

Credible sadness, coercive sadness: Depression as a functional response to adversity and strife

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Abstract

Evolutionary medicine offers the insight that many unpleasant conditions such as physical pain are not harmful in themselves but are evolved mechanisms to mitigate harm. A major goal of the field is to distinguish illnesses from aversive conditions that are evolved defenses. There are numerous evolutionary theories of depression, but many fail to account for the central role of conflict and cannot explain suicidality. We review evidence from Western and non-Western societies that depression is caused by adversity, conflict, and powerlessness. The most parsimonious theory is that depression evolved, in part, as a form of psychological pain that functions to mitigate harm, credibly signal need, and coerce help when the powerless are in conflicts with powerful others.

Key terms: depression, suicide, mental health, cross-cultural, evolutionary medicine

For of men one can, in general, say this: They are ungrateful, fickle, deceptive and deceiving, avoiders of danger, eager to gain. As long as you serve their interests, they are devoted to you. They promise you their blood, their possessions, their lives, and their children, as I said before, so long as you seem to have no need of them. But as soon as you need help, they turn against you. Machiavelli (Wootton Edition, 1995, 52).

Introduction

Many of life's problems cannot be solved alone. Sadness and grief therefore are expressed in the face, posture, gait, tone of voice, and by crying (Grosbras et al., 2018; Sebe et al., 2006), all of which likely evolved to signal need to family, friends, and other important social partners, who in turn provided essential assistance. As we will describe here, depression, too, involves distinctive behavioral patterns, including reduced activity, eye gaze, tone of voice, posture, and gait, which readily communicate dysphoria. This suggests that depression might also involve signals of need. Yet depression alienates others (Coyne, 1976a, 1976b; Segrin & Dillard, 1992) and is stigmatized in some societies (Larkings & Brown, 2018). How, then, could it possibly elicit help?

In examining emotions under the lens of evolution, we confront the discomforting specter that emotions sometimes motivate socially undesirable behaviors. Emotions solve a wide range of adaptive problems (Tooby & Cosmides 1990; Al-Shawaf et al., 2016), many of which involve conflicts with social partners over, for instance, the distribution of resources and access to mates. Anger is aversive to the self and others, yet likely evolved to garner concessions from social partners when there were conflicts of interest (Sell et al., 2009). Men probably evolved to angrily threaten their wives when paternity was at stake, for example, or when wives diverted resources to offspring of other men (Buss & Duntley, 2011; Wilson & Daly, 1998). Similarly, mothers did not evolve to distribute resources equally among their offspring and probably evolved to not form attachments to newborn infants under poor environmental conditions (Hagen, 1999). Many social institutions such as police forces and courts have culturally evolved to reduce these socially undesirable behaviors, e.g., by punishing norm violators. But as Cosmides and Tooby (1999) observed, "...what is desirable or harmful are rarely matters of harmonious consensus or intersubjective agreement, much less matters of fact (p. 456)." This applies to individuals within society and across societies. Wife-beating is a punishable offense in Western society, but it is an acceptable, if not normative, practice in many societies (Rauchholz, 2016; Uthman et al., 2009). Indeed, institutions of social control in these societies sanction women who complain or attempt to escape (Jolly et al., 2012). Many critics have argued that psychiatry is, in part, an institution designed to control certain socially undesirable behaviors by labelling them as illnesses (see Syme & Hagen, 2020, for review).

Our approach to depression, which emphasizes its ability to elicit and coerce benefits from others by imposing, or threatening to impose, aversive costs on them, similar to anger, has been criticized for exactly this reason by other experts in evolutionary approaches to mental illness (Allen & Badcock, 2003; Nesse, 2006; Nettle, 2004). If one needed help from others, would not "normal" low mood, sadness, and grief suffice, leaving depression as a pathological extreme (Wolpert, 1999)? We hope to convince you that the answer is *no*, that although sadness and grief work well when there is little conflict with family, friends, co-workers or employers, they will not work when there is substantial conflict. The difference between depression and 'normal' sadness or grief is not psychopathology, we will argue, but is instead the degree of conflict in one's key relationships. Psychiatry might classify depression as a mental illness not because it is a brain dysfunction but because it is aversive to all involved.

What is Major Depression?

A diagnosis of Major Depression (MD), according to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), requires that an individual experience five or more of nine symptoms in a two-week period. Symptom criteria include: subjective feelings of sadness, hopelessness, or emptiness; diminished interest in activities; sleep changes; fatigue; inability to concentrate; weight changes; feelings of guilt; suicidal thoughts; and psychomotor agitation or retardation (APA, 2013). Most researchers regard these symptoms, taken individually, as normal responses to adversity, at least to some degree (suicidality would be the exception). Under the DSM-5, when the number and duration of symptoms exceed the above threshold, however, depression is considered to be a dysfunction regardless of environmental conditions.

Most MD and suicidality are caused by adversity

Ordinary sadness and grief are caused by adverse events. A common view about MD is that it is fundamentally different, striking without cause. Contrary to this common view, there is a consensus that MD is caused in large part by adverse events, and that more severe events increase the risk and severity of MD. Many early studies found that about 80% of cases of MD had evidence of at least one adverse event, compared to a much lower rate among non-cases (Mazure, 1998). See Figure 1.

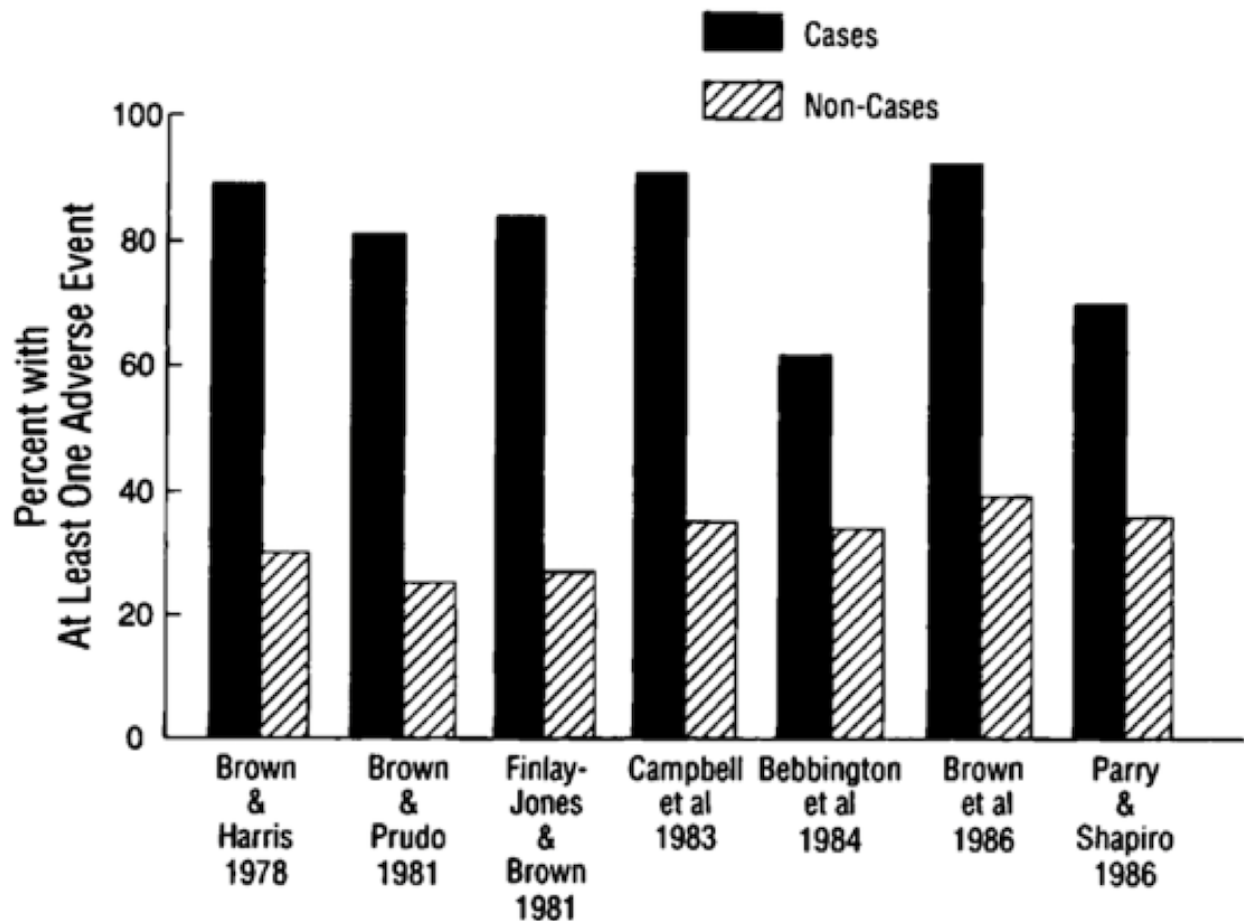


Figure 1: Life events and onset of major depression. Figure and caption from Mazure (1998).

Kendler et al. (1999) investigated if stressful life events *cause* MD by assessing the dates of stressful life events and the dates of onset of depression (if any) in a large sample of US female twins. Stressful life events were categorized as either dependent on participants' behavior (e.g., divorce) or independent of participants' behavior (e.g., death of a loved one). A close temporal association of an independent stressful life event with depression onset was taken as strong evidence of a causal effect of the former on the latter. Kendler et al. conclude that "Stressful life events have a substantial causal relationship with the onset of episodes of major depression. However, about one-third of the association between stressful life events and onsets of

depression is non-causal, since individuals predisposed to major depression select themselves into high-risk environments” (p. 837). See Table 1.

Life Event	Odds Ratio for Onset of Major Depression ^a			
	In Month of Event	1 Month After Event	2 Months After Event	3 Months After Event
Personal				
Assault	25.36***	4.31	— ^b	— ^b
Divorce/separation	5.22	— ^b	— ^b	— ^b
Financial problems	5.85***	2.75	4.31**	2.36
Serious housing problems	7.24***	2.71	1.25	1.26
Serious illness or injury	3.10**	0.70	0.76	0.43
Job loss	3.95*	— ^b	— ^b	— ^b
Legal problems	3.81	— ^b	4.55	10.81**
Loss of confidant	3.17*	1.05	3.19*	1.28
Serious marital problems	8.39*	14.26***	— ^b	4.29
Being robbed	2.74	1.40	1.42	5.01**
Serious work problems	2.44	1.82	1.87	2.74
In subject's social network				
Serious trouble getting along with an individual	5.04***	2.46	1.79	2.23
Serious personal crisis	2.32***	0.65	0.78	0.74
Death	6.29***	0.81	0.86	1.00
Serious illness	2.50***	1.12	0.98	0.43

^a Odds ratios are calculated from logistic regression of person-months (N=24,648), and their significance is determined by Wald chi-square test (df=1).

^b Not estimable because of small cell sizes.

* p<0.05. **p<0.01. ***p<0.001.

Table 1: Odds Ratios for the Onset of DSM-III-R Major Depression in 1,898 Female Twins in the Month of, and 1–3 Months After, Stressful Life Events Rated as Probably or Definitely Independent of Respondent Behavior. Table and caption from Kendler et al. (1999).

Non-Western evidence

Findings from non-Western samples further support that various forms of adversity are strongly associated with, and likely cause, depression, anxiety, and post-traumatic stress disorder (PTSD). An estimated one in five people suffers from depression, PTSD, anxiety disorders, and related disorders, in conflict-affected countries compared to one in 14 worldwide (Charlson et al., 2019). In a study of individual-level data from 160 countries, Elgar et al. (2021) analyzed the association of relative and absolute food insecurity with the number of mental health symptoms: physical pain, worry, sadness, stress, and anger. Higher levels of both relative and absolute food insecurity were associated with higher numbers of mental health symptoms. There were stronger associations for relative vs. absolute food insecurity, indicating the importance of social comparisons in experiences of distress.

Suicidality, an important symptom of depression, is also associated with adversity across populations. Multi-country studies show that violence against women is a strong predictor of emotional distress and suicidality (Devries et al., 2011; Ellsberg et al., 2008). Bullying from peers is another risk factor for suicidality in geographically diverse populations (Alavi et al., 2017; Butler et al., 2019; Ford et al., 2017; Wang et al., 2020). In a Norwegian adolescent sample that included indigenous Sami and non-Sami ages 15-16 years, suicide attempters reported more than twice as many adversities compared to non-attempters (Reigstad and Kvernmo, 2017). A comprehensive review of the ethnographic record found that discussions of suicidality commonly described threats to the victim's fitness (i.e., adversity), such as forced or thwarted marriages and physical abuse (Syme et al., 2016).

The weak evidence that MD in community populations is a brain dysfunction

Almost all research on MD uses either the DSM criteria or the *International Classification of Diseases* (ICD) criteria, which is informed by the DSM. The current DSM-5 criteria are basically the same as those in DSM-III, which ushered in the modern era of depression research. These criteria are listed in the right-most column in Table 2:

Study Authors or Criteria Set, Year				
Stone and Burris (15), 1950	Cassidy et al. (11), 1957	Feighner et al. (1), 1972	Research Diagnostic Criteria, 1975	DSM-III Criteria, 1980
Feeling depressed	Depressed mood	Dysphoric mood	Dysphoric mood	Dysphoric mood
Sleeplessness	Insomnia	Sleep difficulty (insomnia or hypersomnia)	Sleep difficulty or sleeping too much	Insomnia or hypersomnia
Appetite loss	Anorexia	Poor appetite or weight loss	Poor appetite or weight loss; or increased appe- tite and weight gain	Poor appetite or weight loss; or increased appetite and weight gain
Weight loss	Weight loss			
Agitation	Wringing hands, pacing	Agitation or retardation	Psychomotor agitation or retardation	Psychomotor agitation or retardation
Self-deprecation		Self-reproach, guilt	Self-reproach or guilt	Worthlessness, self-reproach or guilt
Psychomotor retardation	Slowed thinking			
	Loss of concentration	Diminished ability to think or concentrate	Diminished ability to think or concentrate	Diminished ability to think or concentrate
Suicidal thoughts	Suicidal ideation	Thoughts of death, suicide	Thoughts of death or suicide	Thoughts of death or suicidal ideation
	Constipation			
	Fatigue	Loss of energy	Loss of energy, fatigue	Loss of energy, fatigue
	Decreased sex interest	Loss of interest or decreased sex drive	Loss of interest or pleasure	Loss of interest or pleasure

Table 2: Historical origins of the symptomatic criteria for MD: Criteria proposed 1950-1980. Table and caption from Kendler et al. (2010).

Everyone will experience at least one of these symptoms at some point in their lives, and the majority of people will probably experience most of them. *Prima facie*, none of these symptoms indicate a brain dysfunction.

Depression criteria come from studies dating back to the 1950s conducted among groups of individuals determined to suffer from a variety of severe psychiatric disturbances (or physical

illnesses). The goal of these studies was to develop criteria that would enable different psychiatrists to reliably provide the same patient with the same diagnosis, such as bipolar disorder or schizophrenia. Importantly, these studies were *not* designed to distinguish the mentally ill from the healthy.

The DSM-III criteria for MD can be traced to Stone and Burris (1950), a clinical study of 50 selected cases; Cassidy et al. (1957), a quantitative study of 100 manic-depressive patients compared to 50 medically sick controls; Feighner et al. (1972), a study of 314 psychiatric emergency room patients and 87 psychiatric inpatients; and Spitzer et al. (1975), which tested the reliability of the Research Diagnostic Criteria (RDC) with 218 psychiatric inpatients. See Table 2.

None of the studies that defined MD as we understand it today included any healthy participants, nor any identified as experiencing only ordinary sorrow, sadness, or grief. There is thus no reason to believe that, when applied to the general population, the criteria developed in these studies would effectively distinguish the small minority of individuals with a genuine brain dysfunction from the much larger number of individuals who were suffering ordinary low mood, sadness, or grief in response to adversity (see also Horwitz et al., 2016). Yet they are widely used for this purpose.

The implausibly high prevalence of putatively disordered affect

It is no surprise, then, that when DSM criteria were first applied to the general population in the Epidemiologic Catchment Area (ECA) program, they generated implausibly high prevalence rates of mental illness. Over a quarter of the population (28.5%) was identified as suffering a mental illness in the last year, and nearly half the population (48%) was identified as having suffered a mental illness in their lifetimes. For MD, up to 10% were identified to have suffered an episode in the last year, and 17% to have suffered MD in their lifetime (Regier et al., 1998).

If MD were a genuine brain disorder, its epidemiology might resemble that of other genuine brain disorders, such as developmental brain disorders, which occur early in life, or the brain disorders related to aging, i.e., those that occur late in life. To compare MD with three common developmental brain disorders, we use data from the 2017 Global Burden of Disease Study. See Figure 2.

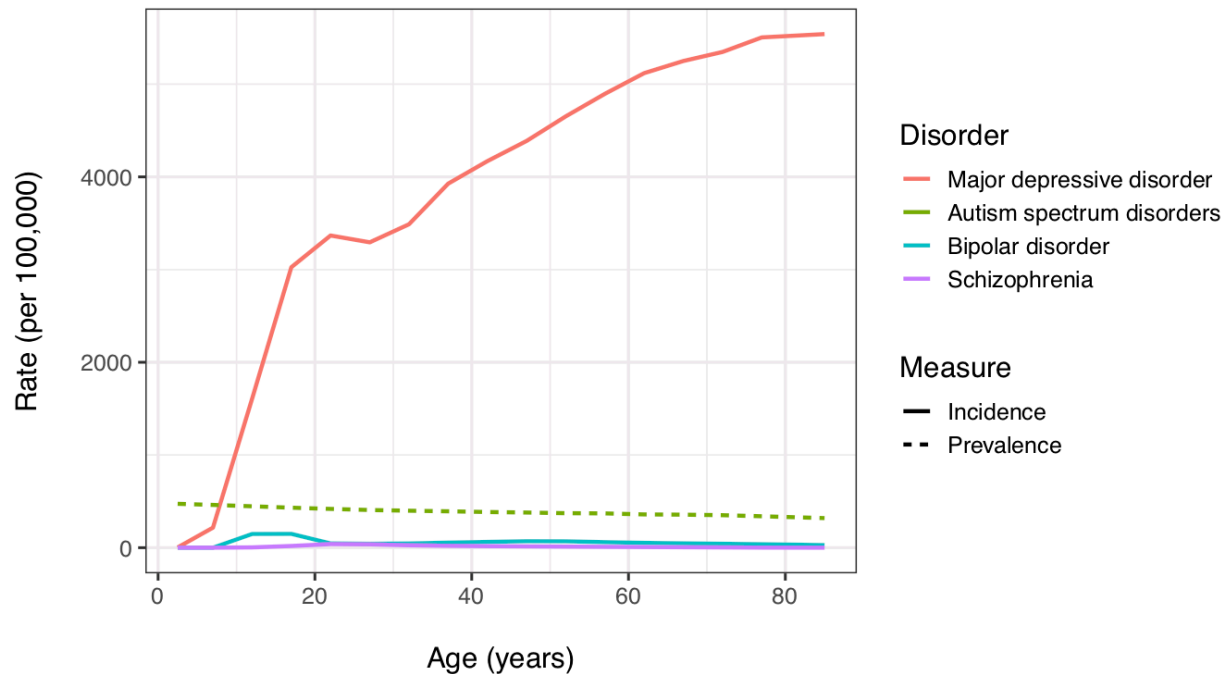


Figure 2: Major depression incidence compared to epidemiology of brain disorders that appear to be due to developmental disruption. X-axis: age (0-100 years). Y-axis: incidence/prevalence (rate per 100,000). Data from Global Burden of Disease (2017) and healthdata.org.

New cases of MD are common starting in adolescence and throughout adulthood and, as we discuss in more detail below, most individuals fully recover within several months to a year. In comparison, autism spectrum disorders are lifelong conditions that are relatively rare and present at birth (hence prevalence rather than incidence is reported). Bipolar disorder and schizophrenia are also lifelong and relatively rare, with peaks in incidence rates in late adolescence and early adulthood. Similarly, although incidence of MD increases with age, it does not resemble other brain disorders related to aging, such as dementias and Parkinson's disease (which do not remit), or stroke, all of which are rare until after the age of 40 or 50 years. See Figure 3.

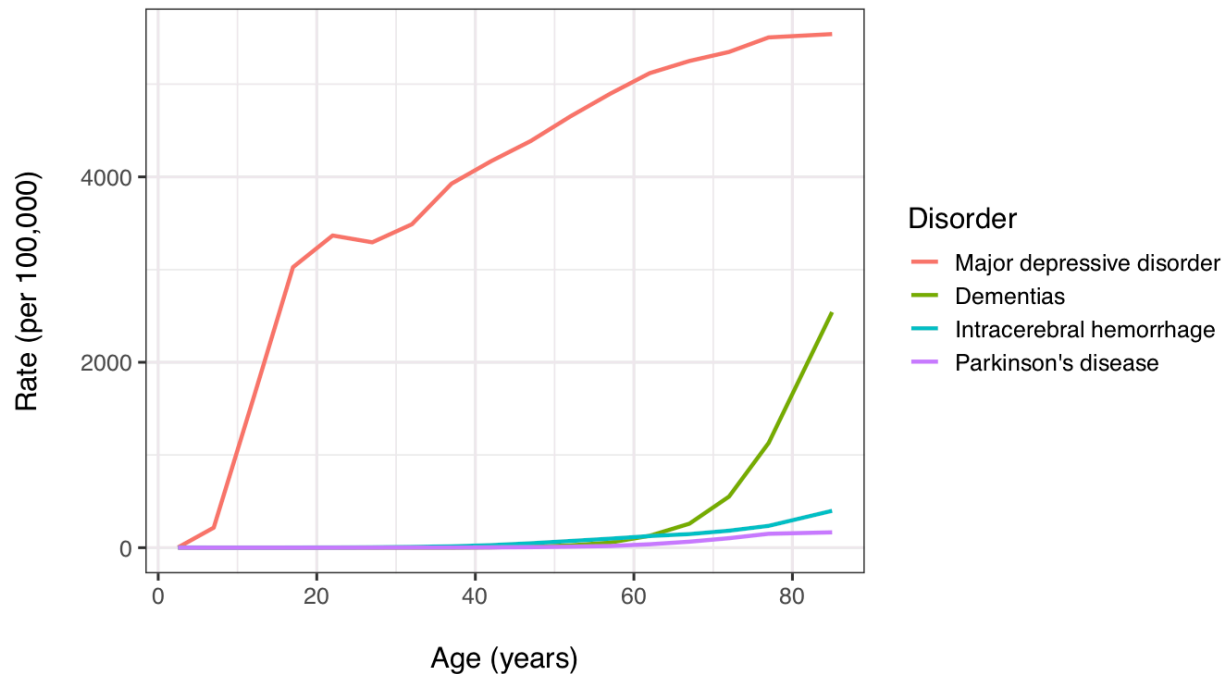


Figure 3: Major depression incidence compared to epidemiology of brain disorders that appear to be due to aging. X-axis: Age (0-100 years). Y-axis: Incidence (rate per 100,000). Data from Global Burden of Disease (2017).

Starting in adolescence, the incidence of MD is about an order of magnitude higher than other mental illnesses. Regier et al. (1998, p. 114) acknowledged that these high rates call into question the validity of “diagnoses” based on DSM criteria in community populations (Reiger initiated the ECA and was Vice-Chair of the DSM-5 task force):

Although it is possible that all of these community-based disorders are simply milder cases of essentially the same disorders seen in clinical settings, there are other possibilities as well. Based on the high prevalence rates identified in both the ECA and the NCS, it is reasonable to hypothesize that some syndromes in the community represent transient homeostatic responses to internal or external stimuli that do not represent true psychopathologic disorders. The human organism has a limited repertoire of response patterns to various physical, biological, and emotional stresses. Transient changes in blood pressure, pulse rate, body temperature, anxiety, or mood are not always indicators of pathology but of appropriate adaptive responses. It is possible that many people with currently defined mental syndromes (*in particular among the affective and anxiety disorders*) not brought to clinical attention may be having appropriate homeostatic responses that are neither pathologic nor in need of treatment — e.g., other equivalents of grief reactions that meet clinical criteria but are not considered pathologic if they are time-limited. (emphasis added)

Spitzer, leader of the DSM-III effort, expressed similar concern for the potential of high false positive rates for MD (Spitzer & Wakefield, 1999).

No evidence that MD is caused by a chemical imbalance

The first two drugs found to decrease depression symptoms both acted on monoamine pathways to increase monoamine concentrations, which motivated the ‘chemical imbalance theory’¹ of depression. Although depression, like all mental states, has a biological basis, decades of research has found no evidence that depression is caused by a simple imbalance of serotonin or any other neurotransmitter (Ruhé et al., 2007). The chemical action of antidepressants is a weak foundation for the ‘chemical imbalance’ model. After all, aspirin reduces headaches but headaches are not caused by an aspirin imbalance. Further, antidepressant effects appear after several weeks even though these drugs increase monoamines within minutes after ingestion (Frazer & Benmansour, 2002; Harmer et al., 2009).

Importantly, antidepressant drugs are only modestly efficacious at best (e.g., Kirsch & Sapirstein, 1999). Adjusting for unreported studies, effect sizes indicated a modest advantage of treatment over placebo (Cohen's $d = .31$ to $.32$) corresponding to less than two points on the Hamilton Depression Scale (HAM-D), which ranges from 0 to 52 (Cipriani et al., 2018; Kirsch, 2008; Turner et al., 2008). A 3-point difference on the HAM-D is a criterion for clinical significance for depression treatment (Kirsch, 2014).

Depression symptoms occur on a continuum

Depression symptomatology exists on a continuum, with little evidence for discrete non-depressed vs depressed states (Prisciandaro & Roberts, 2005; see Figure 4). A recent meta-analysis of studies that compared categorical (e.g., depressed vs. non-depressed) with dimensional (i.e., continuous) approaches to psychological traits supported the dimensional over the categorical approach by a factor of 5 to 1 (Haslam et al., 2020). Putatively dysfunctional MD is continuous with normality. Moreover, the severity and frequency of the stressors that cause depression predict the severity of the depression, in a dose-response relationship (Bustamante et al., 2020; Jenness et al., 2019; Tennant, 2002), albeit with possible differences due to age and sex (e.g., Ge et al., 1994; Kendler et al., 2004). When Cassidy, developer of one of the historical antecedents to the DSM-III MD criteria (see Table 2), was asked how he decided on the threshold number of symptoms to diagnose MD, he replied, “It sounded about right” (Kendler et al., 2010). There is no principled reason to conclude that higher scores indicate brain disorder instead of more severe sadness, grief, or low mood.

¹ The chemical imbalance model is also known as the catecholamine, monoamine, or serotonin deficiency hypothesis.

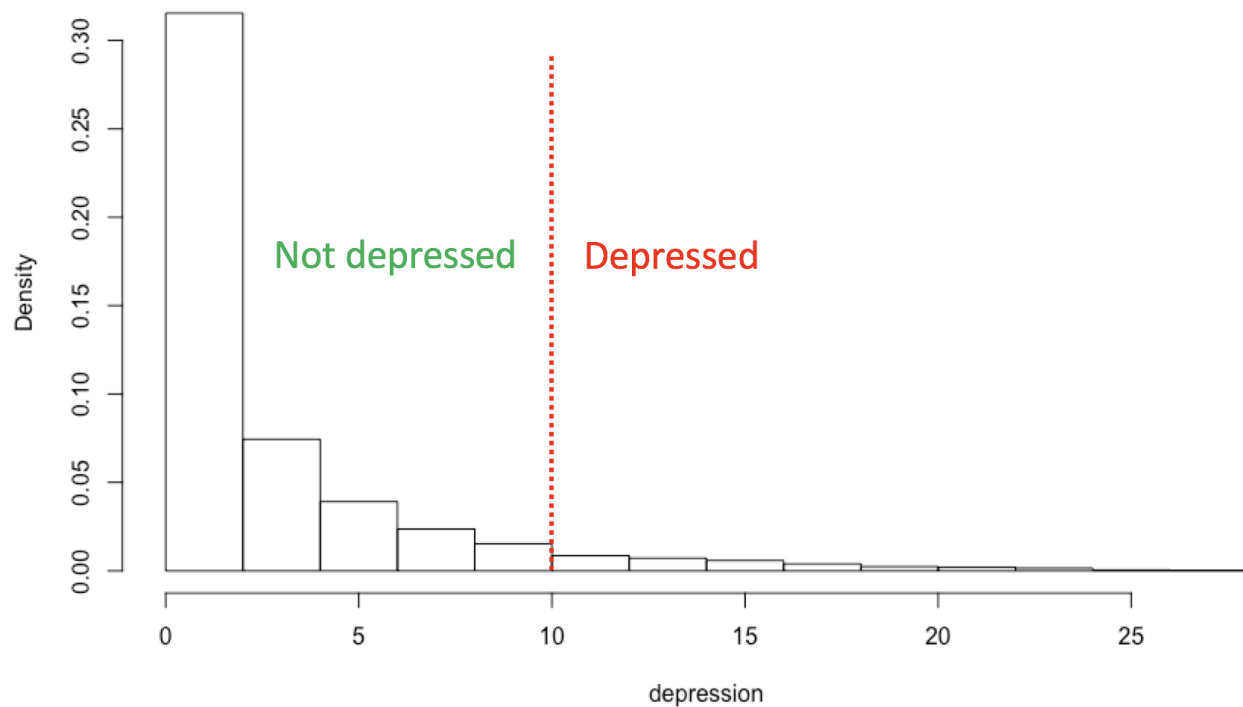


Figure 4: Distribution of depression scores from the Patient Health Questionnaire (PHQ-9), with a conventional threshold of 10 to diagnose MD. Nationally representative US data from NHANES 2011-2012.

Most depression is not chronic

Despite the frequent claim that MD is a chronic disease, the median duration of MD in a recent study of a nationally representative community sample was six months, and about 75% of cases remitted within a year (ten Have et al., 2017; see also Richards, 2011; Steinert et al., 2014). Only about 12-15% of individuals with at least one episode of MD have chronic MD (Eaton et al., 2008; ten Have et al., 2018). See Figure 5.

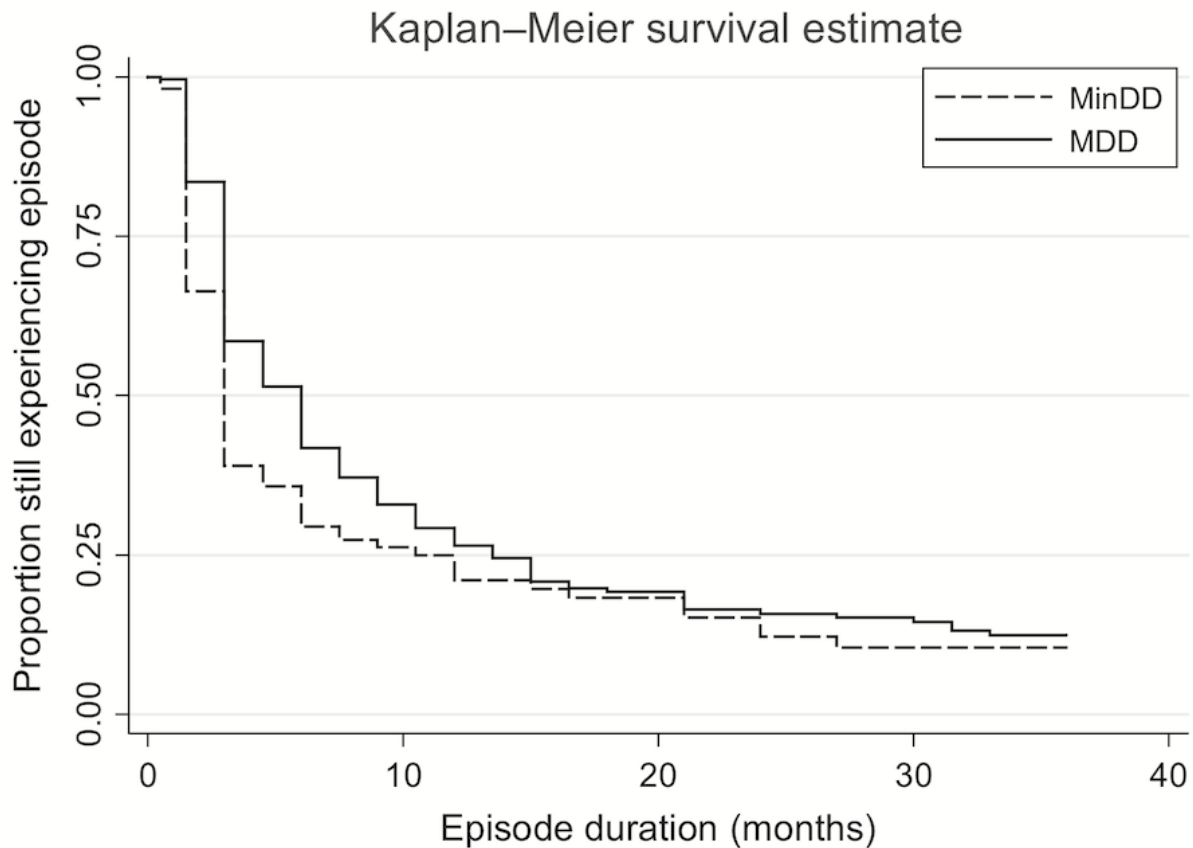


Figure 5: Survival curves of a cohort ($n = 393$) with newly originated (first or recurrent) depressive episodes in the general population; +, censored cases. MinDD: minor depressive disorder. MDD: major depressive disorder. Figure and caption from ten Have et al. (2017).

Indeed, in a US population study, about half of individuals who ever suffered MD had only one episode in their lifetime (Eaton et al., 2008). Similarly, in a Netherlands population study, only 27% of remitted MD cases had a recurrence within 20 years (ten Have et al. 2018).

Are most community cases of MD false positives?

Leaders in the field of psychiatry and related disciplines have warned that psychiatry, in general, and the DSM, in particular, increasingly medicalizes normal, healthy conditions. Allen Frances, Chair of the DSM-IV effort, has criticized an excess of diagnostic labels for variations of normal behavior (Frances, 2013). If depression, anxiety, and PTSD are adaptive responses to adversity, then labeling them as disorders is a problem of conceptual validity (Wakefield, 1992, 2013). On this view, most cases of MD in community populations are false positives.

The DSM-III and DSM-IV included a *bereavement exclusion*: a diagnosis for depressive symptoms occurring within two months following the loss of a loved one was excluded (unless symptoms of worthlessness and suicidality were also present). Depression in this circumstance was seen to be a normal grief response (Wakefield, 2013). Critics proposed that the bereavement exclusion should be expanded to include other forms of adversity that often cause depression,

such as interpersonal conflicts, job loss, and physical or sexual abuse (Kendler & Gardner, 2016; Mazure, 1998).

Members of the DSM-5 depression workgroup instead wanted to remove the bereavement exclusion because MD caused by the death of a loved one is similar to cases of MD caused by other stressors (Kendler et al., 2008; Wakefield et al., 2007). Their proposal sparked one of the most contentious debates over the medicalization of normal human experience in recent years (see Wakefield, 2013).

Ultimately, the bereavement exclusion was removed in the DSM-5, largely because bereaved individuals are at increased risk of suicidality, relative to individuals who have not recently experienced a stressful event, and therefore need treatment (Zisook, 2013). (As Wakefield, 2013, noted, however, the bereavement exclusion never applied to suicidal individuals anyway.) Nevertheless, because most cases of MD in the general population are caused by recent adversity, involve common symptoms like sadness, loss of interest, and changes to appetite and sleep, and resolve within several months to a year, it is unlikely that they represent brain dysfunctions.

Because any evolved function can dysfunction, some cases of MD, such as those seen among psychiatric inpatients, might reflect genuine disorders. Spitzer and Wakefield (1999, p. 1863) suggested that to identify genuinely disordered MD, DSM criteria should be altered to something like the following:

“The symptoms are not better accounted for by a normal reaction to a psychosocial stressor (e.g., loss of a loved one, terminal medical illness in self or loved one, loss of relationship); i.e., *the disturbance is judged to be markedly excessive in intensity or duration relative to the nature of the stressor.*” (emphasis added)

The real mysteries of depression

The primary scientific questions about MD are not what causes it. Adversity causes MD. The primary questions are, first, why, after experiencing what would seem to be the same degree of adversity, do different individuals experience widely varying levels of negative affect, some of which exceeds the threshold for MD diagnosis? See Figure 4. Few studies consider adversity from an evolutionary perspective. Divorce is a stressful life event, for example, but it might represent a benefit to individuals with good mating prospects and a severe adversity to individuals with poor mating prospects. Physical assault could be very adverse for weak individuals but might be much less adverse for formidable individuals. Death of a child might be more adverse for mothers than fathers (e.g., due to paternity uncertainty and/or the shorter female reproductive lifespan). Circumstances that inflicted fitness costs over much of human evolution, on the other hand, such as late or delayed reproduction, small family size, and failure to form a long-term mateship, might not be experienced as adversities by many people and therefore are not included on life event questionnaires yet may be extremely adverse for some. Thus, some differences in depression levels for similar adversities could be attributable to the fact that the degree of adversity depends on difficult-to-assess individual circumstances.

The second primary question is what are the evolved functions of depressed mood, if any, and especially of mysterious depression symptoms like suicidality and profound loss of interest in virtually all activities?

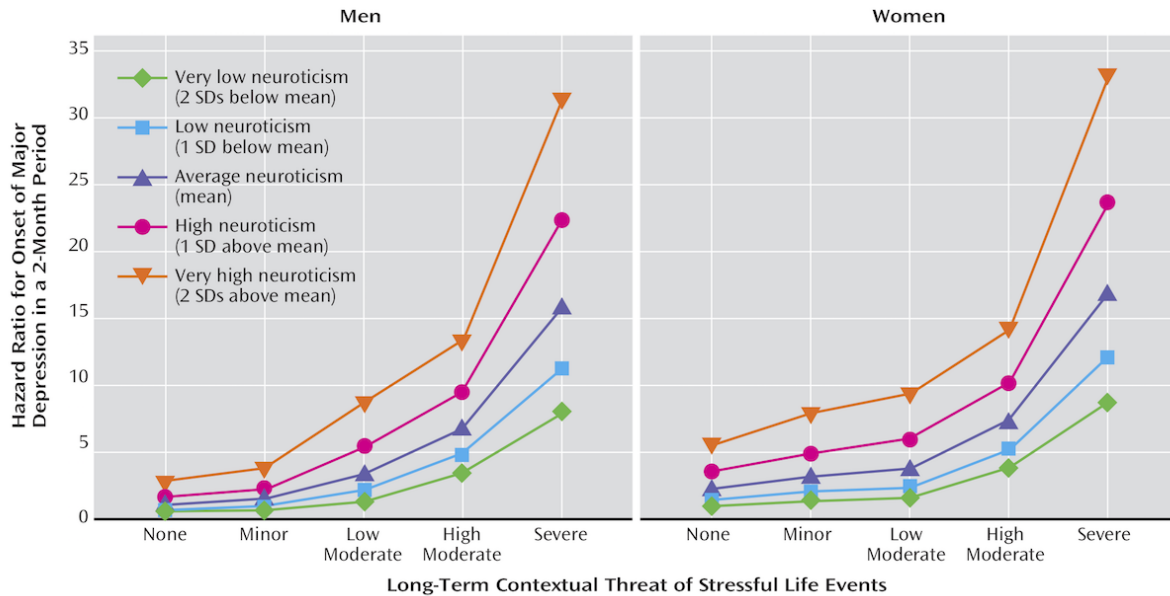
Many studies of depression are poorly designed to resolve its mysteries

Studies of MD commonly employ the following, fatally flawed case-control design: a group of participants that meet diagnostic criteria for MD -- cases -- are compared to a group of “healthy” controls, i.e., individuals without MD. Most of the individuals diagnosed with MD are (1) experiencing sadness or low mood (one of two necessary symptoms according to the DSM), and (2) have suffered recent adversity. Most members of the control group, in contrast, are not experiencing sadness or low mood, and have not suffered recent adversity. Hence, MD is almost completely confounded with sadness and recent adversity. Thus, any differences between the cases and controls could be due to differences in adversity or differences in ordinary sadness rather than the presence or absence of putatively pathological MD.

Studies of “vulnerability” and “resilience” are an important exception to the foregoing in that these studies often recruit individuals who have suffered a common adversity, such as a particular severe physical illness (e.g., spinal cord injury) or loss (death of a loved one), and then investigate why some individuals suffer lower or higher levels of depression, anxiety, and/or PTSD than others, i.e., are vulnerable or resilient (e.g., Bonanno et al., 2012; Maccallum et al., 2015; for review see Galatzer-Levy et al., 2018; Kalisch et al., 2019; for critique of high rates of resilience, see Infurna & Luthar, 2018).

The gold standard criteria for studies of vulnerability and resilience include a prospective design in which a large sample is assessed at baseline (T1) on the outcome measure (e.g., depression levels), as well as on a number of sociodemographic, psychosocial, or other factors that are thought to confer vulnerability or resilience, and is then exposed to a stressor at T2. The key term to estimate is the moderation effect of the putative vulnerability or resilience factors on the relationship between stressor levels and outcome. Kalisch et al. (2017) identified only 13 such resilience studies that met these criteria (albeit in a non-systematic review), many of which investigated the extent to which various factors moderated the effect of war zone deployment on subsequent PTSD in soldiers. Effect sizes were generally small, with several null results.

Two factors that have been consistently associated with higher vulnerability to MD in response to adversity are neuroticism and female sex (see Figure 6). We address the evolutionary significance of neuroticism and the sex difference later.



^a A hazard rate of unity was defined as the risk level for a man with a mean score on the 12-item neuroticism scale from the shortened Eysenck Personality Questionnaire (21) and no exposure to stressful life events. Descriptions of the types of stressful life events and the scoring of long-term contextual threat are given in text.

Figure 6: Hazard Ratios Indicating Risk of Onset of Major Depression for a Population-Based Sample ($n = 7,517$) Classified by Sex, Neuroticism, and Stressful Life Events. Figure and caption from Kendler et al. (2004).

Toward a comprehensive evolutionary model of depression

Adaptations are traits that evolved because they had positive effects on the fitness of an organism's ancestors. To identify psychological adaptations, evolutionary theorists consider the following: (1) evidence of a longstanding computational problem in past environments; (2) evidence that solving this computational problem would have increased the biological fitness of ancestors with the trait relative to those without it; (3) evidence that humans maintain the computational ability to solve this problem; and (4) evidence that the computational ability reliably develops in nearly all humans of one or both sexes during one or more stages of development absent any disruptive environmental mismatches (Williams, 1966; Tooby & Cosmides, 1990).

Evolutionary models of MD take as their starting point that MD is caused by adversity, proposing that it is either 1) mostly a functional response to adversity continuous with sadness and low mood (with only a minority of cases representing genuine dysfunction) or 2) dysregulated sadness and low mood (for reviews, see Allen & Badcock, 2006; Del Giudice 2018; Durisko et al., 2015; Hagen, 2011; Nesse, 2000, 2019; Nettle, 2004). Thus, the correct evolutionary account of MD requires understanding the evolved functions of sadness and low mood, to wit, if and how the latter increased fitness in the wake of adversity. (For simplicity, we will use the term "negative affect" to refer to the entire range of low mood, sadness, grief, and depressive symptoms, but excluding other negative emotions such as anger, anxiety, and

jealousy. We will use the term “depression” to refer to a range of sub- and supra-threshold depression symptoms. We will reserve the term MD for Major Depression as defined in DSM-5).

Before continuing, it is critical to reiterate that nothing in DSM-5 (and almost nothing in DSM-III and DSM-IV) predicates a diagnosis of MD on the experience or lack of experience of adversity (the exception is the bereavement exclusion in DSM-III and DSM-IV). Many studies of MD therefore do not measure adversity. Consequently, it is difficult to relate many facts about MD to the nature of the adverse circumstances that cause it. This makes it difficult to evaluate evolutionary models of depression, most of which incorporate adversity. One evolutionary model, for instance, defines depression as inactivity even when environmental conditions are good (Trimmer et al., 2015). But it is unknown how many, if any, individuals with MD fit that description.

The evolutionary significance of adversity is that it reduced fitness, and any psychological adaptations to adversity would function to mitigate these costs and avoid future costs from similar adverse events. There are many hypotheses for the fitness benefits that negative affect provides to victims of adversity, including energy conservation (Anders et al., 2013; Kinney & Tanaka, 2009; Raison et al., 2006; Raison & Miller, 2013, 2017), submission (Gilbert & Allan, 1998, 1998; Price et al., 1994), risk reduction (Allen & Badcock, 2006; Allen & Badcock, 2003; Badcock et al., 2017), and goal disengagement (Nesse, 2000, 2006).

Hypothesized functions of depression can be divided into two major categories. Some focus on the emotional and cognitive features, such as sadness and rumination, without specifying particular types of adversity or behavioral response to adversity. Others attempt to link specific behavioral features of depression, such as reduced activity, to specific types of adversity, such as loss of resources. Although there is considerable overlap between the two categories, the most general and promising approach to the evolution of depression belongs to the first category.

The psychological pain hypothesis

Alexander (1986) argued that “mental pain” serves a function similar to physical pain, i.e., it evolved to help solve the problem that caused the pain. Building on this suggestion, Thornhill and Thornhill (1989, p. 78) developed their *psychological pain* hypothesis:

Our hypothesis is that humans will have evolved to experience mental distress in direct relation to the actual or potential occurrence of circumstances, especially social, that historically would have reduced inclusive fitness (Alexander, 1986; Thornhill and Thornhill, 1983; 1987; Thornhill et al. 1986). This hypothesis views the evolutionary function of mental pain as analogous to the evolutionary function of physical pain. Physical pain serves to draw an individual’s attention to some aspect of anatomy that needs tending and can be fixed by the individual’s attention. Mental pain seems to focus an individual’s attention on the significant social events causing the pain and the evaluation of future courses of action.

Psychological pain, like physical pain, signals fitness threats, and functions by way of being aversive (Thornhill & Thornhill, 1989; Tooby & Cosmides, 1990).

The psychological pain hypothesis is not specific to MD, nor does it address the claim that MD is an adaptation. Instead, it is a general model that applies to a wide spectrum of low mood and negative affect. As we reviewed above, however, MD is caused by adversity, is characterized by psychological pain, and the most common and most depressogenic forms of adversity were probably experienced by most individuals for most of human evolutionary history, and plausibly inflicted inclusive fitness costs. These include assault, loss of one's mate, loss of resources, serious illness or injury, and death of a loved one (see Figure 5). It would be remarkable if humans had not evolved psychological mechanisms to mitigate the negative fitness consequences of adversity.

Given that most cases of MD are not pathological, and that symptoms occur on a continuum, the psychological pain hypothesis is a compelling account of depression, including MD. The psychological pain hypothesis does not single out specific types of adversity, nor specify a particular behavioral response to adversity, because different types of adversity and different individual circumstances will require different behaviors to mitigate costs.

There are few direct tests of the psychological pain hypothesis. Thornhill and Furlow (1998) and Hagen (1999) independently evaluated postpartum depression (PPD) as a case study, finding that PPD was strongly associated with circumstances that would have inflicted fitness costs on ancestral mothers of newborns, including low paternal and social support, lack of resources, and poor infant and maternal health, and in some circumstances motivated disinvestment in the newborn (see also Hagen, 2002; Hagen & Barrett, 2007; Hagen & Thornhill, 2017). Thornhill and Thornhill also evaluated the hypothesis in the case of rape victims (Thornhill & Thornhill, 1991; Thornhill & Thornhill, 1990a, 1990b, 1990c).

Divorce is a good test case for adaptive theories of depression because loss of a mate could have a profound impact on biological fitness. Rosenström et al. (2017) developed mathematical models of four competing causal, mechanistic theories of the relationship between divorce and depression: an adaptationist model similar to the psychological pain hypothesis (in which the risk of divorce triggers depression, which yields benefits by decreasing the risk of divorce, but increases the risk of death, e.g., from suicide), and three stress-diathesis models (e.g., a stress-relief model in which the period before divorce is depressogenic and the period afterwards is not). These models were used to generate four quantitative predicted temporal distributions of depressive symptoms relative to divorce. Rosenström et al. then tested this model using national registry data on 304,111 Finns that examined the association of divorce with antidepressant use as a proxy for depression (Metsä-Simola & Martikainen, 2013). Their study afforded an unprecedented temporal resolution of one depression prevalence estimate every three months for 10 years with no bias from attrition or repeated interactions with researchers. Rosenström et al. quantitatively assessed how well each predicted distribution of depression relative to divorce fit the empirical distribution, finding that the adaptationist model best fit the data.

Analytical rumination hypothesis

One of the most important extensions of the psychological pain hypothesis, the analytical rumination hypothesis (ARH), was developed by Paul Andrews. Depressed individuals

commonly ruminate about the problems that triggered their depression, which they perceive to be complex, severe, and difficult to solve. Their rumination is intrusive, persistent, resistant to distraction, and difficult to suppress, yet, as the psychological pain hypothesis predicts, depressed people believe it gives them insight into their problems (Andrews & Thomson, 2010).

Because depressive rumination decreases performance on other cognitive tasks, mainstream researchers have interpreted it as a cognitive dysfunction (e.g., Nolen-Hoeksema, 1991). Andrews and Thomson (2010) instead argue that there is a necessary tradeoff: because cognitive resources are limited, individuals experiencing challenging problems should concentrate on solving them rather than allocating attention to solving unrelated problems in laboratory experiments. The more complex and intractable the problem, the more intense the rumination should be.

There is a small but growing literature testing the ARH (Barbic et al., 2014; Bartoskova et al., 2018; Maslej et al., 2019). A recent study by Sevcikiova et al. (2020) that included depressed inpatients and community controls found that depressed patients reported a greater number of problems of greater complexity relative to controls, and engaged in more causal analysis but not in more problem-solving analysis. Depressed inpatients who engaged in more problem-solving analyses relative to other depressed inpatients at the start of hospitalization showed a decrease in symptoms at the end of hospitalization; however, there was no relationship with the likelihood of remission or a decrease in symptoms at follow-up.

There is a critique of the ARH from within evolutionary medicine and psychiatry that cites evidence that interventions to suppress rumination produces positive outcomes for patients (Kennair et al., 2017). Another limitation of the current formulation is that rumination is only moderately associated with depression, and is also moderately associated with anxiety (Olatunji et al., 2013). Rumination, worry, and repetitive negative thinking are similar experiences that follow “emotionally evocative” experiences (McEvoy et al., 2013). Thus, the ARH might not be specific to depression but instead apply to a broad range of difficult situations that could inflict costs or yield benefits, and therefore it is probably associated with a range of negative and positive emotions (e.g., Li et al., 2017).

The association of depression and suicidality with strife and anger

The psychological pain and analytical rumination hypotheses do not account for all the important symptoms of depression, such as suicidality and profound loss of interest in virtually all activities. In the remainder of this chapter we argue that these mysterious symptoms are best explained as strategic responses to adversity *under strife*.

Much adversity does not involve social conflict. The death of a young child from an infectious disease is a tragedy that requires painful rumination to promote understanding that can help prevent the deaths of other children, but it does not necessarily involve conflict. Although Thornhill and Thornhill (1989), in their development of the psychological pain hypothesis, emphasize the negative fitness consequences of adversities that involve conflict, such as loss of

status and sexual assault, and Andrews and Thomson's (2010) ARH similarly notes that solving social dilemmas often requires considerable reflection, psychological pain and rumination function to mitigate adversity regardless of conflict, and thus are expected to operate in a broad range of conflictual and non-conflictual conditions.

It is an empirically well-supported but poorly recognized fact, however, that MD is closely associated with social conflict and anger (Cassidello-Robbins & Barlow, 2016). Many of the most potent risk factors for MD, such as physical and sexual assault, serious marital problems, legal problems, robbery, and job loss, are *prima facie* instances of social conflict (see Table 1). Many studies of MD confirm that sufferers are often embroiled in interpersonal conflict. In a study of a large nationally representative US sample ($n = 4688$), Bertera (2005) found strong and consistent support for the association of social negativity, as measured by items such as "How frequently do you argue with friends (spouse or relatives)" and "How often do friends (spouse or relatives) make you feel tense," with the number of mood disorders. Whisman and Uebelacker (2009), in a two-year prospective study of an English population-based sample ($n = 1869$ couples), found that baseline marital discord predicted subsequent depression, and that baseline depression predicted subsequent marital discord, i.e., there is a bi-directional relationship between conflict and depression. Among the Tsimane, Amazonian horticulturalists, depression was significantly associated with social conflicts, especially with non-kin (Stieglitz et al., 2015). A meta-analysis of 165 studies of the relationship between bullying in children and adolescents and mental health outcomes that included samples from high-, middle-, and low-income countries, indicated a causal relationship between bullying victimization and anxiety, depression, poor general and mental health, non-suicidal self-injury, suicide attempts, and suicide ideation (Moore et al., 2017, p. 72). In a probability sample from 11 European countries ($n = 4779$ couples), marital conflict was associated with both self-reported and partner-reported depressive symptoms in both sexes (albeit with a weaker association for women in Northern Europe compared to women elsewhere) (Salinger et al., 2020). A longitudinal study of a representative sample of German couples ($n = 1273$ couples), comprising three age cohorts, found that for both sexes, financial conflict and depressive symptoms were related initially and over time, that depression in one partner predicted future perceptions of conflict in the other partner, and that men's perception of financial conflict was associated with increasing depression in their partners over time (Morgan & Lim, 2020).

Fatal and non-fatal suicidal behavior also commonly occurs in the wake of social conflict, particularly with family members or spouses. Firth (1961) characterized acts of suicidal behavior among the Tikopia as a form of protest. In many Muslim-majority countries, suicidal behavior among women is regarded as recourse against oppressive kin or restrictive social structures (Billaud, 2012; Canetto, 2015), and the anthropological literature abounds with comparable models of suicide as a form of protest, revenge, and/or appeal (e.g., Malinowski, 1985; Niehaus, 2012). Among the Toraja, Hollan (1990, p. 371) reported that suicide is rooted in "intense feelings of injustice and mistreatment" when, for instance, adolescents "feel their own legitimate needs and desires have been neglected by their parents and relatives." Staples and Widger (2012) highlighted protest as a central theme of the ethnographic literature on suicide. Syme, Garfield, Hagen (2016) and Syme and Hagen (2019), in studies of suicide across 53 traditional cultures, found that conflict was a central theme.

Death of a loved one, one of the most common causes of MD and suicidality, does not at first glance seem to involve social conflict. There are solid theoretical reasons, however, backed by accumulating empirical evidence, to believe that loss of an important social partner often does cause conflict with others and probably did so over human evolution. Social partners provide critical benefits, such as food, protection, information, and assistance with child rearing and other tasks. Individuals who lose these benefits due to the death of a social partner must acquire them from other social partners. Ethnographic research on grief among children and adolescent farmers and foragers in the Congo Basin, for example, shows that the death of a parent or other close family member is tied to the loss of vital resources (e.g., food, clothes), and life can be challenging for bereaved youths. Hewlett (2017) reported that living kin members would console them by offering clothes, money, and other resources, which relieved some of their painful sadness and made it possible for them to grow up and have children of their own. Social network research has found that deaths of family members and confidants can result in profound changes in social networks (Cornwell & Laumann, 2018; Wrzus et al., 2013). But social partners, who have other relationships, cannot always easily compensate for one individual's loss of benefits. A study of 156 Australian families that had suffered the death of a parent found that about one-third of families were characterized by conflict six weeks after the death, one-quarter at six months, and 15% at 13 months (Kissane et al., 1996). A systematic review found that, after a loss, family conflict was a major risk factor for complicated grief (Delalibera et al., 2015).

The association between inflammation and depression (Maes, 1999; Stieglitz et al., 2015) also supports the role of social conflict in depression. Although researchers have proposed that depressive symptoms might, e.g., conserve energy for the immune system or reduce appetite to avoid exposure to pathogens (Kinney and Tanaka, 2009; Anders, Tanaka, & Kinney, 2013; Raison and Miller, 2017; Raison, Capuron, & Miller, 2006; Raison & Miller, 2013), there is considerable evidence that social threats activate the immune system (Slavich & Irwin, 2014). Social conflict, no doubt, frequently led to physical harm throughout human evolutionary history as it does today. For instance, one study reported that 85% of Tsimane endured physical abuse from their spouses (Stieglitz et al., 2011), and another in the same population found associations between depression and immune biomarkers (Stieglitz et al. 2015). Further, assault is a potent risk factor for depression (Kendler et al., 1999). It is likely that increased immune activation in the depressed is a consequence of either their elevated risk of suffering physical attack due to severe conflicts, or prior or ongoing physical abuse.

There is an equally clear association of depression with anger. Riley et al. (1989) found that depressed subjects experienced moderately abnormal levels of anger and tended to suppress instead of express their anger. A study of patients with MD ($n = 73$), anxiety disorders ($n = 67$), somatoform disorders ($n = 47$), and healthy controls ($n = 215$) found that patients with MD were more likely to have higher anger, hostility, and aggression than the other groups (Koh et al. 2002). Gilbert et al. (2004) conducted interviews with 50 depressed inpatients and outpatients and found that 82% reported suppressing anger (the researchers did not ask about expressed anger) and 88% reported feelings of entrapment. Nearly all reported reasons for suppressing anger involved avoiding losing social standing or harming important relationships. Similarly, participants commonly reported feeling trapped for social reasons, e.g., losing a relationship that would be hard to replace. A study of a nationally representative sample of Australians ($n = 8841$) found that, controlling for demographics and comorbidity, several mood, anxiety, and substance

use disorders were independently associated with symptoms of anger (E. L. Barrett et al., 2013). Among US patients with MD at intake ($n = 536$), overt expression of irritability/anger was associated with greater severity and longer duration of the MD episode (Judd et al., 2013). Bodner et al. (2018), in a laboratory study of interactions between parents and their adolescent children (the latter either depressed or non-depressed), found that families of depressed adolescents expressed more anger than families of nondepressed adolescents during the interaction, and expressions of anger co-occurred and interacted more, potentially creating a self-sustaining network of angry negative affect. Ou and Hall (2018), in a systematic review of the literature on postpartum depression and anger, found that anger and depression were often linked, with anger elevated in depressed vs. nondepressed mothers, and that anger was associated with powerlessness. In a large study of psychiatric outpatients ($n = 3800$), anger and overt aggression were common, particularly among those diagnosed with MD, intermittent explosive disorder, and cluster B personality disorders (e.g., antisocial, borderline, histrionic) (Genovese et al., 2017). In a 25-year longitudinal study of depression and anger among Canadians ($n = 944$), Galambos et al. (2018) found a stable relationship between higher anger and higher depression both between individuals, and within individuals over time. In a meta-analysis of 491 studies ($n = 235,085$) examining the associations of depression, anger, anxiety, and negative affect among employees, Ng et al. (2019) found that these emotions strongly correlated with each other (weighted average correlation = 0.69), and that they were similarly associated with job-related stressors. A study investigating personality traits among suicide attempters and non-attempters among the Idu Mishmi ethnic group in India found that attempters scored significantly higher on aggression and impulsivity than non-attempters (Singh & Rao, 2018). In summary, those who have suffered adversity are often embroiled in conflict and are angry, depressed, and sometimes suicidal.

Some non-Western concepts incorporate elements of depression, conflict, and anger

DSM criteria for major depression are a good index of the clinical syndrome of depression. But...this depressive syndrome is not entirely constituted by the DSM criteria. (Kendler et al., 2016, p. 780)

There is a direct line of descent from Hippocrates's writings on melancholia, a condition characterized by extreme sadness, to the modern psychiatric model of depression (see Jackson, 1986). It is noteworthy that many languages do not have direct translations of depression (Littlewood, 2002). There are, however, many terms in non-Western languages that overlap with depression, and in addition to sadness or anhedonia, these terms often denote conflict and anger. In rural Trinidad, the term *tabanka* refers to a state of lassitude accompanied by feelings of low self-worth, anger, anhedonia, appetite loss, and suicidal behavior. Unlike depression, this state is specific to males who have recently lost a spouse or romantic partner to a competitor (Littlewood, 1985). Among Chon Chuuk Micronesians, the term *amwunumwun* denotes a state of anger and despondency that usually arises when one, often an adolescent or young adult, is rebuffed by an elder kinsmen who is both loved and respected. *Amwunumwun* is associated with behavioral withdrawal including avoidance, refusal to eat or speak, running away, and suicide (Hezel, 1987). The Hopi word *qövisti* is a state of moodiness and sullenness in young women

facing intractable conflicts with parents and other elders and can end in a 'willful death' (Titiev, 1944).

Powerlessness

Resolving conflicts in one's favor typically requires some advantage over adversaries. Depression, though, is associated with feeling powerless (Gilbert, 1992; Gilbert et al., 2004; Gilbert & Allan, 1998; Price et al., 1994). The depressed perceive themselves unable to unilaterally change things for the better. There is robust evidence of an association between depression and defeat (Gilbert & Allan, 1998), hopelessness (Abramson & Metalsky, 1989; Abramson & Seligman, 1978), external locus of control (Rotter, 1966), and entrapment (Brown et al., 1995). A meta-analysis found that external locus-of-control and depression were significantly related, that the relation was moderately strong, and that it was consistent across studies; in addition, belief that events were controlled by powerful others and chance was associated with higher levels of depression (Benassi et al., 1988). A meta-analysis of the helplessness models found that attribution of positive events to external causes (i.e., those not under one's control) was associated with depression (Sweeney et al., 1986). A study of 7322 adult twins found that higher ratings of loss and humiliation predicted onset of MD and mixed MD-Generalized Anxiety Syndrome (MD-GAS) and high ratings of entrapment predicted onset of MD-GAS (Kendler et al., 2003).

Although it could be the case that feelings of powerlessness are illusory, there is substantial variation in physical and social formidability in humans and other animals. One interpretation of the association between depression and powerlessness, which draws on comparative evidence from non-human primates and other non-human animals, is that depression is an *involuntary subordinate strategy* following aggressive contests between competitors. According to this *social competition hypothesis*, depression is caused by a specific type of adversity -- social defeat -- and involves specific behavioral responses that signal de-escalation and submission. Specifically, these models focus on the behavioral signs of depression such as slumped posture, lowered eye gaze, and social withdrawal, noting that these resemble submission strategies exhibited by a diverse range of species (Price et al., 1994; Price et al., 2007).

The social risk hypothesis similarly sees the social withdrawal of depression as a harm-reduction strategy. Using a model analogous to risk-sensitive foraging, Allen and Badcock (2003) proposed that individuals with a robust social network can afford to take risks, whereas individuals with a weak social network risk ostracism and cannot take social risks. A more recent version of the model incorporates the free-energy principle into their model, but the emphasis on reducing social risk remains (Badcock et al., 2017).

There are many types of adversity that cause depression, however, such as death of a loved one or an ill newborn that are not losses of social competitions. Submission is also not a universally beneficial response to all types of adversity that cause depression, nor even to all types of social competitions, even when engaged with a formidable adversary. Finally, the social competition hypothesis does not make sense of all the symptoms of depression, such as suicidality. Nevertheless, powerlessness appears to be an important characteristic of depressed individuals.

As we explain next, the powerless do have the means to change others who are reluctant to change, but sad expressions and crying are not enough -- the powerless often have to pay a cost.

Bargaining: Credibly signaling need under conflict

Suicidality, one of the DSM diagnostic criteria for MD, is not well-explained by most evolutionary theories of depression, nor is pronounced loss of interest in virtually all activities, one of the defining features of MD. In our view, these perplexing symptoms are best explained by considering the confluence of adversity, conflict, and powerlessness. In these circumstances, individuals are in need, but conflict with social partners and powerlessness prevents their needs from being met. We argue that depression, like other emotions, involves characteristic signals, specifically *credible* signals of need.

Signals are necessary if the signaler's state is unknown to receivers, i.e., if the signaler's state is *private information*. Signals thus function to reveal private information to receivers.

Signaling between organisms is ubiquitous across the tree of life: bacteria coordinate via molecular quorum sensing signals (Jayaraman & Wood, 2008); plants signal pollinators and the natural enemies of insect herbivores, and possibly communicate with each other (Karban, 2015; Woźniak & Sicard, 2018); and animals send and receive signals from offspring, mates, and competitors (Godfray, 1991; Skyrms, 2010). In humans, characteristic facial expressions signal emotions such as anger and sadness, albeit with some variation across cultures (Darwin, 1916; Ekman & Cordaro, 2011; cf. Barrett et al., 2019). In a study of six million online videos, for example, Cowen et al. (2020) found that 16 types of facial expressions, including anger, pain, and sadness, systematically co-varied with specific social contexts across 144 countries. A study across 12 countries that highlighted variation in the facial recognition of seven emotion categories, such as sadness and anger, nevertheless found that most participants accurately classified facial expressions into emotion categories (the primary exception was that fear was often misclassified as surprise; Quesque et al., 2020)

Tears and crying by infants, children, and adults have long been interpreted as signals of need (Balsters et al., 2013, 2013; Lummaa et al., 1998; Trivers, 1974). An eye-tracking study found that tears were a magnet for visual attention, and a single tear running down a cheek was associated with increased emotional inference and greater perception of sincerity (Picó et al., 2020). It has been proposed that infected individuals experience the emotion of lassitude, which generates protective sickness behavior, including signals of need (Schrock et al., 2020; see also Steinkopf, 2015; Tiokhin, 2016).²

Deceptive signaling is also rife in nature (Mokkonen & Lindstedt, 2016). When there are conflicts of interest between signalers and receivers, signalers can benefit by sending deceptive signals that exploit receivers. Mimicry and crypsis are widespread across taxonomic groups

² It is important to distinguish cues from signals. Cues provide information about an organism's state to others, but did not evolve to provide that information. Vomiting, for instance, is a cue of illness but not a signal of illness. Crying after stubbing one's toe, on the other hand, is probably a signal because the tears do not serve to heal the injured toe. Reliable cues of organism state can evolve into signals if sending that information increases the fitness of the signaler by changing the behavior of the receiver (e.g., Maynard Smith and Harper 2003; Tiokhin 2016).

(Starrett, 1993). Morphological and behavioral mimicry of ants has evolved independently at least 70 times, for example, and is known in 2000 species belonging to 200 genera in 54 families (McIver & Stonedahl, 1993).

Highly cooperative species are vulnerable to social parasites who mimic cooperative signals to receive benefits. To defend against social parasites, ants have evolved a sophisticated chemical recognition system enabling them to behave altruistically towards nestmates and reject non-nestmates (Lenoir et al., 2001). Despite their recognition system, ant species are parasitized by a number of arthropods, including butterflies, beetles, and even other ants. In fact, of the 10,000 or so known ant species, more than 200, or about 2%, parasitize other, often closely related, species, and in the unusually well-characterized ant fauna of Switzerland, about 1/3 of the species are parasitic (Buschinger, 1986). Social parasites are known in a wide variety of other invertebrate and vertebrate species. In vertebrates, well-studied examples include avian brood parasites, such as cuckoos and cowbirds, that lay eggs in the nests of other species to avoid the costs of brood care (Rothstein & Robinson, 1998). Deception is also widespread in primates (Bryne and Corp 2004).

Humans that have recently suffered adversity and are in need, but who also have conflicts with important social partners, face the formidable challenge of convincing them their need is genuine. A wife whose husband has died, for instance, might need assistance with childcare from her sister, or she might instead have enough money to hire help -- her true level of need is private information. Because assistance from her sister would be beneficial, she has an incentive to send a signal of need whether or not she is genuinely needy. Providing regular childcare is time consuming, so the sister would only want to provide it if the need is genuine. If the sisters had a history of conflict, one might be skeptical of the other's level of need.

Theorists have devoted considerable attention to mechanisms that can ensure signal credibility despite conflicts of interest. Costly signals are one well-studied mechanism to ensure the credible signaling of private information (Grafen, 1990; Spence, 1973; Zahavi, 1975). In brief, if the benefits of sending a signal necessarily outweigh the costs for signalers in (private) state A but not signalers in state B (e.g., because the benefits are the same for both but the costs are necessarily lower for signalers in state A), then agents can evolve to send the signal when in state A but not when in state B, and receivers can evolve to infer that the signaler is in state A, and respond to the signal in a way that benefits them both.

Hagen and colleagues have proposed that depression (Hagen, 1999, 2002, 2003, 2011; Hagen & Barrett, 2007; Watson & Andrews, 2002), self-harm (Hagen et al., 2008), and suicidality (Andrews, 2006; Nock et al., 2008; Rosenthal, 1993; Syme, Garfield, Hagen, 2016; Syme & Hagen, 2019) are costly signals that can credibly reveal the true level of need when conflicts with social partners render cheap signals, such as verbal requests, sad faces, or crying, unreliable. The basic logic is that for individuals whose fitness prospects are poor due to, e.g., loss of a mate, important social partner, or job, a dramatic reduction in productivity or putting one's life at risk, as seen in MD, is relatively low cost, whereas for individuals whose fitness prospects are good, these behaviors are quite costly. Thus, only individuals with poor fitness prospects can afford to send the signal, and skeptical social partners will only help those who send the signal.

A further consideration is that humans evolved in small, highly interdependent groups that included close kin, affines, and non-kin, and individuals had both shared and competing interests (e.g., Hagen 2003; Tomasello et al. 2012, Hill et al., 2011). Group members cooperated to raise their children and to provide food and protection. Individuals who reduced their productivity, or put their own lives at risk, imposed costs on the rest of the group (Hagen 1999; 2002; 2003). Theoretical models of bargaining show that if need is public information, such as clear indicators of physical injury, then interdependent social partners should immediately provide care (Rubinstein, 1982). If need is private information, however, and there are conflicts of interest that incentivize exaggerating need, costly signals are required to reveal the genuine level of need before social partners will help. Labor strikes provide an analogy: workers who forego their salaries during a labor strike are honestly signaling that their salary is too low. For workers making good salaries, a strike is too costly. But for workers whose salaries are genuinely insufficient, a strike has low costs because foregoing a poor salary has little cost. At the same time, the strike imposes costs on employers, who therefore have an incentive to increase wages (Kennan & Wilson, 1993). Similarly, depression and suicidality, by putting one's ability to provide benefits to others at risk, incentivize interdependent social partners to provide support that they might not otherwise provide. See Hagen (2003) for a thorough discussion of the bargaining model of depression.

The bargaining model is not a comprehensive model of MD or depressive states. Our comprehensive model of depressive states combines the psychological pain hypothesis with credible signals of need (bargaining). Most individuals experiencing adversity would experience a degree of psychological pain, but not all of them would need to send credible signals of need to social partners. Credible signals of need are only required when (1) individuals need help from social partners, but (2) have severe conflicts of interest with social partners that prevent cheap signals, such as verbal requests, sad expressions, or crying, from being believed or eliciting the necessary help.

Suicidality as a credible signal of need

Killing oneself is unlikely to be an adaptive strategy, especially for young and healthy individuals. Most suicidal thoughts and behaviors, though, do not result in death. In 2013, the U.S. adolescent suicide rate was about 8 per 100,000 per year, yet about 1500 times as many adolescents (12%) reported suicidal ideation (Nock et al., 2013). In low- and middle-income countries as many as 17% of adolescents report suicidal ideation in a 12-month period (Uddin et al., 2019). In the U.S., the ratios of suicide attempts to suicide deaths are approximately 100:1 and 10:1 among young adult women and men, respectively (Syme, Garfield, Hagen, 2016). This high survival rate is likely not entirely a product of advances in lifesaving technologies. Among the Bimin-Kuskusmin of Papua New Guinea, where as many as 10% of deaths over six generations were due to suicide, Poole (1985) reported that females attempted suicide twice as often as they died by suicide. In one Pacific island community, a local clergyman estimated that as many as 30-50% of the adult male population had attempted suicide (Rubinstein, 1995), whereas the rate of suicide deaths was about 40 per 100,000 per year (Hezel, 1989).

If suicidal behavior is a credible signal, it must only arise when less costly signals such as verbal communication are ineffective. Specifically 1) the victim faces of a fitness threat (adversity)

prior to suicidal behavior; 2) there is a conflict of interest between the victim and invested social partners such that the social partners are not otherwise willing to provide support based on less costly signals alone (e.g., verbal communication, sad expressions, crying); 3) the victim is otherwise powerless to single-handedly mitigate the fitness threat; 4) the suicidal behavior involves a low but non-zero risk of death; 5) social partners interpret suicidality as the victim being truly in need; and 6) the social partners assist the victim in mitigating the fitness threat.

In our research investigating suicidal behavior in 53 cultures using the Human Relations Area Files (Syme, Garfield, Hagen, 2016; Syme & Hagen, 2019), we found substantial cross-cultural support for each of the foregoing predictions: those who turned to suicidal behavior had suffered adversity such as abuse, forced and thwarted marriages, and status loss; were relatively powerless individuals caught in intractable conflicts with more powerful others or groups; and who, if they survived, received important benefits including social pressure against an abuser, avoidance of an unwanted marriage, or forgiveness for violating a taboo. The non-zero risk of death is an honest signal of the low value that victims place on their current circumstances. From this perspective, suicidal behavior is a last resort of the truly powerless.

Loss of interest in virtually all activities as a credible signal of need

If depression has an essence it is loss of interest in virtually everything. To diagnose MD according to DSM criteria, one symptom must be either loss of interest in virtually all activities, or depressed mood. 'Virtually all activities' could include one's job, relationships with friends, spouses, and children, as well as the basics of living, such as eating, grooming, bathing, and sex. In concert with other symptoms of depression, like fatigue, loss of energy, hypersomnia, and psychomotor retardation, loss of interest should have a substantial impact on one's ability to be productive and contribute to cooperative ventures.

One way to assess the effects of these symptoms is to estimate the impact of depression on economic productivity. Using a variety of methods, several studies have found that depression has a significant, negative impact on unemployment, absenteeism (missed days of work), and presenteeism (reduced productivity at work, usually measured in missed day equivalents). In a large, cross-cultural study, for instance, Ormel (1994) found that depression was associated with a two-fold increase in days of disability. Stewart (2003) found that, compared to the mean 1.5 hours per week of lost productive time among the general population of U.S. workers, depressed workers reported 5.6 hours per week of lost productive time, 81% of which was due to reduced productivity at work. In a nationally representative sample of U.S. adults, Kessler (2006) found depression accounted for 8.7 days of absenteeism and 18.2 days of presenteeism per year, for a total of 27.2 days of lost productivity per depressed worker per year. Beck et al. (2011) found that, among 771 depressed individuals, each 1-point increase in depression scores was associated with a 1.65% loss of productivity, from approximately a total 30% loss of productivity at the lower range of depression scores to about a 50% loss at the upper range. Khansa et al. (2020), in a random proportionate sample of Lebanese workers, found a strong positive association between depression scores and impairment in work and activity. In a global systematic review and meta-analysis of studies of the monetary cost of depression, König et al. (2020) found that the mean cost of reduced or lost productive was 128% higher in the depressed compared to non-

depressed group (the later included both healthy controls and individuals with, e.g., physical disorders).

A recent review of these and similar studies concluded that presenteeism rather than absenteeism accounted for more lost productivity, with more severe depression symptoms increasing the loss. The predominance of presenteeism's negative impact on productivity shows that depression is costly not only to the sufferer but also to co-workers and employers. In the U.S., the estimated value of this lost productivity exceeds \$84 billion/year (Evans-Lacko & Knapp, 2016). Evidence indicates that risk factors for workplace depression include job strain, low decision latitude, and bullying (Theorell et al., 2015). These results are consistent with the hypothesis that, under adverse conditions, depression serves as a bargaining strategy by imposing costs on the signaler as well as his or her social partners to compel them to make concessions.

Because depression hurts companies and not just individuals, much of the research on depression and work productivity has aimed to make a 'business case' for depression treatment, arguing that the benefit of increased worker productivity would outweigh employers' cost of treatment (Donohue, 2007). If depression is a genuine disease, this makes sense: treating it is in everyone's interest. If depression is not a disease, however, then treatment raises an important ethical issue. What if an employee were depressed because she was exploited by her employer, as the evidence suggests? In this case, chemically treating the employee's depression symptoms without improving her work environment would simply allow her employer to more effectively exploit her.

Sex differences in depression and bargaining

Females are at higher risk of depression than males (Kuehner, 2017). Hagen (2003) proposed that physical aggression and depression were complementary strategies to resolve conflicts. Physical formidability, in the form of upper body strength, provides an advantage in conflict, as does having social allies (Sell et al. 2012). Upper body strength is positively associated with propensity to anger, a history of physical fighting, and feelings of entitled access to resources (Hess et al., 2010; Sell et al., 2009; Sell et al., 2012). Men in many cultures use violence against their wives in service of their own fitness interests (Stieglitz et al., 2011; Stieglitz et al., 2012).

Hagen and Rosenström (2017) hypothesized that the dramatic sex difference in physical formidability help explain the dramatic sex difference in depression. Specifically, the physically formidable (often men) are able to resolve conflicts in their favor with physical threats, whereas the unformidable (often women) must more often resolve conflicts with depressive signaling that imposes costs on others by imposing costs on oneself. They found that in a nationally representative U.S. sample of adults, grip strength (an index of formidability) was negatively associated with depression and that sex differences in grip strength explained a substantial fraction of the sex difference in depression. Kerry and Murray (2018) similarly found that, in a sample of U.S. undergraduates, grip strength was negatively correlated with neuroticism, an important risk factor for depression, and that the sex difference in grip strength accounted for the sex difference in neuroticism.

Social partners interpret depression as an aversive signal of need

Several evolutionary researchers have questioned the bargaining model of depression because depressive behaviors are aversive to social partners (Allen & Badcock, 2003; Nesse, 2019; Nesse, 2000; Nettle, 2004; Myers et al. 2016). These researchers have failed to appreciate the central role of conflict in the bargaining model. Anger, an adaptive emotion, is also aversive to social targets because it threatens to impose costs on them, and that is precisely its evolved function (Sell et al. 2009). Under the bargaining model, depression and suicidality, like anger, function in part to impose costs on interdependent social partners so as to resolve conflicts in the signaler's interest; aversiveness is an evolved feature.

Stereotypical behaviors in depression and suicidality are stereotypical because they are signals that must be correctly interpreted by social partners. After confirming Coyne's (1976a, 1976b) finding that depression alienates others, including strangers in brief interactions (for a meta-analysis, see Segrin, 1992) researchers began to explore what it is that depressed people do or fail to do that leads to others' hostility and rejection. A large literature has since accumulated documenting differences in interpersonal interactions by depressed and non-depressed individuals. Findings include differences in communication 'style,' such as vocal characteristics, eye gaze, and gestures, and differences in 'content,' such as preferred topics and themes of conversation (Segrin, 1994; Segrin, 2000).

With regard to style, e.g., paralinguistic qualities like low rate, low volume, and low monotone pitch, depressed people appear to be "very 'skilled' at communicating sadness and despair" (Segrin, 2000, p. 385). With regard to content, depressed individuals are eager to self-disclose personally relevant negative issues, often at what are judged to be inappropriate times, and they view such topics as more appropriate for discussion than do the non-depressed (Segrin, 2000). Such self-disclosures have been shown to be a key ingredient in the rejection of depressed persons by others, and "may appropriately be understood as an attempt to elicit social support from targets" (Segrin, 1994, p. 657). In interactions with spouses, depressed individuals express anger and aggression, and make frequent demands for help. Segrin (1994) speculates that

The discussion of negative well-being, dysphoric feelings, and frequent demands for help may impose on other people, making them feel either responsible or obligated to offer assistance or find out the cause of the depression. This feeling of obligation or responsibility could potentially prove to be aversive and motivate withdrawal and rejection of the depressed person. (p. 660)

Excessive reassurance seeking (ERS) is another well-studied factor implicated in depression and rejection by others. ERS is defined as repeatedly requesting reassurance that one is lovable and worthy despite previous attempts by others to provide such reassurance. According to Joiner (1999)

The key idea is that the aversive properties of depression are not interpersonally aversive unless clearly and repeatedly signaled to other people and, further, signaled in such a way that others are both implicated in the development of the problem (e.g., "you don't really

love me anymore, do you?"), as well as overwhelmed by demands, whether implicit or stated, to solve the problem. (p. 272)

Our view is that excessive reassurance-seeking predisposes people to the development of depressive symptoms. Initially, this may occur as a function of growing demoralization of the high reassurance-seeking person, who has struggled to gain -- but has been disappointed with -- others' responsiveness. (p. 273)

A meta-analysis of 38 studies found ERS to be moderately associated with depression ($r = .38$) and weakly but significantly associated with rejection ($r = .14$), although the relationship with rejection could be stronger when the seeker is depressed (Starr, 2008).

Because depression alienates others, however, differences in the style and content of interpersonal interactions of the depressed vs. non-depressed, rather than being seen as an aversive signal of need, are interpreted as *deficits*, such as social skill deficits or 'excessive' reassurance-seeking (Segrin, 1994; Joiner et al., 1999). Yet infant cries, clear signals of need, motivate caregiving, in part by being so aversive that caregivers work to terminate the cries. The noxiousness of these cries can also evoke abuse or avoidance by caregivers (Owings & Zeifman, 2004).

Normal sadness can also alienate others. Young children, for instance, find sad people to be rather unlikeable (Glasberg, 1981; Glasberg, 1982), and report controlling their own sadness, especially around peers, because they fear negative interpersonal consequences (Zeman, 1996). Adults commonly regulate their emotions too, including sadness, in part because there are negative social consequences to emotional expression, in addition to positive ones (for reviews see Gross, 1998; Gross, 2002; Ochsner, 2005).

Why would others respond negatively to signals of need? From an evolutionary perspective the real question is, why would others respond *positively* to signals of need? As Machiavelli recognized 500 years ago, self-interested agents frequently turn against those in need because helping them means paying a cost with no guarantee of a compensating benefit. Needless to say, people do help the needy, at least some of the time, and have done so over the course of human evolutionary history (otherwise signals of need, like infant cries and universal facial expressions of sadness and grief, would probably not have evolved), but social partners will only provide help when it is in their fitness interests to do so. If there are few conflicts among social partners, and relationships are valuable, then each will see it in their benefit to provide help to a child, spouse, relative, friend, or co-worker when he or she is in need. In this case, cries, sadness, grief, and simple verbal requests should work well to signal need. But like aggression, signals of need do not always end in an outcome in the signaler's favor.

A reasonable summary of results to date is that depressed people have a distinctive style of communication that is easily recognized by others and is effective at expressing sadness and despair (but not other emotions), and that the style and content of their conversations and interactions signal, correctly in our view, that they have problems and needs and want to talk about them and gain reassurance and support.

Evidence that depression elicits benefits

If depression functions to signal need and to bargain for more care from social partners, then it should elicit increased help and other benefits from them, at least some of the time. Because depression is almost universally regarded as an illness, very few studies have explored possible benefits of depression. Nevertheless, several studies have shown that they exist.

The literature on depression and family dynamics shows that depression elicits benefits from others, so much so that many researchers worry these benefits 'reinforce' the depression. An advantage of several of these studies, compared to most interpersonal studies of depression, is that they observe depressed individuals interacting with social partners in real time, sometimes in their own homes, coding the temporal sequence of depressive behaviors, such as complaints, downcast eyes, and crying; aggressive behaviors, such as threats and angry gestures; facilitative behaviors, such as approval, smiles, and a warm voice; and so forth.

Biglan et al. (1985), for instance, asked couples to discuss two important marital problems for 10 minutes each. For couples in which the wife was depressed, a good deal of the discussion focused on her and her complaints, with the husband proposing most of the solutions, whereas in couples without depression, husbands and wives proposed about the same number of solutions. For those couples experiencing marital distress, the wife's depressive behavior reduced her husband's subsequent aggressive behavior. His facilitative behavior, in turn, reduced his wife's subsequent depressive behavior (as did his aggressive behavior). Hops (1987), in one-hour observations of this same sample in their homes, confirmed that mothers' dysphoric behavior reduced subsequent aggressive behavior by fathers and children. Caring behavior on the part on children and fathers, in turn, reduced mothers' dysphoric behavior. Responses by third parties to both written and video depictions of dysphoric and distressed behavior provide further evidence that although these induce negative feelings in others, such behaviors also prompt others to be solicitous and refrain from aggression (Biglan et al., 1989).

These results have been extended to depression in children and adolescents. Behavioral observations of depressed juveniles interacting with their parents found that depression elicited sympathetic, facilitative, and problem-solving behaviors from parents and suppressed parental aggression, which in some cases depended on the sex of the parent (Dadds et al., 1992; Sheeber et al., 1997).

To experimentally control the depressive behaviors thought to be responsible for interpersonal effects, Stephens et al. (1987) trained confederates to enact depressive, anxious, and normal roles. A sample of female college students then interacted with one of the confederates in a 15-minute conversation before completing questionnaires about the experience. As expected, subjects viewed the 'depressed' confederates more negatively than they did the 'anxious' or 'normal' subjects. Despite their aversive impact, depressed confederates elicited more verbal support and advice and less negative feedback than the other confederates. Subjects appeared to feel obligated to help the depressed and withhold negative reactions.

In another study of college students, Hokanson et al. (1986) compared roommate relationships in which one roommate was dysphoric to relationships with two non-depressed roommates. They

found that dysphoric students initiated the roommate relationship with relatively dependent behaviors, which increased over the three months of the study, and that the roommates showed some evidence of progressive increases in caretaking behaviors. Finally, In a 25-year longitudinal study of depression and anger among Canadians ($n = 944$), Galambos et al. (2018) found that higher anger at ages 18 and 20 years predicted increased social support one and two years later; and that higher depression at age 19 years predicted greater social support one year later (but somewhat older men with higher depression experienced subsequent decreases in social support a few to many years later).

In summary, this research found that depression in naturalistic and laboratory settings elicits attention, concern, potential solutions to problems, and other facilitative behaviors, and care, at least some of the time, and suppresses aggression in situations that frequently involve conflict. Privately, people are alienated by depression, but their actual behavior, at least over the short-to-medium term, aims to help the dysphoric and distressed.

Many researchers have expressed concern that these positive effects of depression could reinforce depressive behavior (e.g., Biglan et al., 1985; Biglan et al., 1989; Hops et al., 1987, Sheeber et al., 1997; Sheeber et al., 2001; Siegel, 1990). Given that the depressed almost always have serious, genuine problems, however, a fact strangely missing from much of the literature on interpersonal relations and depression, eliciting problem-solving behavior and care from social partners is, in our view, a valuable function of depression. Reducing aggression in families plagued by conflict, furthermore, strikes us as a good thing.

The alienating qualities of depression could diminish the enthusiasm of social partners to continue their relationship with the depressed. In situations where switching partners is relatively easy, the depression strategy will therefore frequently fail. Switching partners, however, is often difficult. A father of a newborn with a depressed mother can't simply find a different, non-depressed mother to help care for the newborn. Parents of a depressed adolescent can't simply decide to have another child to replace the unhappy one sitting at their dinner table. Even college students are 'stuck' with their depressed roommates for at least a few months, and feel obligated to offer care.

Think about the screaming infant. It is the very aversiveness of the screaming that motivates rapid attention to the infant's needs by parents who can't exchange it for an infant that doesn't scream. In fact, there are several game theory models of begging by juvenile animals that are conceptually similar to our model of depression. In these models, juveniles want more food from parents than parents are inclined to provide. To increase feeding, juveniles threaten their own survival by crying loudly, potentially attracting predators to the nest or expending excessive energy, thereby blackmailing or credibly signaling their parents to increase feeding (Zahavi, 1977; Bergstrom & Bergstrom, 1999; Eshel & Feldman, 1991; Godfray, 1991; Godfray, 1995; Godfray & Johnstone, 2000; Royle et al., 2002; Soltis, 2004; empirical evidence for begging costs is mixed; for review, see Moreno-Rueda, 2007).

Unlike screaming infants, however, whose needs for food, attention, or a diaper change are, in most cases, easily met, the depressed often have serious problems whose solution might require substantial restructuring of existing social arrangements, leaving social partners expending more

effort and/or accepting less return. If depression were not burdensome and aversive to social partners, they would have little incentive to make these changes.

Improved conditions are associated with remission

If the bargaining model is correct, depression should lead to long-term life improvements, at least some of the time, after which depression would cease. In support, there is increasing evidence that *positive* life events closely precede, and may well cause, recovery from depression. In a study of depression in working class women, for instance, Brown et al. (1988) found that a reduction in life difficulties, presence of difficulty-reducing events, and the occurrence of 'fresh start' events were correlated with recovery. Fresh starts are events, such as getting a job after months of unemployment or starting a new relationship after many months single, that seem to promise new hope against a background of deprivation (Harris, 2001). Similarly, Brown et al. (1992) found that women's recovery from depression was associated with a prior positive event. These events were characterized by one or more of three dimensions: anchoring, involving increased security; fresh start, involving increased hope arising from a lessening of a difficulty or deprivation; and relief, involving amelioration of a difficulty without a fresh start. Fresh starts were particularly associated with recovery from depression.

In primary care patients with anxiety or depression, Leenstra et al. (1995) found a two-fold excess of positive life changes in the three months prior to recovery compared to base rates, with difficulty reduction as the most important recovery-enhancing factor. They conclude that positive life change facilitates recovery but is neither a necessary nor sufficient condition for remission. In an analysis of a sample that overlapped with the previous study, but restricted to those with depression, Oldehinkel et al. (2000) explored whether positive life change interacted with sex, neuroticism, self-esteem, coping style, and other factors to facilitate remission. They found that positive life-change reduced time to remission most for women and those with high levels of neuroticism. In yet another analysis of the data from Leenstra et al. (1995), Neeleman et al. (2003) found that irrespective of diagnosis and symptom intensity, positive life change increased remission rates 2.9-fold. Remission rates were higher in patients with larger social networks and who sought more help and this was partly because such persons experienced more positive life change earlier during episodes. In a naturalistic, six-month prospective study of adult patients in general practice with depressive, anxiety, or panic disorder, Ronalds et al. (1997) found that at follow-up the most important predictor of improvement was reduction in marked difficulties over the prior six months. In a nine-year longitudinal study of 2052 participants with anxiety or depression, or healthy controls, Hovenkamp-Hermelink et al. (2019) found that more positive life events at T2, T4, and T9 (but not at T6) predicted a subsequent decrease in depression severity.

Our model of depression requires that sufficiently improved circumstances cause depression to remit. These studies provide evidence that they do. In some cases, the improvement came about because the depressed individuals themselves initiated positive life changes, such as starting school after years as a housewife (Harris, 2001). This is consistent with the adaptive rumination hypothesis, which proposes that depression engenders a sustained focus on solving one's problems. In other cases, however, the positive life changes were the result of a new development not under the direct control of the depressed subject's agency or control, which, in one study, was true of 76% of the instances of fresh starts and difficulty reductions (Brown et al.,

2009). Of the latter, it was sometimes obvious that the subject's depression played no role in the positive event, such as learning that a cancer diagnosis was incorrect. Other times, however, such as starting to work in a boyfriend's shop, it seems possible that depression might have beneficially influenced the social network.

The high fraction of positive events not under direct control of the depressed subject, combined with the finding that more positive life changes occurred to depressed subjects with larger social networks (Neeleman et al., 2003) could mean, speculatively, that depression causes social partners to make changes beneficial to the depressed. On current evidence it is also possible, of course, that depression has no effect or even decreases the probability that a positive life change will occur. However, we have already seen that depression does elicit short-term helping behavior from social partners, so it might also elicit behavior from social partners that provides long-term benefits involving reduction of difficulties or fresh starts, which, as these studies strongly suggest, then lead to remission.

Concluding remarks

The adversities that cause MD, such as death of a loved one, loss of a mate, loss of resources, and physical attack, were ubiquitous over the course of human evolution. Successfully responding to adversity required a properly functioning nervous system, and brain dysfunctions during common adverse events would have been strongly selected against. Mainstream psychiatry's claim that common adversities cause brain dysfunctions in a sizable fraction of the population is remarkable and unlikely to be true.

Adversity can unfavorably tip life's balance of effort vs. reward, often causing low mood, sadness, or grief. This aversive psychological pain motivates a search for a solution that will favorably rebalance reward vs. effort. Many individuals in such circumstances can unilaterally redress imbalances by rearranging their lives. Some individuals, though, lack sufficient options, resources, or physical or political power to solve their problems by themselves, and need help from relatives, friends, and other social partners, which they obtain by asking or by signaling need with facial expressions, crying, and other behaviors. Yet if key relationships are marked by anger and conflict and their partners do not hold them in high esteem, it is difficult to solve these problems because social partners either are skeptical that the needs are real or do not want to provide additional help. Without their help, however, the problems might be impossible to solve. Folks in this situation feel helpless, hopeless, and trapped. Instead of responding with 'normal' sadness, they become depressed.

Depression, we propose, is in part a credible signal of need involving distinctive behaviors and reduced effort in collective enterprises such as work and parenting. This signal is credible because reduced effort is more costly to those whose lives are going well than it is to those whose lives are not going well -- only the genuinely needy can afford this signal. This dysphoric behavior is easily recognized by social partners, and the message is clear: the depressed have problems, want to talk about them, and need help. Privately, social partners react negatively to this signal of need, probably because it entails reduced benefits from the depressed person and the redirection of more time and effort toward the depressed person.

Nevertheless, social partners do try to help, suppressing their anger and aggression, and offering attention, care, potential solutions to problems, and other facilitative behaviors. Perhaps, despite conflict in the relationship, the depression has convinced them that the needs are genuine. Yet in the short-term this help rarely leads to immediate recovery from depression. It is the depressed's lack of response to offers of help, in fact, that many researchers believe is one of the most aversive qualities of depression. Just as workers will not end a strike until their employer offers a significant increase in salary, most initial offers of help do not lead to recovery from depression because the depressed have serious problems and the initial offers of help are 'too small' to solve them. The depressed need to compel substantially better offers from reluctant social partners.

In our model, depression facilitates positive developments that eventually lead to recovery. Perhaps intense rumination has uncovered a solution to a social dilemma or makes the depressed more attentive to new opportunities, for instance, or prolonged anhedonia or suicidality has extracted substantial concessions from social partners. When difficulties are reduced or new profitable opportunities arise, the depressed recover.

We have not proved our case. Far from it. Serious limitations in our argument include that although most of the correlations we cite are well-established, the causal relationships we assume are less so. The causal relationships we assume, however, are usually the ones for which depression researchers have found supporting evidence. The primary missing piece in our argument is whether depression brings about reductions in difficulties or other positive life events that are thought to lead to recovery. Given that depression does elicit help from others, however, it is reasonable to hypothesize that, in many cases, it might elicit enough help to change one's life for the better.

Our main point is not that depression must be an adaptation, it is that there is little reason to believe most cases of MD are brain dysfunctions, and we can account for most of the facts about MD, including its aversiveness and undeniable costs such as suicidality and reduced productivity, with a relatively parsimonious theory that makes four simple, intuitive assumptions: (1) psychological pain is an aversive but functional response to many forms of adversity, (2) in many situations, problems cannot be solved alone, (3) there are conflicts with social partners, and (4) need is private information. In these circumstances, depression as credible sadness emerges as a functional, not pathological, response to adversity.

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