

Steps to a design-based understanding of depression

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Guinea Pig pups cry when separated from their mothers [4]. The biological basis of this response is linked to central serotonin receptors [4]. The hypothesised evolutionary explanation for separation distress in Guinea Pig pups (and human infants) is that it is an evolutionary adaptation with the function of providing security because the cries bring about caregiver proximity [2, 8, 10]. Human anti-depressant medications interfere with this stress response in Guinea Pigs, stopping attachment separation related crying before proximity has been gained. Moreover, testing whether novel compounds can terminate Guinea Pig pup separation distress is part of screening for new anti-depressants [4, 5].

Could anti-depression medications and talking therapies interfere with adaptive stress responses to depression in humans? A number of researchers have suggested that depression is an evolutionary adaptation, selected for fitness in the human environment of evolutionary adaptation (EEA) [1, 6, 3, 4]. Some researchers take the view that whilst sadness and low mood and subclinical depression have clear adaptive benefits, major depressive disorders are dysfunctional. Others suggest the boundary between subclinical and clinical depression provided by current diagnostic criteria does not constitute the correct dividing line between functional and dysfunctional symptoms, and that even in some major depressive disorders depressive symptoms can have significant adaptive benefits for the depressed person because of the impact on their social relations and thinking style [4]. This view resolves the 'high rate problem' in depression research by suggesting that only the most extreme cases of major depressive disorder constitute a dysfunctional response to stress and adversity [4]. Specific theories for the adaptive function of depression include:

- signalling a need for help from adversity [4]
- yielding in a conflict with a more powerful person [4]
- regulating investments patterns for energy conservation, including for the purpose of implicit social bargaining (in analogy to a going on a 'labour strike' until a more powerful person provides a better deal') [3]
- fostering disengagement from commitments to high level long term goals that have become unreachable [6]
- promoting analytic rumination that may provide insight into how to solve the problems that are causing the adversity [1]

Of course, there are many costs for an individual who has a major depressive disorder. Depressed people show poor concentration and motivation, become socially isolated, and experience anhedonia. Whilst some manifestations of disease have no utility, phenomena such as pain are viewed as subjectively aversive adaptations shaped by natural selection. Sadness, low mood and depression can be viewed as psychological pain that result from an individual experiencing stress and adversity. Andersen and Thomson [1] argue that

impairments associated with depression should not in themselves be used as conclusive evidence of disorder because they might also be caused by appropriate and adaptive responses to stress and adversity. For example, depressed people can experience ruminations about their situation which interfere with the ability to solve problems in laboratory conditions, and so these ruminations might be viewed as maladaptive impairments. But the kinds of ruminations that interrupt performance in the laboratory may help depressed people gain insight into their problems in 'real-life' outside of the laboratory. This is certainly what some depressed people claim [1].

Since it is a fundamental principle in medicine that it is more effective to treat the cause of an illness rather than the symptoms, attempting to change depressed people's cognitions from negative to positive may not be the most fruitful approach to treatment [1]. Instead, a better approach may be to help people understand and solve their problems. In addition, if sadness and low mood are adaptive responses to stress and adversity then studies of people with problematic depression should not use non-depressed people as control subjects, as this confounds pathological depression with adversity [4]. Studies instead need to have control groups with individuals currently showing an adaptive response to stress and adversity.

Limitations of adaptationist theories of depression include weakness in explaining the extended time course of depression. This is particularly the case for Nesse's theory that depression fosters disengagement from commitments to high level long term goals that have become unreachable [6, 9], and Andersen and Thomson's theory that depression promotes analytic rumination that may provide insight into how to solve the problems that are causing the adversity [1]. A contribution of this paper is to suggest this limitation is resolved by augmenting Nesse's and Andersen and Thomson's theories with elements of a design-based information processing model of grief set out by Wright, Sloman and Beaudoin [11]. This model describes how, within the human cognitive architecture, there is constant re-locating and transforming of motivators and references to internal and external entities. Important and influential control states linked to high level goals circulate around becoming gradually distributed in myriad control states through a process of diffusion. The most important percolate up a hierarchy of dispositional control states [11]. When someone interacts with another who they are attached to, the processes of circulation, percolation and diffusion give rise to a rich and highly distributed structure of attachment related motive generators, plans, preferences, predictive models, reflexes and automatic responses. So this attached individual will possess information in many places across their cognitive architecture about those they are attached to. This information will interrelate with other many other control states in complex ways as it diffuses through the architecture. Grief occurs in response to loss of the attachment figure because this diverse collection of control states cannot be quickly dismantled and will therefore for some time continue to be triggered and gain atten-

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tion. Almost any information that a person receives may activate a reference to the lost attachment figure [11, 7]. Adapting this model to the context of depression explains how analytic rumination and disengagement from high level goals often will not occur in a short space of time. In this view, depression is extended in time because it takes time for a distributed network of references and motivators relevant to old goals and current problems to be transformed and updated across the cognitive architecture.

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