Biology and Philosophy (2005) 20:745–766
DOI 10.1007/s10539-004-2279-3

Can evolution explain insanity?

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Received 9 March 2004; accepted in revised form 16 August 2004

Key words: Adaptation, Depression, Evolutionary psychology, Phobia, Psychopathy

Abstract. I distinguish three evolutionary explanations of mental illness: first, breakdowns in evolved computational systems; second, evolved systems performing their evolutionary function in a novel environment; third, evolved personality structures. I concentrate on the second and third explanations, as these are distinctive of an evolutionary psychopathology, with progressively less credulity in the light of the empirical evidence. General morals are drawn for evolutionary psychiatry.

Introduction

Evolutionary explanations of mental illness, like evolutionary explanations of many other parts of human nature, are on the rise. In this paper I taxonomize them, draw some general conclusions, and relate evolutionary psychopathology to more general issues. There are three types of evolutionary explanation of psychopathology. They are not mutually exclusive, since one theory could use all three (e.g. Murphy and Stich 2000).

A *breakdown* explanation sees mental illness as the failure of some component of the mind/brain to fulfill its evolutionary function. Now, the mind has been shaped by natural selection, but accepting that platitude is consistent with many theses about the importance of evolution to psychopathology. It is also consistent with diverse views on psychological organization: mainstream evolutionary psychologists often assert the essential similarity of all human psychology, whereas evolutionary psychiatry tends to stress interpersonal variation (McGuire and Troisi 1998).

A *mismatch* explanation cites a mechanism that was once adaptive but is no longer adaptive because of changes in the environment. This view does not identify a cognitive system within a person as malfunctioning, but locates the pathology in a mismatch between the ancestral environment and our current environment.

Persistence explanations appeal to the theoretical possibility that some putative disorders are adaptive even in the current environment. This is uncontroversial for much of our psychology. Our perceptual systems, working memory and capacities for common-sense psychological explanation, if they are adaptations, are still adaptive. It is much more controversial to claim that

what we think of as mental illnesses include some phenomena that are in fact adaptive in the current environment.

Mismatch and persistence explanations could apply to either a putative disorder or to a behavioral trait that is closely enough linked to a disorder that the presence of the trait in the population inevitably causes the disorder to occur as a side-effect. Call these the *direct* and *indirect* strategies. In the interests of space, I will restrict my remarks to direct strategies.

Mismatch and persistence explanations are often taken to distinguish evolutionary psychiatry from other approaches. However, an evolutionary psychiatry that only employed those two explanations would be in the unfortunate position of arguing that none of our psychopathology involves something going wrong with our minds, and nobody should deny that our evolved nature suffers from a variety of malfunctions and other pathologies.[1] The question, then, is how much human psychopathology comprises mismatches or persistent adaptations. I will argue that these two forms of explanation show little promise (with some possible exceptions among the anxiety disorders) because they are unfaithful to the psychology they try to explain. I discuss a number of places where evolutionary theories of particular mental illnesses make contact with established findings about those illnesses, and I try to indicate points at which the theories can be tested by work that is as yet undone.

I begin with mismatches, starting with the anxiety disorders, notably phobias, which may express once-adaptive predispositions to develop a fear of stimuli that were dangerous in ancestral environments but are no longer dangerous. This is the best current candidate for an evolutionary explanation of mental disorder.

My second example of a mismatch is a group of theories that see depression as an evolved response to loss of status in small social groups like those our ancestors lived in, but not the larger and more fluid groups we live in Price et al. (1994), Nesse and Williams (1995). I will argue that the symptoms of depression just do not fit the theories, although the theories may tell us something about the role of some emotions.

Having looked at mismatch explanations I will move on to persistence explanations. I present a theory of psychopathy as an adaptive game-theoretic strategy. I show that the evolutionary story is rudimentary, and that the psychopathology is speculative.

I conclude with some sketchy remarks suggesting, first, that breakdown explanations should be favored, and hence that others bear the burden of proof, and second, that theories in abnormal psychology exhibit some features that make consilience especially important. An evolutionary perspective may

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[1] I shall throughout use the accepted disease categories of modern psychiatry. This is a controversial thing to do, but I don't have the space to go into detail about psychiatric classification here. For a full length treatment, see Murphy (forthcoming).

be additionally valuable as a source of hypotheses that can be integrated among others.

Anxiety disorders as expressions of adaptive reflexes

The anxiety spectrum

Many diverse conditions fall under the umbrella term 'Anxiety Disorders', including Panic Disorder, Obsessive Compulsive Disorder and Post-Traumatic Stress Disorder. The relevant conditions for our current purposes are Agoraphobia and the specific phobias (although I will introduce panic disorder in a moment). Agoraphobia is not a fear of open spaces but the fear of situations in which escape is difficult or assistance unavailable if one panics or develops incapacitating or embarrassing physical symptoms (Craske and Barlow 2001).

Specific phobias are persistent, excessive or unreasonable fears of a specific object or situation (Antony and Barlow 2002). DSM-IV-TR, psychiatry's current diagnostic 'bible' (American Psychiatric Association 2000, p. 450) lists five subtypes of specific phobia: animal type, natural environment (such as heights, storms or water) type, blood-injection-injury type, situational type (such as airplanes, enclosed places or elevators) and 'other'. The text does not distinguish situational and other types in a theoretically principled way. The examples (p. 445) are not helpful – fear of elevators is a 'situational' subtype but fear of falling down if one moves away from a wall is a subtype of 'other' phobias. The distinction seems to boil down to an intuitive difference between stereotypical phobias (like the fear of heights) and more unusual ones. 'Other' type phobias in fact look like those that are acquired by conditioning to features of the modern world, and not those for which an evolutionary explanation is credible – as we will see, not all specific phobias could be vestigial adaptations.

Phobia and anxiety

Marks and Nesse (1994) argue that the sorts of situations that typically cause phobias are the ones that would have been dangerous in ancestral environments, where a fear of public places or of being far from home might well have been adaptive responses guarding 'against the many dangers encountered outside the home range of any territorial species' (p. 251). Similarly, they argue that a fear of heights accompanied by 'freezing instead of wild flight' (p. 251) would have had obvious adaptive value. In a modern urban environment, however, people who become extremely anxious away from home or in public places will find it all but impossible to lead a normal life. And people who become extremely anxious in high places will find it difficult or impossible to travel in airplanes, ride in glass enclosed elevators or work on higher floors. Thus, because the modern environment is so different from the ancestral

environment, people who are toward the sensitive end of the distribution of phenotypic variation may be incapable of coping with many ordinary situations despite the fact that all of their mental mechanisms are functioning just as natural selection designed them to function.

It is not always clear how we are to read some of the claims Marks and Nesse make. I will develop the argument in what I take to be its strongest form, as a mismatch explanation, but will note alternative interpretations. Then I will look at the evidence.

Marks and Nesse argue that the nervous system 'has been shaped so that anxiety arises in response to potential threats' (1994, p. 254). In effect, we are biologically predisposed to find certain stimuli scary on very little basis: 'minimal cues that reflect ancient dangers' (p. 254). This is an important point, because it aligns the hypothesis with a well-attested body of comparative research, reviewed below, on the speed with which associations can occur.

Evolved fears prepare us for the appropriate response (freezing in some cases, flight in others, for example). As Nesse (1999, p. 262) notes, it is an important source of support for the theory that the form taken by different phobias match their purported evolved function. He argues that almost every aspect of the flight/fright response matches what is needed physiologically to respond to danger, and that the increase in heart rate associated with anxiety is the result of the sympathetic nervous system increasing blood flow to the muscles. For instance, the only phobia associated with fainting is the fear of blood. Marks (1987) argues that fainting is an adaptive response to the sight of one's blood, due to a drop in blood pressure designed to minimize further blood loss. So the argument is that the functional forms of the phobias were once adaptive even if they are often a hindrance in the current environment.

The suggestion that anxiety-producing mechanisms are overactive in phobias can be given two readings. Sometimes the argument seems to be that phobics suffer from broken anxiety-producing mechanisms, with the result that they become unduly anxious. That makes the theory a breakdown explanation. The mismatch interpretation starts from the claim that anxiety, like most traits, shows phenotypic variation. Individuals toward the sensitive end of the distribution, who become anxious more readily, might have functioned quite normally in ancestral environments. But they suffer in contemporary environments where the stimuli are unreliable guides to danger: most of our modern perceptions of ourselves as considerably above the ground come from looking out of windows, not peering over clifftops.

The mismatch reading seems to be what is usually intended. Marks (1987), for example, says that blood-injury phobics 'may be at the extreme end of a normal continuum of cardiovascular responsiveness to tissue-damage stimuli that is genetically influenced.' (p. 46). The second reading is preferable. We should expect phenotypic variation in anxiety, as in any evolved system. Whether or not the system is in fact an adaptation, though, is another story.

I read Marks and Nesse as attributing to phobics evolved mechanisms that produce extreme amounts of anxiety. Their theory, then, might be supported

by more detailed work on what the underlying mechanisms might be. With that in mind, we can look at Clark's influential cognitive theory of panic disorder, which, though not adaptationist in tone, exploits similar reasoning (Clark 1986, 1988, 1997). Although many people experience panic attacks, persistent attacks are rare. Clark suggests this is because only a few people perceive autonomic nervous system activity as indicating impending disaster. Clark contends that panic attacks are triggered and sustained by catastrophic misinterpretations of bodily signals, chiefly those associated with anxiety but including some others. For example, minor palpitations might be perceived as evidence of a heart attack, or the pulsing of blood in the temples as a sign of imminent brain hemorrhage (Clark 1988, p. 149). The misinterpretation causes heightened anxiety that in its turn intensifies the physiological effects, thus increasing the sense that something is badly wrong and inducing further panicking. A feedback loop occurs in which 'the crucial event is a misinterpretation of certain bodily sensations' (Clark and Ehlers 1993, p. 132).

If the susceptibility to panic is a matter of an over-tuned cognitive system, it invites an extension of the idea that excessive anxiety is a matter of oversensitivity to environmental stimuli. And Clark's theory has considerable experimental support (summarized in Clark 1997. See also Ehlers and Breuer 1992). In one study, panic patients and normal controls were asked to read pairs of words. In the crucial condition the pairs consisted of a word denoting a sensation followed by one denoting a horrible outcome (e.g. 'palpitations' plus 'dying'). The combinations were designed to mimic the thoughts attributed to panic sufferers by Clark's model, and based on ratings of anxiety symptoms it appears that during the read-aloud study panic attacks struck 10 out of 12 panic patients, but no controls. Ex-panickers, who had been treated with cognitive therapy to help them understand the causes of their symptoms, also did not panic in that situation. Other experiments have shown that panic patients who inhale a mixture of oxygen and carbon dioxide are much less likely to panic if it is explained to them that the sensations they will undergo are caused by the gas, or if they are duped into thinking they can manipulate the flow of gas (by turning a dial that in fact has no effect). The alternative explanation and the illusion of control each provide a cognitive asset that overrides the natural process of misinterpretation, providing alternative reasons for the sensations. This reduces the chance of an attack.

Both phobias and panic attacks, then, might be extreme manifestations of underlying systems that evolved to respond to dangerous situations, and not as expression of systemic malfunction. Let me now try to integrate this idea with other well-attested aspects of our psychology.

Lurking fears

Even if we have this tendency to get afraid, why suppose that it cannot simply be explained in terms of conditioning? Watson (1924) argued that classical

conditioning could transform any stimulus into one that elicited fear, but (Valentine 1930) rebutted him with studies suggesting that conditioning a fear response in human infants was very much easier with some stimuli than with others. Valentine called the quickly conditioned fears 'lurking fears'. The distinction between conditioned fear responses and lurking fears related to specific evolutionary dangers is now widely accepted on the basis of many studies (Gray 1987). Pictures of stimuli like snakes and spiders that are assumed to make a adaptive sense elicit a much greater galvanic skin response when paired with electric shock than do pictures of unthreatening stimuli like flowers, but they also cause a greater response than pictures of stimuli like guns that are dangerous in the contemporary environment but did not exist in the ancestral environment (Ohman 1979).

Many species exhibit lurking fears. Garcia and Koelling (1966) showed how easy it was to condition rats to associate nausea with foodstuffs, and pain with electric shocks, but not vice-versa. Mineka et al. (1980, 1984, 1989) showed that young rhesus monkeys develop an enduring fear of snakes after seeing film of a conspecific reacting fearfully to a snake. But juvenile monkeys did not develop fear of flowers when the film was edited to show another rhesus reacting fearfully to flowers. If the juveniles saw adults show fear in the face of flowers, they were unmoved, and did not quickly acquire a fearful reaction of their own. Mineka suggested that the difference lies in the evolutionary heritage of the response; it makes sense to be adapted to fear snakes if you're a monkey, but not to be afraid of flowers. Her explanation was that, like Garcia's rats, monkeys are quick to form associations that make evolutionary sense.

A consensus has built up on two points: first that many different lineages, including our own, display this readiness to respond to some stimuli rather than others, and second that the stimuli are ones which are or would have been adaptive to avoid. Humans seem to share this general capacity; for example, it is not at all uncommon for us to avoid certain substances after they have made us ill just once, as anyone who drank too much Southern Comfort as a teenager can testify. Marks and Nesse rely on the assumption that humans share this general animal tendency, and add the claim that it is manifested in phobias, which are typically acquired in response to situations or objects that would have been dangerous in ancestral environments, rather than situations or objects which are currently dangerous, or random stimuli.

Other etiologies

Although the distinction between conditioned fear responses and lurking fears is widely accepted, there are dissenters who argue that phobias 'can develop toward almost any object or situation', like the 'tennis player who wore gloves because he was afraid of fuzz, and tennis balls are fuzzy' (Goodwin and Guze 1996, p. 161). However, everyone, including Goodwin and Guze, notes that much the commonest phobias are those cued by animals, heights (which is

really a fear of falling) crowds, and so on. Moreover, the view of phobias as expressions of evolutionary hangovers is not obliged to predict that no phobias would develop towards other stimuli. The claim that some associations are pre-programmed by evolution does not imply that no other associations can form. Given the right conditioning it may indeed be that anything can become a source of phobias. The evolutionary hypothesis need only argue that the common, readily acquired fears and phobias can be explained in terms of phenotypic variation in prepared responses to features of the ancestral environment. It may also be the case that malfunctions in anxiety-producing mechanisms can produce some phobic responses: any system can malfunction, and a malfunction in a system underpinning prepared fears might result in phobic reaction to atypical stimuli.

Another, more speculative, explanation for the occurrence of phobias directed at non-prepared stimuli is the possibility that a prepared system could be triggered by an input it was not designed for. Sperber (1994) distinguishes a module's actual domain and its proper domain. The proper domain of a module is the information which the module has the biological function of processing. Its actual domain, on the other hand, is all the information in the current environment, which, having been processed by other (input) modules, satisfies the input conditions of the module. This means that features of the modern, rather than ancestral, environment may nonetheless trigger systems designed to produce anxious responses. In Sperber's example, an 'org' has a module with the function of enabling flight in the face of elephants, but oncoming trains also trigger the module. The trains are not part of the proper domain of the module but they are part of its actual domain.

So the evolutionary picture of phobia can cope with alternative etiologies. I now move on to the question whether the observed nature of phobias, rather than their underlying mechanisms, can be squared with the evolutionary scenario. Things here are more complicated than Marks and Nesse think.

How scary should a spider be?

The evolutionary theory offers an explanation for the broad pattern of phobia; the question is whether it can deal with the details. Why, for instance, are specific phobias so much more common among women? Fear of spiders is a common phobias, but it is unclear why spiders should have been such a danger in the ancestral environment, unless our ancestors lived a long time around numerous deadly spiders (and similar creatures), and this might be something we can find out. But our ancestors also lived a long time around some very dangerous large predators, and phobia (as opposed to plain old fear) of big predators seems uncommon. Not enough work has been done to reassure us that the commonest types of phobia have exactly the right objects to fit the theory: a fear of animals and insects is suggestive, but it would be helpful to have more information about exactly which animals and insects. This is

different from the point about form; animal phobias do seem to have the form that one would expect of adaptations for fear of dangerous organisms. The present question is whether the organisms that these functional elements react to are, in fact, the ones that endangered our ancestors.

In sum, there is good but not conclusive support for the idea that at least some of the most common phobias, and some other anxiety disorders, are (1) expressions of oversensitive prepared evolved responses towards stimuli that (2) it was once adaptive to fear. However, Marks and Nesse also seem to believe that (3) fear of these stimuli is no longer adaptive in the current environment, which makes them exponents of a mismatch explanation.

This third point might be correct for most contemporary city dwellers, but there may still be human populations for whom fear responses that are two or three standard deviations above the mean might be useful. In that case, however, we might be disinclined to regard the anxiety response as a disorder at all: Marks and Nesse are attuned to the DSM-IV-TR understanding of a disorder as necessarily interfering with normal life. In that context, their view of phobias as mismatches makes sense, as it is aimed at people whose capacity for normal functioning is hampered. It is the distress and inconvenience of phobias that makes them disorders. And the distress and inconvenience, on this view, is a product of a mismatch between evolutionary and contemporary environments. In snake-infested contemporary environments, however, a fear of snakes may not be a handicap.

Conclusion

There are numerous causal routes to the phobias. Evolution may be one common one. A similar evolutionary story seems plausible for panic disorder, but the overall view still faces some challenges. Depending on how one reads some central ideas of the theory, it can be read as a breakdown-type explanation (if anxiety-producing mechanisms are presumed to be dysfunctional); as a mismatch explanation (if we assume phenotypic variation rather than dysfunction but lack of adaptiveness in the modern environment); or as a persistence explanation (if we assume, as I did at the end, continued utility in some environments).

With this extended example in hand, I will move more briefly through my other examples. I begin with theories that attempt to explain depression as a mismatch. Then I will discuss persistence theories of psychopathy. Then I will sum up.

Adaptive theories of depression

Introduction

The most plausible reading of the adaptive construal of anxiety disorders sees phobics as owners of ultra-sensitive or overactive threat-detection and

anxiety-production systems. In a modern society, says the theory, these are a hindrance rather than a help, leading to a selection/environment mismatch. I turn now to some recent work on depression that offers a much more speculative mismatch explanation and, I argue, fails to capture the psychological phenomena adequately.

Mismatch theories of depression view it as an adaptative response to the problems of living in a small, status-obsessed social, group. One version assumes that in the small bands in which we evolved it was adaptive, having lost status, to accept lowered rank (Price et al. 1994, Stevens and Price 1996). Another story Nesse and Williafms (1995) is that upon being outcompeted one should change one's previous behaviors and look for a new way to shine and attract mates, or at least ruminate upon one's problems (Watson and Andrews 2002). The theories concur that depressed mood provides us with an introspectable marker indicating that self-evaluation in the light of social failure is in order.

To evaluate these theories of major depression[2] I begin with the comparative neurological evidence that the theory often stresses, which relates amine levels to social rank. Then I will discuss whether the theory extracted from those findings can account for the symptomatology of major depression.

Get thee behind me, crustacean: pharmacology and dominance hierarchies

Many animals change their behavior depending on their place in a dominance hierarchy. The shift is accompanied by changes in serotonin levels. This pattern occurs in lobsters (Kravitz 2000) and crayfish (Drummond et al. 2002) as well as in animals closely related to humans. Vervet monkeys show a connection between serotonin, aggression and rank. Lowered serotonin is seen in monkeys of low rank or who have recently lost status. Higher ranking Vervets show increased serotonin levels, correlated with the amount of submission they receive (McGuire and Troisi 1998, pp. 172–174). Manipulation of serotonin can affect behavior, and, indirectly, rank. Injecting a subordinate crayfish with serotonin, for example, causes it to become more aggressive (Drummond et al. 2002).

Social competition theories of depression exploit these links between serotonin, rank and depression. Various measures suggest that serotonin, at least at presynaptic sites, is abnormally low in at least a large subpopulation of depressives, although attempts to correlate serotonin levels with established clinical subtypes have been unsuccessful (Thase and Howland 1995). But it

[2]Note the restriction to major depression. More local theories try to explain particular kinds of depression in evolutionary terms. The best worked out is the defection hypothesis of postpartum depression (Hagen 1999), which maintains that in the ancestral women limited their investment in the new child when, because of social, biological or environmental factors, a major investment in a particular infant would likely reduce their total number of offspring

does seem that among suicidal and gravely depressed patients there is a low level of serotonin release, binding and downstream processing, correlated with excessively negative self-assessment and extreme pessimism (Meyer et al. 2003).

The idea behind the social models of the function of depression is that depressed mood is nature's way of telling you to accept that your current behavior will not improve your reproductive lot and motivating you to try behaving differently.[3] If you are living in a small group switching strategies might result in greater social success. The suggestion is that in ancestral environments, communities were small enough to leave some niches unfilled in which one might prosper. Nesse and Williams (1995) think this is now a mismatch: depression is no longer adaptive, since the social setting has changed in modern society, making an affect-lowering response to change in status profoundly dysfunctional. Other theorists seem to think that in so far as depression fixes one's mind on social problems, it might still be adaptive (Watson and Andrews 2002). That is a persistence version.

The mismatch theory of depression is that we have inherited a mechanism that's triggered when we believe ourselves to be outcompeted. The mechanism will fire frequently in the modern world, as we are inundated with information about people who are better than we are. But, of course, in the modern world it is far more likely that the mechanism will fail to achieve the goal it was selected to attain. If the mechanism is set off by the realization that one is not even close to being the best at anything in the global village of the information age then getting depressed is not likely to be an effective reaction. For it is typically the case that there is no other strategy to adopt – no other niche one could fill – in which one would do much better in that global competition.

If this is right, why isn't everyone depressed? Nesse (1999) notes that depression is on the rise worldwide and is in fact third leading cause of disability-adjusted lost years, according to the World Health Organization. However, given the large groups in which we now live it is unclear why there isn't even more of it if humans compare themselves to everyone rather than just their immediate social group. Whether depressives do compare themselves to global rather than local populations ought to be testable.

A more general objection, on either a persistence or a mismatch reading, is that the evolutionary theories of depression suggest that it should cause a re-evaluation of one's life and a change in one's behavior. But although depressives do seem to review their own behavior more than non-depressives (Pyszczynski and Greenberg 1987), and have a larger repertoire of negative than positive ways of evaluating themselves (Woolfolk et al. 1995) it is unclear

[3]Nesse (2000, p. 17) has more recently argued that depressive cognition might have the function of making one stay in a bad situation, rather than looking for a new one (that might be worse). He presents no evidence, and no reason to think that determination and optimism wouldn't be better aids to seeing things through. More generally, Nesse suggests in this paper that depression is an adaptation for coping with unpropitious circumstances. This is very vague, but (as I note later on) depressive coping styles do not seem to be effective. Furthermore, most people who are in adverse circumstances do not become clinically depressed (as opposed to just ordinarily miserable).

whether their self-evaluation does what the alleged social function of depression requires of it. The social function demands that brooding is good for you, or at least might have been good for you once, but this needs to be shown. For example, there are no studies suggesting that people who have suffered a depressive episode subsequently re-arrange their lives. Furthermore, the non-affective symptoms of depression seem hard to explain on the social function view. In the next section, I will discuss the psychology of depression, and then more briefly discuss the other symptoms.

Depression and motivation

If the function of depressed cognition is to get one to reconsider one's options and try something new, then it seems that the best combination would be negative (or at least objective) self-evaluation coupled with positive affect, or at least some effect on motivational structures that causes the changes in behavior that the theory predicts. This effect on behavior *is what depression is for*, according to the theory. We do not find it.

If we examine the psychology of depressives, we do not find features that take them from introspection to self-reforming behaviors. Depressives do not take up new activities. One frequent symptom is an inability to think, make a decision or concentrate (DSM IV-TR, p. 350), which also fits poorly with the idea of depression as a source of cognitive regeneration. A number of studies have also found that depressives engage in wishful thinking, avoidance and escapism, and do not cope with their difficulties by trying to solve problems (Cronkite and Moos 1995, p. 579–580)). Indeed, depressives do not use cognitive self-control as a coping strategy, whereas the use of escapism as a coping strategy is associated with current and future depression (Rohde et al. 1990). Watson and Andrews (2002) argue that the hopelessness that characterizes depressive cognition is really desperation, indicating that an important problem needs to be solved. They argue that if depression is an adaptation designed to force one to reflect on social problems, it should 'abate when a problem is perceived to be truly insoluble (p. 5)', since there is no longer any function for it. But this prediction is false. Depression does not abate under these circumstances, which is a main reason for the increased risk of suicide which depression carries. Rumination on social shortcomings is indeed a hallmark of depression, but without the motivational effects or cognitive properties that the social function theory requires.

If, as on another version of the theory, depression is an adaptation to make lowered rank palatable, it is unclear why brooding over one's failures is necessary – the actual symptoms of depression do not look like they have been designed to reconcile us to loss of status: depressives aren't *content* with lower status. It is hard to know what a clear prediction might be, based on this view. However, you might expect that either that (1) people might suffer depression, or a distinct form of depression, following a loss in status, or that (2) once

depressed, they would be reconciled to their lot (since, on this variant of the theory, such reconciliation is what depression is for). Neither of these predictions appears to be confirmed.

The stressors that might cause reactive depression are not obviously status-related, since they include just about anything that one might find stressful. And it is well known that people who have been once depressed are at a greater risk of future depression than the general population. Keller et al. (1992) found that 70% of patients who have had a major depressive episode will have at least one subsequent episode, and about 20% of patients with affective disorders develop a chronic condition. We have to assume then, to save the theory, that people who have lost status once are more likely to do so again: if depression is an adaptation designed to make them function better in society, it isn't working.

Other problems

There are many symptoms of depression left unexplained by the social function view: rapid alterations in weight, physical pain, sexual dysfunction, changes in sleeping patterns, psychomotor agitation or retardation and the greatly increased risk of suicide (DSM-IV-TR, pp. 349–352). Nesse (2000) for example, only discusses mood when asking whether depression is sometimes adaptive. There is some evidence (Cheung et al 2004) that loss of social rank, degree of rumination and amount of shame are correlated, and shame may have a social function and some causal connection to depression. But this is really a finding about emotions, rather than depression. The connection between depression and negative self-evaluative emotions like shame and guilt is apparent, but poorly understood. And there is more to depression than lowered mood or feelings of shame. The other symptoms are central to our clinical understanding of depression, and the evolutionary theorists have neglected them.[4]

The social function theories fail the test that, as Nesse stresses, the phobias seem to pass, which is the matching of putative evolved function with the actual form of the psychological phenomena. In effect, the theory takes one aspect of depression – its pharmacology, and perhaps some cognitive properties – and relies on comparative evidence to find an adaptive role for that one aspect without explaining the other features of depression. This is a recurrent problem with evolutionary and sociobiological approaches: explaining only a caricature of a trait, or just a few properties that may seem to have an evolutionary rationale. Some of the data that the social competition theory relies on is actually quite rigorous, unlike the many sociobiological accounts that have relied on impressionistic studies. The problem is the social competition theory is attached rigorously to only one of the phenomena it needs to explain.

[4] Watson and Andrews (2002) are the exception: they argue that the depressive syndrome as a whole has a signaling function, designed to extract care from others (either honestly or as a form of extortion).

In light of these problems, we should question the motivation for approaching depression in the light of evolution. We know that many mental disorders are quite highly heritable, and Nesse (1999) thinks that low mood is so common that it must somehow increase reproductive success, which suggests that the burden of proof is on the side of the anti-evolutionary theorist. But it is equally possible that human beings are just fragile and prone to breakdown, and that brain development in particular offers numerous opportunities for things to go wrong. We have reason to believe that many disorders are the result of psychological malfunction, so a theory that looks for an explanation in non-malfunctioning terms is surely the one that bears the burden of proof.

It may seem that the point of using a broadly adaptationist take on cognitive science to explain psychopathology is an attempt to argue that mental disorders are adaptations. As we saw, that's much too simple. An evolutionary psychology can make use of explanations in terms of psychological breakdowns just as readily as any other orientation on the problem. But the idea that some psychopathologies are or were adaptations is certainly a position in logical space, and mismatch theories are one way to fill it. As well as traits that are selection/environments mismatches, though, we might also ask about adaptations to features of the ancestral environment that are still adaptive today; that is, persistence as well as mismatch explanations.

Persistence explanations

Anti-social personality disorder

The persistence hypothesis says that putative psychopathologies are adaptive in the current environment, just as they were in the ancestral environment. McGuire and Troisi (1998) suggest that antisocial personality disorder and histrionic personality disorder may represent adaptive behavioral strategies. I'll discuss antisocial personality disorder, which is characterized by disregard for the wishes, rights or feelings of others. Subjects are impulsive, aggressive and neglectful of responsibilities. 'They are frequently deceitful and manipulative in order to gain personal profit or pleasure (e.g. to obtain money, sex or power)'. They are indifference to the harms they cause and 'believe that everyone is out to 'help number one' ' (DSM IV-TR, pp. 702–703).

To evaluate this claim we need to distinguish the relevant population. Unfortunately, the terminology is terribly confused. Mealey (1995), who also offers a sociobiological account of antisocial personality disorder, calls it 'sociopathy'. Mealey regards 'primary sociopaths', who lack prosocial emotions like empathy, as designed by evolution to cheat in social exchange situations. Secondary sociopaths exhibit antisocial behavior but do display the prosocial emotions.

Mealey's primary sociopaths are more or less equivalent to 'psychopaths'. Although there is no DSM diagnosis of psychopathy, many researchers use the

term to refer to a distinct condition. Such researchers regard the diagnosis of antisocial personality as too inclusive, because it is purely behavioral or otherwise flawed (e.g. (Cleckley 1988; Hare 1970, 1999; Lykken 1995)). Many career criminals are antisocial but not psychopathic. Psychopaths are behaviorally antisocial, but in virtue of a distinct pathology, whereas antisocial personalities are normal but nasty.

I use 'psychopath' to refer to the population that Lykken, Hare and Cleckley examine, and also the population of evolved defectors posited by McGuire, Troisi and Mealey (see also Harpending and Sobus 1987). I assume the persistence hypothesis tries to explain only genuine psychopathy and not mere antisocial behavior. Psychopathy is diagnosed using Hare's Revised Psychopathy Checklist (see Table 1) which was originally based on a development of Cleckley's sixteen characteristics of the psychopath (Hare 1999) The checklist comprises twenty items.

The explanation

An influential view construes the function of a psychological unit as the effect it has in virtue of which it is copied in successive generations (Godfrey-Smith 1994; Millikan 1989; Neander 1991). If it is indeed true that some personality disorders are adaptive strategies, we can give precisely this causal-historical explanation of the mechanisms that subserve psychopathy. The antisocial behaviors are produced as the proper, selected functions of their peculiar

Table 1. The Psychopathy Checklist.

1. Glibness/superficial charm
2. Grandiose sense of self-worth
3. Pathological Lying
4. Conning/Manipulative
5. Lack of guilt or remorse
6. Shallow affect
7. Lack of empathy, callous
8. Failure to accept responsibility
9. Need for stimulation/proneness to boredom
10. Parasitic lifestyle
11. Poor behavioral controls
12. Early behavior problems
13. Lack of realistic, long-term goals
14. Impulsivity
15. Irresponsibility
16. Juvenile Delinquency
17. Revocation of Conditional Release
18. Sexual Promiscuity
19. Many short-term marital relationships
20. Criminal Versatility

psychological mechanisms. So psychopaths do not, on this view, have malfunctioning mental mechanisms. Nor is there reason to believe that the environment has changed in relevant ways since the time when the system was selected. The relevant environment in this case is social, and the current social environment, like the ancestral one, offers many opportunities to cheat and exploit one's fellows. The persistence hypothesis claims that psychopaths are adapted to exploit others via their sensitivity to, but moral disregard for, the system of social exchange; they can live by the norms if they need to, but are astute enough to know when to defect.

For this to be plausible it must turn out that, other things being equal, psychopathy is an effective way getting one's genes into the next generation. If it is, then a population with a percentage of psychopaths may be in an evolutionary stable state. But the idea that a population with a minority of psychopaths is in equilibrium has not really been developed with any precision. There are both mathematical and psychological obstacles to overcome. First, since we are ignorant of the original state of the system (we don't know how many psychopaths were around in the stone age) it needs to be shown that the system has the right properties to make the equilibrium likely to emerge from a wide set of initial states (D'Arms et al. 1998; Skyrms 2000). Of course manipulative and other antisocial behavior is widespread, given how nasty people are to each other, and the existence of defection, distributed throughout the whole population, is consistent with game-theoretic models in population genetics. However, explaining the evolution of defection is not the same as explaining the existence of a distinct population of defectors.

The psychological obstacles are also formidable. Let me now turn to two properties of the psychology of psychopaths that are *prima facie* difficulties for the view that they are successful defectors: violence and irrationality.

Violence

Psychopaths are more violent than non-psychopaths, but their violence is also of a different kind. It's both more dispassionate and businesslike than 'normal violence' and more impulsive and sadistic. It is also much more likely to be directed against strangers (Hare 1999, pp. 88–93). (The fact that psychopaths are impulsive and dispassionate at the same time says a lot about them.) The game-theoretic view must then assume either 1) grossly abnormal levels of callous violence make for a successful social strategy or 2) there is a population of Machiavellian 'successful psychopaths', who lack empathy, are socially deceptive, but do not rely on violence to get their own way. The first option looks unattractive – the point about successful social deception is that it is hard to detect. It is easy to detect when someone has been beaten up, especially in the small, isolated bands in which we are presumed to have evolved. Moving

freely among human groups to escape the consequences of one's acts is a lot easier now than it would have been in the ancestral environment.

The population we need, then, is the white-collar criminal one, not the violent criminal one. This view, as we shall see, has some (uncertain) support, but it has to surmount another obstacle. Despite the tendency among philosophers to portray psychopaths as rational amoralists (Nichols 2002), psychopaths have cognitive deficits.

Irrationality

Although they are at least as intelligent as nice people, psychopaths are impulsive, irresponsible and poor at long-term planning. Experimental work on psychopaths suggests they 'have the capacity for genuine judgment and sound affect but that a cognitive deficiency interferes with their ability to integrate the products of these faculties with ongoing behavior' (Newman 1998, p. 83). Hare (1999) argued that information-processing problems in frontal systems may cause these cognitive shortcomings. The cognitive deficits cause trouble for the persistence hypothesis, because the cognitive mechanisms underlying what is putatively an adaptive strategy are ones that appear to be far from functional.

The persistence theorist of psychopathy could argue that these apparent shortcomings are in fact adaptive cognitive styles. Alternatively, the persistence theorist might hypothesize a population with all the amorality and skill at manipulating others which psychopaths possess, but without their cognitive deficits. What is needed if the persistence explanation is to apply to psychopaths is a population with the affective profile and desire to prey on the rest of us that psychopaths normally exhibit, but with better instrumental rationality. Such successful psychopaths must be able to inhibit some of the impulsiveness characteristic of their kind. They could have benefited, for example, from a middle-class upbringing that has stressed the gains to be had from delayed gratification (Lykken 1995). The existence of such a population remains speculative, but widely credited. Factor analysis of the psychopathy checklist reveals that the cognitive and affective dimensions of the syndrome do come apart. Items 1–8 and items 9–17 can be distinguished using factor analysis (items 18–20 load about equally on both factors). This raises the prospect of affectively abnormal but cognitively normal subjects.

There is impressive agreement among psychopathy researchers that successful psychopaths exist: (Hare 1999) thinks they are a genuine public menace. But all we have are hunches and anecdotes, and it is hard to reach such people for experimental purposes. ('Dear CEO, your ex-wife and former employees have raised interesting questions about your psychological makeup. Would you, in the interests of science, be prepared to undergo a Hare Countdown test?')

But the possibility that a middle-class upbringing could produce a successful rational psychopath does not just support an evolutionary game-theoretic explanation. It is consistent with an explanation like (Lykken's 1995) 'low fear' hypothesis, which has considerable experimental support. Lykken thinks that psychopaths are fearless and hence hard to socialize via punishment, since they are not afraid of punishment. He does, though, think that rational psychopaths could work out for themselves that they had better obey the rules if they want to get ahead, or that some of their cognitive deficits can be compensated for in other ways.

In conclusion: even if we ignore the mathematical difficulties involved in getting the formal dynamics of the explanation straight, the psychological difficulties it faces means that the game-theoretic view of psychopathy is no better off than the view that psychopathy is a neuropsychological disorder. In fact, the more the theory finesses these psychological obstacles, the more it turns into a theory about rational amoralists rather than actual psychopaths, and the closer it gets to being merely a speculative theory that evolution could produce Hume's 'Sensible Knave'.[5]

The morals

The theories I have reviewed tend to fit only some symptoms of a disorder – psychopaths are too impulsive and violent to be successful at deception and depressed people seem too unmotivated to be plotting a new strategy, or too miserable to be reconciled with their lot. The failure is one of psychological form not matching alleged biological function. In the case of some types of anxiety, though, the match between form and function is at least in prospect.

The adaptive theory of phobia is actually one of the best adaptationist theories of any aspect of our psychology. There are two main reasons for this: functional plausibility, as I have noted, and also consilience. Because the phobias seem to have forms that fit their alleged functions, the theory stands some chance of explaining the whole symptomatology, rather than just one or two favorable symptoms. Second, the theory can be integrated with various other inquiries that seem to mutually buttress each other. Let me conclude by looking at these two issues of functional plausibility and consilience. I will try to convey what may be their general significance for evolutionary psychology more generally.

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[5]The 'sensible knave, in particular incidents, may think that an act of iniquity or infidelity will make a considerable addition to his fortune, without causing any considerable breach in the social union and confederacy. That *honesty is the best policy*, may be a good general rule, but is liable to many exceptions; and he, it may perhaps be thought, conducts himself with most wisdom, who observes the general rule, and takes advantage of all the exceptions.' (Hume 1777/1975, pp. 282–283).

First, function must match form. Arguments about evolutionary scenarios in psychology often focus on biological plausibility, but the psychological predictions the scenario makes are just as important and usually easier to test, so why not start by evaluating them? This is a general point about evolutionary psychology, indeed about psychology. Evolutionary scenarios ought to make predictions about psychology and, at a minimum, be consistent with the known facts of the relevant psychology. Theories that fail these tests can be dispensed with. Before we wonder how some bit of our psychology could have evolved, we should double-check to see whether it's actually there. If form is to fit function, all the symptoms of a disorder (or all the manifestations of an aspect of our non-pathological psychology) must be explained. This is a very demanding standard for any theory to meet.

One of the besetting sins of evolutionary psychology is the tendency to take some crude characterization of a human capacity and try to explain that. This is encouraged by the search for analogies with animal models. Animal models may be very well worked out, like the model of serotonin-mediated aggression in crustacea. The problem is that an animal model will typically correspond, at most, to some part of a human trait, and the use of comparative evidence encourages the selective promotion of some aspects of human psychology at the expense of others. Comparative evidence is helpful, but it can often only speak to broad psychological similarities between humans and other species, given our lack of close relatives. The exception is the study of highly conserved, relatively circumscribed mechanisms, such as lurking fears. But when we study the central psychological systems that underline human affective and rational life, animal models are likely to be little more than suggestive, because the relevant high-level capacities, such as those impaired in depression or psychosis, are just missing in other species. Failure to appreciate this, I conjecture, is what has led some evolutionary theorists of depression to develop a theory that fits the comparative pharmacological data but is wholly unsuitable as an explanation of depression overall.

Consilience is important as a pointer to how evolution might help us to understand mental illness. It seems that facts about the evolution of the mind ought to be of some use in understanding its frailties, yet our evolutionary theories of mental illness seem very tenuous. A confirmation of several independent predictions should increase our confidence that we have a genuine process that different theories are tracking. This is why the case for the adaptive story of phobia looks much stronger if, as seems possible, it can be integrated with comparative, cognitive and neurological evidence.

More generally, evolutionary considerations might help deal with a general feature of theories in abnormal psychology; they are typically, in Lykken's (1995, p. 148) term, 'low-powered'. Usually, they do not single out some categorical difference, such as a lesion or the presence of a particular behavior, such that all and only subjects who meet a diagnosis are expected to exhibit that difference. Usually, we predict that some naturally continuous quantity

will be higher among subjects than among controls or that one variable should be positively correlated with another. But such statistical effects can seem frighteningly cheap; a prediction that manic depressives are taller than average probably stands a 50% of being true just because any group of people we single out and measure carefully is likely to be taller or shorter than average. And even when we have a reliable result, we are often unclear why it obtains: it is easy to see why a biopsy score or an MRI image is a good predictor of the health of a prostate gland or the chance of recovery from knee injury. It is hard to see why an interest in mechanics magazines should be a good predictor of paranoia (Dawes 1994).

So not only are psychological predictions low-powered, we often don't understand why their predictions come true. A neglect of evolutionary thinking in psychology may have been part of the reason for this. Our understanding of what the component parts of our psychology do is still rudimentary, and taking seriously the fact that the mind evolved may be an important source of knowledge. If we find out what the mind's functions are, we may have a better grip on how it can fail to perform those functions, and taking evolution more seriously may help us figure out what our minds are for.

So the significance of evolution for psychopathology is likely to be at one remove, as a source of extra lines of inquiry and constraints on theory choice, and to occur in the context of the neuropsychological basis of the variables distinguishing patients from controls. That is, breakdown explanations may be the likeliest form for psychiatric theories to take, but understanding the breakdowns might require a grasp of our normal psychology that evolution can help to furnish. The evolutionary theories of depression I have looked at are not, I think, going to succeed as theories of depression. But they have raised interesting issues about the social role and significance of emotions like shame that might not have been raised without an evolutionary perspective on human social conflict (Gilbert 2003; Nesse 1998). Understanding the social emotions should tell us more about the nature of the systems involved in depression, even if adaptive theories of depression cannot be defended. Psychiatry must try to understand the mind that evolution, among other causes, left us with. We are animals with an evolutionary past, and understanding our normal psychology will likely involve ideas about how that past has shaped it. Hence, understanding breakdowns in that psychology will be, in part, based on our understanding of how evolved minds are organized. Evolutionary hypotheses may at least have heuristic value in the development of testable predictions about behavior and testable assumptions about the functions of the mind/brain. And if we can link the functions of the mind/brain to facts about behavior, development and society, we may be able to develop more high-powered predictions, and even establish why paranoiacs have a taste for engineering.

Acknowledgements

Thanks to Fiona Cowie, Jim Woodward and a reviewer for Biology and Philosophy for helpful comments on earlier drafts. This paper is a more skeptical amplification of some of the themes in Murphy and Stich (2000); I remain indebted to Steve Stich for his help back then, and at many other times.

References

American Psychiatric Association. 2000. Diagnostic and Statistical Manual of Mental Disorders, 4th edn, textual revision. American Psychiatric Association, Washington, DC.

Antony M.M. and Barlow D.H. 2002. Specific phobias. In: Barlow D.H. (eds), Anxiety and Its Disorders, 2nd edn. Guilford Press, New York, pp. 380–417.

Cheung M.S-P., Gilbert P. and Irons C. 2004. An exploration of shame, social rank and rumination in relation to depression. Pers. Indiv. Differ. 36: 1143–1153.

Clark D.M. 1986. A cognitive approach to panic. Behav. Res. Ther. 24: 461–470.

Clark D.M. 1988. A cognitive model of panic attacks. In: Rachman S. and Maser J. (eds), Panic: Psychological Perspectives. Erlbaum, Hillsdale, NJ, pp. 71–90.

Clark D.M. 1997. Panic disorder and social phobia. In: Clark D.M. and Fairburn C.G. (eds), The Science and Practice of Cognitive Behavior Therapy. Oxford, Oxford University Press, pp. 121–153.

Clark, D.M., Ehlers, A. 1993. An overview of the cognitive theory and treatment of panic disorder. Appl. Prevent. Psychol. 2: 131–139.

Cleckley H. 1988. The Mask of Sanity, 5th ed., First published 1976. E. S. Cleckley, Augusta, GA.

Craske M.G. and Barlow D.H. 2001. Panic disorder and Agoraphobia. In: Barlow D.H. (ed.), Clinical Handbook of Psychological Disorders, 3rd ed. Guilford Press, New York.

Cronkite R.C. and Moos R.H. 1995. Life context, coping processes and depression. In: Beckham E.E. and Leber W.R. (eds), Handbook of Depression, 2nd ed. Guilford, New York, pp. 569–587.

D'Arms J., Batterman R. and Gorny K. 1998. Game theoretic explanations and the evolution of Justice. Philos. Sci. 65: 76–102.

Dawes R. 1994. House of Cards: Psychology and Psychotherapy Built On Myth. Free Press, New York.

Drummond J.M., Issa F.A., Song C.-K., Heberholz J., Yeh S.-R. and Edwards D.H. 2002. Neural mechanisms of dominance hierarchies in crayfish. In: Wiese K. (ed.), The Crustacean Nervous System. Springer, Berlin, pp. 124–135.

Ehlers A. and Breuer P. 1992. Increased cardiac awareness in panic disorder. J. Abnorm. Psychol. 101: 371–382.

Garcia J. and Koelling R. 1966. Relation of cue to consequence in avoidance learning. Psychon. Sci. 4: 123–124.

Gilbert P. 2003. Evolution, social roles and the differences in shame and guilt. Soc. Res. 70: 1205–1230.

Godfrey-Smith P. 1994. A modern history theory of functions. Nous 28: 344–362.

Goodwin D. and Guze S. 1996. Psychiatric Diagnosis, 5th ed. Oxford University Press, New York.

Gray J.A. 1987. The Psychology of Fear and Stress, 2nd ed. Cambridge University Press, Cambridge.

Hagen E.H. 1999. The functions of postpartum depression. Evol. Human Behav. 20: 325–359.

Hare R.D. 1965. Psychopathy, fear arousal and anticipated pain. Psychol. Rep. 16: 499–502.

Hare R.D. 1970. Psychopathy: Theory and Research. Wiley, New York.

Hare R.D. 1999. Without Conscience. Guilford, New York.

Harpending H.C. and Sobus J. 1987. Sociopathy as an adaptation. Ethol. Sociobiol. 8(suppl.):563–572.

Hume, David 1777/1975. Enquiry Concerning the Principles of Morals. Oxford University Press, Oxford.

Keller M.B., Lavori P.W., Mueller T.I., Endicott J., Coryell W., Hirschfeld R.M.A. and Shea T. 1992. Time to recovery, chronicity and levels of psychopathology in major depression – A 5 year prospective follow up of 431 subjects. Arch. Gen. Psychiat. 49: 809–816.

Kravitz E.A. 2000. Serotonin and aggression: insights gained from a lobster model system and speculations on the role of amine neurons in a complex behavior. J. Compar. Physiol. A 186: 221–238.

Lykken D. 1995. The Antisocial Personalities. Erlbaum, Hillsdale, NJ.

Marks I.M. 1987. Fears, Phobias and Rituals. Oxford University Press, Oxford.

Marks I.M. and Nesse R. 1994. Fear and fitness: an evolutionary analysis of anxiety disorders. Ethol. Sociobiol. 1: 247–261.

Meyer J.H., McMain S., Kennedy S.H., Korman L., Brown G.M., DaSilva J.N., Wilson A.A., Blak T., Eynan-Harvey R., Goulding V.S., Houle S. and Links P. 2003. Dysfunctional attitudes and 5-HT2 receptors during depression and self-harm. Am. J. Psychiat. 160: 90–99.

McGuire M. and Troisi A. 1998. Darwinian Psychiatry. Oxford University Press, New York.

Mealey L. 1995. The sociobiology of sociopathy: an integrated evolutionary model. Behav. Brain Sci. 18: 523–599.

Millikan R. 1989. In defense of proper functions. Philos. Sci. 56: 188–202.

Mineka S., Davidson M., Cook M. and Keir R. 1984. Observational conditioning of snake fear in rhesus monkeys. J. Abnorm. Psychol. 93: 355–372.

Mineka S., Keir R. and Price V. 1980. Fear of snakes in wild and laboratory-reared rhesus monkeys. Anim. Learn. Behav. 8: 653–663.

Mineka S. and Tomarken A. 1989. The role of cognitive biases in the origins and maintenance of fear and anxiety disorders. In: Nilsson L. and Archer T. (eds), Aversion, Avoidance and Anxiety: Perspectives on Aversively Motivated Behavior.

Murphy D. (forthcoming). Psychiatry in the Scientific Image. MIT Press, Cambridge, MA.

Murphy D. and Stich S. 2000. Darwin in the madhouse: evolutionary psychology and the classification of mental disorders. In: Carruthers P. and Chamberlain A. (eds), Evolution and the Human Mind. Cambridge University Press, Cambridge, pp. 62–92.

Neander K. 1991. Functions as selected effects: the conceptual analyst's defense. Philos. Sci. 58: 168–184.

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

Nesse R.M. 1999. Testing evolutionary hypotheses about mental disorders. In: Stearns S.C. (ed.), Evolution in Health and Disease. Oxford University Press, New York, pp. 261–266.

Nessee R.M. 2000. Is depression an adaptation? Arch. Gen. Psychiat. 57: 14–20.

Nesse R.M. and Williams G.C. 1995. Why We Get Sick. Times Books, New York.

Newman J. 1998. Psychopathic behavior: an information processing perspective. In: Cooke D.J., Forth A.E. and Hare R.D. (eds), Psychopathy: Theory, Research and Implications for Society. Kluwer, Dordrecht, pp. 81–104.

Nichols and S. 2002. How psychopaths threaten moral rationalism, or is it irrational to be amoral? Monist 85: 285–304.

Price J., Sloman L., Gardner R., Gilbert P. and Rohde P. 1994. The social competition hypothesis of depression. Br. J. Psychiatr. 164: 309–315.

Pyszczynski T and Greenberg J. 1987. Self-regulatory perseveration and the depressive self-focussing style: a self-awareness theory of reactive depression. Psychol. Bull. 102: 122–138.

Rohde P., Lewinsohn P.M., Tilson M. and Seeley J.R. 1990. Dimensionality of coping and its relation to depression. J. Pers. Soc. Psychol. 58: 499–511.

Skyrms B. 2000. Stability and explanatory significance of some evolutionary models. Philos. Sci. 67: 94–113.

Sperber D. 1994. The modularity of thought and the epidemiology of representations. In: Hirschfeld L.A. and Gelman S. (eds), Specificity in Cognition and Culture. Cambridge University Press, Cambridge, pp. 39–67.

Thase M.E. and Howland R.H. 1995. Biological processes in depression. In: Beckham E.E. and Leber W.R. (eds), Handbook of Depression, 2nd ed. Guilford Press, New York.

Valentine C.W. 1930. The innate bases of fear. J. Genet. Psychol. 37: 394–419.

Watson J.B. 1924. Behaviorism. W.W. Norton, New York.

Watson P.J. and Andrews P.W. 2002. Towards a revised evolutionary adaptationist analysis of depression: the social navigation hypothesis. J. Affect. Dis. 72: 1–14.

Woolfolk R.L., Novalany J., Gara M., Allen L. and Polino M. 1995. Self-complexity, self-evaluation and depression: an examination of form and content within the self-schema. J. Pers. Soc. Psychol. 68: 1108–1120.