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Psychometric and neurobiological assessment of resilience in a non-clinical sample of adults

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Social support

Summary
Background: Resilient individuals are capable of adjusting and coping successfully in the face of adversity. Efforts to assess resilience and its biomarkers have focused on individuals with a history of trauma and related disorders.
Objective: To psychologically assess resilience in a non-clinical community population through questionnaires, and analyse the associations between the psychological parameters and salivary cortisol and dehydroepiandrosterone sulphate (DHEA-S) as putative biomarkers of resilience.
Method: An opportunistic sample ($n = 196$) completed a cross-sectional survey assessing resilience, self-reported depressive symptoms and anxiety, and possible correlates. A sub-sample ($n = 32$) selected in order to maximise variation of mental health, provided saliva samples for enzyme-linked immunoassay (ELISA) detection of cortisol and DHEA-S.
Results: Resilience correlated negatively with depressive symptoms, trait anxiety and early life stress, and positively with self-efficacy, optimism, social support and wellbeing (all $r > 0.40$; all p-values ≤ 0.001 except for early life stress: $r = -0.20$; $p \leq 0.05$). Resilience and DHEA-S concentrations correlated significantly ($r = 0.35$; $p \leq 0.05$); this relationship remained stable after adjustment for demographics. Gender differences were observed for DHEA-S and cortisol ($p \leq 0.05$).
Conclusion: Resilience is associated with positive aspects of psychological health and salivary DHEA-S, suggesting the latter can be treated as a biomarker of resilience in a non-clinical sample of adults.

1. Introduction

Resilience as the ability to withstand adversity and successfully adapt in the face of adversity has received considerable attention by researchers in the fields of child abuse, war trauma and post-traumatic stress disorder (PTSD) (Charney,

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2004; Bonanno et al., 2011). However, researchers are debating operational definitions of resilience, details of suitable research designs and measurement strategies (Windle et al., 2011; Bonanno, 2012). Furthermore, the study of resilience in non-clinical and community populations using questionnaires and linking resilience to putative biomarkers has received less attention so far.

One major line of enquiry into resilience understands it as a stable personality trait. Kobasa (1979) in her work on workplace stress introduced the term hardiness to describe a person with strong commitment, exerting control and rising to challenges — characteristics which facilitate coping with stress. Hardy individuals experience less serious illness for a given level of stress compared to non-hardy individuals. Although Kobasa did not use the term resilience itself, Connor and Davidson (2003) draw on her work when conceptualising resilience as a personality factor as measured by their CD-RISC resilience scale. These authors define resilience as the ability to ''thrive in the face of adversity'' (Connor and Davidson, 2003, p. 76). In their work they also draw on the research by developmental psychologists including Rutter (1985) whose work aims to understand factors protecting individuals, particularly children and young people from PTSD. Resilience apart from the hardiness component includes close attachment, patience and adaptability to change; it is a fairly complex concept and the CD-RISC and other resilience questionnaires reflect this complexity. This psychometric tradition of resilience has led to a number of relatively short questionnaires with the CD-RISC showing most promise in terms of reliability and validity (Windle et al., 2011). Importantly this approach studies resilience in general and student population samples, and in people with chronic conditions rather than focusing on those who have been exposed to traumatic events (Connor and Davidson, 2003).

Bonanno (2012) taking a functional approach, conceptualises resilience as the absence of psychopathology subsequent to a potentially traumatic event. While Bonanno (2012) distinguishes resilience from recovery, other authors view resilience as 'bouncing back' e.g. from depression through social support (Netuveli et al., 2008). Elsewhere, focus has been placed on the positive effects of adverse experience on what Tedeschi and Calhoun (1995) termed post-traumatic personal growth. These individuals, following a traumatic event, undergo a constructive transformation rather than developing PTSD or other chronic mental health problems. This positive development manifests in the individuals' perception of themselves as being psychologically stronger, an appreciation of clearer personal priorities and relationships with others becoming more meaningful. For the purpose of the present paper, resilience is viewed as the protection of good mental health or its recovery within a relatively short time.

Protection is not only sourced from psychosocial factors; neurobiological factors also play a role in alleviating risk and adversity (Charney, 2004). Thus, Connor and Davidson (2003) suggest that a number of factors such as age, gender, cultural background and context can influence resilience, indicating that both resilience and vulnerability can adjust circumstantially throughout the lifespan.

While resilience is primarily identified by psychological measures, evidence demonstrates that specific peripheral biomarkers are associated with resilience, and may represent the biological basis of this phenomenon. Those include the anxiolytic neuromodulators oxytocin and neuropeptide Y (NPY) (Ozbay et al., 2008; Yehuda et al., 2006) and the steroid hormones cortisol and dehydroepiandrosterone (DHEA) (Charney, 2004; Simeon et al., 2007). Oxytocin, a neurohormone released into the general circulation from the posterior pituitary gland, facilitates social and emotional behaviour in humans and rodents, including enhancement of trust and bonding, and reduction of aggression and anxiety (e.g. review by Heinrichs and Domes, 2008). Oxytocin has anxiolytic effects in human volunteers exposed to psychosocial stress, through enhancing the effects of social support and decreasing anxiety (Heinrichs and Domes, 2008). There is a growing consensus that oxytocin plays a role in building and/or maintaining resilience (e.g. Ozbay et al., 2008).

NPY has been considered as a biomarker of resilience as a result of animal and clinical studies, which have demonstrated that this peptide prevents the anxiogenic actions of the corticotrophin releasing factor (CRF), which is the major central regulator of the stress reaction via the hypothalamic—pituitary—adrenal (HPA) axis (e.g. review by Morales-Medina et al., 2010). Plasma NPY is negatively correlated with psychological symptoms of dissociation, and soldiers with high levels of plasma NPY exhibit better performance under severe stress (Morgan et al., 2000). Thus NPY has been suggested to be a biological correlate of stress resistance/resilience (e.g. review by Yehuda et al., 2006). Amongst the putative neurobiological substrates of resilience there are also central biogenic amines such as dopamine, which regulates motivation and reward mechanisms and serotonin, that sets the balance of anxiolytic versus anxiogenic effects (review by Charney, 2004).

Against the complexity of the neurobiological underpinnings of resilience, the present study focuses on the neuroendocrine stress response mediated by the HPA axis, and the glucocorticoid hormones involved in stress and resilience, namely cortisol and dehydroepiandrosterone (DHEA), respectively (Charney, 2004). DHEA and its sulphated ester DHEA-S — together known as DHEA(S) — are endogenous steroid hormones mainly secreted by the adrenal gland in response to HPA axis activation. DHEA(S) exerts neuroprotective effects by inhibiting glucocorticoid-induced neurotoxicity (Kimonides et al., 1999), antagonising the dexamethasone-induced suppression of lymphocyte proliferation (Blauer et al., 1991) and dampening cortisol concentration (Wolf et al., 1997).

There is a consensus that a physiological response to acute stress entails the swift activation of the HPA axis and its effective return to pre-stress conditions thereafter through glucocorticoid feedback (De Kloet et al., 2005). Chronic stress can lead to HPA axis dysregulation resulting in excessive concentrations of cortisol in cerebrospinal fluid, plasma and saliva, and increasing susceptibility to disease (O'Connor et al., 2000), whilst DHEA concentrations decline. A disturbance to the equilibrium of cortisol and DHEA(S) may influence metabolic changes that contribute to the pathophysiology of stress and/or protection in resilience (Maninger et al., 2009). Thus, the ratio of DHEA(S) to cortisol has been suggested as an indicator of stress-dependent neuroendocrine activity and mental wellbeing (Young et al., 2002; Goodyer et al., 2001).