Invited commentary on . . .
Psychiatric resilience: longitudinal twin study†
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Summary
The study of resilience may lead to the identification of new targets for prevention and intervention, yet there has been little research on why some people, but not others, show resilience after facing stressful life events. New research in this issue shows that resilience is equally explained by genetic and environmental influences, and that individual experiences and situational factors are both important in shaping resilient responses to stress. These findings could inform the development of interventions that enhance psychiatric resilience after exposure to adversity.

Declaration of interest
None.

Resilience in psychiatry
As mental health practitioners and researchers, a large part of our work consists of caring for people who have mental disorders, and of studying the pathways leading from biopsychosocial risk factors to the development of psychopathology. The paper on resilience by Amstadter et al in this issue of the Journal is a refreshing change from this preoccupation with risk and disorders, because it highlights the ‘optimistic side of the risk–psychopathology equation’: that some people are able to maintain a better level of functioning than others, despite having been exposed to the same level of adversity.

By studying resilience to developing internalising symptoms after exposure to stressful life events, Amstadter et al address a question of utmost clinical importance, since a better understanding of the processes underlying resilience could help us design interventions to prevent the development of mental health problems. This is particularly relevant to depression, where many risk factors are not easily modifiable or escapable, such as a genetic predisposition or the presence of chronic and uncontrollable stressors.

Measuring resilience
Resilience is a difficult construct to define and to measure. In their study, Amstadter et al operationalise resilience by using the residuals from a regression analysis predicting people’s symptoms (on the internalising subscales of the Symptom Checklist-90, which include predominantly depressive, anxiety and somatisation symptoms) from the number of life events experienced in the previous year. The resulting measure reflects a continuum from resilience to vulnerability, where individuals whose symptom score is lower than predicted are considered resilient, whereas those with a symptom score higher than predicted would be considered vulnerable. Therefore, the findings are as much applicable to explaining resilience as they are to explaining vulnerability.

Using this measure is appealing, because it captures what has been considered to be the essence of resilience by many authors: showing a better outcome than others exposed to the same risk. However, there are implications for the interpretation of Amstadter et al’s findings. First, unlike the positive connotation carried by ‘resilience’ suggests, experiencing fewer symptoms than predicted is not necessarily equal to being well or symptom-free; the symptoms might still be enough to reflect clinically significant distress. Second, using this ‘residual’ approach, a person who experiences fewer symptoms than predicted is considered resilient even if the difference between their actual and expected symptoms is very small. However, there might be qualitative differences between individuals who are doing far better than expected, and are thus very resilient, and those who are doing just slightly better than expected, and are therefore just a little bit resilient. Both of these points highlight the need for future research on psychiatric resilience to incorporate clinical parameters, such as level of functioning and need for care, to complement measures based on a purely statistical approach.

Genetic and environmental influences on resilience
The main finding of this paper is that variance in resilience to internalising symptoms is equally explained by genetic and environmental influences. Thus, innate factors are as important as the environment in explaining who will develop fewer (or indeed more) internalising symptoms than expected, after experiencing stressful life events. This finding has important clinical implications because it opens the possibility of identifying factors that affect resilience and could be targeted in preventive or therapeutic interventions. For example, previous findings suggest that genetically influenced personality traits, such as self-acceptance and environmental mastery, are associated with resilient responses to stress, and explain some of the heritability of resilience. Fostering these qualities in vulnerable individuals, for example through self-efficacy training, stress-inoculation exercises or mindfulness interventions, could be an effective prevention strategy.

In the search for specific environmental factors that influence psychiatric resilience, Amstadter et al show that all of the environmental influence on resilience is the result of non-shared environmental effects, not shared environment. One of these environmental factors could be childhood experiences that are unique to a member of a family, and indeed childhood maltreatment has been shown to shape an individual’s response to stressful events until well into adulthood.

†See pp. 275–280, this issue.
environmental contributions. This is because estimates of non-shared environmental effects indicate not only the direct influences of individual experiences, but also the effects of interactions between an individual’s environment and their genes. The important implication for clinical practice is that modifying an individual’s environment might have varying effects for different individuals, depending on their genotype.

When Amstadter et al assess resilience at different time points, they also find that a large part of the environmental influence is ‘occasion-specific’, and thus affects resilience in a transient fashion. This finding highlights the importance of conceptualising resilience not as a fixed personal characteristic, but as a changing and composite quality, influenced by situational factors. These factors are likely to be those that affect our ability to cope with stressful life events, such as supportive relationships with friends and partners, socioeconomic situation or physical health.

How stressful is ‘stressful’?

Another such ‘occasion-specific’ situational factor could be the type and severity of the stressful life event itself. The life events in this study seem to be of quite a severe nature, but they still reflect different types of stressors, ranging from major financial problems to violent assault. Different types of stressful life events have been shown to vary considerably in the risk that they carry for the subsequent development of internalising problems. Moreover, even the same event might differ in its level of contextual severity, that is, severity evaluated in relation to an individual’s set of living circumstances.7 In addition, the amount of time that has passed since a life event has been experienced is likely to play a role too, in that life events that are experienced more recently carry a higher risk for the development of internalising problems.7

Future studies should integrate measures from these different domains and examine their dynamic interactions: this will greatly advance our understanding of this fascinating construct, thereby improving identification of targets for prevention and intervention, and increasing its translation into clinical practice.

The need for a multidomain approach

Finally, a myriad of studies has shown that stressful life events, in addition to increasing the risk for psychopathology, also affect multiple other health domains, such as immune, endocrine and cardiovascular functions.9 In order to gain a comprehensive measure of resilience that truly captures its complexity, it would be important to incorporate indicators of resilience relevant to each of these domains.7 This is because individuals might display resilience in one domain, but not in others. For example, some people may experience low levels of psychopathology while at the same time displaying abnormal health-relevant physiological measures, such as heart rate and blood pressure reactivity.10

References

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References
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