



A resilience framework for promoting stable remission from depression



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HIGHLIGHTS

- Resilience may be improved in people in remission from depression.
- We present targets for improving resilience in remitted people.
- We present appropriate assessments of potential changes in resilience.
- We review several examples of validated resilience interventions.

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ABSTRACT

A significant proportion of people in remission from depression will experience a recurrence of depression. One theoretical mechanism for this recurrence is that with each additional episode of depression, people become more sensitive to the deleterious effects of less powerful stressors. We propose that research on resilience – the ability to adapt to and recover from stress – can inform interventions to prevent recurrence in people in remission. We conceptualize resilience as a dynamic process that may be deficient in people in remission from depression, rather than as a static personal quality that is unattainable to people who have experienced psychopathology. The three aspects of resilience that we suggest are the most important to target to prevent recurrence are (1) improving stress recovery from minor daily stressors that may aid remitted people in coping with major stressors, (2) increasing positivity, like promoting positive emotions during stress, and (3) training flexibility—the ability to identify different demands in the environment and employ the appropriate coping strategy to meet those demands. We offer suggestions for the appropriate assessment of changes in resilience in remitted people and provide some examples of effective resilience interventions.

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1. Introduction

Depression is a highly prevalent, severe mental illness that is related to substantial individual suffering. In terms of disability, estimations suggest that major depressive disorder (MDD) is among the leading causes of burden of diseases worldwide (e.g., [Demyttenaere et al., 2004](#)). Despite the availability of a wide variety of treatment options (psychological, pharmacological, and neurostimulation interventions) and strong investments in treatment research, major challenges in the treatment of depression remain. One particularly pervasive problem is the frequent recurrence of depression after remission.

Research shows that recurrence of MDD (i.e., experiencing a depressive episode after having exhibited full and/or partial remission from a previous depressive episode) is high in the general population (35% after 15 years), and even higher in those treated at specialized mental health centers (60% after 5 years and 85% after 15 years; [Hardevald, Spijker, De Graaf, Nolen, & Beekman, 2010](#)). In this research, recurrence is best predicted by the number of previous episodes and subclinical residual symptoms ([Solomon et al., 2000](#)). [Solomon et al. \(2000\)](#) found that 2/3 of people with one episode will experience another depressive episode within 10 years, and that after each episode, the likelihood of subsequent episodes increases by 16%. Not exhibiting full recovery also increases rates of recurrence. People with even mild residual symptoms after a depressive episode are at a much higher risk of relapsing, even when controlling for number of previous episodes ([Judd et al., 1998](#)).

Such findings have spurred a number of theories to explain increasing vulnerability after initial depressive episodes. A major theory is the stress kindling hypothesis ([Monroe & Harkness, 2005](#); [Post, 1992](#)). Post noted that major life stressors have less of an impact on recurrent episodes of depression than they do on the first episode of depression and proposed that through stress kindling or sensitization new episodes can develop more autonomously from stress compared with first episodes of depression. [Monroe and Harkness \(2005\)](#) carefully elaborated on this idea and distinguished between possible explanations. *Stress kindling/sensitization* suggests that this dissociation is due to the increased influence of lesser amounts of stress to cause recurrence, and *stress autonomy* suggests that depression just becomes independent of stress episodes, whatever their intensity. A recent longitudinal study of late-adolescent women provided support for the stress sensitization model over the stress autonomy model. The impact of non-severe life events on the likelihood of having an onset of a depressive episode was greater in individuals with a history of depression compared to those with no history of depression ([Stroud, Davila, Hammen, & Vrshek-Schallhorn, 2011](#)). This stress sensitization is also apparent in response to discrete lab stressors ([Heim et al., 2000](#)). Investigators found that increased emotional reactivity to a sad mood induction (listening to sad music while thinking about a sad time in their life) predicted earlier time to recurrence in people being treated for their depression ([Van Rijsbergen et al., 2013](#)). Investigators have also found that increased cognitive reactivity to sad mood inductions predicts increased risk of relapse even when controlling for number of previous depressive episodes ([Segal et al., 2006](#)).

Although there is substantial heterogeneity in depression and mechanisms underlying depression (e.g. [Hasler & Northoff, 2011](#)), the evidence does indicate that increasing sensitivity to smaller stressors is a potential cause of recurrence (for a review see [Monroe & Harkness, 2005](#)). This implies that therapies and interventions that increase resistance to stress may be particularly effective at reducing the risk of relapse. First line psychological treatments such as CBT (e.g., [Beck, 1976](#); [Clark, Beck, & Alford, 1999](#)) and interpersonal therapy (IPT; [Klerman, Weissman, Rousaville, & Chevron, 1984](#)) attempt to change stress reactivity by focusing on the interpretation of personally relevant stressful stimuli or events (e.g., feeling rejected after criticism). However, these therapies are mainly aimed at alleviating negative affect and pay only limited attention to building other skills related to resilience in overcoming stress. The past decades have witnessed a surge of interest in the psychobiological factors underlying resilience to stress and in line with others (e.g., [Dunn, 2012](#); [Padesky & Mooney, 2012](#); [Southwick, Vythilingham, & Charney, 2005](#); [Wood & Tarrier, 2010](#)) we propose that given the nature of recurrent depression, treatments should increasingly focus on building resilience.

In this article we argue that the knowledge gained from resilience research can improve our understanding of recurrent depression and its treatment. We view the capability to effectively handle stress as a unipolar dimension with increasing stress sensitivity on the negative end and increasing stress adaptability on the positive end (similar to ego-brittle vs. ego-resilience; [Block & Kremen, 1996](#)). [Fig. 1](#) illustrates how remitted individuals may change in their capability to handle stress over time. With increasing time/depressive episodes, remitted individuals become more vulnerable to depression via increasing stress sensitivity. The goal of a resilience intervention is to reverse this process – increasing people's ability to handle stressors, and therefore their resilience, thus decreasing their stress sensitivity and increasing their stress adaptability. Important to our conceptualization, however, is that

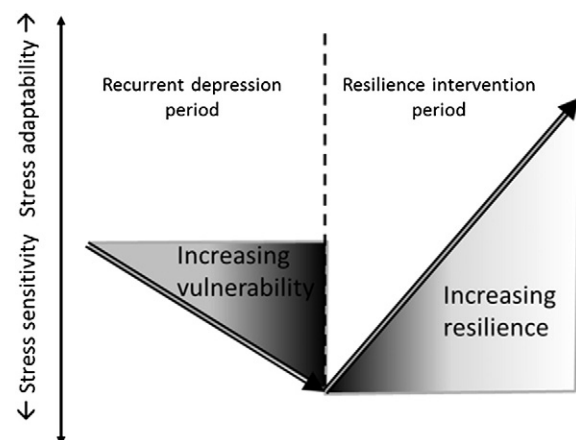


Fig. 1. Hypothesized relationship between resilience/vulnerability and stress sensitivity/adaptability during periods of recurring depressive episodes or resilience interventions.

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