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Review article

Darwinian models of depression: A review of evolutionary accounts of mood and mood disorders

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Abstract

Over the last ten years, there has been increased interest in the evolutionary origins of depressive phenomena. The current article provides a review of the major schools of thought that have emerged in this area. First, we consider important Darwinian explanations of depressed mood, including an integrative social risk hypothesis recently proposed by the authors. According to the social risk hypothesis, depression represents an adaptive response to the perceived threat of exclusion from important social relationships that, over the course of evolution, have been critical to maintaining an individual's fitness prospects. We argue, moreover, that in the ancestral environment, depression minimized the likelihood of exclusion by inducing: (i) cognitive hypersensitivity to indicators of social risk/threat; (ii) signaling behaviours that reduce social threat and elicit social support; and (iii) a generalized reduction in an individual's propensity to engage in risky, appetitive behaviours. Neurobiological support for this argument is also provided. Finally, we review three models that endeavour to explain the relationship between the adaptations that underlie depressed mood and clinically significant, depressed states, followed by a consideration of the merits of each.

Keywords: Darwinian medicine; Depression; Evolutionary psychology; Mood; Social risk

Contents

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1. Introduction

In the past, theoretical and empirical approaches to depression have been dominated by investigations targeting the symptomatology and treatment of severe, clinically-significant depressed states, and questions of etiology have been largely reserved for precipitants of immediate relevance to the depressed individual. While such proximate causes are undoubtedly of central importance to an understanding of depressive phenomena, more recently an expanding body of literature has focused on evolutionary, or functional, explanations. Arguably, Darwinian explanations of depression (e.g., Allen and Badcock, 2003; Bowlby, 1980; Gilbert, 1992; Nesse, 2000; Price et al., 1994; Watson and Andrews, 2002) are of practical and theoretical importance for three reasons. First, an understanding of the role of depressive phenomena in an evolutionary context provides insight into why people experience depression. If depressed states evolved in response to certain adaptive problems in our evolutionary history, they may be said to perform a particular adaptive function. By isolating that function, one stands to introduce a new tier of explanation — an understanding of depression rooted in the lives of our ancestors. Notably, such an approach is also likely to yield direct, practical implications with regards to treatment (Price et al., 1994; Watson and Andrews, 2002). Second, an evolutionary account of depression challenges the more traditional view that depressed states are intrinsically associated with dysfunction (Gilbert, 1998). If it can be shown that it played an adaptive role in our past, it follows that in some situations, depression provided reproductive benefits for the individual or their kin. Third, and finally, a Darwinian framework provides a powerful heuristic for generating testable hypotheses concerning psychological phenomena (Cosmides and Tooby, 1994), and facilitating broadly integrative explanations of them (Buss, 1995).

The idea that depression has evolved in response to specific adaptive problems is by no means a theoretical novelty. The argument enjoys a six-decade history (McGuire and Troisi, 1998), and has drawn contributions from a broad range of theorists (e.g., Allen and Badcock, 2003; Bowlby, 1980; Gilbert, 1992; Nesse, 2000; Price et al., 1994; Watson and Andrews, 2002). Generally, explanatory frameworks that have emerged from this expanding body of literature can be meaningfully grouped into two broad categories: explanations of normative, depressed mood, and explanations that explicitly target more severe, clinically significant depressed states. We shall now consider each of these prevailing schools of thought in turn.

2. Darwinian models of depressed mood

Although the concept of adaptation should not be reflexively applied to biological and behavioural features without sound logical reasons, we propose that mood states are appropriate targets for an evolutionary analysis. First, they are ubiquitous human capacities indicating a considerable degree of specialization (Cosmides and Tooby, 1994). Second, they are activated by specific contexts, suggesting that their input is specialized (Oatley, 1992). Finally, mood states are characterized by complex but coordinated sets of output in the form of physiology, overt behaviour, and conscious experience.

While there has been some very useful work on the distinct adaptive functions of other negative mood states like anxiety (Öhman et al., 1985), the adaptive significance of depressed mood has proven more elusive. For example, there is considerable evidence that the essential psychological "theme" of anxiety is threat, whereas the "theme" for depression is loss (Clark and Beck, 1989) or defeat (Price, 1972; Gilbert, 1992). Despite such distinctions, the argument that anxiety is an adaptive response to threat (facilitating early detection and responses) is more widely accepted than any evolutionary claim regarding depression. Indeed, it is not even clear what kind of loss is most relevant to depression (McGuire et al., 1997).

Nesse (1998) has argued that circumstances involving the loss (or the threat thereof) of a reproductive resource are likely to shape negative mood states, while circumstances involving the gain (or potential gain) of such resources are likely to shape positive ones. Thus, an evolutionary understanding of depression should depend on: i) identifying recurrent situations in the ancestral environment typically associated with biologically significant loss or gain; ii) describing the selection pressures in such situations (i.e., the particular social-reproductive goals that they would have threatened); and iii) isolating the features of depressed mood that would have enabled the organism to cope with these pressures (Nesse, 1990). While this approach has engendered a range of propositions, three prevailing schools of thought characterize the literature: the conservation of resources, social competition, and attachment theories of depression.

2.1. Theories of resource/energy conservation

Conservation of resource theories assert that the inhibition of appetitive functions associated with depression (i.e., low levels of energy, pleasure, and appetitive motivation) is likely to be adaptive by allowing an individual to conserve resources and later redirect them towards more productive endeavors. According to such views, depressed mood is instigated by a low rate of positive reward or insufficient control over rewards or punishments. Seligman's (1975) learned helplessness theory, for example, was founded on studies of animals exhibiting helpless behaviour when subjected to uncontrollable aversive events. Nesse's (2000) resource allocation model concentrates more on low rates of rewarding outcomes. Here, depressed mood represents an adaptive response to the propitiousness of situations by adjusting resource allocation (e.g., energy and investment) to inhibit investments in poor pay-off activities. In a similar vein, incentive disengagement theory (Klinger, 1975, 1993) proposes that depressed states disengage an organism from unobtainable incentives or goals, whilst Leahy's (1997) "sunk costs" model suggests that depression occurs when people persevere too long with behaviours resulting in low or diminishing rewards. Consistent with such models, Champion and Power (1995) have argued that depression-prone people tend to over-invest in a limited number of goals and, when such goals fail, there is a collapse in an individual's incentive and motivational systems.

2.2. The social competition theories

A central claim of evolutionary theory is that an individual's access to reproductive resources will vary according to his or her position, or rank, within the wider social group (Buss, 1999). Accordingly, humans are powerfully motivated to acquire such status through competitive encounters with others (Buss, 1991).

Perhaps the first author to apply this argument to depression was Price (1967), who has developed a Darwinian explanation of depressive states based on ritual agonistic (or fighting) behaviours (e.g., Price, 1998, 1992; Price and Sloman, 1987; Price et al., 1994). Price has argued that in any agonistic encounter between competitors, the stronger contestant will typically adopt an escalating strategy to increase its chances of success (i.e., continuing to participate in the contest, possibly threatening or attacking its opponent); whilst the losing contestant will adopt a de-escalating strategy, characterized by subordinate or yielding behaviour (Price, 1998). This latter strategy represents a withdrawal from the fight, reducing the risk of physical incapacity or death by sending 'no-threat' signals to deactivate the aggressive behaviour of the attacker (Price, 1984). Here, depression is conceptualized as an evolved, involuntary de-escalating strategy — enabling the individual to acknowledge defeat in ritual agonistic encounters, and adapt to the resulting loss in social rank (Price et al., 1994).

However, while fighting and intimidation are effective competitive strategies for most species, this is not always the case with humans and other primates (de Waal, 1989). Instead, resource acquisition and the formation of important social defenses rely heavily upon an individual's ability to elicit help from others. Thus, ritual agonistic behaviours have been replaced, at least partially, by competition through attraction — such that an individual's fitness prospects will depend largely upon his or her social value, prestige and attractiveness (Barkow, 1989). Gilbert and co-workers (e.g., Gilbert, 1997; Gilbert et al., 1995) embrace this argument in their notion of Social Attention Holding Power (SAHP).

SAHP refers to "the ability to elicit positive attention and social rewards, in the form of approval, praise, acceptance, respect, admiration, desire, etc." (Gilbert, 1997, p. 118). Gilbert suggests that humans compete for status through bestowing benefits on others to maximize SAHP, and that status differences are attributable to differences in the degree and quality of attention conferred by others (Buss, 1999). The biological significance of high SAHP has been highlighted by recent research that has shown that rhesus macaques will sacrifice a fluid reward in order to view images of high status conspecifics, but require an *overpayment* of fluid rewards in order to view images of low status conspecifics (Deaner et al., 2005). These findings suggest that even in non-human primates, social attention is differentially allocated according to status.

Importantly, agonistic strategies to cope with conspecific challenges have not disappeared, but are overlaid with SAHP-oriented systems. Consequently, not only may aggression be a common response to being deprived of the approval, support, admiration, respect or love to which one thinks oneself is entitled (Baumeister et al., 1996a,b), but loss of control over such signals may activate the defensive, subordinate, and social defeat responses of loss of confidence, anxiety, and depression.

2.3. The attachment theory of depression

Central to the attachment theory of depression is the proposed fitness consequences of affective bonding. Indeed, according to Buss (1991), mate retention, reciprocal dyadic alliance formation and coalition building are among the principal social selection pressures that have shaped human evolution.

One of the first proponents of the attachment model of depression was Bowlby (1969, 1973, 1980), who forwarded an evolutionary argument for interpersonal attachments based on parent–child interactions. Given the gradual maturation and protracted helplessness of human infants, the survival and emergent reproductive success of one's offspring necessitates an intensive contribution of time, commitment, energy and resources (Ingram et al., 1998). Bowlby (1988) argues that affective bonding ensures that a parent will provide the necessary commitments to safeguard the survival, and latent reproductive success, of his or her child(ren), and thus, the survival and perpetuation of that parent's genes.

It has also been suggested that affectional bonding between adult sexual partners has evolved to ensure the contribution of protection and resources from the father during the mother's pregnancy, and periods when the female must focus on the care of offspring (Ainsworth, 1991). Affiliative relationships are also seen to play a central role in the formation of social defenses and the acquisition of resources, as they enable the individual to elicit assistance from conspecifics (Ainsworth, 1991).

In specific relation to depression, the attachment model suggests that behaviours designed to maintain proximity to caregivers are instigated when significant affectional bonds are threatened (Gilbert, 1992). The model attributes depressive onset to the loss or dissolution of significant interpersonal relationships (Ingram et al., 1998), and has germinated several hypotheses concerning the adaptive function of depressive states. Some, for example, have suggested that depression inhibits exploratory or risk-laden activities in the absence of secure attachment bonds, and instigates appeasement-related behaviours designed to maintain relationships (Gilbert, 1992). Others have argued that the depressive response serves as a distress call (Frijda, 1994), provokes a search for the lost relationship (Averill, 1968), or motivates the sufferer to avoid further deterioration of preexisting bonds (Ingram et al., 1998).

2.4. The social risk hypothesis: an integrative view

While the perspectives outlined above provide important, complimentary insights into the adaptive significance of depression, we have suggested elsewhere that they are not mutually exclusive (Allen and Badcock, 2003). The conservation of resources views, while explaining the inhibition of reward seeking behaviour that is prominent in depression, do not explicitly account for the features of depressed mood that are specifically associated with social cognition and behaviour, especially self-depreciation and the fact that depression is

specifically associated with withdrawal from social contexts. Furthermore, they do not explain why precipitants of depression are more typically social than asocial in nature (e.g., Monroe et al., 1999; Rudolf et al., 2000). Moreover, although the idea that depression evolved as a response to the loss or dissolution of attachment bonds does point to the social origins of depressive phenomena, its explanatory power is limited by the fact that only a proportion of human relationships (especially amongst adults) can be validly characterized as attachment relationships (Fiske, 1991; Haslam, 1994), and that the contexts that elicit depression often involve humiliation and entrapment rather than interpersonal loss (Brown et al., 1995; Gilbert and Allan, 1998). Also, the idea that depressed states motivate an individual to re-establish lost relationships or seek out new ones does not fit well with the demobilization and social withdrawal that characterise of depression. Finally, although the argument that depression evolved from more primitive subordinate strategies does explain many of its features and has led to some supportive research findings (Gilbert and Allan, 1998), previous accounts have not explicitly explored the way in which this rank-oriented strategy has been adapted to the human social environment, especially in terms of the diversity of human social relationships (Fiske, 1991; Haslam, 1994), and the dramatic advances in social cognition associated with evolution whereby humans (and possibly some other primates) developed the capacity to think about the mental states of others (Bradshaw, 1997; Byrne and Whiten, 1986; Mithen, 1996). We have proposed that many of the important insights of these approaches can be integrated and extended by an analysis of social risk assessment in depression (Allen and Badcock, 2003).

Wiggins and Trapnell (1996) have argued that all forms of interpersonal relatedness can be understood in terms of two fundamental dimensions: agency (or power) and communion (affiliation). These dimensions of relatedness are emphasized by the social competition and attachment views, respectively. However, both of these domains of interpersonal behaviour present social risks, that is, risks to one's social circumstances, well-being and reputation. The dimension of agency, for example, presents risks of defeat, humiliation, and entrapment, while the dimension of affiliation presents risks of rejection and shunning. The social risk hypothesis (Allen and Badcock, 2003) suggests that depressive phenomena can be conceived as a defensive psychobiological response to increased risk within either one of these interpersonal domains.

Finally, another issue with the theoretical perspectives proposed to date is that they are often not explicit about which aspects of depressive phenomena, or forms of depression, they conceive of as reflecting species-wide adaptive functions, and which aspects they consider maladaptive. There is a diversity of opinion, for instance, about whether clinical forms of depression, such as Major Depressive Disorder or Dysthymia, reflect evolutionary adaptations or adaptive mechanisms operating outside of their adaptive range. We will return to this issue later in the paper.

2.4.1. Avoiding social exclusion

Many theorists have argued that depressed states are most essentially related to reductions of positive affect (anhedonia being a key defining feature), and that the regulators of positive affect are embedded in *social* cognition and behaviour (Allen and Badcock, 2003; Gilbert, 1989, 1992; Gotlib and Hammen, 1992; Joiner and Coyne, 1999; Watson, 2000). Critical to our functional or evolutionary view of depression is the proposition that there are various biological processes that guide individuals to enact certain social roles. There are many clues in the research literature that suggest that social processes (both in terms of social cognition and interpersonal behaviour) play a critical role in the aetiology and maintenance of depressed states. Critical empirical observations here include findings that depression is often precipitated by interpersonal events (as noted above), and that interpersonal processes regularly mediate the exacerbation or resolution of depressive episodes (Joiner and Coyne, 1999). Stressful interpersonal contexts are amongst the most reliable predipitants of depressed states (Kendler et al., 2003; Monroe et al., 1999) and certain interpersonal behaviours, such as excessive reassurance seeking, are strong and specific predictors of risk for depression (Joiner and Metalsky, 2001).

Our model of the function of depressed states seeks to explain why there is such a close link between social cognition, social behaviour, and depressive phenomena. The *social risk* hypothesis of depression (Allen and Badcock, 2003) suggests that depressed mood (i.e., down-regulation of positive affect and confident engagement in the world) evolved to facilitate a risk-averse approach to social interaction in situations where individuals were typically at risk of exclusion from social contexts (i.e., dyadic relationships or groups) that were vital to dealing with adaptive, socio-reproductive challenges.

As we have already noted, evolutionary models emphasize that an individual's access to non-plentiful, fitness-enhancing resources depends largely upon his or her position in a social context. Indeed, given that human groups contain concentrations of particular reproductive resources, including potential mates, kin to whom altruism can be directed, and non-kin with which to exchange resources, the effect of social exclusion on various proximal adaptive tasks can be considerable (Buss, 1990). Furthermore, in the Pleistocene period, social exclusion may have threatened one's survival by excluding the individual from group-based benefits in terms of protection from predators or foraging for food.

Consequently, a critical matter for the individual is to detect when the danger of exclusion from currently beneficial social relationships is high. The argument that individuals are highly sensitive to how they are perceived and valued by others, and that this sensitivity is based on an evolved human drive for social belonging, has previously been forwarded by Baumeister and Leary (1995), Bowlby (1969) and Tooby and Cosmides (1996), and has also received some empirical support (see Gardner et al., 2000; Williams and Sommers, 1997). The social risk hypothesis maintains that the loss or dissolution of significant interpersonal relationships, and/or experiences implicative of low status (such as defeat or humiliation), can be categorised more broadly as signals that throughout evolutionary history have been associated with the kind of lowered social value that could lead to ostracism from important social contexts. Indeed, we would argue that precipitants of

depression do not just involve loss or defeat, but rather, any socially relevant experience that indicates (or has indicated through evolutionary history) to an individual that his or her ability to successfully negotiate important social contexts is critically low. Such precipitants may include, for example, negative interpersonal experiences (such as losses or rejections); the failure of an important goal; loss of social rank or status; and/or perceptions of a lack of control in social situations (as is the case with experiences of entrapment). Once the depressive mechanism is activated, the usual opportunity-seeking social investment strategy shifts towards a risk-averse strategy, namely, that of depressed mood.

2.4.2. Ecological actions of the depression mechanism

As others have also proposed (Leahy, 1997; Nesse, 2000), the social risk hypothesis suggests that the adaptive function of the depressive state is to protect an individual's fitness prospects by minimizing behaviours that put social connections at risk, and ensuring the reduction and avoidance of further threats to reproductive opportunities.

Arguably, under the threat of social exclusion, those who were able to *cautiously* increase their social value, while minimizing the risk of further reductions, were more likely to have preserved their participation in adaptive social contexts, and were favoured by natural selection. On the other hand, those who did not adjust their social behaviour in the face of low social value were at even greater risk of exclusion from critical social alliances.

Thus, we have argued that the depression mechanism controls aspects of both social perception and behaviours to reduce the likelihood of further, critical reductions in an individual's social value. The mechanism affects social-perceptual processes in that the individual becomes hypersensitive to indications of social risk. In terms of social behaviour, the mechanism influences both communicative behaviour (signalling in order to reduce threats and to elicit safe forms of support), and resource acquisition behaviours (a general reduction in behavioural propensities towards high-risk investments that may result in interpersonal conflict or competition).

2.4.3. Neurobiological evidence consistent with the social risk hypothesis

In this paper, we have briefly outlined a model of depression that we believe successfully integrates many of the previous arguments concerning the adaptive significance of depressed mood states. In the following section, we explore how this hypothesis is consistent with findings that have emerged from neurobiological research concerning depressive phenomena.

First, it is interesting to note that a neurochemical explanation for the link between negative social experiences and onset of depression has been forwarded by Deakin (1996). Deakin (1996) has argued that 5-HT_{1A} receptors mediate coping responses which minimise the impact of psychosocial adversity by modulating the operation of medial temporal lobe memory circuits. Through a mechanism of dissociation and denial, these circuits function as a 'resilience' system that reinstates normal behaviours in the face of chronic or repeated adversity. Deakin (1996) contends that social contact is critical to maintain the functioning of 5-HT_{1A} resilience mechanisms, including those that preserve self-esteem. Thus, in contexts involving minimal social contact, psychosocial adversity is likely to interfere with 5-HT neurotransmission, causing a breakdown in this resilience system and thereby resulting in low self-esteem and depression.

Consistent with this, inhibition of monoamine function, including serotonergic function, has long been associated with depression, and is thought to be the principal mechanism of the action of some antidepressant drugs (Lambert et al., 2000; Stahl, 2000). Furthermore, work on the effects of serotonin on social behaviour in non-human primates has also implicated this neurotransmitter system in social vigilance, revealing an association between low levels of CNS responsiveness to 5-HT and the devotion of a high percentage of time to inter-animal vigilance (McGuire et al., 2000).

Additional work has pointed to a relationship between neurobiological correlates of depression and hypersensitivity to social risk. For example, the amygdala, a brain region that has a critical role in the detection of threatening and fear-related stimuli, is known to be hyperactive during depressed states (Cahill et al., 1996; Davidson and Irwin, 1999). Most relevant to our argument, however, is that fact that the amygdala has also been shown to have a critical role in complex social judgments, especially judgments of socially threatening stimuli (Adolphs, 1999). It has been demonstrated, for example, that lesions to the amygdalae are associated with impairment in judging the emotional and social characteristics of faces, especially those that imply social danger such as untrustworthiness, and inapproachability (Adolphs et al., 1994, 1998). Amygdala lesions have also been associated with impairment in the ability to attribute social meaning to stimuli (Adolphs, 1999). Interestingly, lesion (Fine et al., 2001) and fMRI (Baron-Cohen et al., 1999) studies have also implicated the amygdala in the development or mediation of "theory of mind", the ability to attribute mental states to the self and others, and predict and understand others' behaviour on the basis of their perceived mental states (Premack and Woodruff, 1978). Although it is not currently clear whether the amygdala serves some specifically social cognitive functions, or is simply recruited in the detection of threat regardless of context, the finding that the amygdala is necessary for the detection of social danger (Adolphs et al., 1998) and the operation of theory of mind suggests that hyperactivity of these structures would be expected during states of high sensitivity to social risk.

Finally, there exists some neurobiological evidence for the proposal that depression is associated with reduced social risk taking. However, before examining such evidence, it is first important to consider some relevant aspects of the psychology of risk taking.

Much of the research in this area has tested economic models of risk, requiring participants to make choices between gambles with a known monetary value and a known probability (Lopes, 1987). Although this situation is considerably removed from the social one of interest here, we believe that this work has revealed general principles regarding human risk perception that are useful in understanding socially risk-averse behaviour.

In order to explain a number of findings that diverged from the predictions of expected utility theory, Kahneman and Tversky (1979) have proposed an alternative view of decision-making under risk called "prospect theory." They propose that "prospects" (i.e., expected outcomes) are "edited" by various psychological processes, and that it is only after this editing stage that the decision-maker chooses the prospect of highest value. The overall value of an edited prospect is determined by two functions. The first function associates the *probability* of an outcome with a decision weight that determines the impact of this probability on the overall value of the prospect. The second function assigns a subjective *value* to each outcome. Kahneman and Tversky (1979), and subsequent researchers, have shown how these functions can alter the value of prospects, and result in decisions under risk that are surprising according to normative criteria (Lopes, 1987; Slovic, 1995). More recently, Loewenstein et al. (2001) have emphasized the crucial role of affect in determining both the outcomes of risk decisions, and the way in which prospects are edited.

The relevance of this to depression is that we expect that depressed mood will be associated with an alteration in psychological processes that reduces the behavioural propensity to choose particularly risky options. The most risk-averse behaviour will occur when probability judgments under-weigh the likelihood of gains and over-weigh the likelihood of losses, and when judgments concerning the value of outcomes consider gains to be of low value and losses to be of high (negative) value.

These principles predict some of the neurobiological correlates of depression. For example, many of the neurobiological systems disordered in depressed states are functionally related to the perception and valuation of future outcomes, especially rewards. Lesion, electrophysiological and neuroimaging studies have all established that depression is associated with hypoactivation of regions in the left prefrontal cortex (Davidson and Irwin, 1999; Davidson et al., 2000). The dorsolateral regions of the prefrontal cortex in particular have long been understood to be involved in working memory, including working memory used to maintain reward expectancy (Watanabe, 1996). Davidson et al. (2000) have proposed that this kind of "affective working memory" may involve the evocation of actual emotion when emotional stimuli are not present. Regions of the prefrontal cortex, therefore, are critical to risk taking behaviour, where the propensity to take risks relies on the value assigned to future outcomes. Inhibition of such functions, especially in the left frontal region (which appears to show some specificity to the representation of positive future outcomes; see Tomarken and Keener, 1998), may be an essential feature of the risk inhibition functions of depressed states. Notably, hypoactiviation of brain activity in the left frontal region is also hypothesized to be associated with low positive affect and appetitive motivation (Davidson, 1993). This is consistent with the notion that depression involves an emotional/motivational editing process that inhibits the value of positive outcomes and potentiates the value of negative ones.

Here, it is worthwhile noting that an important aspect of judging the value of outcomes when evaluating prospects is that the outcomes being evaluated are in the future. The social risk hypothesis predicts that depressed states should be chiefly associated with insensitivity to future positive consequences, rather than insensitivity to immediate stimuli. To elaborate, a classic maxim of low risk behaviour, "a bird in the hand is worth two in the bush", suggests that evaluative mechanisms designed to minimize risk should undervalue future positive outcomes in favour of more immediate stimuli. In support of this view, there is some evidence to suggest that the prefrontal hypoactivity observed in depressives might be implicated in decision-making. Bechara et al. (1994) have found, for example, that patients with damage to the ventro-medial region of the prefrontal cortex are insensitive to the future consequences of their behaviour, and instead, appear to be guided by immediate reward prospects alone.

Finally, support for the social risk hypothesis can also be gleaned from neurochemical findings. As we have discussed, inhibition of monoamine function has long been associated with depression (Stahl, 2000). Two of the monoamine systems, the dopaminergic and serotonergic, are though to have functions that specifically affect an individual's capacity to engage in risk-taking behaviour. For instance, dopamine is understood to be crucial for incentive motivation and reward seeking (Depue and Collins, 1999; Ikemoto and Panksepp, 1999). Serotonin, on the other hand, is typically associated with emotional constraint, and is thought to have an inhibitory effect on dopaminergic and noradrenergic function (Depue and Spoont, 1986). Nevertheless, the capacity for emotional constraint has also been associated with the capacity to motivationally engage with longer-term goals and more distal stimuli (Katz, 1999). In other words, serotonin can be understood to moderate the impact of immediate affective stimuli so that behaviours associated with more distal long term goals can prevail.

In order to take risks, one must value a potential future outcome in favour of the status quo (i.e., the two in the bush must be worth more than the bird in the hand). However, if one is in a situation where risk taking is not propitious, it makes sense for the organism to adjust the functioning of these fundamental affective neurochemical systems in order to inhibit engagement with future rewards (i.e., dopaminergic hypoactivity) and to increase responsiveness to immediate affective experience to the detriment of behaviour oriented towards future goals (i.e., serotonergic hypoactivity). Indeed, the principal behavioural mechanism by which serotonergic drugs alleviate severe depression might relate to the fact that they potentiate re-engagement with long-term goals and thus facilitate adaptive risk taking.

2.4.4. Summary

Arguably, the theoretical contribution of our social risk hypothesis is, at the very least, twofold. First, the model illustrates the utility of Darwinian theory when generating unique, explicative and testable hypotheses relating to depression. Second, it provides a comprehensive explanatory framework that readily integrates previous evolutionary accounts, and a wide range of research findings concerning etiological, neurobiological and psychosocial factors associated with depressed mood (for a comprehensive discussion of these

issues, see Allen and Badcock, 2003).That said, given our view that depressive illness does not represent an adaptation, but rather a maladaptive outcome of the psychobiological mechanism operating outside of its adaptive range, the social risk hypothesis does not endeavour to provide an evolutionary explanation for clinically-significant depressed states. However, some models extend the Darwinian framework to deal explicitly with clinical depression, and it is to these that we shall turn now.

3. Evolutionary explanations of clinical depression

According to Nettle (2004), conventional evolutionary accounts of *clinical* depression can be broadly divided into two categories. The first type of model, the *dysregulation* view (to which our social risk hypothesis, and other models such as Nesse's, 2000, resource-conservation view and more recent developments of the social competition hypothesis belong), sees clinical depression as a form of dysregulation, chronic overactivation or inappropriate evocation of the mechanism upon which depression is based (Nettle, 2004). Although dysregulation views provide insight into the core psychological mechanisms responsible for depression, Nettle (2004) argues that they do not directly address the issue of individual variations in depressive vulnerability.

By contrast, *adaptation* models claim that clinical depression itself is adaptive. Examples of this latter view include early forms of the social competition hypothesis (see Price et al., 1994), and the models advanced by Watson and Andrews (2002) and Hagen (1999), which emphasize the capacity of severely depressed states to elicit resources from the social environment. Notably, a third class of evolutionary theory has recently been proposed by Nettle (2004), one that focuses upon individual, personality differences to explain variation in peoples' susceptibility to depression. Three important models corresponding to each school of thought are reviewed below.

3.1. Failure of the mechanism: chronic stress and clinical depression

According to Gilbert (2001), severe depressive illness represents a maladaptive response emerging from exposure to heightened and/or chronic stress. He begins with the argument that evolution has equipped human beings with an extensive repertoire of defensive responses (e.g., fight, flight, submit) that help us deal with particular threats to inclusive fitness. If the defensive response is effective, the individual either escapes/avoids the stressor or accommodates/adjusts to it. However, if the defence fails, the individual is in danger of entering a dysregulated state — an exacerbation of symptoms that falls beyond the normal range of adaptive functioning (McGuire and Troisi, 1998). Gilbert (2001) explains many of the features of clinical depression by suggesting that severe depressed states represent a maladaptive pattern of dysregulated defenses. The link between chronic stress and depressive illness is well established (Monroe and Hadjiyannakis, 2002), and suggests that depression is associated with heightened and prolonged arousal of ineffectual or arrested defensive responses.

Notably, Gilbert's (2001) view that clinical depression results from a chronic state of stress where a number of psychobiological defenses are aroused but ineffective is compatible with our own, social risk model in a number of ways. Both arguments assert that normative levels of depressed mood represent an evolved, *defensive* psychobiological strategy associated with a number of fitness-enhancing functions. Gilbert also sees the social environment as critical when attempting to understand the etiology and function of depressed states. Furthermore, both models interpret the increasing prevalence of major depression in terms of the incompatibility between mechanisms that have evolved during Pleistocene conditions and sociocultural conditions in the modern world (see Allen and Badcock, 2003).

3.2. The social navigation hypothesis

Watson and Andrews (2002) have forwarded a Darwinian conceptualisation that sees clinical depression itself as adaptive. They argue that clinical depression is an appropriate target for Darwinian analysis for two reasons. First, it is highly prevalent, with estimations of lifetime risk in the US as high as 20% (Kessler et al., 1994). Second, there is evidence to suggest that the potential to experience clinically-significant depressed states may be cross-culturally universal (Nesse and Williams, 1994).

According to their social navigation hypothesis, the onset of depressive illness is specifically associated with critical social problems and conflicts. Furthermore, the adaptive significance of clinical depression lies in two complimentary, adaptive functions. The first, social rumination, function refers to the proposal that depression generates cognitive changes that both focus and enhance an individual's ability to analyse and solve crucial social problems (Watson and Andrews, 2002). Here, it is argued that when highly complex, socially embedded problems are sufficiently critical, it is likely to be adaptive to shut down hedonic interests and the use of cognitive resources on physical activities to focus exclusively on the problem at hand. This view is consistent with findings that depression is associated with a preoccupation with social information (Marsh and Weary, 1994; Swallow and Kuiper, 1993; Yost and Weary, 1996). Another important component of the social rumination function is that depression should be characterised by improved social problem-solving. In support of this view, Watson and Andrews (2002) cite research demonstrating that depressives perform better than non-depressives on certain social problem-solving tasks (Yost and Weary, 1996; Lane and DePaulo, 1999); are less likely to make the fundamental attribution error (Yost and Weary, 1996); and are more accurate in assessments of personal control over contingent outcomes (Ackermann and DeRubeis, 1991; Alloy and Abramson, 1979). Interestingly, they also refer to literature specifying that low serotonin, which is well known to be associated with depression, retards physical activity (Jacobs and Fornal, 1997), and improves performance on demanding cognitive tasks (Buhot, 1997).

The second function of clinical depression proposed by the social navigation hypothesis is a social motivation function. Here, through honest signalling and/or passive, unintentional fitness extortion, the high costs associated with depression are

seen to motivate reluctant coalitional partners to provide the depressive with further investments or concessions (Watson and Andrews, 2002). In relation to signalling, Watson and Andrews (2002) argue that depression and suicidality might operate as a high credibility "cry-for-help", motivating conspecifics who have a positive fitness interest in the depressive to provide such help. This idea has also been explored by Lewis (1934) and Stengel (1974). In regards to fitness extortion, the considerable risk of death associated with depression provides kin and allies with a potent incentive to care for the depressive. In other words, depression is likely to elicit help from social partners because it is better to help, and attempt to resolve the depressive episode, than continue to suffer the escalating costs (Watson and Andrews, 2002). In this way, depression may well represent a strategy for extorting increased investment from an entire social network or specific coalitional partners. Notably, a similar notion has previously been forwarded by Hagen (1999) in regards to post partum depression.

3.3. Nettle's individual difference model

Another important model emerging from evolutionary conceptualisations of clinical depression has been forwarded by Nettle (2004), one that appeals to individual, personality differences. Nettle rejects adaptationist claims regarding clinically depressed states for a number of reasons. First, the well-established heritability of depression (Sullivan et al., 2003; NIMH, 1999) contradicts the evolutionary proposition that, as an adaptation, the capacity for depression should be ubiquitous among all human beings, and that heritable variation in the capacity for depression should have been, through a process of natural selection, reduced to approximately zero (Nettle, 2004).

Second, if we accept that clinical depression is an adaptation, the mechanism should be activated by appropriate environmental circumstances, and should only be active when such circumstances occur. However, Nettle (2004) cites a number of studies that call into question the etiological significance of negative life events in the development of depression. For example, Weissman (1987) has demonstrated that the strongest risk factor for depression is the presence of a previous depressive episode, far outweighing other social and biological causes. Moreover, research into environmental causes of depression is methodologically limited. Chiefly, since depression tends to be chronic or recurrent (Pakriev et al., 2001), and given that it often causes problems in social relationships (e.g., Coyne, 1976; Reich, 2003), it is difficult to distinguish between negative life events resulting from previous clinical or sub-clinical episodes, and negative life events responsible for depression. Also, in light of the chronicity or common recurrence of depressive illness, and research showing that depressive relapse can be weakly, if not completely unrelated, to life events (Lewinsohn et al., 1999; Paykel, 2002; Post, 1992), it appears that depressogenic phenomena can indeed be present in the absence of appropriate environmental triggers.

Third, Nettle (2004) refutes the adaptation view on the grounds that clinically significant depressed states do not provide evidence of good design. In this vein, there exists considerable evidence to suggest that the depression mechanism fails to perform the key adaptive functions that adaptationists claim have led to its selection. For example, in regards to Watson and Andrews' (2002) proposed social rumination function, the literature is unmistakably equivocal. Indeed, while there is some support for improved social problem-solving amongst depressives (Lane and DePaulo, 1999; Yost and Weary, 1996), the majority of work in this area has demonstrated that depression is associated with reduced interpersonal problem solving skills (Cooley and Nowicki, 1989; Gotlib and Asarnow, 1979; Watkins and Baracaia, 2002; Persad and Polivy, 1993). In fact, it is likely that there is a curvilinear relationship between the severity of depression and the likelihood that depressive biases in information processing will facilitate social problem solving. For example, although some recent studies have shown that mild depressed states facilitate both social reasoning (Badcock and Allen, 2003) and performance on theory of mind tasks (Harkness et al., 2005), other studies using the same assessment procedures have found that in clinical populations, these advantages are absent or even reversed (Badcock and Allen, in preparation; Lee et al., 2005). Moreover, Nettle (2004) points out that with the exclusion of post partum incidences of depression, there exists little empirical support for a social motivation function, with some studies demonstrating a relationship between severe depression and the loss of social support (Coyne, 1976; Monroe and Steiner, 1986).

Fourth, and finally, if clinical depression is inherently adaptive, individuals who lack the mechanism due to chance mutation or disrupted ontogeny should have reduced fitness when compared to a normal population (Nettle, 2004). However, not only is the potential for clinical depression absent in most people, those who suffer from the condition exhibit impaired psychosocial functioning, increased mortality rates and poorer physical health (Angermeyer et al., 2002; Klerman, 1989), making groups who in any way benefit from clinical level depressive symptomatology extraordinarily difficult to identify (Nettle, 2004).

On the basis of these considerations, Nettle (2004) considers the view that clinical depression is adaptive implausible. How, then, are clinically significant depressed states explained from an evolutionary point of view? In order to address this issue, he turns to the personality dimension of neuroticism.

Nettle (2004) argues, like the authors of the current paper and others previously (e.g., Nesse, 1998; Price et al., 1994; Gilbert and Allan, 1998), that affect systems are adaptations that enable an individual to negotiate complex human relationships by instantiating behaviours that minimise the impact of circumstances damaging to inclusive fitness, or capitalise on opportunities likely to enhance it. However, Nettle (2004) maintains that while the design features of affect systems are common to all human beings, individuals vary in regard to the reactivity of such systems. He points out, moreover, that variation in the lability of negative affect is reflected in neuroticism (Watson and Clark, 1984), which, given the complexity of neurobiological mechanisms responsible for emotional reactivity, is characterised by a polygenic continuum, an approximately normal distribution, and considerable heritable variation (Tellegen et al., 1988).

Nettle (2004) claims that while high neuroticism has been shown to be related to poor health and relationship failure (Kelly and Conley, 1987; Neeleman et al., 2002), evidence also exists to suggest that increasing neuroticism may be associated with certain reproductive benefits. For example, a positive relationship has been identified between neuroticism and competitiveness (Ross et al., 2001), and neuroticism has been shown to predict success among university students when individuals are able to cope with its negative aspects (McKenzie, 1989; McKenzie et al., 2000). To explain such findings, Nettle (2004) argues that "[h]aving a fairly reactive negative affect system causes people to strive hard for what is desirable and to avoid negative outcomes, and this may well be associated with increasing fitness" (p. 98).

In short, then, Nettle (2004) proposes that increasing neuroticism may have been selected for in the evolutionary environment because it engendered striving behaviours in interpersonal contexts, until a point where its negative consequences outweighed its benefits. This would result in a normal distribution around an adaptive peak, with considerable heritable variation and a significant proportion of the population falling on either side of this optimal point (Nettle, 2004). According to this view, individuals with excessive neuroticism are vulnerable to all sorts of affective disorders, including clinical depression. More specifically, Nettle (2004) contends that depression results from the tendency of the affect system to spiral into a self-reinforcing cycle, locking the mechanism in a state of maladaptive negativity, and over-riding the typical self-correcting tendencies of normal emotional mechanisms.

3.4. Conclusion

It is our view that the models reviewed above provide valuable insights into the possible evolutionary processes that underlie depressed states. Gilbert's (2001) chronic stress model, for example, provides a promising explanation of how the adaptive capacity for depressed mood may, given appropriate environmental circumstances, exacerbate into more severe, maladaptive depressed states. Moreover, and in particular reference to the social risk hypothesis (Allen and Badcock, 2003), Gilbert presents a plausible account of what might occur when an individual's estimated social value remains critically low despite the operation of the proposed depressive mechanism. On the other hand, Nettle's (2004) model specifies how individual differences in trait vulnerability may impact upon evolved, adaptive systems. Given the prevailing objective of evolutionary psychology to establish universally applicable principles of psychological phenomena, the interaction between species-typical adaptation and individual differences is commonly overlooked in the field. At this point, it should also be noted that Nettle's position and the dysregulation views are by no means incompatible. As Nettle (2004) himself suggests, dysregulation models elucidate the core psychological mechanisms activated in depression, and explain the occurrence of normal levels of depressed mood that we all experience from time to time, while the individual difference view highlights why there exists individual variation in peoples' susceptibility to clinical depressed states. In closing, while we would agree with Nettle (2004) that clinically significant depressed states are unlikely, in themselves, to represent adaptations, the social navigation hypothesis does emphasise the socially-embedded nature of depression, and provides numerous suggestions concerning how the depressive mechanism may assist the individual in negotiating complex social problems. Moreover, we would agree with Watson and Andrews' (2002) assertion that the practical implications of evolutionary conceptions in relation to the treatment of depressive illness should not be overlooked. Regardless of whether clinical depression represents an adaptive strategy in and of itself, we contend that consideration of evolutionary explanations of depressive phenomena is likely to be fruitful in identifying key, socially-embedded precipitants of depressive illness, and has direct consequences in terms of choosing efficacious treatment options. Indeed, given that evolutionary models explicitly address the way in which social cognition and behaviour emerge out of biological adaptations that were, in turn, shaped by the social context throughout human history, they provide a strong basis for understanding and extending the important synergies that are likely to be obtained by combining biological and psychosocial interventions (Keller et al., 2000; TADS, 2004).

References

Ackermann R, DeRubeis RJ. Is depressive realism real? Clin Psychol Rev 1991;11:565–84.

Adolphs R. Social cognition and the human brain. Trends Cogn Sci 1999;3: 469–79.

Adolphs R, Tranel D, Damasio H, Damasio A. Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. Nature 1994;372:669–72.

Adolphs R, Tranel D, Damasio A. The human amygdala in social judgment. Nature 1998;393:470–4.

Ainsworth MDS. Attachments and other affectional bonds across the life cycle. In: Parkes CM, Stevenson-Hinde J, Marris P, editors. Attachment across the life cycle. London: Routledge; 1991. p. 33–51.

Allen NB, Badcock PBT. The social risk hypothesis of depressed mood: evolutionary, psychosocial, and neurobiological perspectives. Psychol Bull 2003;129(6):887–913.

Alloy LB, Abramson LY. Judgment of contingency in depressed and nondepressed students. Sadder but wiser? J Exp Psychol Gen 1979;52: 366–78.

Angermeyer MC, Holzinger A, Matschinger H, Stengler-Wenzke K. Depression and quality of life: results of a follow-up study. Int J Soc Psychiatry 2002;48:189–99.

Averill JR. Grief: its nature and significance. Psychol Bull 1968;70:721–48.

Badcock PBT, Allen NB. Adaptive social reasoning in depressed mood and depressive vulnerability. Cogn Emot 2003;17:647–70.

Badcock PBT, Allen NB. Mood and domains of risk taking. (in preparation).

Barkow JH. Darwin, sex and status: biological approaches to mind and culture. Toronto: University of Toronto Press, 1989.

Baron-Cohen S, Ring HA, Wheelwright S, Bullmore ET, Brammer MJ, Simmons A, et al. Social intelligence in the normal and autistic brain: an fMRI study. Eur J Neurosci 1999;11:1891–8.

Baumeister RF, Leary MR. The need to belong: desire for interpersonal attachments as a fundamental human motivation. Psychol Bull 1995;117: 497–529.

Baumeister R, Smart L, Boden J. Relation of threatened egotism to violence and aggression: the dark side of high self-esteem. Psychol Rev 1996a;101: 5-33.

Baumeister RF, Smart L, Boden JM. Relation of threatened egotism to violence and aggression: the dark side of high self-esteem. Psychol Rev 1996b;103: 5–33.

Bechara A, Damasio AR, Damasio H, Anderson SW. Insensitivity to future consequences following damage to human prefrontal cortex. Cognition 1994;50:7-15.

Bowlby J. Attachment and loss. vol. 1. Attachment. New York, N.Y.: Basic Books, 1969.

Bowlby J. Attachment and loss. vol. 2. Separation, anxiety and anger. New York, N.Y.: Basic Books, 1973.

Bowlby J. Attachment and loss. vol. 3. Loss: sadness and depression. New York, N.Y.: Basic Books, 1980.

Bowlby J. A secure base: parent–child attachment and healthy human development. New York, N.Y.: Basic Books, 1988.

Bradshaw JL. Human evolution: a neuropsychological perspective. Hove, UK: Psychology Press, 1997.

Brown GW, Harris TO, Hepworth C. Humiliation and entrapment among women developing depression: a patient and nonpatient comparison. Psychol Med 1995;25:7-21.

Buhot M-C. Serotonin receptors in cognitive behaviours. Curr Opin Neurobiol 1997;7:243–354.

Buss DM. The evolution of anxiety and social exclusion. J Soc Clin Psychol 1990;9:196–201.

Buss DM. Evolutionary personality psychology. In: Rosenzweig MR, Porter LW, editors. Ann rev of psychol, vol. 42. Palo Alto, CA: Annual Reviews; 1991. p. 459–91.

Buss DM. Evolutionary psychology: a new paradigm for psychological science. Psychol Inq 1995;6:1-30.

Buss DM. Evolutionary psychology: the new science of the mind. New York, N.Y.: Allyn & Bacon, 1999.

Byrne RW, Whiten A. Machiavellian intelligence: social expertise and the evolution of intellect in monkeys, apes, and humans. Oxford: Oxford University Press, 1986.

Cahill L, Haier RJ, Fallon J, Alkire MT, Tang C, Keator D. Amygdala activity at encoding correlated with long term free recall of emotional information. Proc Natl Acad Sci USA 1996;93:8016–21.

Champion LA, Power MJ. Social and cognitive approaches to depression. Br J Clin Psychol 1995;34:485–503.

Clark DA, Beck AT. Cognitive theory and therapy of anxiety and depression. In: Kendall PC, Watson D, editors. Anxiety and depression: distinctive and overlapping features. San Diego: Academic Press, 1989.

Cooley EL, Nowicki S. Discrimination of facial expressions of emotion by depressed subjects. Genet Soc Gen Psychol Monogr 1989;115:449.

Cosmides L, Tooby J. Beyond intuition and instinct blindness: toward an evolutionarily rigorous cognitive science. Cognition 1994;50: 41-77.

Coyne JC. Depression and the response of others. J Abnorm Psychology 1976;85:186–93.

Davidson RJ. The neuropsychology of emotion and affective style. In: Lewis M, Haviland JM, editors. Handbook of emotions. New York, N.Y.: Guilford Press; 1993.

Davidson RJ, Irwin W. The functional neuroanatomy of emotion and affective style. Trends Cogn Sci 1999;3:11–21.

Davidson RJ, Jackson DC, Kalin NH. Emotion, plasticity, context, and regulation: perspectives from affective neuroscience. Psychol Bull 2000;126: 890–909.

de Waal FMB. Peacemaking among primates. Cambridge, MA: Harvard University Press, 1989.

Deakin JFW. 5-HT, antidepressant drugs and the psychosocial origins of depression. J Psychopharmacol 1996;10:31–8.

Deaner RO, Khera AV, Platt ML. Monkeys pay per view: adaptive value of social images by rhesus macaques. Curr Biol 2005;15:543–8.

Depue RA, Collins PF. Neurobiology of the structure of personality: dopamine, facilitation of incentive motivation, and extraversion. Behav Brain Sci 1999;22:491–569.

Depue RA, Spoont MR. Conceptualizing a serotonin trait: a behavioral dimension of constraint. Ann NY Acad Sci 1986;487:47–62.

Fine C, Lumsden J, Blair RJR. Dissociation between 'theory of mind' and executive functions in a patient with early left amygdala damage. Brain 2001;124:287–98.

Fiske AP. Structures of social life: the four elementary forms of social relationship. New York, N.Y.: Free Press, 1991.

Frijda NH. Emotions are functional, most of the time. In: Ekman P, Davidson R, editors. The nature of emotion: fundamental questions. New York, N.Y.: Oxford University Press; 1994. p. 97-177.

Gardner WL, Pickett CL, Brewer MB. Social exclusion and selective memory: how the need to belong influences memory for social events. Pers Soc Psychol Bull 2000;26:486–96.

Gilbert P. Human nature and suffering. Hove, U.K.: Lawrence Earlbaum Associates, 1989

Gilbert P. Depression: the evolution of powerlessness. New York, N.Y.: Guilford Press, 1992.

Gilbert P. The evolution of social attractiveness and its role in shame, humiliation, guilt and therapy. Br J Med Psychol 1997;70:113–47.

Gilbert P. Evolutionary psychopathology: why isn't the mind designed better than it is? Br J Med Psychol 1998;71:353–73.

Gilbert P. Depression and stress: a biopsychosocial exploration of evolved functions and mechanisms. Stress: The Internat J of the Biol of Stress, vol. 4. 2001. p. 121–35.

Gilbert P, S.Allan S. The role of defeat and entrapment (arrested flight) in depression: an exploration of an evolutionary view. Psychol Med 1998;28:585–98.

Gilbert P, Price J, Allan S. Social comparison, social attractiveness and evolution: how might they be related? New Ideas in Psychology: an Internat J Innovat Theory in Psychol, vol. 13. 1995. p. 149–65.

Gotlib IH, Asarnow RF. Interpersonal and impersonal problem-solving skills in mildly and clinically depressed university students. J Consult Clin Psychol 1979;40:1190–3.

Gotlib IH, Hammen CL. Psychological aspects of depression: towards a cognitive interpersonal integration. Chichester: John Wiley and Sons, 1992.

Hagen EH. The functions of postpartum depression. Evol Hum Behav 1999;20: 325–59.

Harkness KL, Sabbagh MA, Jacobson J, Chowdrey N, Chen T. Sensitivity to subtle social information in dysphoric college students: evidence for an enhanced theory of mind. Cognit and Emot 2005;19:999–1025.

Haslam N. Categories of social relationship. Cognition 1994;53:59–90.

Ikemoto S, Panksepp J. The role of nucleus accumbens dopamine in motivated behavior: a unifying interpretation with special reference to reward seeking. Brain Res Rev 1999;31:6-41.

Ingram RE, Miranda J, Segal ZV. Cognitive vulnerability to depression. New York, N.Y.: Guilford Press, 1998

Jacobs BL, Fornal CA. Serotonin and motor activity. Curr Opin Neurobiol 1997;7:820–5.

Joiner Jr TE, Coyne JC. The interactional nature of depression: advances in interpersonal approaches. Washington D.C.: American Psychological Association, 1999

Joiner Jr TE, Metalsky GI. Excessive reassurance seeking: delineating a risk factor involved in the development of depressive symptoms. Psychol Sci 2001;12:371–8.

Kahneman D, Tversky A. Prospect theory: an analysis of decision under risk. Econometr 1979;47:263–91.

Katz LD. Dopamine and serotonin: integrating current affective engagement with longer term goals. Behav Brain Sci 1999;22:527.

Keller MB, McCullough JP, Klein DN, Arnow B, Dunner DL, Gelenberg AJ, et al. A comparison of nefazodone, the cognitive behavioral-analysis system of psychotherapy, and their combination for the treatment of chronic depression. N Eng J Med 2000;342:1462–70.

Kelly EL, Conley JJ. Personality and compatibility: a prospective analysis of marital stability and marital satisfaction. J Pers Soc Psychol 1987;52: 27–40.

Kendler K, Hettema J, Butera F, Gardner C, Prescott C. Life event dimensions of loss, humiliation, entrapment, and danger, in the prediction of onsets of major depression and generalised anxiety. Arch Gen Psychiatry 2003;60: 789–96.

Kessler RC, McGonagle KA, Zhao S, Nelson CB, Hughes M, Eshleman S, et al. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: results from the national comorbid survey. Arch Gen Psychiatry 1994;57:14–20.

Klerman GL. Depressive-disorders — further evidence for increased medical morbidity and impairment of social functioning. Arch Gen Psychiatry 1989;46:856–8.

Klinger E. Consequences of commitment to and disengagement from incentives. Psychol Rev 1975;82:1-25.

Klinger E. Loss of interest. In: Costello CG, editor. Symptoms of depression. New York, N.Y.: J. Wiley; 1993. p. 43–62.

Lambert G, Johansson M, Agren H, Friberg P. Reduced brain norepinephrine and dopamine release in treatment refractory depressive illness. Arch Gen Psychiatry 2000;57:787–93.

Lane JD, DePaulo BM. Completing coyne's cycle: dysphorics' ability to detect deception. J Res Pers 1999;33:311–29.

Leahy RL. Depression and resistance: an investment model of decision-making. Behav Ther 1997;20:3–6.

Lee L, Harkness KL, Sabbagh MA, Jacobson JA. Mental state decoding abilities in clinical depression. J Affect Disor 2005;86:247–58.

Lewinsohn PM, Allen NB, Seeley JR, Gotlib IH. First onset versus recurrence of depression: differential processes of psychosocial risk. J Abnorm Psychology 1999;108:483–9.

Lewis AJ. Melancholia: a clinical survey of depressive states. J Ment Sci 1934;80:1-43.

Loewenstein GF, Weber EU, Hsee CK, Welch N. Risk as feelings. Psychol Bull 2001;127:267–86.

Lopes LL. Between hope and fear: the psychology of risk. Adv Exp Soc Psychol 1987;20:255–95.

Marsh KL, Weary G. Severity of depression and sensitivity to social information. J Soc Clin Psychol 1994;13:15–32.

McGuire MT, Troisi A. Prevalence differences in depression among males and females: are there evolutionary explanations? Br J Med Psychol 1998;71:479–91.

McGuire MT, Troisi A, Raleigh MM. Depression in evolutionary context. In: Baron-Cohen S, editor. The maladapted mind. Hove, U.K.: Psychology Press, 1997.

McGuire M, Fawzy F, Spar J, Troisi A. Dysthymic disorder, regulation–dysregulation theory, CNS blood flow, and CNS metabolism. In: Sloman L, Gilbert P, editors. Subordination and defeat: an evolutionary approach to mood disorders and their therapy. Mahwah, NJ: Lawrence Erlbaum Associates; 2000. p. 71–93.

McKenzie J. Neuroticism and academic-achievement — the Furneaux Factor. Pers Individ Differ 1989;10:509–15.

McKenzie J, Taghavi-Khonsary M, Tindell G. Neuroticism and academic achievement: the Furneaux Factor as a measure of academic rigour. Pers Individ Differ 2000;29:3-11.

Mithen S. The prehistory of the mind: the cognitive origins of art, religion and science. New York, N.Y.: Thames and Hudson, 1996

Monroe SM, Steiner SC. Social support and psychopathology: interrelations with pre-existing disorder, stress and personality. J Abnorm Psychology 1986;95:29–39.

Monroe SM, Hadjiyannakis KK. The social environment and depression: focusing on severe life stress. In: Gotlib I, Hammen C, editors. Handbook of depression. New York, N.Y.: Guilford; 2002.

Monroe SM, Rohde P, Seeley JR, Lewinsohn PM. Life events and depression in adolescence: relationship loss as a prospective risk factor for first-onset of major depressive disorder. J Abnorm Psychology 1999;108:606–14.

Neeleman J, Sytema S, Wadsworth M. Propensity to psychiatric and somatic ill-health: evidence from a birth cohort. Psychol Med 2002;32: 793–803.

Nesse RM. Evolutionary explanations of emotions. Hum Nat 1990;1:261–89.

Nesse RM. Emotional disorders in evolutionary perspective. Br J Med Psychol 1998;71:397–415.

Nesse RM. Is depression an adaptation? Arch Gen Psychiatry 2000;57:14–20.

Nesse RM, Williams GC. Why do we get sick? The new science of Darwinian medicine. New York, N.Y.: Times Books; 1994.

Nettle D. Evolutionary origins of depression: a review and reformulation. J Affect Disord 2004;81:91-102.

NIMH. Report of the National Institute of Mental Health's genetic workshop. Biol Psychiatry 1999;45:559–602.

Oatley K. Best laid schemes: the psychology of emotions. New York, N.Y.: Cambridge University Press, 1992.

Öhman A, U.Dimberg U, L.G.Öst LG. Animal and social phobias: biological constraints on learned fear responses. In: Reiss S, Bootzin RR, editors. Theoretical issues in behaviour therapy. New York, N.Y.: Academic Press; 1985.

Pakriev S, Shlik J, Vasar V. Course of depression: findings from a cross-sectional survey in rural Udmurtia. Nord J Psychiatry 2001;55:185–9.

Paykel ES. Which depressions are related to life stress. Acta Neuropsychiatr 2002;14:167–72.

Persad SM, Polivy J. Differences between depressed and nondepressed individuals in the recognition of and response to facial emotional cues. J Abnorm Psychology 1993;102:358–68.

Post RM. Transduction of psychosocial stress into the neurobiology of recurrent affective-disorder. Am J Psychiatry 1992;149:999-1010.

Premack D, Woodruff G. Does the chimpanzee have a theory of mind? Behav Brain Sci 1978;4:515–26.

Price J. The evolutionary model of psychiatric disorder. Arch Gen Psychiatry 1984;41:211.

Price J. Accentuate the positive, eliminate the negative: the role of boosting and putting-down signals in mental health. In: Trent D, editor. Promotion of mental health, vol. 1, 1991. Aldershot, U.K.: Avebury; 1992. p. 89-101.

Price J. The adaptive function of mood change. Br J Med Psychol 1998;71:465–77.

Price J. The dominance hierarchy and the evolution of mental illness. Lancet 1967;7502:243–6.

Price JS. Genetic and phylogenetic aspects of mood variations. Int J Ment Health 1972;1:124–44.

Price JS, Sloman L. Depression as yielding behaviour: an animal model based on Schjelderup–Ebbe's pecking order. Ethol Sociobiol 1987;8:85S–98S.

Price JS, Sloman L, Gardner R, Gilbert P, Rohde P. The social competition hypothesis of depression. Br J Psychiatry 1994;164:309–15.

Reich G. Depression and couples relationship. Psychotherapeut 2003;48:2-14.

Ross SR, Stewart J, Mugge M, Fultz B. The imposter phenomenon, achievement dispositions, and the five factor model. Pers Individ Differ 2001;31:1347–55.

Rudolf KD, Hammen C, Burge D, Lindberg N, Herzberg D, Daley SE. Toward an interpersonal life-stress model of depression: the developmental context of stress generation. Dev Psychopathol 2000;12:215–34.

Seligman MEP. Helplessness: on depression development and death. San Francisco: Freeman and Co., 1975.

Slovic P. The construction of preference. Am Psychol 1995;50:364–71.

Stahl S. Essential psychopharmacology: neuroscientific basis and practical applications. Cambridge, U.K.: Cambridge University Press, 2000.

Stengel E. Suicide and attempted suicide. New York, N.Y.: Penguin, 1974.

Sullivan PF, M.C.Neale MC, K.S.Kendler KS. Genetic epidemiology of major depression: review and meta-analysis. Am J Psychiatry 2003;157:1552–62.

Swallow SR, Kuiper NA. Social-comparison information in dysphoria and nondysphoria: differences in target similarity and specificity. Cogn Ther Res 1993;17:103–22.

Tellegen A, Lykken DT, Bouchard TJ, Wilcox KJ, Rich S. Personality similarity in twins reared apart and together. J Pers Soc Psychol 1988;54:1031–9.

Tomarken AJ, Keener AD. Frontal brain asymmetry and depression: a self-regulatory perspective. Cogn Emot 1998;12:387–420.

Tooby J, Cosmides L. Friendship and the banker's paradox: other pathways to the evolution of adaptations for altruism. Proc Br Acad 1996;88:119–43.

Treatment of Adolescents with Depression Study (TADS) Team. Fluoxetine, cognitive behaviour therapy, and their combination for adolescents with depression. J Am Med Assoc 2004;292:807–20.

Watanabe M. Reward expectancy in primate prefrontal neurons. Nature 1996;382:629–32.

Watkins E, Baracaia S. Rumination and social problem-solving in depression. Behav Res Ther 2002;40:1179–89.

Watson D. Mood and temperament. New York, N.Y.: Guilford Press, 2000.

Watson PJ, Andrews PW. Toward a revised evolutionary adaptation analysis of depression: the social navigation hypothesis. J Affect Disord 2002;72:1-14.

Watson D, Clark LA. Negative affectivity: the disposition to experience aversive emotional state. Psychol Bull 1984;96:465–90.

Weissman MM. Advances in psychiatric epidemiology: rates and risks for depression. Am J Publ Health 1987;77:445–51.

Wiggins JS, Trapnell PD. A dyadic-interactional perspective on the five-factor model. In: Wiggins JS, editor. The five factor model of personality. New York, N.Y.: Guilford Press; 1996. p. 88-162.

Williams KD, Sommers KL. Social ostracism by one's coworkers: does rejection lead to loafing or compensation. Pers Soc Psychol Bull 1997;23:693–706.

Yost JH, Weary G. Depression and the correspondent inference bias: evidence for more effortful cognitive processing. Pers Soc Psychol Bull 1996;22:192–200.