resilience processes and individual differences therein.

Methodological Demands

From the above it is also clear that the theoretical possibilities for modeling resilience that we describe here place a significant burden on empirical research practice. First, they require extensive longitudinal monitoring of study subjects. One-time (cross-sectional) assessments (e.g., a questionnaire, a brain scan, genotyping) will in no case deliver the data needed for dynamic HSR modeling and will increasingly turn out to be unsatisfactory (Kalisch et al., 2017). Second, as already pointed out, monitoring will have to take place at a high frequency and incorporate stressors, symptoms, and resilience factors. Hence, HSR modeling will flourish only to the extent that modern information technology for subject monitoring is applied and further improved.

It should also be mentioned that there are, in theory, different ways of how HSR models can be mathematically implemented. These include parametric stochastic Lotka-Volterra systems (van Nes & Scheffer, 2004) that allow for the detection of early warning signals for critical transitions, applying bifurcation theory (Scheffer et al., 2012) or autoregressive models (Scheffer et al., 2009). Furthermore, nonparametric approaches such as regularized partial correlation analysis for continuous variables (J. Friedman, Hastie, & Tibshirani, 2008) or Ising models for binary data (van Borkulo et al., 2014) are applied. To propose or recommend a concrete implementation is not the point of this article. Nevertheless, it is worth keeping in mind that different ways of mathematical implementation always require different amounts and quality of data. This again emphasizes the need for proper subject monitoring.

Limitations

One important limitation of our approach linked specifically to the amounts of data needed for network modeling is that there are mathematical and computational boundaries as to how many nodes, or more generally, parameters, can be modeled. This is problematic specifically in light of the large number of potential resilience factors identified in the literature so far (see above; Bonanno et al., 2015; Kalisch et al., 2017). Again, although the problem will ease to the extent that data sets grow (both in the temporal dimension and in terms of number of subjects), a different way to address this issue may be to use theoretical considerations and empirical insights to pinpoint what we have earlier called *resilience mechanisms* (Kalisch et al., 2015) and to use these as nodes in HSR networks. Resilience mechanisms are hypothesized common end paths, or mediators, of the actions of the myriads of proposed resilience factors on resilient outcomes and are, according to positive appraisal style theory of resilience (PASTOR), likely to be located in the neurocognitive processes that govern stressor appraisal. Because many resilience factors are supposed to converge in only a few common resilience mechanisms to thereby determine resilience outcomes, resilience mechanisms are necessarily fewer in number and thus more amenable to inclusion in HSR networks. As a matter of fact, our above example of reappraisal as a resilience factor would be an example of a key resilience mechanism, if PASTOR is right (Kalisch et al., 2015).

A specific potential limitation of discrete time models is with continuous time or data on different time scales. Therefore, in cases in which discrete time models are used for such data, important temporal structures might potentially be missed.

Our suggestion to extend pure symptom networks to also include resilience factors raises the questions of (a) how exactly those new nodes are defined and (b) which of those possible new nodes one chooses to include in a given model. Both the exact definition and measurement of a resilience factor and any network design choice will obviously strongly influence outcomes. Reaching a consensus on what resilience factors are and how they are best determined is an endeavor that—like the efforts that were and are being made to define psychiatric symptoms—will require much further empirical and theoretical work and intense exchange between resilience researchers. A platform explicitly created for such work is the International Resilience Alliance. Given the computational restrictions on node numbers discussed above, hypothesis-driven a priori choices of included resilience factors and of their links to other network connections will be inevitable for the foreseeable future. This implies that HSR modeling is unlikely to soon become a tool for massive exploratory research and that it will not be useful without the incorporation of expert knowledge.

Conclusions

We have proposed a formalized way of describing and testing how putative resilience factors protect against stressor-evoked symptoms of psychopathology and contribute to the stabilization of individuals' mental health. We completely abandon the notion of resilience as a biological, psychological, or sociological entity by deconstructing it into separable resilience factors. Within a general-symptom network framework, resilience factors affect symptoms by dampening symptom autoconnections or symptom–symptom connections. They can themselves be affected by symptoms, other resilience factors, or stressors. Further, the effectiveness and efficiency of resilience factors are allowed to vary in time. The resulting HSR networks have the potential to mathematically express the complex and dynamic processes (resilience processes) through which some individuals successfully adapt to challenges and manage to maintain or rapidly regain mental health (resilience as an outcome). This concept of dynamic HSR networks is abstract and generic. As for pure symptom networks, different mathematical methods are theoretically available to implement HSR networks, and the concrete architecture of an HSR network will depend on the population and type of adversity studied as well as on the available measures of symptoms and resilience factors. We hope that HSR networks will better explain intra- and interindividual variance in symptoms than pure symptom networks and permit the mathematically precise characterization of the protective

effects of resilience factors. Another promise, albeit in the more distant future, is the use of HSR networks in precision medicine, offering individualized disease prognosis and prevention. In particular, in cases in which dense sampling of stressors, symptoms, and resilience factors is possible, HSR networks informed by such data may be used to predict future symptom course and to detect critical periods of potential-state transition that warrant intervention. Further, they may allow for simulating the effects of preventive interventions that target and enhance a resilience factor, thereby guiding clinical decision making.

Action Editor

Laura A. King served as action editor for this article.

Author Contributions

R. Kalisch and A. O. J. Cramer contributed equally to this manuscript. R. Kalisch, A. O. J. Cramer, and A.-L. van Harmelen conceived and wrote the manuscript. All of the authors contributed to the writing of the manuscript, providing critical feedback and individual elements, and approved the final manuscript for submission.

Declaration of Conflicting Interests

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

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