Review

Evolutionary origins of depression: a review and reformulation

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Abstract

There has been a recent surge of interest in the evolutionary basis of depression. One approach argues that the affective mechanisms that are dysregulated in depression are adaptations, whilst a second approach argues that depression itself is an adaptation. The evidence relating to whether depression could itself be an adaptation is reviewed. Adaptations generally have four hallmarks; they lack heritable variation, show evidence of good design, are evoked by appropriate triggers, and fitness is reduced where they are absent. Depression shows none of these hallmarks. It is characterized by heritability, recurrence, cognitive impairment, and poor social outcome. In an alternative evolutionary formulation, I argue that evolution has produced a continuous population distribution of affective reactivity that is subject to stabilizing selection. Individuals vulnerable to depression are at the upper end of this distribution. This conceptualization, in which depression itself is not selected for, is compatible with the known clinical and epidemiological facts.
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1. Introduction

The recent 'adaptive turn' in psychology has led to evolutionary explanations being sought for an increasing number of pervasive aspects of human thought and feeling (Barkow et al., 1987; Buss, 1995). Depression is sufficiently widespread through history that it is quite fitting to ask how the capacity for it evolved, and this has indeed been the topic of a number of investigations. All these investigations share an interest in useful functions of apparently negative emotional states, but broadly speaking, they can be divided into two categories.

The first category, 'dysregulation' models, uses evolutionary ideas to elucidate the nature of the mech-

anisms that are activated in depression, without claiming that depression itself either is now or ever has been adaptive. For example, Nesse (2000) hypothesizes that normal low mood is an adaptation to circumstances where a life goal cannot be achieved and should be disengaged from. Similarly, Gilbert and Allan (1998) explore the idea that the mechanism activated in depression is one designed to cope with threatening circumstances where flight is impossible. This builds on the idea that low mood is an inherited mechanism for accepting social subordination without threatening higher ranking individuals or expending energy on futile status competition (Sloman et al., 2003). Authors in this category hypothesize that clinical depression usually results from some kind of dysregulation, chronic over-activation, or inappropriate evocation of mechanisms (in the mood or affect system) that are adaptations. The dysregulation might occur for a num-

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ber of reasons, including the fact that the environment in which we live is so different from that in which we evolved (Wilson, 1998). The dysregulation literature is strong in its elucidation of the core psychological mechanisms involved in depression, but does not generally address the question of why some people rather than others are liable to become depressed.

The second category of evolutionary approach ('adaptation' models) makes the bolder claim that depression itself is an adaptation. Price et al. (1994) was an influential early statement of such a position. More recently, Hagen (1999) has argued that post-partum depression is a mechanism by which mothers extort greater investment from their social network, especially their partners, by making themselves unable to cope with nurturance of the child without such investment. Watson and Andrews (2002) generalize the adaptationist stance to all cases of depression, not just those which occur post-partum. In what they call the 'social navigation hypothesis' (SNH), Watson and Andrews characterize depression as an evolved response to unpropitious social circumstances.

The purpose of the present paper is 2-fold. First, I review the logic and evidence relating to the idea that depression is an adaptation. The SNH forms the focus of this review, since it is the most clearly stated explicitly adaptationist theory of depression. However, several of the issues raised are also of relevance to the post-partum depression model of Hagen (1999) and other adaptationist approaches. Having presented the SNH and the evidence for it (Section 2), I then consider the characteristic hallmarks of biological adaptations in general, and investigate the extent to which depression fulfills these criteria (Section 3). In the final part of the paper (Section 4), I present an alternative formulation, in which depression is seen not as an adaptation but nonetheless as the outcome of evolutionary processes. The implications of this view, and its place with respect to other models, are considered.

2. An adaptationist model: the social navigation hypothesis

2.1. The hypothesis

The SNH proposes that depression evolved as a context-evoked suite of psychological changes that

enhances fitness (or did so in an ancestral environment) by serving two functions. The first is a *social rumination function*. The depressive is usually faced with a difficult social problem, and depression aids its resolution by switching energy away from normal social and physical activities to focus exclusively on the task in hand. A suite of cognitive changes designed to enhance the solution of life problems is argued to be characteristic of the depressive cognitive style. The second function is *social motivation*. Depression serves to signal the need for more investment from coalitional partners. Indeed, it may be a strategy for extorting such investment, since left to its natural course it entails significant risk of death. Thus, conspecifics with a positive interest in the fitness of the depressive have an incentive to care for her, given that she is failing to care for herself. Depression thus, when it works, provokes increased, fitness-enhancing investment from the depressive's kin and allies.

2.2. Evidence for the SNH

Watson and Andrews provide a plausibility argument for the SNH by noting a number of features of depression that either require adaptive explanation, or make sense only in the light of the SNH.

2.2.1. Features of depression which require adaptive explanation

Watson and Andrews point out two 'good reasons to consider adaptationist hypotheses for depression': 'depression is very prevalent' and 'the capacity for it may be cross-culturally universal' (p. 2). It is certainly true that depression is too common to be accounted for by a single deleterious mutation (Wilson, 1998). However, this does not allow strong inference that depression itself is an adaptation.

Maternal death during childbirth has been found across all cultures and ages, and is a significant health risk in all countries today. The lifetime risk in the least developed countries today is around 6.25%, which is of the same order as the incidence of depression (WHO, 2003). In the ancestral environment, the incidence would have been higher. However, no adaptationist account of maternal mortality is plausible. Rather, it is a non-adaptation that arises in a law-like way from an evolutionary dynamic. There is strong selection to increase brain size. This can be

done by post-natal growth, but this makes the new-born infant very vulnerable, or pre-natal growth. As a result there is selection to increase the pre-natal growth of the baby. However, once the size of the baby, especially the head, passes a certain size, both mother and baby are placed at risk (Graafmans et al., 2002). There is an adaptive peak where the baby is at the maximal size consistent with safe birth. However, due to multiple genetic and environmental variables, there is variation around this adaptive peak, which means that both low birthweight babies and maternal mortality are maintained at significant rates. Neither is in adaptation; they both reduce fitness, but they arise from an evolutionary process operating on a complex landscape.

Watson and Andrews argue that types of pain other than depression have been profitably interpreted from an adaptationist perspective. Physical pain protects the soma. Types of emotional pain such as jealousy (Buss, 2000) draw attention to possible threats to fitness in the environment, so that the individual can take remedial action. Given that these cases can be given a plausible adaptive story, it seems a priori reasonable to extend the mode of explanation to depression.

However, there is a difference between depression and the other types of pain that seem plausibly to be adaptations. All normal human beings have the capacity to feel physical pain (indeed, those rare individuals born without this ability are likely to die from damage to the soma, providing strong evidence for the adaptationist account (Rosemberg et al., 1994)). Similarly, the experience of emotions is universal. However, there is no evidence that all individuals have the capacity to become clinically depressed. Rather, it seems likely that most depression is the result of an inherited diathesis borne by only a minority of the population (see Section 3, below).

2.2.2. Features of depression explained by the SNH

Watson and Andrews point out, following a long line of earlier investigators, that depression tends to be brought on by social problems, most significantly loss, failure to attain an important goal, or conflict in interpersonal relationships. They argue that such situations are precisely those where the depressive needs to come up with a new strategy, and evoke more investment from allies and kin to help her. Thus, both

social rumination and the social motivation of others would be fitness enhancing under such circumstances.

As further evidence that depression subserves a social rumination function, Watson and Andrews cite findings that depressives are better at some kinds of social judgement and problem solving than non-depressives. Thus it would appear that depression is both elicited by the right triggers and designed in the right way to be an adaptation for solving difficult social problems. However, a rather selective review of phenomena is presented, and there are many other research findings about depression that do not fit so easily into this picture. I review these in Section 3.

3. Hallmarks of adaptations and characteristics of depression

To consider whether depression might plausibly be considered an adaptation, a clear definition of an adaptation is needed. Buss et al. (1998) define an adaptation as 'an inherited and reliably developing characteristic that came into existence as a feature of a species through natural selection because it helped to directly or indirectly facilitate reproduction during the period of its evolution'. The key characteristics would thus seem to be that an adaptation (1) arose in an ancestral population; (2) enhanced the fitness of those individuals carrying it relative to those who did not; and (3) thus spread to reach fixation in the gene pool of the relevant species.

Identifying an adaptation may not be straightforward in practice. For example, adaptations need not be present in every individual all the time. Having two eyes is an adaptation that is visible from birth in all intact individuals. Other adaptations may appear only at a later point in life or only when the appropriate environmental triggers are present. Thus, it is possible for an adaptation to be universal in the make-up of the species without the related behavior being manifest in all individuals. This is presumably what Watson and Andrews have in mind for depression, which affects only a minority of people.

However, natural selection fixes adaptations in the species, by gradually increasing their frequency until all individuals in the population carry them. In the process, heritable variation is used up (Fisher, 1930), and thus it is a hallmark of adaptations that there is no

heritable variation left with respect to the presence of the characteristic in question (though there may be residual variation around the adaptive peak for quantitative traits). The only exception would be characteristics maintained at sub-fixation levels by frequency-dependent selection. Watson and Andrews give no indication that they see depression as a frequency-dependent, sub-fixation adaptation. Since they treat depression as a discrete trait, the following seem to be implications of their theory: (1) that the capacity for depression should be characteristic of all intact human beings; and (2) that heritable variation in the capacity for depression, having been used up by selection, should be close to zero.

Adaptations are generally identified using two methods. The first, as Watson and Andrews point out (p. 2), is reverse engineering or functional design analysis. This is the examination of characteristics of the trait at hand to investigate the extent to which they represent good design for the proposed adaptive function. If the trait has features that fit the function to a high degree of specificity and cannot easily be explained in other ways, the argument that the trait evolved under selective pressure to fulfill the function is plausible. A corollary of good design is appropriate evocation; the characteristic should be evoked under appropriate circumstances. For example, gazelles leap into the air in the presence of predators, a behavior known as stotting. The function of stotting is thought to be to signal good condition to the predator to make it choose a different victim. Stotting shows evidence of good design, since only animals in very good condition can do it (Fitzgibbon and Fanshawe, 1988), and appropriate evocation, since it is done when and only when there are predators in the vicinity (Caro, 1986). The second way of identifying adaptations is to show that, in environments significantly resembling the ancestral environment, individuals who by chance or experimental manipulation lack the trait, have reduced fitness. For example, West Africans carrying a rare genetic factor that causes albinism tend to die in the second or third decade from skin cancer, which provides evidence of the adaptive, protective effects of melanin (Aquaron, 1990). It would thus seem to be an important prediction of the SNH that, to the extent that the contemporary environment resembles the ancestral one, then any individuals who lack the capacity to become de-pressed when there are appropriate cues in the environment should suffer reduced fitness.

In the rest of this section, I go through each of the criteria just discussed which are generally held to be hallmarks of adaptations (No heritable variation, Appropriately triggered, Good design, and Reduced fitness when absent), assessing the extent to which depression fulfills the criterion.

3.1. No heritable variation

The most significant risk factor for the development of a first episode of affective disorder is having a family member with an affective disorder. In a recent meta-analysis, the odds ratio for the occurrence of major depression in first degree relatives of depressives compared to general controls is estimated at 2.84 (95% CI 2.31–3.49) (Sullivan et al., 2000). Twin studies suggest a heritability of around 0.37 (95% CI 0.33–0.42) for unipolar depression, with a higher value for bipolar disorder (NIMH, 1999). The heritability in the unipolar case is higher if the family history is severe, early onset or recurrent. The best estimate of the shared environment component of variability from twin studies is 0 (Sullivan et al., 2000).

Life-event studies do suggest that depressive episodes can have a social trigger (see below). However, all such studies agree that although most depressed individuals report the occurrence of a stressful life event before the onset of their episode, most individuals who experience such an event do not become depressed (Kessler, 1997). Genetic factors that vary between individuals affect the likelihood of life stress becoming clinical depression (McGuffin et al., 1991).

It thus seems unlikely that the capacity for depression is fixed in identical form in all members of our species, and triggered only by external events. Instead, there is an inherited diathesis, which makes some people liable to the depressive response to life events, which would trigger only normal mood changes in most of the population. This diathesis may well be a polygenic continuum close to what is measured by the neuroticism personality trait (Watson and Clark, 1988), and thus rather than conceptualizing individuals as carriers or non-carriers, it may be more sensible to think of a spectrum of risk. Nonetheless, there is too much heritable variation in affective

response to straightforwardly describe depression as a discrete adaptation. Very similar comments apply to the post-partum case, since there is evidence for genetic vulnerability (Treloar et al., 1999), and a history of affective disorder (post-partum or otherwise) is an important risk factor (Beck, 2001).

3.2. Evoked by appropriate triggers

If depression has been crafted by natural selection as an adaptive response to unpropitious circumstances, then it should be reliably elicited by those circumstances. There are two sides to this; as an adaptation, it should appear whenever needed (the reliable appearance criterion). On the other hand, since depression is highly costly, it should only appear when the circumstances demand (the appropriate absence criterion).

On the side of reliable appearance, the SNH is prima facie on strong ground, since common experience suggests that depression is brought on by adverse life events. However, the scientific evidence for social causation of clinical depression (as opposed to normal mood fluctuations) is much less strong than commonly thought (Kessler, 1997), due to a number of methodological difficulties. One of these is that since depression tends to be chronic or recurrent at either a clinical or sub-clinical level, it is hard to separate negative life events that are the result of previous clinical or sub-clinical depressive behavior from life events that cause a depressive episode. In the epidemiologic catchment area study, by far the strongest risk factor for depression is having been depressed before (Weissman, 1987). This has an odds ratio of 40 compared to no history of depression, dwarfing all other social and biological causes. Since depression and neuroticism often cause marital and occupational difficulties, as well as leading to further episodes, the direction of causality in life event studies is hard to disentangle. Watson and Andrews cite the ECA as the source of the remarkable statistic that being in an unhappy spousal relationship increase the odds of depression 25-fold (p. 4). However, the review from which Watson and Andrews derive this datum (Weissman, 1987) explicitly states that since the interviews in the study were simultaneous, neither direction of causality can be preferred to the other. Depression also causes marital difficulties (Reich, 2003).

Very similar points can be made about the findings that social support has a protective effect, and the lack of it is a risk factor for depression. In fact, depression impairs social functioning in such a way as to produce a situation of low social support. Thus when an association between social support and depression at a point in time is found, the direction of causality is hard to infer (Monroe and Steiner, 1986). Monroe and Steiner show that when pre-existing disorder is controlled for statistically, the strength of the association between social support and depression is reduced, and in some studies abolished altogether.

Turning now to appropriate absence, the SNH sees depression as a context-evoked discrete defense. Once it has achieved its function, it should disappear completely. However, depression tends to be a recurrent or even chronic disease (Pakriev et al., 2001). Although around 80% of patients respond to treatment, only around 50% of them achieve complete remission, which leaves around 30% or 35% of individuals becoming long-term chronic depressives (Bondolfi, 2002). Amongst those who recover, about 50% relapse within 2 years (Belsher and Costello, 1988). In long-term studies, the rate of continuous freedom from illness is very low; 20% over 20 years (Kiloh et al., 1988), or 11% over 25 years (Brodaty et al., 2001). Even post-partum depression, which is argued by Hagen (1999) to be a special, childbirth specific adaptation, entails an extremely high rate of subsequent non-post-partum psychiatric illness (Robling et al., 2000). These results taken together have been argued by Brodaty et al. (2001) to necessitate a 'paradigm shift' in thinking about depression, such that it is viewed as a chronic illness, with recurrence of the norm. Depression may also be a risk factor for dementia (Kessing and Nilsson, 2003).

Watson and Andrews might argue that the relapse statistics beg the question, since the depression in these studies was generally treated, and one of their principal arguments is that treating depression may interfere with its beneficial, adaptive course. There is some suggestion that relapse rates are lower in community settings than psychiatric institutions (van Weel-Baumgarten et al., 2000), though this might simply reflect less severe illness. However, in treatment trials with a placebo arm, the rate of relapse is invariably even higher where only placebo is given

(Geddes et al., 2003) than when there is active treatment.

Furthermore, as depressive episodes continue, the triggers required to provoke a further bout of illness seem to become smaller (Bondolfi, 2002; Mitchell et al., 2003). Indeed, there is some evidence that over time recurrent episodes may become only weakly related or even completely unrelated to life events (Paykel, 2002; Post, 1992). This is probably due to kindling of the neurobiological mechanisms involved. Given abundant evidence that depression decreases social attractiveness (Coyne, 1976), and neuroticism and depression greatly increase the risk of failing to maintain relationships (Bouchard et al., 1999; Kelly and Conley, 1987; Reich, 2003), then such a tendency to recurrence appears unlikely to be adaptive. It has all the hallmarks of a progressively deteriorating and socially disastrous dysfunction of the central nervous system rather than an adaptation that has achieved its goal.

3.3. Good design

If depression is an adaptation, it should show evidence of good design. That is, its symptoms should be efficient at achieving the functions, which are posited for it. This leads to two predictions; depressives should be good at solving difficult social problems, and they should be effective (at least most of the time) in attaining investment from their social contacts.

In general, cognitive functioning is impaired in depression, with deficits in processing speed, memory, learning and shifting of attention (Austin et al., 1992; Tsourtos et al., 2002). However, as Watson and Andrews are correct to point out, these impairments have usually been demonstrated in non-social domains, and thus do not invalidate the SNH. Instead, under the hypothesis, the depressive is supposed to switch cognitive resources towards ongoing intractable social problems, and away from other cognitive domains. Watson and Andrews provide evidence for this adaptive switch of cognitive resources. They cite two studies showing 'that depressives out-perform non-depressives on difficult tasks that tap social problems' (p. 6), and allude to the depressive realism effect, whereby depressives show more accurate judgment on contingency tasks than the non-depressed.

This literature view seems somewhat partial. First, one of the studies they cite as showing that depressives outperform non-depressives on a task tapping social problems (Lane and DePaulo, 1999) is selectively reported. The authors of that study investigated the detection of deception by dysphorics and specifically state in conclusion that 'the prediction... that dysphorics will be more skilled... at detecting deception and reading true feelings... found no support' (p. 323). Instead, the dysphoric group more often detected phoniness as it squared with their negative schemata for interpersonal interaction, but were no more accurate overall.

The second study cited by Watson and Andrews did indeed show that depressives are, under certain circumstances, less likely to commit the fundamental attribution error than non-depressives (Yost and Weary, 1996). The authors argue that this is due to their willingness to invest greater processing resources in the task. It is an extremely interesting result; however, there is no evidence that as a consequence, depressives are better at solving social problems. The fundamental attribution error may in fact be an effective heuristic for choosing coalition partners (Andrews, 2001).

Watson and Andrews' literature reporting is selective by omission when it comes to social cognition in depression. Depressives are slower and less accurate than controls at reading non-verbal social cues (Cooley and Nowicki, 1989; Feinberg et al., 1986; Persad and Polivy, 1993). They show impaired social skill (Libet and Lewinsohn, 1973). In what may be the most direct tests of the social rumination function of depression yet devised, depressives perform *worse* than controls on tasks designed to tap inter-personal problem solving skills (Gotlib and Asarnow, 1979; Watkins and Baracaia, 2002). The indecisiveness, hostility and poor decisions they make are of major clinical concern. From this evidence, depression does not appear well designed for solving social problems.

As for the depressive realism effect, the very review that Watson and Andrews cite (Ackermann and DeRubeis, 1991) concludes that there are almost as many studies finding that depressives are less realistic as there are finding that they are more realistic. The outcome depends on the task; on tasks where the normal population is unrealistically positive, depressives are closer to realism, but on tasks

where the normal population is reasonably accurate, depressives are unrealistic, usually in a negative direction. This includes the ability to accurately evaluate the self, which one would think would be highly necessary for solving severe personal problems.

The second prediction of the SNH was that depression should be effective, at least part of the time, at eliciting greater investment from social partners. Hagen (2002) has provided data to support this view for the specific post-partum case. No such data exist in the general case. Indeed, depressive symptoms make others more hostile and rejecting (Coyne, 1976), and tend to lead to the loss of social support (Monroe and Steiner, 1986). Depression and neuroticism are predictors of marital failure (Kelly and Conley, 1987; Reich, 2003). For Watson and Andrews' adaptive account to work, there might be a sub-group for whom depression leads to such poor social outcomes, but there would have to be a larger group for whom depression ameliorated their social situations. There seems to be no evidence that such a group exists.

Finally, it is worth noting that the theory of Watson and Andrews has no account of mania or hypomania. This would not appear as a strong objection in the light of the conventional dichotomous division of unipolar and bipolar disorders, with their theory applicable to the former only. However, this dichotomy is increasingly being questioned (Akiskal, 2003). Although the diagnosis of bipolar I, where clinical mania is the dominant feature, accounts for only about 1% of the general population and perhaps 1 in 10 or 15 cases of affective illness, the diagnosis of bipolar II, where depression is the dominant feature but is interspersed with (often sub-clinical) hypomanic periods, may be five times more common (Akiskal, 2003; Angst et al., 2003). Indeed, there may be hypomanic episodes in as many as 50% of all cases of unipolar disorder (Hantouche et al., 1998). Given that a bipolar family history increases the risk of unipolar disorder, it seems likely that these different affective illnesses are on a spectrum. If this view becomes dominant, it presents a serious challenge to any functional design analysis of the depressive state based on low mood only.

3.4. Absence associated with reduced fitness

For some adaptations, it is possible to show that those few individuals who, due to mutation or disrupted ontogeny, lack them have reduced fitness relative to the normal population. This is a useful ancillary string to the adaptationist's bow. A human example is congenital anesthesia, the inability to feel pain, which leads to terrible damage to the body and usually death (Rosemberg et al., 1994). From this we can infer that the ability to feel physical pain is an adaptation.

Depression, at least of clinical severity, it is absent in most people. Moreover, there is widespread evidence that those who suffer depression have impaired psychosocial functioning, excess mortality and poorer physical health than those who do not, even when symptoms are judged to have remitted (Angermeyer et al., 2002; Cuijpers and Smit, 2002; Klerman, 1989). Watson and Andrews would rightly argue that the relevant comparison group would be a cohort of individuals who had had the same life stressors that precipitate depression, but lack the depressive response. However, the stressors that precipitate depression are things that happen to almost everyone at some point in life, and most people who experience them may become sad but do not become clinically ill, so it could reasonably be argued that the entire population is such a comparison group. And compared to the entire population, depressives do badly. The SNH predicts that, since depression is a costly signal, at least some individuals employing it should fare very badly. However, the theory also requires that a sizeable group does very well, to keep the adaptation in the population, and it is that group which is very hard to locate.

3.5. Evaluation

We have thus reviewed the evidence and argumentation for the SNH. Watson and Andrews have made an important contribution in focusing attention on the puzzling question of how depression could have become or remained so prevalent in human experience, and drawing out all the evidence and implications of an adaptationist theory. However, this author judges the idea that depression is itself an adaptation implausible, briefly, because depression is heritable, recurrent or chronic, cognitively impairing, and socially damaging. The insights of the SNH, in as much as they are valid, seem more likely to apply to normal, non-clinical low mood, rather than clinical depression.

In other words, though it purports to be an 'adaptation' theory of depression, it may in fact be more useful as a contribution to the 'dysregulation' literature. Of course, it is possible that some cases of clinical depression represent the proper functioning of adaptive emotional mechanisms (for example, under extreme life circumstances); the SNH attempted to argue that all depressions could be characterized in this way, and it is this generalization that seems implausible. In the next section, I outline an alternative formulation, which attempts to square Watson and Andrews' concern with evolutionary forces with the evidence about depression reviewed thus far.

4. An alternative formulation

Persuasive Darwinian explanations can be given for disease states (Nesse and Williams, 1994). One sub-class of such explanation is the demonstration that the apparent pathology is in fact an adaptation. However, this is not the only possibility, and where it is not supported, there are other avenues the evolutionist can pursue.

Diseases can also arise because a population is adapting to a complex fitness landscape, and/or trading-off several different design features against each other. Consider height, for example. For men, reproductive fitness increases steeply with increasing height, until a certain point, above which the prevalence of musculo-skeletal and other health problems becomes very much greater (Nettle, 2002). In addition, very tall men are large babies, and this causes birth complications and increases perinatal morbidity and mortality (Gage, 2002; Graafmans et al., 2002). Thus, men are trading off the social and mating advantages of tall adult height against the health risks as babies and as adults. A thin adaptive peak must be aimed for between the two sets of disadvantages. Many genes affect final height. The consequence of the evolutionary dynamic is that considerable heritable variation persists in the population relating to height, with the mean height close to the adaptive peak. However, every generation, there is a normal distribution of statures around that peak, and a significant minority of men who have stature-related health problems, whether of over or under height.

Human beings live in highly complex, dynamic social groups to which affect systems are adaptations. Affect systems enable the negotiation of rank and relationship, and divert attention away from things that are damaging to fitness and towards opportunities likely to be fitness enhancing (Price et al., 1994; Sloman et al., 2003; Tooby and Cosmides, 1989). Though the design features of affect systems are adaptations that are common to all human beings, individuals vary in the *reactivity* of their affect control mechanisms. That is, the same interpersonal events cause a larger and longer perturbation of affect in some individuals than others. This variation in lability of negative affect systems is captured by personality dimensions such as neuroticism or negative emotionality (Watson and Clark, 1984).

The neurobiological mechanisms governing the reactivity of the affect system are no doubt complex. They involve a network of interconnected neurotransmitters and associated receptors, enzymes and hormones, including cortisol, norepinephrine, dopamine, serotonin, and monoamine oxidase (Depue, 1995; Lesch, 1998; McCleery and Goodwin, 2001; Roy, 1999). Thus the number of genes in which mutations can cause changes in reactivity is very high, and so neuroticism is underlain by a polygenic continuum. In the population, it has a roughly normal distribution, and retains a fair degree of heritable variation (Tellegen et al., 1988). Where the number of genes involved in a trait (and hence the number of mutational targets) is large, significant genetic variance can be maintained, even when selection is strong (Houle, 1998).

High neuroticism is known to be associated with poor physical and mental health and failure to maintain relationships (Kelly and Conley, 1987; Neeleman et al., 2002). Negative effects of very low neuroticism have been less thoroughly investigated. However, increasing neuroticism is associated with increasing competitiveness (Ross et al., 2001), and neuroticism is a strong predictor of success in attainment (generally studied amongst university students) amongst those who are resilient enough to cope with its negative effects (McKenzie, 1989; McKenzie et al., 2000). Having 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 increased fitness.

Table 1
Summary of the main types of evolutionary model of depression

Type of model	Product of natural selection	Cause of depression	Examples
Adaptationist	Mechanisms that produce depression given appropriate triggers	Proper functioning of mechanisms	Hagen (1999); Price et al. (1994); Watson and Andrews (2002)
Dysregulation	Mechanisms underlying normal (non-clinical) mood variation	Dysregulation of mechanisms	Gilbert and Allan (1998); Nesse (2000); Sloman et al. (2003); Wilson (1998)
Individual differences	Optimal reactivity of affect systems, with a normal population distribution around this optimum	Being in the upper tail of the population leads to vulnerability	Nettle (2001), this paper

Thus it is plausible to argue that increasing neuroticism is selected for, because of its beneficial effects on striving in interpersonal contexts, until the point where the negative effects of mental and physical illness outweigh the marginal benefits. The results, as for height, would be a normal distribution around the adaptive peak, considerable retained heritable variation, and a significant fraction of the population in each generation falling one side or the other of the optimum position. Those on the high neuroticism side would be vulnerable to affective disorders of all kinds. Depression arises because of the tendency of the affect system, in extreme deviations from center, to go into a self-reinforcing cycle, at both the neurobiological and psychological level, which traps it at pathological negativity, and over-rides the usual self-correcting tendencies that emotional mechanisms have over time.

In fact, the adaptive landscape is probably even more complex than the single peak just described. There is a noted excess of affective illness, both unipolar and bipolar, amongst very high achievers in creative domains, entrepreneurship and public life (Andreasen, 1987; Jamison, 1989, 1993; Nettle, 2001; Richards et al., 1988). It seems that the capacity to experience hypomania (though not full clinical mania) can be very beneficial in arenas that lead to high public acclaim, and thus, perhaps, were associated in the past with high reproductive success (Jamison, 1989). Thus at least some individuals with unusually labile moods are extremely successful. However, in many other individuals with this temperament, the negative effects, principally the depression, poor physical health, suicidality and impaired social functioning, outweigh any positive ones. Which predominate probably depends on complex interactions of other inherited capacities and the environment. Nonetheless, the association of high affective vulnerability and leadership is

another reason why variation in mood reactivity may be retained in the population, despite the negative consequences of depression.

5. Conclusions

The main approaches to the evolutionary psychology of depression are set out in Table 1, which gives, for each approach, exactly what is considered to be the outcome of natural selection, and what is thought to be the immediate cause of depression. Note that a number of authors discuss several possibilities, or combine elements of all three approaches (for example, Gardner, 1982; Gilbert, 1992). This paper has argued that strongly adaptationist models are implausible, and, in Section 4, set out an alternative based on the population distribution of individual differences. Dysregulation and individual-differences models are generally compatible with each other, answering the complementary questions of what the mechanisms activated in depression are, and why some people are more vulnerable than others to becoming depressed. Dysregulation and individual difference models are just as evolutionary as adaptationist accounts. However, the evolutionary process is complex, and because of trade-offs and complex adaptive landscapes, it produces individual dysfunctions and by-products as well as adaptations. Depression could be an example of such a dysfunction.

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