Evolutionary Theories of Depression: A Critical Review

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We critically review evolutionary theories of major depressive disorder (MDD). Because most instances of MDD appear to be caused by adversity, evolutionary theories of MDD generally propose that sadness and low mood evolved as beneficial responses to adversity, and that MDD is dysfunctional sadness and low mood. If so, MDD research should focus much more heavily on understanding the healthy functions of sadness and low mood to better understand how they dysfunction. A debate about the boundary between healthy sadness and MDD is then reviewed. In part, this debate turns on whether MDD's costliest symptoms could provide unknown benefits. Therefore, the review concludes by discussing 2 theories that explore possible benefits of prolonged anhedonia and suicidality.

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Clinical Implications

- The clinical implications depend on which, if any, of these evolutionary theories of MDD is correct. In general, if MDD is dysfunctional sadness or low mood, then the primary goal of psycho- or chemotherapy would be to restore healthy sadness and low mood, and not simply to reduce MDD symptoms. Conversely, if MDD is functional, then therapy would aim to solve the social problem that triggered the MDD.
- In particular, if MDD is an involuntary subordinate strategy, then therapy should involve encouraging patients to let go of important but seemingly unobtainable life goals that involve conflicts with higher-status people. However, if the analytical rumination hypothesis is correct, then therapy should instead focus on identifying and solving the complex life problem that triggered the MDD. In some patients, and contrary to the involuntary subordinate strategy hypothesis, this may involve redoubling efforts to achieve a seemingly unobtainable life goal.
- Finally, all psychosocial theories of MDD imply that it may be possible to prevent MDD by identifying and avoiding social risk factors. For instance, the credible signalling hypothesis highlights the potent MDD risk posed by the confluence of social conflict, powerlessness, and adversity, each of which, in principle, could be reduced with appropriate interventions.

Limitation

- The primary limitation of all evolutionary theories of MDD is that, although each is a reasonably parsimonious account of known facts about MDD, few of their novel predictions have been explored. In other words, these theories are largely untested.

Key Words: *evolution, depression, sadness, low mood*

Nature has placed mankind under the governance of two sovereign masters, pain*, and* pleasure*. It is for them alone to point out what we ought to do, as well as to determine what we shall do.—Jeremy Bentham*[1]

By rejecting [Cartesian] dualism, we accept that all psychiatric disorders are biological. But so then are all mental processes, pathological or otherwise.—Kenneth Kendler[2]

MDD has long been claimed to be a common disease that is caused, in part, by a simple imbalance of neurotransmitters, and that is effectively treated with drugs correcting this imbalance.[3–5] (MDD will be used as defined in DSM-III or -IV, or depressive episode in the International Classification of Diseases, 10th Revision, excluding bipolar and psychotic depression. Sadness and low mood will be used to refer to a range of negative emotions that would not be classified as MDD.) This mainstream story has been undermined by a failure to prove the monoamine deficiency hypothesis or any other theory of MDD,[3,6] long-standing concerns about a high false-positive rate in diagnoses of MDD,[7,8] the poor performance of ADs relative to placebo,[9–11] failure to improve AD efficacy since the 1950s,[12] biased publication of AD trials,[13] and accusations of ethical violations by leading MDD researchers.[14–16]

However, the failure of the mainstream approach to either successfully explain MDD or improve its treatment does not mean that MDD has no biochemical basis. As Kendler[2] points out, the brain is a biochemical machine: all brain activity, including thoughts, emotions, and memory, arise from biochemical changes in the brain. Hence MDD is (and must be) biological or chemical. The failure of the mainstream approach to MDD suggests, instead, that MDD researchers may benefit by considering an alternative theoretical framework that incorporates *all* levels of analysis: genetics, biochemistry, neurobiology, cognition, emotion, and social relationships.

We explore the clinical utility of one such framework, evolutionary theory, and a key concept, adaptation, for understanding MDD. Adaptations, also known as biological functions, are organism traits whose properties are best explained by the positive effects they had on the reproduction of the organism's ancestors over evolutionary time; that is, which evolved by natural selection. They usually exhibit evidence of design to promote the survival and reproduction of the organism.[17] Examples include hearts, lungs, ovaries, the immune system, vision, and physical pain. Most evolutionary accounts of MDD reviewed propose some biological function for sadness and low mood, with depressive illness then arising as dysregulated or malignant sadness.[18,19]

It is important to keep in mind that adaptations are typically universal in a species. Thus, when considering the evolved functions of emotions, such as sadness and low mood, the focus is on the expression of these emotions in the day-to-day lives of the entire human species, and not simply their expression in clinical populations.

Abbreviations

AD	antidepressant
DSM	Diagnostic and Statistical Manual of Mental Disorders
MDD	major depressive disorder

We summarize evolutionary theories of sadness and low mood. We discuss implications of these theories for MDD research. Implications for MDD diagnosis, including a debate about the boundary between healthy and pathological sadness and low mood, are also discussed. In part, this debate turns on the correct explanation of MDD's costliest symptoms, such as prolonged anhedonia and suicidality. If these symptoms are pathological, as virtually all mental health researchers believe, this solidifies the case that MDD is pathological. However, if these symptoms turn out to have evolved functions, then, contrary to the consensus in the mental health sciences, much MDD may be a healthy response to adversity. Therefore, we review 2 theories that propose evolved functions for prolonged anhedonia and suicidality.

What Are the Evolved Functions of Sadness and Low Mood?

Adversity is strongly associated with, and is probably a cause of, MDD,[20–22] but adversity more commonly triggers healthy, adaptive psychological responses, such as sadness and low mood. Therefore, most evolutionary theories of MDD first consider how sadness and low mood could be adaptations to adversity—that is, how they could provide benefits to people experiencing adversity—and then consider how MDD arises as dysregulated or malignant sadness.[18,19]

The evolutionary theories of sadness and low mood discussed in this section draw primarily on hedonism, a venerable idea that, in its motivational or psychological variant, holds that people seek pleasure and avoid pain, and in its normative variant, holds that only pleasure and pain have value and disvalue, respectively. Developed by ancient Greek philosophers, hedonic principles formed an important part of the philosophies of later British Enlightenment thinkers, were a foundation of Freudian psychoanalysis, and are a basic assumption across much of psychology and neurobiology.[23,24] Most evolutionary theories of MDD are grounded in evolutionary theories of psychic pain or distress as a motivator.

A second core idea, that adversity elicits cries for help, has played an important role in theories of pain, distress, and psychopathology, especially MDD and suicide.[25–27] This idea is especially important for theories that propose evolved functions for even the costliest symptoms of MDD, such as prolonged anhedonia and suicidality.

These 2 ideas suggest that evolved responses to adversity comprise both internal signals influencing one's own behaviour, and external signals, such as facial expressions, cries, and perhaps suicidal behaviour and prolonged anhedonia, that are meant to influence others' behaviour.

Darwin and Bowlby, Attachment and Loss

For Darwin, the evolution of sadness was rooted in the mother–infant relationship—in numerous species, infant cries functioned to elicit care from the mother. However,

Darwin focused less on the function of sadness than on the function of associated facial expressions, such as the firm closing of the eyelids when crying (to protect the eyes from being too much gorged with blood, according to Darwin).[28]

By far the most influential evolutionary theorist of sadness has been Bowlby,[29] who, like Darwin, rooted sadness in the attachment patterns of the infant and its primary caretaker (usually the mother). Anxiety and sadness were evolved reactions to temporary losses, such as separation from the mother, that served to re-establish physical proximity.[29] Grief after the death of a loved one, which obviously could not serve to re-establish proximity, was then a costly byproduct of evolved attachment mechanisms[30] (but see Nesse[31,32]).

According to Bowlby,[29] MDD is maladaptive sadness or mourning (see also Henderson[27] and Henderson et al[33]), an idea expressed by Western thinkers dating back to Hippocrates.[34] Bowlby closely aligned his views of MDD with those of Beck,[35] who argued that MDD resulted from distorted cognitions about the self. Bowlby and his followers rooted those cognitive distortions in childhood attachment problems.

Evidence for Bowlby's hypothesis is mixed. Childhood adversity, which includes death of a parent, physical and sexual abuse, parental drug abuse and psychopathology, and severe illness, is strongly associated with adult psychopathology. However, parental losses, such as death or divorce, have, at best, a small-to-modest association with adult mood disorder, specifically.[36]

Dominance and Submission

Ethological studies of dominance hierarchies, which noted similarities in the demeanour of animals that had lost rank and depressed people,[37] inspired another approach to MDD. According to the social competition hypothesis,[38,39] MDD is the emotion of submission, an involuntary subordinate strategy functioning to create a subjective sense of incapacity, inhibiting aggression toward higher-ranked people; signal submission; and encourage acceptance of rank loss and promote yielding. Early formulations seemed to argue that MDD itself was functional.[38] Later refinements suggest MDD is a dysfunctioning involuntary subordinate strategy.[40,41]

The cardinal strength of the social competition hypothesis is that MDD is indeed closely associated with powerlessness. A perceived inability to control events—variously termed external locus-of-control,[42–44] helplessness or hopelessness,[45] entrapment,[46] or defeat[47–50]—is clearly implicated in MDD.

However, at least 3 questions can be raised about the social competition hypothesis. First, it has little to say about suicidality as either a dysfunctional or functional aspect of MDD. Why would submission to dominants, which protects a person from harm, be associated with thoughts of killing oneself?

Second, harm is best avoided by yielding quickly, so why would depressive episodes often last for months? According to the social competition hypothesis, prolonged depressed mood is either a pathological failure to yield,[41] or a consequence of conflict within the triune brain.[51] For instance, if the so-called rational neomammalian complex refuses to stop competing, the reptilian complex compels yielding via MDD.[39,40] The triune brain model is controversial, though, and flatly rejected by some textbooks[52,53] (for a defence of McLean,[51] see Cory and Gardner[54]).

Contrary to the social competition hypothesis, the delay in yielding may be due to genuine uncertainty about the best course of action. Choosing between continued competition and yielding requires additional information about, for example, potential allies and competitors' abilities, and prolonged low mood functions to collect and analyze that information, an hypothesis revisited later in my article.

A final question regarding the social competition hypothesis is why MDD occurs in response to adversity that is seemingly unrelated to defeat, such as death of a loved one. One plausible answer is that, in humans and many other primates, the loss of an ally often entails a loss of rank.[55] A second answer has been to integrate the social competition hypothesis with Bowlby's attachment theory of MDD, which highlights loss.[41,56]

Social Risk Hypothesis

The social risk hypothesis for MDD[57] was inspired by work on risk-sensitive foraging.[58–60] Consider an animal with an energy reserve threshold, below which it will not survive the cold night, facing a choice of foraging in a patch of either blueberries or strawberries, both with the same mean energy return

$$\mu_{blue} = \mu_{straw}$$

but with different daily variations in return, that is, risk

$$\sigma_{blue} < \sigma_{straw}$$

If energy reserves are well above the threshold, then either patch would suffice. However, if they are only slightly above the threshold, then choosing high-variation strawberries means a higher risk of not finding enough to survive the night, so the animal should be risk-averse, choosing blueberries.

Allen and Badcock[57] similarly argue that people with successful social relationships can tolerate some social risk-taking. However, people with poor social relations cannot, and their low mood reduces social risk-taking, reducing the threat of ostracism. Allen and Badcock[57] marshal considerable evidence that MDD is caused by social problems and associated with perceived low social value (low self-esteem), attentional biases toward social threats, lessened expectations of success, and heightened expectations of failure.

One challenge to the social risk hypothesis is that the literature on risk-sensitive foraging predicts people in poor condition will often be risk-seeking. Reconsider the berry patch: an animal well below the energy reserve threshold would not survive the night if it foraged on low-variance blueberries because the mean return would be insufficient. Instead, it should forage on high-variance strawberries because this provides at least some chance of exceeding the reserve threshold. A mathematical model of MDD inspired by this logic predicts risk aversion for people close to, but above, a critical threshold, and risk-seeking for those below it, a refinement that may illuminate manic states, which can be triggered by adversity.[61]

Although Allen and Badcock[57] make a strong case that low mood is a response to social adversity and heightens social vigilance, the case is weaker for a reduction in social risk, per se. Indeed, there is overwhelming evidence that depressed people work less and parent less,[62–67] which would seem to reduce the mean payoffs of these critical social relationships (μ) rather than reducing their variation (σ); this fact may require incorporating a trade-off between risk and return[68,69] into the theory.

The evidence Allen and Badcock[57] present is also consistent with other theories that explain sadness and low mood as functional responses to social adversity. Their own comparison of the social risk hypothesis to the social competition hypothesis notes few differences. The social risk hypothesis also does not explain why suicidality, a high-risk behaviour, emerges as either a function or a dysfunction of a risk-aversion mechanism, although, as we shall see, the risk-sensitive foraging literature provides a clue. Finally, sadness and low mood are painful. Physical pain, which we discuss next, certainly encourages cautious behaviour, but it appears to have other important functions, such as threat avoidance and learning, and the same is likely to be true of psychic pain.

Psychic Pain Hypothesis

Other researchers see sadness and low mood serving functions analogous to physical pain, which, though aversive, informs an animal of injury, indicates its location, motivates withdrawal from the source of injury, discourages activity to promote healing, and conditions avoidance of similar circumstances in the future. According to the psychic pain hypothesis, sadness and low mood have similar evolved functions, except in response to social adversity rather than physical injury.[70–75]

The relatively few empirical tests of the psychic pain hypothesis have focused on predicted relationships between socioecological conditions and the experience of psychic pain. One controversial group of studies examined the severity of psychic pain following rape, finding, for instance, that reproductive-aged women suffered more from rape than nonreproductive-aged women, predicted because reproductive-aged women are exposed to unwanted pregnancy in addition to physical injury, social stigma, and other harmful consequences of rape.[74,76–78]

Another test involved emotional reactions to newborns. Because there is a trade-off between investing in new offspring and investing in existing or future offspring, mothers should not always invest in new offspring.[79] Hagen[80–82] and Thornhill and Furlow[83] independently realized that postpartum depression may not be a simple endocrine problem, as commonly believed,[84] but instead functions to reduce investment in offspring who, during human evolution, would have been unlikely to survive to adulthood. In support, postpartum depression is probably caused by low social support, which reduces offspring viability; mothers with postpartum depression reduce investment in the baby and often do not love or want it, and even have thoughts of harming it; and remission is associated with improved social support. All this suggests that mothers are modulating investment in the new offspring in response to predicted socioecological conditions.[80–83,85]

However, the psychic pain hypothesis does not explain suicidality, and its proponents disagree on whether MDD itself is functional or dysfunctional.

Integration of Psychic Pain and the Psychology of Affect

Nesse integrates the psychic pain hypothesis with important literature on the psychology of affect that holds that basic emotions, such as sadness and joy, are best viewed as regions in an underlying 2-dimensional affective space whose axes are labelled valence and arousal[86] or, alternatively, positive affect and negative affect.[87] Positive affect is thought to subserve an evolved behavioural activation system that promotes an approach toward natural rewards, such as food and sex. Negative affect subserves a distinct behavioural inhibition system that promotes avoidance of potential threats and dangers,[88–92] similar to the psychic pain hypothesis. In the synthesis of Nesse[71,93–95] and Nesse and Ellsworth,[96] high mood promotes profitable investment and risk-taking in propitious circumstances, whereas low mood disengages effort and motivates consideration of other ways to reach the goal in unpropitious circumstances, an idea Nesse traces to Klinger.[91]

Keller and Nesse[97] move beyond most theories discussed so far in recognizing that different types of adversity may call for different affective responses, which they term the situation–symptom congruence hypothesis (see also Higgins [98]). Keller and Nesse[97,99] found that crying, which may be advantageous in the wake of a social loss, was prominent following the death of a loved one or romantic loss, whereas pessimism and fatigue, which would conserve resources and decrease initiative, were prominent in response to failure, stress, and wintertime. An analysis of data from the Virginia Adult Twin Study found broadly similar patterns.[100]

For Nesse,[95] prolonged depressive symptoms often result from the pathological pursuit of unreachable goals, akin to

the pathological failure to yield in the social competition hypothesis. However, refusal to let go is not due to conflicts in the triune brain but to dysregulation of low mood, which, in turn, has various possible causes, including mismatches with the modern environment, infection, autoimmune reactions, constraints, trade-offs, and inherent properties of human social life that often trap people in fruitless endeavours.[95]

Despite its strengths, Nesse's theory shares some of the weaknesses of the social competition hypothesis (which can be viewed as a special case of Nesse's theory). For example, it does not explain the association of MDD and suicidality.

Implications for MDD Research

The possibility that MDD is dysregulated sadness or low mood has at least 2 important implications for MDD research. First, MDD research should focus much more heavily on understanding healthy sadness and low mood. After all, how is it possible to understand dysregulated sadness and low mood if sadness and low mood themselves are poorly understood?

The second, related implication is about the choice of an appropriate healthy control group. To understand disease etiology, medical researchers often compare people with disease with healthy control subjects. Important differences between the 2 groups provide insights into etiology. If, as many evolutionary theorists suspect, sadness and low mood are healthy responses to adversity, and MDD is dysfunctional sadness, then the appropriate control subjects for MDD research would usually be either people experiencing sadness and low mood, or matched on recent exposure to adversity. Comparison of depressed people with sad people could reveal the essential differences in unhealthy and healthy responses to adversity.

Unfortunately, control subjects in MDD research almost always comprise nondepressed people, most of whom are not sad and, as a group, have not recently experienced much adversity. For instance, a review of 7 community-based studies of MDD onset in women found that whereas the percentage of those with MDD preceded by at least 1 adverse event ranged from 61% to 94%, among those without MDD, the percentage exposed to at least 1 adverse life event during a comparable time period was only 25% to 36%.[20] In studies using nondepressed people as control subjects, MDD is confounded with adversity.

Adversity is a common experience for most members of most species, including humans. Hence there are likely to be numerous, adaptive, healthy responses to it, and these responses would likely involve multiple and substantial changes in the brain and body. Significant differences in the neurophysiology of depressed and nondepressed people, such as mean differences in hypothalamic–pituitary–adrenal axis function, hippocampal size, or brain-derived neurotrophic factor, could therefore be related to MDD[6] or could be due to myriad healthy neurobiological, psychological, and behavioural responses to adversity.

Implications for MDD Diagnosis: The High Rate Problem

The lifetime prevalences of severe mental illnesses, such as schizophrenia, autism, and bipolar disorder, are about 1 in 100 or less.[101–103] Conversely, lifetime prevalence of MDD for US women is more than 1 in 5, and for middle-aged women, more than 1 in 4.[104] What insights does evolutionary theory provide for such extraordinarily high rates of a severe brain disease?

Evolutionary Explanations for the High Rate of MDD

High rates of illness can be caused by infections, physical injury, unavoidable byproducts of adaptations (such as damage caused by healthy inflammation), mismatches between the current environment and the environment in which humans evolved (which may explain, for example, high rates of allergy), and old age.

However, there is little evidence that most MDD is a consequence of infection or physical injury, and it is, if anything, more common in young adults,[104] leaving as possibilities unavoidable byproducts, for example, of inflammatory responses to stress-induced neural microdamage,[105] mismatches,[106] or some combination of all of these.[95] Nettle[107] proposed a special case of the byproduct hypothesis. Humans have complex affective systems, based on many genes. Genetic variation in traits involving many genes can be maintained despite strong selection (see also Keller and Miller[108]). People in the tail of the resulting distribution in emotional reactivity (neuroticism) may be vulnerable to affective disorders.

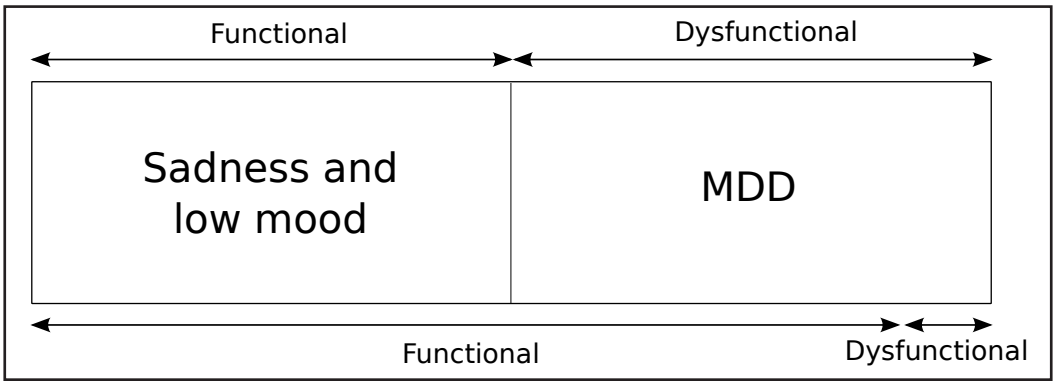
A High Rate of False Positives?

Alternatively, the current means of diagnosing MDD may produce many false positives, explaining the high rate. Although all evolutionary theorists of MDD agree that some MDD is pathological—after all, any mechanism can malfunction—a few believe that most cases of MDD in community samples are actually healthy responses to adversity (Figure 1).[34,81,109–113]

To make their case, these scholars argue, first, that the severity of an emotional response, per se, is not evidence of psychopathology. Severe physical pain in response to severe physical injury, though highly aversive and disabling, is ultimately beneficial. Common precipitants of MDD, such as mate loss, are as serious a threat to fitness as a severe physical injury, thus a severe yet functional negative emotional response to, for example, divorce, is unsurprising.

Horwitz and Wakefield[34] continue this line of reasoning with an historical analysis showing that, from antiquity to modern times, clinicians did not consider severe

Figure 1 Two views of the boundary between function and dysfunction



All evolutionary theorists agree that sadness and low mood are probably adaptations. Most see MDD as a dysfunction of sadness and low mood (top). Some, however, argue that much MDD is also functional (bottom). Because all mechanisms can dysfunction, all agree, though, that at least some MDD is dysfunctional.

depressive symptoms, in and of themselves, to indicate psychopathology, instead weighing symptoms against patients' circumstances. Psychopathology was diagnosed only when symptoms occurred without cause, or were disproportionate to circumstances in severity or duration.

A major change occurred in the 1970s. In the service of reliability, the Feighner et al criteria[114] and the Research Diagnostic Criteria,[115] crystalized in the DSM-III, diagnosed MDD by symptoms only, ignoring patient circumstances, with bereavement the only exception. Severe depressive symptoms lasting more than 2 weeks in relation to, for example, divorce or job loss were now, by definition, pathological. When applied to community populations in large epidemiologic surveys, these criteria revealed surprisingly high rates of psychopathology, raising concerns of false positives[7,8] (a clinical significance criterion was introduced in DSM-IV to reduce false positives by requiring that symptoms cause significant distress or impairment[8]).

To encourage consideration of patient circumstances in the upcoming definition of MDD in DSM-5, Wakefield et al[116] showed, first, that, in uncomplicated cases, the symptom profiles of patients following bereavement are very similar to symptom profiles of those following other losses. Hence, for patients with uncomplicated cases, the bereavement exclusion should be extended to other losses.[116] Second, they criticized the clinical significance criterion by showing that almost everyone with persistent sadness, even in the absence of MDD, is distressed and (or) impaired. The criterion is therefore redundant and cannot reduce false positives.[8,117]

Kendler[118] argues the opposite point: because there is little-to-no difference between MDD following the death of a loved one and other losses, the bereavement exclusion should instead be eliminated from DSM-5. On this view, the constellation of symptoms currently described as MDD is never a normal or healthy response to even severe adversity.

The debate between Wakefield et al[116] and Kendler[118] turns, in part, on whether common but severe symptoms of MDD, such as prolonged loss of interest in virtually all activities and suicidality, could be functional in certain severe circumstances. If so, this strengthens the case for consideration of patient circumstances in the diagnosis of MDD.

Are Prolonged Anhedonia and Suicidality Ever Healthy? (And Why Does It Matter?)

The final theories reviewed here seek benefits of prolonged anhedonia and suicidality that may outweigh their obvious costs.

The Analytical Rumination Hypothesis

In the analytical rumination hypothesis, a refinement of the psychic pain hypothesis, Andrews and Thomson[110] propose that MDD is triggered specifically by complex problems. Choosing the best course of action requires extensive analysis of the problem, diverting time and cognitive resources away from daily activities. What seems to be a pathological loss of interest in virtually all activities is instead a beneficially single-minded focus on, for example, marital problems at the expense of housecleaning, grooming, eating, and sleeping.

In support, Andrews and Thomson[110] note that people with MDD ruminate extensively about their life problems, which they perceive to be complex, severe, and difficult to solve, and that MDD promotes an analytical style of reasoning. Studies seeming to document cognitive deficits in people with MDD erroneously compared their performance to control subjects, that is, those with little recent exposure to adversity. It is not surprising that, for example, a wife whose husband had just died would do worse on abstract reasoning tasks. According to Andrews and Thomson,[110] not one study has examined whether depressive rumination improves responses to the problem that triggered the MDD. Andrews and Thomson[110] propose that it does, but convincing evidence of this is currently lacking.

A Credible Cry for Help, a Demand for Help

To successfully respond to adversity, people often need help from others. It has frequently been suggested that MDD, deliberate self-harm, and suicidality are cries for help, albeit pathological cries, because of their costs.[25–27] Hagen[80–82,109] and Watson and Andrews[113] independently proposed, instead, that costly cries are functional for those enmeshed in interpersonal conflicts.

Evolutionary biologists and economists have long recognized that when senders and receivers have conflicts of interest, and senders therefore have an incentive to deceive, costs can ensure the credibility of signals.[119,120] Applied to MDD and suicidality, the logic is that when life is going well, prolonged MDD or suicide attempts would be very costly because they would interfere with work, marriage, child rearing, and so on, as indeed they do.[62–67] Although nonneedy people may benefit by deceptively signalling need, they cannot afford to send the signal, that is, be depressed or attempt suicide.

For people whose work, marriage, or child rearing are not going well, MDD and suicidality impose few costs because there is little to lose, so they can afford to send such signals. Hence when social partners observe MDD or a suicide attempt they can be confident that the sufferer is genuinely needy, even if interpersonal conflicts would lead them to be skeptical.

Suicide attempts, in particular, are gambles—needy people win if they survive and consequently receive more help; they lose if they actual kill themselves.[121,122] This risky strategy may only be open to people below some critical threshold, as suggested by the risk-sensitive foraging literature. Important for this hypothesis, most suicide attempts fail: globally, there are more than 14 attempts for every completion[123]; for young adult US women, there are more than 100.[124]

Because social partners respond negatively to MDD and deliberate self-harm,[125–127] credible signalling cannot be the whole story. In interdependent relationships, such as work and marriage, where social partners depend on one another to produce shared benefits, a person who harms him- or herself is also harming his or her partners, which motivates them to respond. MDD and suicidality are, at once, credible cries and demands for help, a strategy referred to as bargaining with incomplete information.[128,129]

In this light, it is not surprising that MDD is aversive to social partners. Infant cries, which are obviously evolved signals of need, are aversive.[130] So, too, are internal signals of need—physical and psychic pain. Aversiveness is probably required to motivate otherwise disfavoured behaviours.

In support of these hypotheses, those who become depressed or suicidal in response to adversity are characterized by powerlessness,[42–50] and thus plausibly require help, yet are embroiled in interpersonal conflicts,[131–138] which would inhibit help. Despite these conflicts, social partners provide help in response to depressive behaviours,[134,139–144] so much so that many researchers worry these benefits reinforce MDD.[134,135,139,140,145,146] Cross-culturally, much (but not all) suicidality is a response to conflict that often elicits benefits.[147]

The primary weakness of these hypotheses is that although remission of MDD is associated with important life improvements,[148] there is as yet no evidence that depressive symptoms themselves bring about life improvements, as these hypotheses require.

Avoiding a Brave New World

Depression is a response to adversity. If (and when) it is a dysfunctional response then ADs are therapeutic. However, if much MDD is a functional response to adversity, as some evolutionary theorists believe, suppressing it could be harmful. ADs do interfere with an adaptive stress response in at least one species. Guinea pigs pups, which possess central serotonin (5-HT_{1D}) receptors similar to humans, cry when separated from their mother. ADs suppress these separation cries, interfering with a clearly adaptive stress response. In fact, potential AD compounds are screened precisely for their ability to suppress these functional cries for help.[149,150] It is conceivable that, in humans, ADs are also suppressing a functional cry for help. Unfortunately, there is little ongoing effort in mental health science to determine whether MDD has any such benefits that outweigh its manifest costs.

In summary, evolutionary approaches to MDD, although still in their infancy, could transform research on this affliction. If, as most evolutionary theories propose, MDD is dysfunctional sadness and low mood, then MDD research should focus much more heavily on understanding healthy sadness and low mood, control subjects should be people experiencing sadness or low mood (or matched on recent life events), and the goal of chemo- and psychotherapy should be to restore healthy sadness and low mood, not simply to reduce MDD symptoms. Conversely, if much MDD is actually functional, then MDD, like aggression or grief, is largely a social problem, not a medical problem.

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Résumé : Théories évolutionnaires de la dépression : une revue critique

Nous effectuons une revue critique des théories évolutionnaires du trouble dépressif majeur (TDM). Parce que tous les cas de TDM semblent être causés par l'adversité, les théories évolutionnaires du TDM proposent généralement que la tristesse et l'humeur sombre aient évolué au titre de réponses avantageuses à l'adversité, et que le TDM soit une tristesse et une humeur sombre dysfonctionnelles. S'il en est ainsi, la recherche sur le TDM devrait porter beaucoup plus sur la compréhension des fonctions saines de la tristesse et de l'humeur sombre, pour mieux comprendre comment elles deviennent dysfonctionnelles. Un débat sur la frontière entre une saine tristesse et le TDM est ensuite étudié. Ce débat dévie partiellement sur la question de savoir si les symptômes les plus pénibles du TDM pourraient procurer des avantages inconnus. Par conséquent, la revue conclut en discutant de 2 théories qui explorent les avantages possibles de l'anhédonie prolongée et de la suicidabilité.