**Resilience and psychiatric epidemiology: Implications for a conceptual framework**

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**Resilience and psychiatric epidemiology: Implications for a conceptual framework**

Joseph M. Boden, Ph.D.

Geraldine F.H. McLeod, Ph.D.

Christchurch Health and Development Study

University of Otago, Christchurch School of Medicine and Health Sciences

PO Box 4345

Christchurch, New Zealand

Tel: 64-3-372-0406

Fax: 64-3-372-0407

Email: joseph.boden@otago.ac.nz

**Abstract**

Kalisch and colleagues present a conceptual framework for the study of resilience, using a neurobiological approach. The present commentary examines issues arising for the study of resilience from epidemiological data, which suggest that resilience is most likely a normative function which may operate as a kind of psychological immune system. The implications of the epidemiological data on the development of a neurobiological theory of resilience are discussed.

The target article by Kalisch and colleagues presents a unified conceptual framework for the study of resilience at the neurobiological level. The authors argue that resilience is best understood as a process, and suggest that resilience can be examined via the study of appraisal style, and the extent to which appraisal style protects against stressors and mediates the effects of other factors related to resilience. One key argument made by Kalisch and colleagues is that a better understanding of the processes underlying resilience will contribute to a paradigm shift in psychological and psychiatric research, away from a focus on disorder and psychopathology.

The field of psychiatric epidemiology has a long-standing interest in the concept of resilience, with a range of studies examining the factors that appear to buffer individuals from the effects of exposure to severe stressors (Luthar, 2003). Because these studies employ measures of real-world stressors and psychiatric symptomatology, research in psychiatric epidemiology has important implications for the development of a conceptual framework for the study of resilience.

One critical issue, as noted by Kalisch and colleagues, is that effect sizes for the associations between exposure to severe stressors and psychiatric disorders are relatively modest. For example, several studies of exposure to deadly natural disasters, including hurricanes, floods, bushfires and earthquakes have shown that the increase in risk of mental health disorders attributable to disaster exposure is surprisingly small (Fergusson, Boden, Horwood, & Mulder, in press; Fergusson, Horwood, Boden, & Mulder, 2014). Fergusson and colleagues, using data from a longitudinal birth cohort, found that individuals with the highest level of exposure to a series of earthquakes had adjusted rates of mental disorder that were only 1.4 times higher than those not exposed, and that exposure to the earthquakes accounted for only 10% to 13% of the total mental disorder in the cohort. Similarly, studies of exposure to

severe levels of childhood sexual abuse, which has been shown to be one of the most severe stressors to which individuals may be exposed, have also shown modest adjusted associations between abuse exposure and later psychiatric disorders. For example, Fergusson, McLeod and Horwood (2013) found that exposure to sexual abuse in childhood accounted for 5.7% to 16.6% of mental health problems during the period 18-30 years, with effect sizes (Cohen's *d*) ranging from .24 to .48.

The consistent findings of relatively modest mental health effects attributable to severe stress exposure suggests that, if resilience processes are protecting individuals from more severe symptoms, these processes almost certainly must be operating in a global manner. Such processes could be described as a kind of psychological immune system (Davydov, Stewart, Ritchie, & Chaudieu, 2010; Shastri, 2013), and it is clear that such functioning is a normative psychological phenomenon in humans. Such a system would fit comfortably into the framework developed by Kalisch and colleagues, who posit both general and symptom-specific resilience processes. However, in order to better understand the operation of such a normative system, it is critical to understand factors that compromise the system and cause it to operate less effectively. From the perspective of the psychiatric epidemiology literature, those factors may be identified as covariate factors that exist prior to or contemporaneously with the exposure, and which increase the likelihood of psychiatric symptomatology. Such factors include: adverse socioeconomic and family circumstances; parental and childhood maladaptive behaviour; genetic factors; and individual characteristics/personality factors (e.g. Fergusson et al., 2014; Fergusson et al., 2013). Taking such factors into account in statistical models of the associations between stress exposure and psychiatric symptoms generally reduces the magnitude of the associations, suggesting that exposure to a variety of adverse life circumstances increases the risk of an individual developing psychiatric symptoms following stress exposure.

An important implication arising from this general pattern of associations is that it will prove difficult to develop models of resilience that do not take into account the range of adverse circumstances that individuals may be subject to, and that may compromise the operation of a resilience system or process. In the context of laboratory research on resilience, it will be important for researchers to account for possible mediating effects of adverse life circumstances on appraisal processes. More generally, these considerations imply that, in order to understand the processes that protect individuals from psychopathology, it will still be necessary to focus on factors that put individuals at risk, suggesting perhaps not a paradigm shift, but rather a nuanced view of the factors that mediate the associations between stress exposure and psychopathology.

An additional issue arising from the small effect sizes observed in psychiatric epidemiological studies of severe stressors and mental health is that it will also be difficult to replicate such stress levels in a laboratory environment when working with human participants. This issue has been observed in the literature on the mechanisms of psychological defence, in which it is both impractical and unethical to induce high levels of stress or expose individuals to strong negative emotional material (Draguns, 2004; Hentschel, Draguns, Ehlers, & Smith, 2004). One way of addressing this issue is to study individuals who tend to display exaggerated responses following exposure to laboratory stressors, such as trait repressors (Boden & Baumeister, 1997; Weinberger, 1990). Although it is certainly within the scope of laboratory research to expose individuals to stress at the level of "daily hassles", it is not at all clear that such stress levels would cause an effect of sufficient magnitude for individuals who respond in a normative manner to engage resilience processes (Del Giudice, Ellis, & Shirtcliff, 2011).

In summary, the study of resilience has been a feature of the psychiatric epidemiology literature for some time, and data from the literature suggest that the consistent relatively small effect sizes for the associations between severe stress exposure and mental health symptomatology raise key considerations for neurobiological studies of resilience. The conceptual framework developed by Kalisch and colleagues represents a promising advance in our understanding of resilience processes, but the development of this model should take into account these and related issues.

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