Evolutionary Approaches to Mental Health: Prospects and Limitations

Abstract. Evolutionary psychiatry regards our minds as significantly shaped by processes of natural selection and aims at offering evolutionary explanations for mental disorders. This chapter provides a short introduction to the field and explores two evolutionary approaches to depression, which is one of the most common topics in evolutionary psychiatry. After examining some of the advantages and challenges of these approaches, the chapter will close by indicating how evolutionary psychiatry can yield practical benefits in the form of clinical applications.

Keywords. Evolutionary Psychiatry, Depression

"Pain or suffering of any kind, if long continued, causes depression and lessens the power of action; yet it is well adapted to make a creature guard itself against any great or sudden evil." Darwin (1859/2003, 431)

Darwin’s (1859) theory about the evolving and transmutation of the species has advanced into a pivotal concept in biology and modern medicine, and it has increasingly become a valuable source in both explaining individual behaviors and social structures (Nesse 1984). Important works by Hamilton (1964) and Wilson (1975) have helped to establish evolutionary theory as an independent discipline, but also to making evolution into a basic science that can serve as a foundation for a number of research and clinical areas. For instance, seeking an explanation to why natural selection has left the human body susceptible to disease, “Darwinian medicine” (a phrase that began to be used in the early 1990s) fruitfully narrowed the gap between medicine and evolutionary biology, generating a number of evolutionary applications in medicine that include population genetics and evolution-based modeling to comprehend antibiotic resistance.

Given the prevalence of highly incapacitating (fitness-reducing) mental disorders such as schizophrenia, depression, phobias, anxiety, and obsessive-compulsive disorder, it is not surprising that evolutionary theory has begun to inform psychiatry's attempts to
understand and explain psychopathology. Arguments in favor of the view that at least some mental disorders are adaptations typically emphasize the global prevalence of mental disorder. For instance, it is widely agreed that depression is a highly debilitating condition that is extreme common, with 16.5% of the U.S. population experiencing major depression in their lifetime (Hadley & Patil, 2008; Kohrt et al., 2005; Patil & Hadley, 2008; Watson and Andrews 2009; National Institute of Mental Health). Beside the psychological suffering and distress, cognitive and emotional difficulties, patients with often commit suicide and die childless. Depression is also suitably prevalent throughout history to be appropriate to investigate its evolutionary origins. The prevalence of mental disorders like depression presents a puzzle from an evolutionary point of view, in particular because the risk profiles of individuals affected by incapacitating mental disorders can partly be explained through different genetic makeup (Brune 2006). As Adriaens and De Block (2010, 134) put it:

while ethological psychiatrists are mainly interested in understanding mental disorders by observing psychiatric patients and relating their symptoms to behaviour patterns found in other animal species, the second group of evolutionary psychiatrists considers mental disorders to be evolutionary oddities that need explaining. For why is it, they wonder, that natural selection is so slack in getting rid of mental disorders? Biological psychiatrists invariably assume that there are genes involved in man’s vulnerability to mental disorders – how come such genes have managed to escape natural selection?

In other words, evolutionary-informed researchers are surprised to discover that numerous mental disorders seem to have a genetic basis, while they occur at prevalence rates that are too high to be explained as mutations. It is normally assumed that variants with detrimental effects that hamper the ability to survive or reproduce would not replicate. This raises crucial but notoriously difficult questions, since high prevalence rates are usually taken to reveal that the genetic bases of these mental disorders have been promoted by natural selection. It would be natural to think that given the highly disabling nature of mental disorders the relevant genetic variants (susceptibility alleles) should
The Emergence of Evolutionary Psychiatry

Partly motivated by what appears to be a deeply puzzling fact, researchers have begun to systematically investigate mental disorders within the framework of contemporary evolutionary theory and to apply insights about evolutionary processes to offer new ways to understand the origin of mental disorders. Bringing this research program under a name, the term ‘evolutionary psychiatry’ was coined by MacLean in an influential editorial in *Psychological Medicine* from 1985. As a corrective to the risk of overly focusing on molecular biology, he envisaged that the domain of evolutionary psychiatry could encompass both the microscopic and macroscopic aspects of underlying phenomena.

“Some physicians are concerned that, because of the present emphasis on molecular and genetic biology and the Paracelsan drive to find a chemical to cure every condition, there is a tendency to treat patients as though they could be manipulated impersonally like machines. […] Evolutionary psychiatry provides counteractive leaven for reductionistic views inherent in the molecular approach. […] Evolutionary psychiatry would therefore apply to potentially salutary insights, derived from a better understanding of how the psychencephalon has evolved and how it functions” (MacLean, 1985, 219).

MacLean’s piece was followed by two important publications. Cosmides and Tooby (1987) put forward a highly influential account of evolutionary psychology, which also became an important reference for the emergence of evolutionary psychiatry, together with the publication of *Evolutionary Psychiatry: A New Beginning* by Stevens and Price in 1996. As they maintain in the second edition of their influential book, their work was committed to the idea that “no theory in psychology or psychiatry could hope to possess any lasting value unless it was securely founded on knowledge of the evolution of our species” (Stevens and Price 2000, 275). It might sound surprising to some contemporary
ears that evolutionary psychiatrists were often criticized by opponents for being political motivated by a “right wing” agenda. However, as Stevens and Price (2000, 277) note,

the primary duty of the psychiatrist will remain the same: to put skill, empathy, knowledge, and professional commitment at the service of the patient. To adopt an evolutionary approach is not to espouse a political cause, nor is it an invitation to submit to ‘biological determinism’ or an encouragement to abandon a proper concern with ethical or value-oriented premises. However sophisticated our understanding of neural mechanisms and brain chemistry may become in the years ahead, human beings will continue to live in their minds rather than their brains and the primacy of the psyche will remain paramount in all human endeavours – not least in the treatment of mental suffering.

Since then, there has been a veritable “adaptive turn” and evolutionary explanations are being proposed for an increasing number of aspects of psychopathologies (Nettle 2004; Nesse & Williams 1995, Stevens & Price 1996, McGuire & Troisi 1998). Since MacLean first talked about it, evolutionary psychiatry has emerged as a significant theoretical perspective, and the growing number of studies by researchers in this field is beginning to assume a noticeable presence within psychiatry. At the same time, some of the hypotheses generated in evolutionary psychiatry remain controversial among some psychiatrists, particularly due to scientific concerns about underlying frameworks, to some disturbing implications regarding worries about an overly simplistic picture of human beings as fitness-maximizing genetic machines. Other concerns are related to the fact that the conceptual framework of evolutionary psychiatry is more a metatheory for assisting psychiatry’s understanding the disorders of the human mind and less designed to perform as creating practical applications.

One of the features of evolutionary psychiatry that has fueled the “adaptive turn” was the potential ability of evolutionary theory to offer explanations that identify ultimate causes of disorders. Psychiatric research has mostly focused on proximate explanations: proximate causes explain a process or structure in an individual organism. Evolutionary psychiatry aims to go beyond identifying proximate causes and to pinpoint ultimate
(evolutionary) causes that explain the particular process or structure in all members of a species. Such explanations can be eye opening in many ways, resulting in fuller understanding of the condition and the possibility of integrating different levels of description. Indeed, it seems in many cases plausible that a complete understanding requires integrating both types of explanation.

**Evolutionary Psychiatry and Depression**

In spite of some general agreements between researchers in evolutionary psychiatry, a second look reveals a surprising heterogeneity of theoretical inferences, methodologies, and conclusions. When it comes to the explanations of the applications of evolutionary theory to psychopathology, three types of explanations can be distinguished (Murphy 2005). To provide a brief outline of the structure of evolutionary explanations in psychiatry and to introduce some reflections on the benefits and problems of such approaches, the investigation in this chapter will be limited to addressing two different evolutionary accounts of depression.

1. **Mismatch explanation** (Nesse 2000; Gilbert and Allan 1998): mental disorder is connected to a mechanism that was once adaptive but is no longer adaptive because of changes in the environment.


It is not difficult to see that these accounts proceed in opposing directions, but they share the basic idea that the mechanisms activated in depression evolved to manage hostile situations in which flight was impossible.
The Mismatch Explanation

In a myriad of ways, our contemporary world fundamentally differs from the world of the Pleistocene, which also means that mechanisms which were fitness enhancing might under contemporary conditions be fitness reducing. Our craving for fats, sugars, and salt are plausibly adaptations that were essential but rare in the Pleistocene. But in most societies today, these cravings often lead to fitness-reducing excess, particularly as they are common and relatively affordable. So while on the one hand our cravings are functioning normally as they evolved to, they are malfunctioning in the sense that they are detrimental to our health. Other consequences of such mismatches have been identified in the case of autoimmune diseases, asthma, and allergies (Stearnes 2012).

This is the fundamental idea behind the mismatch explanation of depression. The authors in this camp take seriously that the human mind consists of hierarchically organized systems of very different evolutionary ages, including a “reptilian,” a “palaeo-mammalian,” a “neo-mammalian,” and mechanisms specialized for language and symbolic processing (see Stevens and Price 1996, Ch. 2). On this view, our minds can be understood as composite integrated assemblages that consist of a number of functionally specialized adaptations that evolved as solutions to different adaptive problems. The core of the proposal is that these systems are still active beneath the threshold of consciousness, albeit sometimes acting in conflicting ways (Stevens and Price 1996; Cosmides and Tooby 1999). Like our ancestors, we are predisposed to behave in ways that were adaptive in that original environment. However, in a radically changed environment, those evolved genes and patterns of behavior now promote psychopathological conditions (Baptista et al. 2008). Such a mismatch between inherited predispositions (behavioral, cognitive, affective, etc.) and the current environment with very different social stimuli and affordances might trigger mental disorders. More precisely, the idea of the mismatch explanation is that depression evolved as an adaptive response to specific problems that arise in the small, status-oriented social group of our ancestors (Price et al. 1994; Nesse and Williams 1995). It might actually have served several purposes.

One the one hand, it may be that depression helped assist the restoration of
exhausted resources by forcing the individual to withdraw and ultimately to help to maximize payoffs by resource re-allocation (Nesse 2000; Nesse and Williams 1995; Schmale, 1973). The characteristic sense of capability to fulfill tasks, pessimism, behavioral inactivity, and the well-documented exaggerated interpretation of the difficulty of a task, restrains the depressive from allocating resources in demanding activities with low probability of success (Sloman, Gilbert and Hasey 2002). Similarly, Stevens and Price (2000) argue that certain forms of depression are considered to be constructive human responses to situations in which a desired social goal seems impossible to achieve.¹ Thus, depression could be seen as a defense mechanism to help the person disengage from impossible undertakings that have become such a matter of habit that under normal circumstances it is unfeasible for the individual to abandon them. While this evolved capacity has functioned as a defense mechanism for enabling disengagement from impossible undertakings, the claim is that it now causes conflicts that lead to mental disorders.

On the other hand, it may be that depression is an adaptive response to the loss of status in small social groups. Depression-like states occur in animals and humans who have been defeated and lost rank, and the advantage might be that depressive states help the loss of status and lowered rank (Price et al. 1994, Stevens and Price 1996; Gilbert, 1992, 2000). The authors maintain that depressed states induce self-evaluation and reflection upon weaknesses that might lead to altered behaviors and ultimately to better reproductive chances. Research on down-regulation of serotonin level in depression appears to offer some support for this theory. For instance, when animals change their place in a power hierarchy, their behavioral changes are accompanied by changes in serotonin levels (Kravitz 2000; Drummond et al. 2002; Shively 1999; Grant et al. 1998; McGuire and Troisi 1998).

In all, it is not difficult to see that such an adaptive response might have been a fruitful strategy in the small groups of our ancestors, while recognizing that the affect-lowering response to change in status is no longer adaptive in contemporary societies (Nesse and Williams 1995). As a result, at least in modern Western societies, this

¹ It appears to support this thesis that depression is common in people who are pursuing unattainable goals (Randolph & Nesse 2000; Davis 1970).
mechanism will both fail to accomplish the goal it was selected to achieve and contribute to fitness decreasing.\(^2\)

### The Persistence Explanation

In contrast to the mismatch explanation, the persistence explanation makes the bold claim that depression and the related genetic material is also adaptive in our current environment. The researchers in this camp of course recognize that the relevant mental disorders cause serious pain and distress, but they argue that there is an overall adaptive function to changed circumstances (Price et al. 1994; Hagen 1999; Watson and Andrews 2002). Just as they did in the ancestral environment, some alleged psychopathologies function adaptively in the present environment.

It is well-known that some involuntary responses to environmental challenges like negative emotions can be seen as stress response mechanisms, which coordinate bodily and cognitive resources to manage certain tasks linked to adaptive challenges. Other mechanisms like fever are adaptations that evolved to increase fitness by a specific immune system response, but they simultaneously reduce metabolism, sexual, and social activity (Hasday, Fairchild, & Shanholtz, 2000; Cosmides & Tooby, 2000; LeDoux, 1996). In much the same way, while depression clearly interrupts normal function, its aversive and disruptive characteristics might turn out to be the key to its adaptive capacity. Following the same path of thought, Darwin (1859/2003, 431) himself considered depression as an adaptive function: “Pain or suffering of any kind, if long continued, causes depression and lessens the power of action; yet it is well adapted to make a creature guard itself against any great or sudden evil.”

The persistence explanation comes in several variants, and we may distinguish between individual and social versions. As mentioned, certain forms of depression are conceivable as constructive human responses to situations in which a desired social goal is unachievable. If the distress and the down-regulation of positive affect systems that

\(^2\) In contemporary societies, it is arguable that such a mechanism will be activated inappropriately as our peer group with whom we compete is very large. In fact, it is difficult to imagine a niche one could fill in which one would not be outmatched in global competition (Murphy 2005). This also gives some ground for speculation about a possible link between globalization and the increase of depression in the last few decades.
characterize depression incites the depressed to re-evaluate and abandon impossible or unmanageable undertakings, then depression might be seen as an adaptive response to social circumstances, enhancing our ability to navigate in the social context. This view goes back to earlier work by D. A. Hamburg, who maintained that in a case where the individual estimates that the probability of achieving a goal is very low, “the depressive responses can be viewed as adaptive,” because “feelings of sadness and discouragement may be a useful stimulus to consider ways of changing (the) situation” (Hamburg 1974, 240). Thus, the down-regulation of positive affect, diminished responsiveness, and the lack of motivation may be seen as fostering disengagement from the unachievable challenges, which at the end could harm the individual.

But how can we square this with the widely acknowledge view that depression produces specific impairments, such as maladaptive cognitions (Beck and Alford 2009)? Not only are “depressive ruminations” often extremely resistant to interventions, but they are also considered to play an important part in the development, maintenance, and relapse of recurrence of depression (Treynor, Gonzalez, & Nolen-Hoeksema, 2003; Nolen-Hoeksema, 1990, 1991; Wenzlaff & Luxton, 2003). However, Thompson and Andrews (2009, 623) argue that contrary to this appearance, depressive rumination harbors a beneficial cognitive effect. The point is that depression as a stress response mechanism is triggered by analytically difficult problems, and the sustained analysis of in depressive rumination helps people generate and evaluate potential solutions to the triggering problem. Thus, the adaptive aspect is that the social withdrawal and depressive rumination enables the depressed individual to engage in a profound analysis of the triggering problems. Watson and Andrews (2009) are well aware of the costs of such withdrawal and rumination, but argue that benefits are great enough to compensate for the substantial costs.

The second variant of the persistence explanation takes an entirely different, social path. Focusing on an adaptationist stance on postpartum depression, Hagen (1999) has maintained that depressed mothers obtain greater care from both their partners and their social network. Watson and Andrews (2002) extend this idea to depression generally, and argue that depressive responses have this function of obtaining a re-vitalizing of social relationships. In this view, adults' depression conveys a plea for help
to others, to arouse pity in them interpreted as a communication designed to manipulate others into providing resources. Watson and Andrews (2002) review evidence that depression is associated with social problems and suggest that complementary to focusing limited cognitive resources, it plays a crucial role in motivating close social partners (who have a positive fitness interest in the normal functioning of the depressed) to provide help and to make concessions in favor of the depressed. Disinterest in normal fitness-related activities, anhedonia, and psychomotor perturbation does not only impose costs on the depressed; social partners are aware of the costs imposed on them when a partner is depressed (Segrin and Dillard, 1992). The costs of depression motivate members of the depressive’s social network to make investments that they under normal circumstances would hesitate to make. They might understand the benefits of assisting and ending the depressive episode, to avoid escalating costs. Besides specific partners, depression is also thought to be designed to motivate the entire social network, depending on the configuration of the network and the way the costs of depression are distributed in it. In this sense, depression may be an extortive means to motivate within the network to overcome their reluctance to help (Hagen, 1999). The fact that there is overwhelming empirical evidence maintaining that individuals in conflict with significant peers are more likely to become depressed seems at least partly to support the persistence position. For instance, while interpersonal conflict is commonly associated with depression (Hammen, 1992), it is striking that in unhappily married people the risk for major depression is about 40 times greater than in happily married ones (Weissman, 1987). Overall, even if depression might cause abandonment and sometimes produce social deterioration, the main idea underlying the social navigation hypothesis is that depression has evolved to serve a social motivation function.

Some challenges

Both the mismatch and the persistence explanation provide accounts that productively deploy the resources of evolutionary theory to the field of psychiatry. The type of ultimate causes they aim to offer could provide a helpful contribution to understanding a host of conditions and could complement our knowledge about proximate causes.
Another positive feature is that both accounts have the resources to reflect on how the change of social environments could affect the prevalence of mental disorders. Having explored the main lines of thought underlying the mismatch and persistence explanations of depression, the last part of the chapter will be dedicated to a brief survey of some of the challenges that both types of explanations face. While I have elsewhere provided a fuller assessment of the challenges (Varga 2010), due to special limitations I shall consider only the most important ones here.

Let me start with a worry of a very general nature. We only possess a very sketchy understanding of the myriad of changing selection pressures over millions of years that our ancestors encountered. In lack of detailed knowledge about selective events, caution is warranted when it comes to the conclusions we are able to justifiably draw. This limitation is aggravated by the fact that evolutionary psychiatry displays a comparative deficiency in explaining not only individual but cultural differences.

Further limitations arise from the logic of evolutionary explanations. We have noted in the beginning of this chapter that the prevalence of mental disorder is usually taken to be supporting the view that some mental disorders are adaptations. Nonetheless, as Ravenscroft (2012) notes, this argument contains a problematic and suppressed premise. The premise is that natural selection is a mechanism that not only optimizes systems, but also eliminates imperfections. However, Richard Dawkins (1986) maintains that this is not how selection mechanisms operate. For instance, he demonstrates how the blind spot of the human eye is a maladaptive property that natural selection has not eliminated. In the same way, Ravenscroft (2012, 453) holds that “some mental disorders may be maladaptive features of the human cognitive apparatus which are unlikely be driven from the lineage because the brain is trapped on a local optimum.”

The mismatch model as a specific application of evolutionary theory to depression clearly offers illuminating aspects. However, it also needs to provide convincing answers to some issues. While there are several points to address, let me just mention a crucial one. This is that the mismatch account fails to address individual and social factors. In other words, why are certain individuals afflicted by the condition and not others? And, given the ubiquity of status competitions and status changes in our contemporary societies, why are not more individuals afflicted by the condition? So while it is an
advantage that on the mismatch view it is possible to speculate on the relation between globalized societies and the increase of depression, this advantage risks remaining speculative if the individual factors are not addressed.

The persistence account fairs better integrated on this issue, as it can integrate concrete individual causal factors and social problems. It is compatible with the view that the way our modern societies have evolved has created the conditions under which higher rates of ruminative and motivational depression are likely to occur. For instance, it seems reasonable to think that living in social networks with reduced fitness interests amongst partners may contribute to a growing prevalence of depression. But the main problem of the persistence account may be that the usefulness of depressive rumination is overestimated.

One way of opposing the thesis that depressive rumination is useful is by looking at the typical themes that occur in such ruminations. It appears that typical ruminations such as “Why am I such a bad person?” are often of hypothetical nature, and it is in many cases questionable whether they can be understood as real problems that require analytical attention or that need to be “solved.” Also, depressive rumination may not be triggered by complex social problems that are to be treated and solved in an analytical manner. Often, there is no apparent external trigger for a depressive episode that can be identified and ruminated on. But even if this were the case, social dilemmas might not have analytical structure that is best dealt with by breaking it down and studied with respect to each goal the individual might have (Varga 2010). It might not be the case that such complex choices are adequately treated by analytical processing involving cost and benefit analysis.

But an even more pressing question is whether depressive rumination is actually helpful for the individual. According to an established body of research, instead of helping to provide solutions, rumination actually exacerbates and prolongs distress—particularly depression (Nolen-Hoeksema, 1991)—impairs problem-solving capacities, and hinders instrumental behavior. Longitudinal studies demonstrate that people engaging in rumination as a reaction to stress are more vulnerable to develop depressive disorders and to have prolonged periods of depression (Just & Alloy, 1997; Kuehner & Weber, 1999; Nolan et al., 1998; Nolen-Hoeksema, 2000; for an overview see Nolen-
Hoeksema, Wisco and Sonja Lyubomirsky 2005). Thus, depressive rumination not only fails to be solution-oriented, but directly hampers problem-solving abilities: the ruminating person will be focused on her depressive symptoms, which typically involves negative self-ascriptions that might lead to an incapacity to engage in constructive activities or to lack confidence in their solutions. In fact, rumination tends to result in an assessment of the particular problems as overpowering and impossible to solve, and consequently people do not succeed in problem solutions (Lyubomirsky et al., 1999; Donaldson & Lam, 2004). Thus, it seems that ruminative responses in depression also interfere with good solutions through the inhibition of instrumental behavior.

Assuming that depression is an adaptation, one would still have to account for the fact that every depressive episode dramatically increases the risk of another episode. It is difficult to see for whom this could be an advantage. Rather, it seems to contradict that depression is an adaptation to allow optimal functioning. In addition, it is also difficult to see how this account could be squared with the fact that characteristic traits in depression not only fail to bring about advantages, but increase sexual dysfunction, physical pain, and the risk of suicide (DSM-IV-TR, 349–52).

**Conclusion**

Evolutionary psychiatry has emerged to the status of a major theoretical perspective over the last two decades, and it has already generated a sizeable volume of theoretical and empirical studies. It is understandable that many are attracted to the application of evolutionary principles to psychiatric phenomena in the hope of being able to offer ultimate explanations. But it is helpful to counterbalance a certain biological nativism. As Nesse and Jackson (2011, 194) put it, “campaigns to convince the public and practitioners that depression and anxiety are brain diseases have motivated much useful research and have decreased stigma, but they are biologically naive. An evolutionary approach supports a more medical model in which clinicians recognize many symptoms as defenses shaped by natural selection that are aroused by more primary causes, and others arising from defects in the systems that regulate defenses.”
However, while evolutionary psychiatry is assuming an increasing presence within psychiatry science, the “adaptive turn” has also generated a range of criticisms. Many researchers are able to appreciate the contributions that evolutionary explanations can make for a number of mental disorders, but some point to problems that the different version of evolutionary explanations will have to face. Nevertheless, the challenges of evolutionary theories do not warrant the conclusion that we should reject applications of evolutionary theory to depression. Our understanding of mental disorders like depression is still rudimentary, and there is no general reason why the fact that mental capacities evolved should not be considered as a significant source of knowledge. Such theories have provided perspectives that might not have been raised without an evolutionary perspective on human social behaviors, and their heuristic value in the development of testable assumptions should definitely not be overlooked.

When introducing evolutionary psychiatry in the beginning of this chapter, I have noted that some psychiatrists are concerned that although the conceptual framework of evolutionary psychiatry assists psychiatry’s understanding of disorders, it is not designed to help create practical applications. Although Stevens and Price (2000, 278) close their book by expressing hope that evolutionary psychiatry will eventually be able to help provide effective measures for the prevention and treatment of mental disorders, skeptics may emphasize that it is difficult to claim that progress in this area has been astonishing. But let me finish this chapter with an example that points in the opposite direction by demonstrating a practical application in the field of clinical psychology. In recent years, a treatment for depression has been developed based on the mismatch theory of depression. Taking seriously the enormous differences between ancestor and modern environments, diet and social settings, Ilardi has developed a treatment for depression that simulates ancestral living conditions, emphasizing exercise, exposure to sunlight, good sleep hygiene, and anti-ruminative activity. The therapy had a surprisingly high success rate in reducing the symptoms of depression (Ilardi et al., 2007; Confer, Easton et al 2010). These types of effective clinical applications are still rare. However, they nevertheless indicate that evolutionary psychiatry can yield practical benefits in the form of clinical applications.
References


