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Review

Darwinian depression

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ABSTRACT

Background: The standard evolutionary explanation for depression is that being in an emotionally depressed state is adaptive.
Method: The article first undertakes a critical review of the extant literature. It then provides an alternative evolutionary explanation for event-based depression and elation. It argues that being in a depressed state is not adaptive (indeed, quite the opposite), but that the threat of depression for bad outcomes and the promise of pleasure for good outcomes are adaptive because they motivate the individual toward undertaking effort that increases fitness. The article then explains reasons for failure in the motivation system and the mood disorders that arise as a consequence.
Results: The article explains why motivation depends on both elation and depression and why individual happiness is not permanently improved by winning the lottery (or permanently reduced by becoming wheelchair bound). It explains the comorbidity of bipolar disorder and panic disorder, why mood stabilizers tend to reduce motivation, and when anti-depressants are unlikely to cure "laziness."
Limitations: The evolutionary explanation for depression does not directly provide clinical criteria for determining when major depressive disorder is present nor has it yet provided new treatment strategies for mood disorders.
Conclusions: The theory presented here provides a coherent explanation for depression and elation and leads research in a different direction from previous evolutionary explanations.

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1. Background

The widespread existence of depression across cultures is a puzzle for evolutionary theory. The prevalence of any trait, especially one that appears during reproductive years, should increase fitness. But the listlessness that often accompanies depression appears to be anything but adaptive. A number of authors have proposed a solution to this conundrum by arguing that being in a depressed state helps the individual to solve problems. Watson and Andrews (2002) and Andrews and Thomson (2009) argue that depression allows one to focus on the problem at hand, thereby enabling the depressed person to find good solutions. Hagen (2003) argues that depression signals that the person needs help, which increases reproductive advantage by shifting the burden from the depressed person to others. Stevens and Price (2000) provide a different signaling explanation. They argue that the submissiveness in depression signals to others of higher rank that the depressed person is not a threat to them, thereby preventing the depressed person from engaging in costly challenges to dominant figures. More generally, Nesse (2000) argues that being in a depressed state is useful in that it discourages individuals from undertaking risky behavior when the payoff is likely to be negative.[1]

This article argues that *being* in a depressed state is not adaptive (indeed, quite the opposite), but that the *threat* of depression for bad fitness outcomes and the *promise* of pleasure for good outcomes are adaptive because they motivate the individual toward undertaking effort that increases fitness. The threat of physical pain discourages people from putting their hands too close to a fire; the threat of emotional pain encourages individuals to undertake action that reduces the likelihood of depression, and the promise of emotional reward encourages people to undertake action that increases the likelihood of elation.

This article is neither the first to suggest the parallel between physical pain and emotional pain nor the first to argue that emotions motivate effort towards greater survival. Spencer (1870, 2012, p. 280) was an early proponent of this view.[2] More recently, Thornhill and Thornhill (1989) and Nesse (2000) also use the analogy. But Thornhill and Thornhill concentrate their chapter on what outcomes are most likely to bring on depression (those that are most closely associated with reduced fitness, such as a child dying) and Nesse devotes only a couple of sentences on the analogy before going on to the other explanations mentioned in the introductory remarks. More striking is the fact that the aforementioned surveys of evolution and depression by Alan and Badcock and Gilbert never consider the possibility that the threat of depression could improve fitness. In sum, the literature neither develops a comprehensive theory of depression as a deterrent nor considers its relationship to elation.

This article is organized along the following lines: Section 2 argues against the view that being in a depressed state enables the individual to solve social and psychological problems. Section 3 develops the incentive theory of depression that individuals in normal states discover solutions that reduce the likelihood of bad fitness outcomes (depression) and increase the likelihood of good outcomes (elation). Section 4 considers the possibility that depression and suicide are ways that the individual improves the welfare of kin. And Section 5 takes a critical look at the view that depression fights disease.

The word "depression" has been used to refer to a wide variety symptoms, including grief, major depressive disorder, and so on. Here, depression is divided into two main categories: event-based depression (including grief and sadness) covered in Section 3, and major mood disorders, (including chronic depression, bipolar disorder, and dysthymia) covered in Section 6. Finally Section 7 considers the close relationship between anxiety disorders and depression that is explained by the incentive theory of depression and elation.

2. Does being depressed solve problems

The Social Navigation Hypothesis and related hypotheses argue that being in a depressed state helps the individual to solve social and psychological problems.

2.1. Is depression a time for rumination

To set the stage, it is useful to start off with two quotes from Watson and Andrews (2002, p. 1, 4). "[D]epression induces cognitive changes that focus and enhance capacities for the accurate analysis and solution of key social problems, suggesting a social rumination function." "[T]he inability to feel pleasure may help the depressive to sustain cognitive effort on the problem by preventing cognitive distractions."

An important difference between the social rumination hypothesis just quoted and the argument made in this article that people try to avoid depression is when *cognitive* solutions are *formulated*. Consider first physical pain and pleasure. If someone has tripped on a root and has hurt his or her leg, pain provides immediate feedback as to which movements, if any, are less painful, and, more important, which movements to avoid. However, when one is in pain, one does not really think about how to avoid such accidents in the future; instead, one is focused on the pain, itself. Once healed the person will undertake a number of cognitive tasks to avoid future pain. The person might not walk at night in order to avoid tripping on roots in the dark, might take a stick along to steady him or herself to avoid slipping on rocks, and might avoid certain routes where wild animals might attack. Similarly, sexual pleasure provides immediate feedback as to which movements are more pleasurable. However, one does not use these moments to figure out how to find sexual partners in the future. Rather, the cognitive task of finding sexual partners is done at other times in the hopes of gaining future pleasure. Similarly, when one is depressed, one is primarily aware of the emotional feelings and is not using that time to develop plans as Andrews and Thomson (2009), have argued. When people are in the throes of depression and feeling unwanted, unloved and/or unlovable, they are not coming up with methods of meeting and attracting potential

[1] There are many variations on the theme that being in a depressed state is adaptive for the individual. For surveys, see Gilbert (2005) Allen and Badcock (2006) and Nesse (2009).

[2] "In other words, those races of beings only can have survived in which, on the average agreeable or desired feelings went along with activities conducive to the maintenance of life, while disagreeable and habitually avoided feelings went along with activities directly or indirectly destructive of life: and there must ever have been, other things equal, the most numerous and long-continued survivals among races in which these adjustments of feeling to action were the best …"

partners. Instead they have a broken record of their failures and the hopelessness of their situation. In sum, people use less emotionally charged times to anticipate what might bring them emotional pleasure or pain and plan accordingly. Furthermore, because of either built in circuitry or the ability to generalize and imagine, one does not have to experience pain (depression) from some particular outcome to know that that the outcome will produce physical or emotional pain. A person might never have been harmed by an animal and still know that a rhino could be dangerous and produce pain; and a person may never have had a loved one die and still know that he or she would be depressed if this were to happen.

The argument here is that the amount of thinking that goes on while being depressed is *at best* only a very small fraction of the amount of rumination that goes on to prevent depression. Let us consider the care of children. Because of the elation that children bring, we undertake all kinds of rumination and behavior to prevent their demise. We do not need their demise to inform us that we would be despondent if they were to die. Every day we ruminate about how to protect our children so that they will grow into adulthood. We make sure that they get enough to eat and that they are in a safe place. We ruminate about possible dangers and find ways to mitigate them. We learn from near misses and respond to threats. We do not need to be depressed from the death of a child in order for us to ruminate. Suppose that your child is playing near a cliff and almost falls over. You learn from that experience even though there is no depression associated with a near miss. If your child dies, that is a harder lesson. If she were your only child, it would have been better to anticipate or at least learn from previous near misses. Indeed, a threat to one's happiness, such as impending danger, is what motivates people to ruminate and act. But that is motivation based on the *threat* of depression and the *promise* of elation, which is the argument made here. In sum, it is more fitness enhancing to be proactive than having to engage in postmortems.

When a person is depressed, there is increased rumination, but it is a rumination of a destructive kind, such as dwelling on suicide or on how his or her life is without meaning. Indeed, the weight of the scientific evidence suggests (1) that depressed people are poor at finding solutions to their interpersonal problems and (2) that rumination by dysphoric subjects interferes with problem-solving! Lyubomirsky et al. (1999) and Lyubomirsky and Nolan-Hoeksema (1995) show that inducing dysphoric participants to ruminate results in their seeing their problems as unsolvable and in their failing to come up with effective solutions.[3] Furthermore, Just and Alloy (1997) and Sarin et al. (2005) show that people who engage in rumination when distressed have more prolonged periods of depression.

2.2. Gaining help from others

The second prong to Watson and Andrew's Social Navigation Hypothesis is the argument that depression enables the depressed person to gain help from others (also see Hagen, 2003).

It is true that depressed people get help from those close to them just as people who have broken their leg receive help. But this help mitigates a loss of fitness, not enhances it. Breaking one's leg reduces the joint productivity of the household. Being depressed also reduces the joint productivity of the household because the healthy spouse can only partially make up for the loss of energy by the depressed person. It is the precaution undertaken to avoid breaking one's leg that is fitness enhancing, not the broken leg. It is the precaution undertaken to avoid depression that is fitness enhancing, not the depression, itself.

In general, any benefits that come from being depressed must be *limited.* Otherwise, the benefits would interfere with the deterrent effect of depression. Any adaptationist argument that *being* depressed is helpful runs up against this problem. Despite the very clever and thoughtful arguments by those who find silver linings in the cloud of being depressed, these silver linings can at best be of secondary importance. If being depressed on average made things *sufficiently* better for the person, then it would not serve as a deterrent and, counter to what we observe, people might desire to be depressed as this would make them better off in the future. Depression would then be seen as a form of investment, where costs today are more than made up by increased benefits in the future.

2.3. Does depression keep people out of trouble

Another variation on the theme that being depressed is adaptive is found in the work of Stevens and Price (2000), who argue that being depressed keeps low status people out of trouble because depressed people do not challenge people of higher rank. There is clearly a positive correlation between depression and low status. In general, low status people are more likely to have suffered a bad outcome such as reduced status or chronic debilitating illness. Low status people also die at younger age. And so it is not surprising that low status people and/or their partners are also more likely to be depressed. Indeed, later it is argued that status may be an important determiner of mood. Nevertheless, one might still wonder whether depression is a good way to stay out of trouble as Stevens and Price have argued. The problem is that the passiveness of depression also makes one less likely to resist any challenge, whether the challenge is from people of equal or lower status trying to take advantage of the depressed person or the challenge is from the natural world.

A more convincing argument is found in Nesse (2000). He argues that depression discourages people from undertaking risky behavior when it is not propitious to do so; and on the other side of the emotional spectrum, elation encourages people to undertake risky behavior in good times. The problem is that being depressed is a blunt instrument. One can imagine bad outcomes when the best thing to do is to lay low; but there are other times when the best thing is to take a risk (e.g., crossing dangerous rapids with a child in one's arms to avoid being eaten by a predator after it has attacked and killed another child). In contrast the threat of depression engenders a malleable response depending on the nature of the threat.

3. Depression and elation as incentive devices

The explanations presented at the beginning of this essay only focus on depression, not on the whole picture. By looking only at depression and not its incentive effect on behavior when the person is not depressed, these explanations miss the underlying evolutionary rationale for the punishment and reward system. It is something akin to looking at people in prison and arguing either that they are being rehabilitated to become more productive members of society (prison, like depression, is good for you) or that having prisons is bad because prisoners are not productive while being incarcerated. These arguments, like the arguments that only focus on people in depressed states, ignore the incentive effects of imprisonment (depression) and the possibility that

[3] See Nettle (2004) and Nolen-Hoeksema et al. (2008) for a more detailed review of the research on rumination that counters much of the evidence marshaled by Watson and Andrews (2002). Also see the literature review by Kircanski et al. (2012, p. 309), who state the following: "the cognitive biases and deficits that reliably characterize depression influence maladaptive patterns of emotion dysregulation in this disorder." For evidence that supports the social rumination hypothesis see Andrews and Thomson (2009).

some, but not all, will be deterred from committing a crime (or from underperforming) in the first place. To continue with the prison analogy, just as it is rational to imprison criminals if the benefit of reduced crime is greater than the cost of putting people in prison, it is rational for the gene to inflict costly punishments (depression) if the increase in fitness that is induced by the threat of depression is greater than the reduced fitness from the depression itself that occurs when there are low fitness outcomes (either because of bad luck or insufficient effort on the part of the individual).

And all the evolutionary explanations for depression mentioned in the opening paragraph miss the other half of the incentive system – elation.[4] The possibility of elation incentivizes individual behavior toward increased reproductive fitness just as the threat of depression incentivizes individual behavior away from outcomes that reduce reproductive fitness.

3.1. Motivation

Natural selection means that those individuals who are motivated to undertake behavior that increases fitness will become more prevalent than those individuals who are not so motivated. In turn, individuals are motivated by a system of punishments and rewards. Some of these feedback mechanisms are almost completely hardwired (withdraw hand from the hot stone). Other feedback mechanisms, such as depression and elation, do not arise because pain receptors are stimulated. Instead, certain cognitive connections are made so that less desirable outcomes result in depression, while more desirable outcomes result in elation.

One can remain agnostic concerning which elements lead to reproductive success, but the analysis is easiest to comprehend if status is viewed as a key component of fitness in human societies. Those who are of higher status will gain more mates if they are male and more desirable mates if they are female and both will have more resources to increase the probability of their offspring surviving. Depending on circumstances, status may depend on strength, intelligences, knowledge, bravery, etc. In turn, many of these depend on the motivation of the individual. Physical skill depends not only on inherited muscular and skeletal traits, but also on training. The acquisition of knowledge depends not only on inherited intellect, but also on the time invested in learning. In turn, training and learning depend to a great degree on motivation. If a person has a great desire to be of high status (that is, the person gains great pleasure from high status and endures great pain from being low status), then the person will be motivated to undertake the costs needed to achieve higher status.[5] And so we are back to the role of depression and elation. Finally, the culture determines what it means to be high status.

As argued above, those who are motivated to undertake effort that increases fitness will be more likely to have surviving offspring; in turn, this motivation comes from a system of punishments and rewards. Elation (reward) and depression (punishment) are typically based on outcomes rather than on effort. Parents are depressed when a child dies despite their best efforts. However, punishment and reward can also be based on effort. These incentives are called guilt and pride. Guilt (pride) arises when an individual believes that a bad (good) outcome would not have occurred if the person had acted differently.

3.2. The underlying mechanism

The context in which the individual lives is not fixed but varies over time due to changing cultural and environmental circumstances. And because the environment is not fixed, the incentive system needs to respond to specific cognitive events that change more rapidly than the gene. Hence, the individual's responses to situations is reliably developed, but within the context of specific features of the individual's social ecology. Prehistoric man was not depressed because he did not get into Yale and present-day individuals are not depressed because they did not get into Yale fifteen years ago. Emotions are ultimately designed to respond to changes in fitness, not levels.[6]

To illustrate, suppose that fitness is based on number of surviving children.[7] An increase in period t in the *expected* number of surviving offspring at the end of one's life from three to five will yield greater happiness than maintaining in period $t+1$ the expected number of offspring at the end of one's life at five. In the long run both event-based happiness and event-based depression are fleeting as the person adjusts to new circumstances. Bad outcomes, such as having a leg amputated or being confined to a wheelchair, are initially depressing but generally less so overtime as expectations decrease to match the new reality. On the other side of the ledger, winning the lottery is initially a cause for joy, but the joy does not last because expectations have increased.[8]

3.3. The cost in being either depressed or elated

Just as there is a fitness cost to being in a depressed state (being punished), there can be a fitness cost to being in an ecstatic state (being rewarded). The later is clearly the case when individuals are bipolar and in mania, where the likelihood of suicide and risky behavior is greatly enhanced. Besides the behavioral consequences, there may be direct biological costs involved as abnormal levels of certain chemicals associated with moods, such as cortisol, can have deleterious effects on the body. The question is whether these costs arise when the individual does not have a mood disorder but is responding to various events in the individual's life. To rephrase the question, do the costs of elevated mood rise continuously (possibly at an increasing rate) as the individual's mood becomes more elevated from the normal or do these costs suddenly arise when the person is in mania? The former seems more plausible as the control system is unlikely to be costless, but further research is needed to answer to this question. In both cases, the high fitness costs involved when there is either severe depression or extreme elation means that it would be fitness enhancing for the gene to rely on moderate amounts of both depression and elation rather than employing just one of them as an incentive device.

4. Inclusive fitness and suicide

In a series of papers, de Catanzaro (1980a, 1980b, 1984, 1986, 1991) has argued that suicide and other instances of self-injurious behavior may increase inclusive fitness. In essence, the argument is that when a person's existence reduces the fitness of closely related individuals, suicide may increase inclusive fitness.

[4] The one exception is Nesse (2000), who considers risky undertaking when the individual is elated. See the earlier discussion.

[5] See Stieglitz et al. (forthcoming), who have shown how a decline in functional status and health increases the probability of depression among Amazonian forager-farmers.

[6] It is well established that our sensations are based on relative rather than absolute values. See for example, Kandel et al. (2000).

[7] One could also consider the number of surviving nieces and nephews (appropriately discounted), but that would just make the example more complicated without adding insight.

[8] See Gilbert (2006), for an extended account of this phenomenon.

In "species in which there are no interactions among kin … pressures favoring outright self-destructiveness would never exist. … [I]n highly social species … natural selection could foster … outright self-destructiveness within definable circumstances. … Burdensomeness [to kin] would require no more than the consumption of resources such as nutrients, shelter, and so forth that might otherwise be available to potentially reproducing kin, especially when i's [the individual's] own remaining reproductive potential is low" (de Cantanzo, 1986, p. 171, 172).

In a nutshell, suicide and self-destructive behavior, more generally, are altruistic acts that increase inclusive fitness; and absent an ongoing relationship with kin, there would be no suicide. This is a very clever explanation for suicide and explains the increase in the rate of male suicide across cultures from age 40 or so until old age. And the concept can be used to explain depression, more generally (see Brown et al., 1999). Unfortunately, other data is more contradictory. Consider the following quote from de Catanzaro (1991, p. 24): "[M]ales experiencing longstanding social isolation are an exceptionally high risk group for both accidents and suicide."[9] Longstanding social isolation implies, among other things, that the person either does not have any kin or is not interacting with them. But looking back at the previous paragraph, de Catanzaro argues that when there is no interaction with kin, species would not act self-destructively. Hence, a follower of de Catanzaro's theory would expect that, other things being equal, those who are isolated socially would be less likely to commit suicide than those who are not isolated socially. While both de Catanzaro (1984, p. 81) and Brown et al. (1999, p. 63) show that being a burden on kin increases thoughts of suicide, the socially isolated person is not a burden on kin. So again, one would expect lower rates of suicide among the socially isolated. But the opposite appears to be the case. Of course, the present article has provided reasons why social isolation and/or helplessness and the feeling of being a burden would lead to depression. These are bad fitness outcomes and the threat of depression incentivizes people to undertake actions to avoid them in the first place.

The key to distinguishing de Catanzaro's theory from other theories is to find evidence that is consistent with his theory and not the other theories. Social isolation appears to be the key. de Catanzaro (1986, p. 93) suggests that an aging mother bear whose two cubs are threatened would protect her cubs from a predator even at the risk of her own demise. Presumably, an aging female bear without cubs would avoid the fight. The bear example seems plausible and explains the lengths that human parents might go to in protecting their children. The question is whether this explains the *pattern* of suicide in human societies. But, as already noted, human suicide appears to be more common among the socially isolated.

There is a possible way out of the contradictions posed by the previous paragraphs. Men who are socially isolated are not identical to men who are not socially isolated except for the fact that they are socially isolated. They may be less educated and poorer (these might be controlled for in a multiple regression). They may be prone to self-destructive behavior, which could very well be the cause for the social isolation rather than the result. They may be in great pain and suffering physical debilitation that makes it harder to maintain old relationships or create new ones, especially if they are older. At the same time, the physical debilitation may cause the depression and thoughts of suicide. And making use of de Catanzaro's logic, it is possible that the individual's decision to become socially isolated in the first place was to reduce the burden on kin.

So before de Catanzaro's argument is dismissed, more empirical work needs to be done. In particular, one has to control for the confounding effects discussed in the preceding paragraph. For example, one might determine whether, other things being equal, widowers without kin are less likely than widowers with kin to commit suicide, as de Catanzaro would predict. Unfortunately, de Catanzaro's focus is elsewhere. Hopefully others will take up the gauntlet and undertake more rigorous tests that either confirm or disconfirm his argument.

5. Depression as a defense against infection

Infections appear to be the leading source of mortality throughout human history (see Finch, 2010). The "infection–defense hypothesis" claims that depression reduces both infection and the spread of infection. To the degree that both are true, this could explain the widespread existence of chronic depression, despite its negative consequences outlined elsewhere in this article.

Here is the argument behind the hypothesis. Increased rates of depressive symptoms follow infections (see Murray et al., 2007). Pro-inflammatory cytokines, such as interleukin-1*b* and interferon-*g*, that fight infection, trigger depressive states by affecting the hypothalamic–pituitary–adrenal (HPA) axis. It is therefore not surprising that depressed people often have elevated levels of cytokines (Irwin and Miller, 2007). Furthermore, therapeutic administration of cytokines used for treating people with cancer and a number of infectious diseases leads to major depression in a significant number of patients (see Capuron and Miller, 2004). Depression helps fight infection by resetting "an organism's priorities to adaptively cope with the threat of bodily insult" (Anders et al., 2013, p. 11). According to the infection–defense hypothesis, the characteristics of depression, including listlessness, anhedonia, and *impaired concentration*, represent an organized strategy for fighting infection by conserving metabolic resources and helping the individual to avoid further stressors. Depression also increases social withdrawal, thereby reducing the spread of infection to kin and the likelihood of the individual getting a new infection when so vulnerable.[10]

The infection–defense hypothesis is not necessarily contradictory to the argument made in this article. Being ill reduces expected fitness and thus depression would be a likely result. It would be better to avoid the infection and the depression in the first place. Of course, people do get depressed when infection is not an issue.

At present, the infection–defense hypothesis is not fully developed. Many of the empirical results are based on cross-section studies where longitudinal studies would be more appropriate. There are also certain gaping holes in the argument. As a consequence, some of the "supporting" evidence is contradictory, as can be seen in the following examples.

The rate of depression in children less than five years old is small compared to the rate of depression in adults over seventy. Yet children under five and especially those under two years old are at a high risk for developing infections such as the flu. Old people are the most likely age group to be depressed, but infection is not the most common cause of death, and anyway an older

[9] The social isolation explanation for suicide has been around for a long time. See Durkheim (1951), as well as Linden and Breed (1976) and Meer (1976). For a more recent comprehensive study with similar results, see Qin et al. (2003).

[10] Other explanations for the relationship between depression and infection have been proposed. For example, Raison and Miller (2012) have shown that alleles associated with depression are also associated with known effects on immune function. As another example, Benros et al. (2013) in a study of 3.56 million Danes born between 1945 and 1996, show that hospitalization for infection increases the likelihood for later mood disorders by 62%.

person's contribution to the gene's survival is typically less than a younger person's. To put the depression as infection fighter hypothesis into context, imagine a world where fever were less common in children than in adults and where fever arose in all kinds of situations where there was no infection. If the *only* reason for fever were to fight infection, then under such circumstances the benefits of fever might be outweighed by the costs.

If depression helps to reduce infection, one would expect that depressed people would be less vulnerable to infection. However, a number of studies show that depressed individuals are at increased vulnerability to contracting infections, such as reduced cellular immunity to varicella–zoster (Irwin, 2002). Furthermore, depression appears to reduce NK cell activity (Zorilla et al., 2001; Irwin and Miller, 2007). Both examples suggest that depression weakens the body's ability to fight infection, yet Anders et al. (2013) cite both as supporting the infection–defense hypothesis without explaining the apparent contradiction.

6. Failures in the incentive system

As opposed to the earlier sections where depression and elation are viewed as being adaptive, this section discusses failures in the motivational system such as bipolar disorder and clinical depression.[11] Evolution does not mean that we are born perfect. Not all of us are geniuses and about 30% of children have astigmatism (Kleinstein et al., 2003) even though better eyesight appears to improve fitness; and, with regard to the focus of this article, there may be improper regulation of the motivation system so that there is either hyper or hypo-active implementation of depression and elation.

Going back to the eye, ophthalmologists are not only able to say that certain conditions are suboptimal, but also to explain the impact of the various deviations from the optimal. If there is more curvature in the eye than optimal, then the person will be able to see close objects in focus, but not distant ones and if the curvature of the eye is less than optimal, then the reverse will be the case. In the same way, this article explains how certain results differ when the incentive system is hyperactive as opposed to hypoactive.

6.1. The difficulty in achieving the right balance

The regulation of behavior involves both emotional memory (the recall of past emotional experiences associated with actions and outcomes) and forward thinking (planning) with emotional forecasting (i.e. consideration of potential emotional consequences). Memory and forecasting, when event-based and not too extreme, are part of a healthy system, whereas a chronic mood disorder is a symptom of a feedback system that is not working correctly in some respect. The hedonic feedback system could be not working properly if it is not properly calibrated to goal achievements and failures (so as to produce subsequent problem avoidance and solution-pursuit motivation). A hedonic feedback system could also be not working properly if memories of previously aroused negative or positive affect are not recalled, or not properly appreciated. Likewise, a hedonic feedback system could also be not working properly if it does not simulate and forecast future outcomes or incorrectly forecasts such outcomes (see the later discussion on anxiety and panic disorder).[12] As noted earlier, moods depend on the cognitive understanding of the existing environment, making appropriate regulation more difficult to achieve than when regulation does not involve cognitive connections. It should therefore not be surprising that failures in mood regulation are common.

Evolution must avoid the Scylla of persistent depression, where the person is immobilized and the Charybdis of incessant euphoria, where the person tends to be reckless and subject to substance abuse.[13] And likewise, evolution must avoid the Scylla of being bipolar and the Charybdis of having no changing moods, whatsoever (these will be discussed at further length, below). One can see the difficulty in achieving the right balance by looking at the moodiness of adolescents where the internal monitoring system is a work in progress and there are great emotional swings from reckless euphoria to withdrawn depression.

Because expectations are context dependent, it is much harder to maintain the appropriate emotional equilibrium than producing an appropriately shaped eye, for example. The chemical–biological system needs to provide just the right incentive structure in a changing environment. It should not be surprising that the balance system may itself be out of balance.

6.2. Bipolar disorder

Bipolar I disorder occurs when the person is prone to experience extended periods of extreme euphoria and at other times, extreme depression. While the initial stages of mania are sometimes romanticized, being in a manic state is not adaptive.[14] It is generally the case that manic episodes have a short period of elevated mood followed by a more prolonged period of disorganized thoughts and behavior (including excessive anger), often ending in suicide.

From the viewpoint of this article, a person with bipolar disorder has an overly powerful incentive system that undermines the person's ability to function when in the throes of the disorder, but creates a powerful incentive for extraordinary productivity, otherwise.[15] It is the highly productive behavior during these more or less normal periods that partially compensates for the downside – the periods of mania and depression when the person is not able to function in a productive way. Another way of seeing this is that the destructive obsession with suicide is the downside of the obsessive concentration that occurs during more normal periods. This focus may also exist in unipolar depression.[16]

6.3. Laziness

To obtain a greater appreciation of the punishment and reward system, suppose that a person had no moods whatsoever, a situation that could be considered the mirror of bipolar disorder.

[11] Others have made similar distinctions between event-based depression and clinical depression. See for example, Nesse (2000) and Holzheimer and Mayberg (2011).

[12] See Schniter and Shields (2013) for an extended discussion of how emotions recalibrate behavior.

[13] Although many authorities tie addiction to depression, there is considerable evidence that addiction is tied to the euphoric states. Individuals with bipolar disorder are more than twice as likely to be alcoholics as people with unipolar depression (see Sonne and Brady, 2002). Drugs and alcohol result in a short-lived euphoria for both humans and mice. The latter are not necessarily depressed. Seeing addiction in this light may lead to different therapies to reduce addiction than those that are based on the belief the drinking is caused by depression.Opiates are a way of short-circuiting the reward system. It therefore should not be surprising that taking of opiates interferes with the incentive structure and that those who take such drugs are less likely to strive, except striving for more opiates, while they are addicted.

[14] Although it has been argued that the increased sexuality during mania increases the number of offspring.

[15] See Jamison (1996), for a study of creativity and bipolar disorder. There is some disagreement concerning exactly where in the mood cycle the most enduring work is done.

[16] This suggests the following test. Are people who are obsessed with suicidal thoughts more focused when they are in a normal state than normal people in normal states?

Just as bipolar disorder is recognized as a biological illness and from the viewpoint of this article a failure in the hedonic feedback system, the opposite of bipolar can arise where the incentive system is underperforming and lacking a monitor. As a result, there would be insufficient motivation to act while in a "normal" state.[17] Before continuing, it is important to distinguish the situation where someone is clearly depressed and therefore passive and withdrawn from the situation where someone is on an even keel, but not sufficiently motivated. Here we are focusing on the latter. The former is likely to be resolved by antidepressants; the latter not. Some view a person in the latter category as having a character flaw – the person is labeled lazy. Pop psychology recommendations abound, including a "tough love" approach, where the view is that until things get bad enough the person will not be motivated to change. Others view a person lacking in motivation as being depressed, but if the person is satisfied with his/her situation, the word depressed is inappropriately applied. The observer is saying that the observer would be depressed if in a similar situation, but the unmotivated person is not depressed, just not motivated. It is something akin to telling the depressed person that he/she should not be depressed because his/her objective reality is so good. That is just saying that the observer would not be depressed if facing a similar objective reality, but the observer is not the depressed person. And in a similar way, the observer is not the unmotivated person. In this view, "lazy" individuals have a weak (hypoactive) incentive system and need not be at all depressed by their situation. And once we understand this to be the case, the treatment (if there is to be a treatment in the first place) involves a totally different approach from those presently offered.

Bipolar disorder has a strong genetic component and IQ has a strong genetic component. Therefore, one should not be surprised that motivation is also likely to have a genetic component. Motivation is just more likely to interact with the environment – the possibilities (or lack of possibilities) that the person faces.

6.4. Unipolar depression

Most people do not suffer from major depressive disorder (MDD); nevertheless, a significant number of people do. This article argues that chronic depression does not increase fitness; instead, it is a failure in achieving the right biological–chemical balance in a very delicate system that should respond to certain cognitively understood situations, but not others. Individuals who suffer from chronic depression are often immobilized for long periods of time, lack libido, and have a much higher rate of suicide than those who do not suffer from chronic depression or bipolar disorder. So being chronically depressed does not increase fitness.[18] According to the theory presented here, when these individuals are not suffering from depression, they are likely to be very productive, but unlikely to be so productive as to outweigh the lack of productivity during the periods of depression. In a nutshell, being chronically depressed does not enhance fitness in general.

In DSM-V the word "dysthemia" has been replaced by persistent depressive disorder. A more or less constant mood, at whatever level, means that the regulatory system is not providing the appropriate emotional feedback. A persistent low mood means that all of the negatives associated with being depressed are there without the benefit of depression signaling a bad outcome. It is the lack of event-contingency and the difficulty in getting to a "recovered" state that makes the occurrence of persistent mood disorders so maladaptive. The recovered state (not the depressed state as others have argued) is where the person can make the appropriate cognitive response.

7. The role of the amygdala

To generate further insight, this section takes a brief look inside the brain. The anterior cingulate cortex, the hippocampus and the amygdala have all been viewed as the sources of depressive feelings. Here, we concentrate on the role of the amygdala. In general, increased activation of the amygdala is associated with depression.[19] This should be seen in the light of the amygdala's central role in directing attention by influencing cortical arousal and increased sensory and perceptual processing (see Davis and Whalen, 2001). It therefore should not be surprising that activation of the amygdala is associated with anxiety-based depression (see Davidson et al., 2009).

But why are people anxious in the first place? They are anxious because they are concerned about the outcome, in particular, the potential for physical or emotional pain (and at other times the potential for pleasure). If they did not care about the outcome, they would not be anxious. *The threat of emotional pain or the promise of emotional reward is manifested in our anxiety level.* The closer the outcome is in time and the more impact our behavior might have, the more anxious we become. A certain level of anxiety is not only a sign of motivation, but also helpful in that it makes the person more alert when needed and therefore more fit. However, as we have already seen in discussing bipolar disorder, the motivating system can be hyperactive, in which case it is debilitating, as is the case for excessive anxiety. And speaking of bipolar disorder with its excessive punishment and rewards, it should not be at all surprising that people who suffer from this condition are very likely to be anxious. In their study, Chen and Dilsaver (1995), found that among subjects with bipolar, the lifetime prevalence of panic disorder was 20.8%; among subjects with unipolar depression, it was 10.0%; and among comparison subjects, it was 0.8%. The causal relationship between bipolar disorder and panic disorder suggests that the best treatment for panic disorder for those with bipolar disorder is a mood stabilizer rather than an antidepressant, which typically works for those without bipolar disorder. The evidence appears to corroborate this view (see Perugi et al., 2010).

Anxiety is about anticipation. The person already knows that a fall down steep a cliff will result in physical harm and that the

[17] It should be noted that marijuana is a mood stabilizer and that increased use of marijuana is associated with reduced motivation. See Syed et al. (1991). Those who take medicine to control their mood swings often complain of reduced motivation. Side effects of lithium, the main drug used to reduce the effects of bipolar disorder, include lethargy and poor concentration while the person is in a normal state.

[18] Note that de Catanzaro, whose work was covered earlier, would probably argue that such behavior increases inclusive fitness. Here is the logic behind such an argument. Reduced consumption of scarce resources during depression increases the available food for others, thereby increasing inclusive fitness. This is true if the person is old and already feeble. Then adding depression to the list reduces consumption of scarce resources. But depression in the young and healthy also reduces production (e.g., gathering of food), which to some degree is shared by kin. The problem is that being depressed tends to be scripted for each individual (for example, curling up in a ball and crying). This script may increase inclusive fitness under certain circumstances (when one is ill) and decrease inclusive fitness under other circumstances (when a child dies from a fall, but the other children still need to be taken care of). In contrast, the argument here is that the possibility of bad fitness outcomes impels the individual to think and find the appropriate behavior that reduces such possibilities and the resulting depression.

[19] The hyperactivity of the amygdala appears to be coupled with diminished responsiveness during depressive episodes of regions involved in emotion regulation such as the dorsal anterior cingulate and the prefrontal cortex (see Stoll et al., 2000).

death of a sick child will lead to depression. The fact that unipolar depression is comorbid with having panic attacks (extreme anxiety) and bipolar depression is even more so provides further evidence that depression and elation are incentive devices more than times of learning and reflection.

8. Concluding remarks

Success is not just about IQ (and luck of the draw regarding the environment in which the person lives) but also about motivation. Scientists readily accept that there is a genetic component to IQ and that IQ varies across individuals. In contrast, differing levels of motivation across individuals are rarely attributed to genes. This article seeks to change that perspective by arguing that an individual's strength of motivation depends to a great extent on the individual's inherited punishment–reward system. It is this inherited emotional structure, particularly depression and elation, but also other emotions such as anger, that motivate people to act. Of course, it is the individual capabilities within the context of the particular culture that determines whether this drive is focused on intellectual, physical or other areas of achievement.

Understanding and treatment of major depressive disorder and bipolar disorder is enhanced if we first know the evolutionary basis for event-based depression. This article has provided a theory of motivation that is based on cognitive punishments (in particular, depression) and rewards (elation). The potential for depression and elation motivate the individual to undertake actions that promote greater fitness. Because both elation and depression have biological and behavioral costs, the motivation system cannot rely only on one of them. The punishment–reward system is to a great extent genetic, but it is hard to fine tune and thus may be either hypo or hyper-active. If the punishment–reward system is hyperactive, the individual suffers from the extremes of major depressive disorder and mania; if the punishment–reward system is hypo-active, the individual may lack motivation.

The analysis provided here explains the comorbidity of bipolar disorder and panic disorder. It also explains the lethargy and reduced motivation that often occur when individuals with bipolar disorder take drugs to reduce their mood swings. It is hoped that the approach outlined in this article will lead to other insights into chronic depression, bipolar disorder, and lack of motivation, and ultimately to their cure.

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