

Chapter 22

Evolutionary Approaches to Depression: Prospects and Limitations

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Lay Summary Evolutionary psychiatry has emerged to the status of an important theoretical perspective over the last two decades, and it has 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. Some are attracted by the possibility of providing ultimate explanations for certain mental disorders, while others also think that such an approach can help to counterbalance a naïve understanding of mental disorder. As Nesse and Jackson [1: 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 naïve. 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”. Nonetheless, while evolutionary psychiatry is assuming an increasing presence within psychiatric science, the “adaptive turn” has also generated a range of criticisms. Many researchers appreciate the contributions that evolutionary explanations offer for a number of mental disorders, but highlight serious problems that different versions of evolutionary explanations face. The investigation in this chapter was limited to addressing two evolutionary approaches to depression, the mismatch explanation and the persistence explanation. Although both accounts exhibit deficiencies, the conclusion that we should reject applications of evolutionary theory to depression is not warranted. Evolutionary psychiatry should be considered as a potential source of knowledge, and its heuristic value in the development of testable assumptions should not be ignored.

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22.1 Introduction

Darwin's theory [1] about the evolution and transmutation of the species has advanced into a pivotal concept in biology, and it has increasingly become a valuable source for explaining structural and behavioural variation between species, groups and individuals [2]. Important works by Hamilton [3] and Wilson [4] have helped to establish evolutionary biology as an independent discipline, but it is only recently that this field of research began to narrow the gap between medicine and evolutionary biology [5]. The prevalence of mental disorders such as depression presents a puzzle from an evolutionary point of view, in particular, because the risk profiles of individuals affected by such incapacitating conditions can partly be explained through different genetic make-up [6]. As Adriaens and De Block [7: 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?

The prevalence of highly incapacitating (fitness-reducing) mental disorders such as schizophrenia, depression, phobias, anxiety and obsessive-compulsive disorder raises crucial and difficult questions. Depression is both prevalent enough throughout history to make appropriate the investigation of its evolutionary origins, and it is an extremely common debilitating condition [8]. However, beside the psychological distress, cognitive and emotional difficulties, patients with depression are more likely to develop diabetes and cardiovascular disease, to commit suicide and to die childless [9–11].

Partly motivated by what appears to be a deeply puzzling fact (i.e. genes causative of mental disorders have escaped natural selection), researchers have begun to systematically investigate mental disorders within the framework of contemporary evolutionary theory. Bringing this research programme under a name, the term “evolutionary psychiatry” was coined in 1985 by MacLean in an influential editorial published in *Psychological Medicine*. MacLean [12: 219] argued that “evolutionary psychiatry provides counteractive leaven for reductionist views inherent in the molecular approach” and envisaged that the domain of evolutionary psychiatry could encompass both microscopic and macroscopic aspects. Shortly after, Cosmides and Tooby [13] put forward a highly influential account of evolutionary psychology, which quickly became an important reference, together with *Evolutionary Psychiatry: A New Beginning* by Stevens and Price. As Stevens and Price [14: 275] maintain in the second edition of their influential book, their work is 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”. Because evolutionary approaches were sometimes criticized for being politically motivated,

Stevens and Price [14: 277] explicitly maintain that “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”.

Since the publication of MacLean’s editorial, 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 [6, 14, 15, 16, 17, 18, 19, 20]. One of the attractions of evolutionary psychiatry is its potential to offer explanations that identify the ultimate causes of mental disorders. Traditional psychiatric research has mostly focused on proximate causes, shedding light on the relevant processes or structures in individual organisms. While such explanations seek to understand the mechanisms that underpin a trait or behaviour, ultimate explanations are concerned with evolutionary function [21].

22.2 Research Findings: Evolutionary Psychiatry and Depression

When it comes to the applications of evolutionary theory to psychopathology, several types of explanations can be distinguished [22]. To provide a brief outline of the structure of evolutionary explanations in psychiatry and to introduce some reflections on the benefits and problems associated with such approaches, the investigation in this chapter will be limited to addressing two different evolutionary accounts of depression.

- (1) Mismatch explanation [23, 24]: mental disorder is connected to mechanisms that were once adaptive, but that in our present environment are best seen as maladaptive.
- (2) Persistence explanation [25–27]: some mental disorders qualify as adaptive even in the present environment.

These accounts proceed in opposing directions, but they share the basic idea that the mechanisms activated in depression evolved to manage certain hostile situations. The chapter will not consider another type of evolutionary explanation that is to a certain degree compatible with the mismatch explanation. According to Nettle [18], depression itself is not selected for. Instead, evolution has produced an optimal reactivity of affect systems, with a normal population distribution around this optimum. But individuals situated in the upper tail of the population distribution are vulnerable to depression.

22.2.1 *The Mismatch Explanation*

The authors in this camp propose that the human mind consists of hierarchically organized systems of very different evolutionary ages (including a “reptilian”, a

“palaeo-mammalian” and a “neo-mammalian” system) and mechanisms specialized for language and symbolic processing (see [14], Chap. 2). In 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 (e.g. foraging and mating). The core of the proposal is that these systems are still active beneath the threshold of consciousness, albeit sometimes acting in conflicting ways [28, 29]. The idea is that depression evolved as an adaptive response to specific problems that arise in the small, status-oriented social groups of our ancestors [16, 25]. However, in a radically changed environment, those evolved patterns of behaviour now promote unfavourable and maladaptive conditions [30].

Stevens and Price [14] argue that certain forms of depression were constructive human responses to situations in which a desired social goal appeared impossible to achieve. It was speculated that depression assisted the restoration of exhausted resources by forcing the individual to withdraw, helping to maximize pay-offs by resource reallocation [16, 23, 31]. In small groups of hunter-gatherers, depressed states might have induced reflection on weaknesses leading to altered behaviours and ultimately to better reproductive chances. Research on serotonin-level down-regulation in depression appears to offer some support for this theory. For instance, when animals change their place in a power hierarchy, their behavioural changes are accompanied by changes in serotonin levels [19, 32, 33, 34]. The characteristic sense of incapability to fulfil tasks, pessimism, behavioural inactivity and the well-documented exaggerated interpretation of the difficulty of a task restrains the depressed individual from allocating resources in demanding activities with low probability of success [35]. 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 individual to accept the loss of status and lowered rank [28, 36, 37].

In all, some authors maintain that depression is an adaptive response to the loss of status in small social groups. According to the mismatch explanation, while depression might have been a productive strategy in small groups of hunter-gatherers, in contemporary Western societies it is no longer adaptive [16] and associated with decreased reproductive success [14: 79, 38].

22.2.2 The Persistence Explanation

The persistence hypothesis makes the bold claim that depression and the related genetic material are still adaptive in current Western environments. Researchers in this camp of course recognize that depression causes serious pain and distress, but they argue that depression is the expression of an overall adaptation to changed circumstances [25–27]. It is well-known that whether or not a trait is adaptive is a matter of degree and that some adaptations can be associated with fitness benefits as well as fitness costs. Some mechanisms, such as fever, can be understood as adaptations, while they simultaneously reduce metabolism, sexual and social activity [39–41]. In much the same way, the idea is that while depression clearly

interrupts normal functioning, its aversive and disruptive characteristics might actually help us understand its adaptive function. Following the same path of thought, Darwin (1859/2003, 431) himself considered depression as adaptive.

If the characteristic rumination and the down-regulation of positive affect systems that characterize depression incite the depressed individual to re-evaluate and abandon impossible or unmanageable undertakings, then depression might be seen as an adaptive response to social circumstances. 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” [42: 240]. Thus, characteristic features in depression such as rumination, down-regulation of positive affect, diminished responsiveness and the lack of motivation may be seen as fostering disengagement from unachievable goals, which at the end could harm the individual. For instance, Andrews and Thompson [43: 623] argue that depressive rumination harbours a beneficial cognitive effect. The point is that depression as a response mechanism is triggered by analytically difficult problems, and depressive rumination helps people generate and evaluate potential solutions. Thus, the persistence explanation claims that the adaptive aspect is that depressive rumination enables the individual to engage in a profound analysis of the triggering problems. Watson and Andrews [27] are well aware of the costs of such solitary rumination, but conjecture that the benefits are great enough to compensate for the costs.

Focusing on post-partum depression, Hagen [26] maintains that depressed mothers obtain greater care from both their partners and their social network. Watson and Andrews [27] extend this idea to depression generally, and argue that depressive responses revitalize social relationships. In this view, adults’ depression conveys a plea for help to others and should be understood as involving a type of communication designed to manipulate others into providing resources. Watson and Andrews [27] review evidence that depression is associated with social problems and suggest that depression plays a crucial role in motivating close social partners to provide help and to make concessions in favour of the depressed. Social partners are aware of the costs imposed on them when a partner is depressed [44], and the costs motivate the members of the depressive’s social network to make investments that under normal circumstances they would hesitate to make. In this sense, depression may function like an “instrument” to motivate individuals within the network to overcome their reluctance to help [26]. Overall, despite the fact that depression sometimes causes abandonment and produces social deterioration, the main idea is that depression qualifies as adaptive even in the present environment.

22.2.3 Some Challenges

Having explored the main tenets of the mismatch and persistence explanations, the last part of the chapter will be dedicated to a brief survey of some of the most important challenges that they face (for a more complete evaluation, see [45]).

Let me start with a general concern. In the absence of detailed knowledge about selective events, caution is warranted when it comes to the conclusions we are able to draw. This limitation is aggravated by the fact that evolutionary psychiatry displays a comparative deficiency in explaining both individual and cultural differences in the diagnosis and experience of depression. Further limitations arise from the logic of evolutionary explanations. We have noted in the beginning of this chapter that the prevalence of some mental disorders is usually taken to support the view that they are adaptations. Although the meaning of optimization in biology and medicine is not identical, Ravenscroft [46] notes that this argument contains a problematic and suppressed premise, according to which natural selection is a mechanism that not only optimizes systems, but also eliminates imperfections. However, Dawkins [47] maintains that this is not how selection operates. 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 [46: 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”.

Let us now turn our attention to the mismatch explanation. A crucial concern is that the mismatch account fails to address why under identical environmental conditions only certain individuals will be afflicted by the condition. And, given the ubiquity of status competitions and status changes in our contemporary societies, why are not more individuals afflicted by the condition? The persistence explanation fares better on this issue, as it is compatible with the view that the way our modern societies have evolved has created conditions under which higher rates of depression are likely to occur. But a limitation of the persistence explanation is that it is difficult to see how depression can be understood as an adaptation to allow optimal functioning if it significantly increases the risk of another depressive episode, diabetes, cardiovascular disease, sexual dysfunction, physical pain and suicide [8, 9]. Individuals with depression have a shorter life expectancy than those who are not suffering from the condition, in part because of the significantly increased risk of dying by suicide [48].

In addition, the fitness enhancing nature of depressive rumination may be questioned. When analysing typical themes that occur in typical ruminations (such as “Why am I such a bad person?”), we see that they are often of hypothetical nature, and it is in many cases doubtful that they can be understood as problems that require analytical attention or that need to be “solved”. Also, depressive rumination may not be triggered by complex social problems. 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 requires a particular type of analytical approach. Furthermore, depressive rumination may not be fitness enhancing at all, as it actually exacerbates and prolongs distress in depression [49], impairs problem-solving capacities and hinders instrumental behaviour. Depressive ruminations appear to play an important part in the development, maintenance and recurrence of depression [50, 51]. 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 [52–56]. Thus, depressive rumination not only fails to be solution-oriented, but also directly hampers problem-solving abilities and tends to result in an assessment of problems as overpowering and impossible to solve [57, 58].

22.3 Implications for Policy and Practice

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 does not directly contribute to the creation of practical applications. Although Stevens and Price [14: 278] close their book by expressing hope that evolutionary psychiatry will eventually be able to help providing effective measures for the prevention and treatment of mental disorders, sceptics may emphasize that it is difficult to claim that progress in this area has been satisfactory. Although effective clinical applications are still rare, some recent developments nevertheless indicate that ideas from evolutionary psychiatry can yield practical benefits in the form of clinical applications. For example, in recent years, a treatment for depression has been developed based on the mismatch theory of depression. Taking seriously the enormous differences between past and modern diet, physical environments and social relations, Ilardi has developed a treatment for depression that simulates ancestral (or just pre-industrial) 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 [59, 60].

Glossary

Rumination A way of responding to distress that is characterized by a compulsively focused attention on the symptoms of distress and their possible causes and consequences. Usually, the rumination in individuals with depression is negative in valence

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