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Review

Toward a revised evolutionary adaptationist analysis of depression: the social navigation hypothesis

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Abstract

Evolutionary biologists use Darwinian theory and functional design (''reverse engineering'') analyses, to develop and test hypotheses about the adaptive functions of traits. Based upon a consideration of human social life and a functional design analysis of depression's core symptomatology we offer a comprehensive theory of its adaptive significance called the Social Navigation Hypothesis (SNH). The SNH attempts to account for all intensities of depression based on standard evolutionary theories of sociality, communication and psychological pain. The SNH suggests that depression evolved to perform two complimentary social problem-solving functions. First, depression induces cognitive changes that focus and enhance capacities for the accurate analysis and solution of key social problems, suggesting a *social rumination function*. Second, the costs associated with the anhedonia and psychomotor perturbation of depression can persuade reluctant social partners to provide help or make concessions via two possible mechanisms, namely, honest signaling and passive, unintentional fitness extortion. Thus it may also have a *social motivation function.*

Keywords: Adaptation; Anhedonia; Cognition; Depression; Emotional pain; Extortion; Honest signal; Psychomotor; Parasuicide; Reciprocity; Social analysis; Suicide

1. Introduction

Depression is an affective and psychophysical state that is emotionally painful, affects concentration, reduces the pleasure derivable from activities such as eating and sex (anhedonia), and causes troublesome changes in motor activity and social interaction. Symptoms commonly intensify over time (NIMH, 1994) and may lead to suicidality [American Psychiatric Association (APA), 1994]. While many functional hypotheses for depression have been proposed (Nesse, 2000), the predominant medical view is that depression is a mental disorder (APA, 1994). Even among evolutionary theorists, clinical depression is often seen as maladaptive. Depression

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surely entails costs for the depressive, but this alone does not justify the conclusion that it is maladaptive.

Some psychiatrists are urging that greater empirical and theoretical attention be paid to depression's possible evolved functions (Abed, 2000; Nesse, 2000). There are good reasons to consider adaptationist hypotheses for depression. For instance, unlike other mental phenomena classified as disorders (e.g., schizophrenia), depression is very prevalent and the capacity for it may be cross-culturally universal (Nesse and Williams, 1994). There are reports of depression in traditional societies such as the Ache of Paraguay (Hill and Hurtado, unpublished observation) and the !Kung of southern Africa (Howell, 1979). Moreover, the incidence within cultures is often high. The point prevalence of major depression in the US is approximately 5–10% (Kessler et al., 1993), and the lifetime risk is estimated to be about 20% (Kessler et al., 1994). If depression is an adaptation, some treatments and cultural trends may interfere with functions it evolved to serve.

In this paper we present the *Social Navigation Hypothesis* (SNH) of depression. We begin with a discussion of pertinent evolutionary theory that serves as a background for the hypothesis. Then, we review evidence that depression is associated with social problems. We go on to suggest that depression plays two complementary roles in dealing with particularly important and troublesome social problems by (1) focusing limited cognitive resources on planning ways out of complex social problems and (2) motivating close social partners (friends, mates, family) to provide problem-solving help and concessions, especially in cases where they are initially reluctant to do so. Depression gets its motivational power by virtue of the costs it imposes on the depressive and on close social partners who have a positive fitness interest in the normal functioning of the depressive.

Today's social environments differ from ancestral ones in ways that could affect the prevalence and intensity of depression. Modern social complexity and dynamism probably increases the context for ruminative and motivational depression, because people face an ever-changing array of fitness enhancing opportunities, but are blocked from or do not understand how to access them. Moreover, people tend to have a greater number of positive fitness partners in modern societies and this could increase the incidence of depression. At the same time, these partners become more replaceable and so the average fitness interest amongst them is lower. Reduced fitness interests amongst partners may increase the intensity of depression needed to motivate partners to help.

The ruminative and motivational functions of depression may correlate with so-called minor (subclinical) and major (clinical) depression, respectively. However, we think these functions overlap and complement each other. Our model accords with growing evidence that, while depression varies greatly in severity, it is a continuous phenomenon (Kumar et al., 1998; Tennen et al., 1999). The SNH provides a framework that has the potential to explain the full array of psychological and motor changes typically associated with human unipolar depression.

2. The adaptationist approach to depression

2.1. The adaptationist approach and alternative adaptationist hypotheses

An adaptationist hypothesis proposes that a trait was designed by selection for a specific fitness enhancing function. The burden on adaptationists is like that on other scientists. Before accepting a hypothesis, the adaptationist must show all its alternatives to be very unlikely (Andrews et al., 2001). One way to acquire information about the relative likelihood of alternative hypotheses is by examining the design features and development of a trait to infer its potential adaptive functions (Williams, 1966; Thornhill, 1997), a method called *reverse engineering analysis*. The value of an adaptationist approach is strategic. Adaptationists are not committed to the idea that all traits are adaptations (Andrews et al., 2001). They test adaptationist hypotheses to determine whether *or not* a trait evolved to perform a specific function.

The prevailing medical view is that depression is maladaptive. Depression is overtly costly to the depressive, and it is prevalent (i.e., genes for depression must have undergone substantial evolution). Genes for depression would not have evolved unless they conferred net benefits, and there are two pos-

sible explanations for their evolution. First, depression itself may have had a beneficial effect that outweighed its costs (i.e., depression could be an adaptation). Second, depression may have evolved indirectly because its genetic underpinnings code for other traits (i.e., by pleiotropy) whose beneficial effects outweighed the costs of depression—the maladaptive byproduct hypothesis (MBH). For instance, Akiskal (2001) proposed that depression is the maladaptive byproduct of a human adaptation for sensitivity to suffering; this adaptation may lead to depression as the result of developmental or current traumatic experience.

The MBH is not invalid, but it is extremely difficult to test. Save for some life history traits, there is no general theory, or clear predictions, about which traits are linked. Confidence grows in the MBH *only* to the extent that our confidence in adaptationist hypotheses decreases. We can *only* be sure that depression is not an adaptation to the extent that we consider adaptationist hypotheses, subject them to the rigorous evidentiary standards required under reverse engineering analyses, and systematically reject them. Since many depression researchers are very reticent about considering and testing adaptationist hypotheses for depression, we still do not know whether depression is an adaptation despite decades of research. For this reason, we limit discussion to explicitly adaptationist hypotheses. Moreover, any idea proposing a function for depression is an adaptationist hypothesis. We limit discussion to three major hypotheses as a thorough review of adaptationist hypotheses is beyond the scope of this paper (for a more extensive review, see Nesse, 2000).

2.2. Alternative adaptationist hypotheses

Perhaps the first adaptationist hypothesis of depression posited that it constitutes a ''cry for help'' (Lewis, 1934). As will be seen, our SNH includes a detailed Darwinian elaboration of this view.

The social yielding hypothesis proposes that depression is an adaptation for forcing the loser of a conflict to: (1) cease competing with the winner, (2) accept the fact that he has lost, (3) signal submission and thereby stop oppressive behavior by the winner (Price et al., 1994; Gilbert and Allan, 1998). It proposes that depression forces acceptance of a fitness-limiting situation, especially loss of rank. It sees prolonged depression as a *maladaptive* inability to accept the loss of an unwinnable competition, and it does not consider depressive ruminations to be useful for complex problem-solving. In contrast, the SNH proposes that depression plans ways and motivates help for getting out of complex fitness-limiting social situations (i.e., winning). The SNH suggests that persons who have lost social status may need enhanced social analysis and motivation abilities to mitigate their loss.

The more recent ''strike hypothesis'' (Hagen, 1999) proposes that clinical depression helps the depressive gain greater investment from an exploitative partner. The strike metaphor resembles our independently derived extortionary view of depression. However, the strike hypothesis disavows any ruminative function for clinical depression. Moreover, the strike hypothesis implies that depression is designed to solve intensive, dyad-specific problems, whereas the SNH suggests that motivational depression may play its prime role in solving problems that have a far more systemic basis in the social network.

2.2.1. The functions of emotional pain and pleasure

The SNH derives from a general evolutionary theory about the functions of emotional pain (Thornhill and Thornhill, 1989; Nesse, 2000). This theory has received empirical support in relation to trauma experienced by rape victims (Thornhill and Palmer, 2000) and jealousy (Buss, 2000). The SNH builds on these ideas by providing new or more detailed functional design analyses of depression's core symptomatology and the social problem it is designed to solve.

The evolutionary theory of emotional pain posits that it is analogous to physical pain. Physical pain draws attention to a problem in the environment that threatens damage, or is causing damage, to bodily tissues. It motivates cognition and behavior that, at least in ancestral environments, would tend to remedy the problem. Analogously, emotional pain is thought to draw attention to a problem in the social environment that could, if not fixed, have detrimental fitness consequences, and to motivate action designed to remedy the problem. Moreover, the intensi-

ty of pain is expected to correlate with both the fitness consequences that are at stake and the cognitive difficulty of the social problem. Similar arguments explain the purpose of emotional pleasures—they reward us for fitness enhancing accomplishments (Buss, 1999). We feel pleasure when we have sex or eat nutritious foods because natural selection has wired our nervous system to provide us with a positive feedback mechanism for pursuing goals that enhanced inclusive fitness in ancestral environments.

We also propose that emotional pain forces the individual to consider a wider range of strategic options than would otherwise be considered, including costlier and riskier ones. Thus, the SNH predicts that depressives will mentally simulate a greater number of possible solutions to their social problems than non-depressives, they should consider a greater range of strategic options, and the options that they consider should often be costlier and riskier.

Depression is not only painful, the inability to feel pleasure may help the depressive sustain cognitive effort on the problem by preventing cognitive distractions. Anhedonia may also increase the range of strategic options under consideration by freeing the individual from pleasure based attachments that may impede adaptive changes in social relationships.

Different forms of emotional pain should be designed to solve different sorts of problems (Nesse, 2000); depression is only one form of emotional pain. Whereas many forms of emotional pain are specific to particular social interactions (e.g., jealousy and sexual infidelity; Buss, 2000), the functional domain of depression may be social complexity. The utility of depression for finding ways out of bad social situations may explain why depressives are often co-morbid for other forms of emotional pain. For instance, a mate's infidelity could cause depression and jealousy if the problem is sufficiently difficult to resolve. Co-morbid anxiety may frequently exist because of the dire consequences of misjudging how social partners will respond to a problem solving strategy.

3. Depression is strongly associated with social problems

Social problems are characterized: (1) by social dependency, where the fitness of one social interactant is dependent upon the behavior of another, and (2) by interpersonal conflict of interest. Depressives exhibit many cognitive characteristics of enhanced social dependency (Coyne and Whiffen, 1995), including an enhanced desire for social approval and success (Sheppard and Teasdale, 1996). Moreover, people from more interdependent societies may be more depressed than people from less interdependent societies (Anderson, 1999).

Situational and cognitive indicators of social conflict also are strongly related to depression. Depressives tend to be more openly aggressive toward their spouses than non-depressives (Biglan et al., 1985) and the degree of negative interaction is related to the degree of marital conflict (Schmaling and Jacobson, 1990). While supportive social networks provide some protection against depression (Paykel, 1994), the presence of social conflict is an even better predictor of depression (Coyne and Downey, 1991). For instance, marriage is a mild buffer to depression relative to being single or divorced. However, the odds of depression among people who do not get along with their spouse is an astounding 25:1 (Weissman, 1987).

Finally, the conditions that alleviate depression also suggest that it serves a social problem-solving function. For instance, if depression serves such a function, then it should end when the social problem is solved. Recovery from depression is hastened by improvements in social relationships and strong social support (Brown et al., 1988; Andrews and Brown, 1995; Brugha et al., 1997). Similarly, adaptive depression should abate if the problem is perceived to be unsolvable. Clinicians have long known that depression is sometimes resolved only when the sufferer gives up the pursuit of an unobtainable social goal (Price et al., 1994; Nesse, 2000; Klinger, 1975).

4. Social rumination function

The highly contingent nature of social problems favors those who can accurately predict and influence the behavior of their social partners (Humphrey, 1976). Accurate behavioral prediction sometimes requires accurate inferences about the intentions of others. This can be difficult when multiple

intentions are possible, because it requires the collection and processing of information about all the actor's possible mental states (Andrews, 2001). Similarly, successfully influencing the behavior of a social partner often requires considering how the partner will react to one's own behavioral choices. Moreover, one's choices may have direct and indirect effects on third parties—their reactions also must be anticipated. The successful social manipulator will often be the one who best analyzes the decision-tree (really, a decision-web) of the choices and responses of all parties in the social network. Thus, finding solutions to social problems can require massive cognitive effort.

Cognitively demanding social problems probably were important in shaping some of the unique aspects of human intelligence over evolutionary time (Alexander, 1989; Humphrey, 1976). If so, then people should have evolved psychological adaptations governing the allocation of cognitive resources toward finding solutions. Since cognitive capacities are limited, fewer resources are available for planning and pursuing other activities, goals, and interests as more cognitive resources are allocated to the task of solving a problem. So, the relative importance of a problem, coupled with its difficulty, should determine the proportion of cognitive resources allocated to finding a solution.

Normally, concentration on a given task or problem is achieved by temporarily putting alternative attractors of attention aside, but interest in these alternatives is easily renewed as the situation warrants. Working in this way may be associated with strong positive affect (e.g., interest or inspiration). However, the ability to expend sustained cognitive effort analyzing a major threat or limitation to fitness rooted in a complex stubborn situation may require an enhanced ability to resist and avoid distractions. At some point, such a problem may be so important to solve, yet so difficult, that it pays to shut down hedonic interests until the problem is solved or it becomes clear that further cognitive effort cannot effect a solution. Consistent anhedonia is a hallmark of depression and may reflect the importance of resisting hedonic distractions. Thus, some depression should ensue whenever a person is faced with a social problem so difficult that the flexible pursuit of hedonic interests is likely to delay or interfere with attempts to resolve it. The SNH is the only adaptationist hypothesis that explicitly requires enhanced social analysis to be associated with depression.

Physical activity also requires the use of cognitive resources (e.g., navigation or planning activities like hunting or shelter-building) and may lead to encounters with predators or conspecifics to which further cognitive resources must then be devoted. The reduced activity and sociality that accompany depression may cause the depressive to avoid events that reduce focus on a crucial problem. Supporting this view is the fact that psychomotor retardation in depression is positively correlated with anhedonia (Lemke et al., 1999). Moreover, the degree of psychomotor retardation is a better predictor of the ability to concentrate on novel tasks than is the severity of depression itself (Lemelin and Baruch, 1998).

The social situations that put one at the greatest risk of depression should place great cognitive demands on the depressive. Stressful life events such as unemployment or divorce are well-known antecedent risk factors for depression (Kessler, 1997). By themselves, such events are mild risk factors for depression, but if they lead to the perception of social entrapment or defeat the risk of depression is much greater (Brown et al., 1995; Gilbert and Allan, 1998). Great cognitive effort may be required to find means to escape such entrapping events.

If depressives are faced with social problems that stubbornly resist solution, they should perceive that their social situation is undesirable and difficult to change. This perception is a strong psychological predictor of subsequent levels of depression and non-fatal suicide attempts (Alloy et al., 1999; Baumeister, 1993). Depression researchers refer to this perception as *hopelessness* (Abramson et al., 1989). *Desperation* may be more appropriate because it does not imply that a problem is perceived to be unsolvable. Rather it implies that the problem is important to solve, it so far has resisted solution, and resolution may require risky or unusual strategies. Again, depression should abate when a problem is perceived to be truly unsolvable.

Unlike other psychic states regarded as psychopathologies, depression appears to induce cognitive changes that help depressives build a model of their social situation and plan actions for resolving important problems. For instance, depressives exhibit signs of focusing attention on social problems.

Studies consistently show that depressives are relatively consumed with negative thoughts (Haaga et al., 1991). However, the content of their thoughts is specific: depressives perceive themselves to be in an unenviable social situation and desire to gain more social approval and success (Sheppard and Teasdale, 1996). Depressives also pay closer attention to social information, including social comparison information, and process it more extensively than non-depressives (Gannon et al., 1994; Marsh and Weary, 1994; Swallow and Kuiper, 1993; Weary et al., 1994; Yost and Weary, 1996).

Depression should not only influence the proportion of cognitive resources devoted to a social problem, it should also affect the *style* in which information is processed and interpreted. For instance, if depressives are caught in complex social situations whose outcomes could dramatically affect their fitness, then it should be more important for them to be realistic or conservative about their strategic assets and liabilities, as well as their strategic position and the options they have available to them. However, if the social situations that non-depressives are in are not as complex or important as those of depressives, then they may strategically benefit or suffer smaller costs from being less realistic about such things (Andrews, 2001). These cognitive differences may make depressives appear more pessimistic than non-depressives about themselves and their future.

Depressives may be less biased about their phenotypic quality than non-depressives (Joiner et al., 1994). Moreover, depressives and non-depressives exhibit different attributional patterns for successes and failures. Non-depressives tend to exhibit a *self-serving bias* (SSB; Sedikides, 1993), attributing their successes to ability and their failures to chance or lack of effort. They also are more prone to attribute successes of others to chance or effort and their failures to lack of ability (Sedikides, 1993). Conversely, depressives are less likely to attribute their successes to ability and their failures to chance or lack of effort. The less biased attributional pattern of depressives is called the *depressive attributional style* (DAS; Sweeney et al., 1986). The DAS may be more accurate. The SSB is partly distorted by strategic self-enhancement (Sedikides, 1993; Sedikides et al., 1998), which suggests that the DAS may not be so distorted. Moreover, depressives evaluate themselves and others more even-handedly for personality traits, the reasons for success and failure, and future outcomes (Brown, 1986; Tabachnik et al., 1983; Ahrens et al., 1988; Alloy and Ahrens, 1987), which suggests that the DAS may be more accurate (Ackermann and DeRubeis, 1991).

However, depressives should not be more accurate than non-depressives in all cognitive domains. If depressives are facing difficult problems, then their attention should be biased toward information that addresses the nature and source of their problem. For instance, depressives tend to focus on, and even show a preference for, negative social feedback (Giesler et al., 1996). Thus, their recall of feedback tends to be more negative than it actually was (Ackermann and DeRubeis, 1991). Negative feedback may be useful for identifying social problems and anticipating the full range of potential partner responses to one's efforts to solve a problem, including worst case scenarios.

The performance patterns of depressives on particularly difficult cognitive tasks also indicate that they are facing and primed to solve difficult social problems. Depressives exhibit diminished performance on cognitively demanding tasks of a non-social or abstract nature—intelligence tests, general learning and memory tasks, reading comprehension, and organization and clustering tasks (Hartlage et al., 1993). This is *expected* if their cognitive efforts are focused elsewhere.

Conversely, depressives often outperform non-depressives on difficult tasks that tap social problem-solving skills (Yost and Weary, 1996; Lane and DePaulo, 1999), and are more accurate than non-depressives in judging the control they have over contingent outcomes (Ackermann and DeRubeis, 1991; Alloy and Abramson, 1979). Depressives may be cognitively primed to accurately judge their degree of control over contingent outcomes, because planning a successful solution to a social problem often depends on accurately assessing their degree of control over others.

Making an accurate inference about the mental state of another when several are possible is a demanding task, because one must evaluate all possible alternatives (Andrews, 2001). Depressives are less likely to make the *fundamental attribution*

error (FAE; Yost and Weary, 1996). This is an error of judgment where an actor's mental state and behavior are assumed to correspond to a degree that is logically unwarranted by the situation (Andrews, 2001). Non-depressives may tend to make the FAE because they are less dependent upon others and have little incentive to put out the effort needed to make a logically correct inference. Instead, they may resort to quick, error-prone heuristics to make inferences about the mental states of others. Depressives, because they are more socially dependent, have a greater incentive to make accurate behavioral predictions and may be primed to make more accurate attributions about mental states. Consistent with this reasoning, people tend to avoid the FAE when their own outcomes depend on making more accurate attributions (Vonk, 1998). Moreover, individuals from more interdependent societies are less likely to make the FAE or show the SSB (Choi et al., 1999). There is evidence that people from more interdependent societies are less likely to exhibit the SSB because they tend to be more depressed (Anderson, 1999). This raises the intriguing possibility that culture-specific FAE prevalences are also mediated by depression.

Finally, strong evidence for a rumination function comes from studies indicating that the changes in cognitive and activity patterns associated with depression are modulated by serotonin (5-HT) (Brewerton, 1995). Low serotonin retards physical activity (Jacobs and Fornal, 1997) and enhances performance on cognitively demanding tasks (Buhot, 1997).

The depressive's close social partners will experience costs to the extent that ruminative depression interferes with activities in which they have a positive fitness interest. Much ruminative depression may be voluntarily hidden since critical review of interpersonal relationships is often best done in secret. There should be selection to minimize costs of ruminative depression imposed on social partners when it is important to maintain relational stability at the social planning stage. Yet some imposition of costs imposed on the depressive or social network may be unavoidable, drawing the attention of others. Costly episodes of ruminative depression probably selected for the responsiveness in social partners necessary for motivational depression to evolve.

5. Social motivation function

As depression intensifies, enhanced anhedonia and psychomotor perturbation make the depressive increasingly disinterested in normal fitness-related activities and physically unable to pursue them. This disinterest and incapacitation gradually spreads to more fitness domains, including self care. Yet depression is not only costly to the depressive. Social partners with a positive fitness interest in the normal activities of the depressive necessarily will incur costs to the extent that the depressive episode interferes with those activities. There is evidence that people feel the costs imposed on them when a partner is depressed. Their reactions to the depressive are stronger and more negative than those of strangers (Segrin and Dillard, 1992). There are two mechanisms whereby the costs of depression may motivate members of the depressive's social network to make investments or concessions that they are otherwise reluctant to make.

5.1. Motivation by honestly signaling need

Some have argued that depression and suicidality function as a cry-for-help (Lewis, 1934; Stengel, 1974). Because depression is costly, it could function as an honest signal of need, motivating people with a pre-existing interest in helping an individual who honestly signals the need for help, but not those who falsely exaggerate their need. Since a signal must be costly to reveal the signaler's true state of need (Godfray, 1991), depression as an honest-cry-for-help could motivate social partners to help by virtue of the costs that it imposes on the depressive.

5.2. Motivation by fitness extortion

Depression also may motivate social partners to provide help via the gradually increasing costs it imposes on them (see also, Hagen, 1999). Extortionary depression can elicit help from social partners who perceive that it is better to help, thus stopping the depressive episode, than to continue to endure the escalating costs. Such depression may be designed to motivate the entire social network or specific partners. Resolution of this issue depends on how the costs of depression are distributed among

social partners and the types of problems that typically elicit severe depressions.

5.2.1. Motivating whole networks—the ''niche change'' function of depression

Humans live in groups where diverse goods and services are exchanged according to complex, heavily negotiated social contracts. An individual's ''social niche'' is defined by the interacting reciprocal exchange contracts they hold with each person in their social network, coupled with the skills and strategies they use to create, maintain, and modify those contracts.

Contractual webs secure the individual's role in society. Yet, they also can cause social entrapment. A person's social niche may vastly under-utilize their capacities for maximizing inclusive fitness. Human groups and individual development are so dynamic that severe mismatches between a person's capacities and opportunities for fitness-enhancing activities can arise in many ways. Where people inherit social status or occupation, for example, individuals may find themselves with a severe mismatch. Creative people tend to manufacture their own mismatches by producing novel ideas that social partners are slow to appreciate, or by developing new socioeconomic capacities that threaten to reform existing contracts or impinge on others' niches.

The problem of changing social niche may be a key adaptive context for wholesale extortion of the social network via severe depression. Niche change often will require acquiescence or assistance of the network in the form of political favors, skill training, capital investments, or savvy brainstorming about plausible better niches. Extortive depression is well suited to battling constraints on fitness-enhancing activity issuing from a diffuse social source (e.g., pervasive attitudes, customs, or expectations). Such a problem, if severe, may be solved most efficiently by depression simultaneously broadcasting costs to many social partners. If so, the problems depression is designed to solve should necessitate *special motivation* of much of the social network to help if the depressive is to resolve the problem.

There are several reasons why even loving social partners might stubbornly resist one's attempts at niche change, and so require special motivation. First, niche change necessarily involves modifying the terms of social contracts with many social partners. These partners may be reluctant to support niche change because they have great difficulty predicting the likely net benefits of giving such help *under a reformed and relatively poorly understood contract*. In contrast, the net benefits of providing forms of help under the *status quo* contract are known. Second, even if predictable, the goods and services expected under the new contract may be less beneficial than those under the current contract. Third, the social partner may have a fitness interest in someone else occupying the sought for niche. Partners may use creative rationalizations and diverse ''carrots and sticks'' to keep an individual in their current niche. Well-intentioned mental health providers unwittingly may do the same. Depression should be designed to resist such tactics, and to contingently worsen to a point where the costs imposed on the depressive's social partners overwhelms their resistance to the bid for niche change.

5.2.2. Motivating specific partners

Extortive depression also may serve to motivate specific partners within the network (see also, Hagen, 1999). If this is its main function, then selection should disfavor indiscriminate imposition of costs that endanger relationships that do not need changing. Thus, the activities that depression interfered with could target the partners whose help must be extorted. Functional design for motivating specific social partners could be demonstrated by showing variation in symptomatology that preferentially imposes costs on those partners with whom the depressive is in conflict, and that the costs were imposed to overcome their reluctance to help.

Even if depression indiscriminately broadcasts costs on social partners, it could still function to motivate specific partners. Imposing costs on the entire social network to modify one or a few dyad-specific contracts will be costly. However, this could be favored if the likely benefits outweigh the likely costs of endangering other relationships.

5.3. Evidence for social motivation

Depression may impose costs on the depressive and their partners whether or not the depression is extortionary or an honest signal. There are four

responses partners could take to costs imposed on them: (1) endure the depressive episode until it passes; (2) help or make a concession to try to stop the episode; (3) impose retaliatory costs on the depressive to try to stop the episode; or (4) divest from the relationship with the depressive and develop other relationships. Retaliatory costs imposed by a partner may often fail to stop depression because an individual, by becoming depressed, shows a willingness to endure costs. Thus, people should generally avoid retaliating (see also, Hagen, 1999).

It is not clear how often depression causes abandonment; in the close-knit groups humans probably evolved in this option may seldom have been viable. In any event, depressives may have often been in desperate social situations where the costs of making things worse were low and the benefits of improvement high. Even if depression usually produced social deterioration, it may still have evolved to serve a social motivation function if the expected net benefits were high enough to make the gamble profitable. That depression often elicits rejecting responses from others is consistent with both motivational hypotheses.

Since depression can only motivate partners who have a positive fitness interest in the depressive, both motivational hypotheses predict that people should become more depressed when they are in conflict with partners who have a greater positive fitness interest in them. In one study, caregivers of Alzheimer's patients were asked to rate how upset they currently were with their social network and that network's past helpfulness (Pagel et al., 1987). High levels of current upset coupled with high levels of past helpfulness was a better predictor of subsequent depression than high upset and a history of low helpfulness. The study suggests that people are more likely to become depressed when they are in conflict with partners who, by being helpful in the past, have exhibited a positive fitness interest in them. This finding is surprising given the large literature showing that a supportive social network is a buffer to depression (Paykel, 1994).

Both motivational hypotheses explain features of depression inconsistent with a rumination function. Depression is often self-endangering. It handicaps the immune system (Weisse, 1992), and in unforgiving ancestral environments the depressive's reduced capacity for self-care can be viewed as passive suicidal behavior. Depression can also cause active suicidal behavior: 2.2–8.6% of clinically depressed people commit suicide, compared to less than 0.5% of the non-depressed population (Bostwick and Pankratz, 2000). The incidence of non-fatal attempts must be higher.

Evidence supporting a motivation function comes from studies indicating that suicide attempts stop when relationships improve (Hawton et al., 1982a). It may seem odd to propose that human suicidal behavior is adaptive, but we are not the first to do so (deCatanzaro, 1981). Selection also has forged adaptations for suicide in other species (Alcock, 2001). As an honest signal, the risk of death associated with a suicide attempt could inform partners about the attempter's level of need. Under the extortion hypothesis, suicide attempts impose a risk of loss on all partners with an interest in the attempter's existence. Extortionary attempts threaten repeated, escalating attempts, exposing partners to further risk unless they provide sought for help (see also, Zahavi and Zahavi, 1997).

Treatments that remove depressives from their social group (e.g., hospitalization) are associated with the greatest risk of subsequent suicide (Bostwick and Pankratz, 2000). Such treatments may blunt their ability to motivate social partners, forcing the depressive to produce a ''louder signal''.

To serve a motivational function, suicide attempters sometimes must survive. Yet, the attempter gets more motivational power by incurring a greater risk of death. Thus, the attempter trades off the risk of death against the possible benefits to be gained from motivating close social partners to help. Motivational suicide should be classified as a form of *parasuicide* (suicidality without intent to die, but which may entail a genuine risk of death) (Stengel, 1974; Kreitman, 1977), because it is distinct from suicidal behavior where the intent is to end life with certainty (deCatanzaro, 1981).

5.4. Distinguishing honest signaling and extortion

The two motivational models of the SNH make different predictions about how social partners are motivated. The honest signaling hypothesis predicts

that the costs imposed on self are crucial for eliciting help, while costs imposed on partners are incidental. Conversely, the extortion hypothesis predicts that costs imposed on partners are crucial for overcoming their reluctance to help, and costs imposed on self are incidental or useful for deterring retaliation. Self- and clinician-reported reasons for adolescent suicide support both hypotheses. Sometimes imposition of costs on the self appears to be important (e.g., so others understand how desperate one feels) and sometimes imposition of costs on others appears to be important (e.g., to punish or influence someone) (Hawton et al., 1982b).

The extortion hypothesis predicts that depression will escalate, imposing increasing costs on partners, until they either capitulate or divest, or it becomes unprofitable to escalate further. Depression often escalates, suggesting extortion. However, there are two reasons why escalation could occur even if signalers were only communicating need and not extorting. First, signalers could systematically under-signal their level of need and then escalate until they get a response from receivers. Modeling of this issue suggests that systematic under-signaling and escalation often will be unprofitable and thus rare (Payne and Pagel, 1996). Second, honest signalers could escalate if their need escalates over time. Thus, the case for extortion could be bolstered by showing that escalation of depression does not track changes in need.

5.5. A general test of the social navigation hypothesis

If the SNH is correct, interfering with depression should sometimes prevent improvements in a person's social situation. In our view, a critical test would be to give anti-depressant medication to one group of depressives and a placebo to another, and then monitor their *social* outcomes over time vis a vis an objective standard. Changes in social outcomes should exhibit a unimodal distribution in the medicated group. However, the distribution of changes in outcomes for the placebo group should exhibit greater variation, *with greater chances of a positive outcome*, than in the medicated group. The distribution of outcomes might even be bimodal (one with a positive mean, one with a negative mean). If bimodal, the mean of those with a positive social outcome should be greater than the mean of the medicated group. We suspect a bimodal distribution of good and bad outcomes because: (1) depressives are more likely to be desperate and attempt risky strategies for resolving their social problems, and (2) social partners sometime may respond to extortive depression with persistent rejection. To our knowledge, such an experiment has not been performed.

6. Clinical implications

That depression usually is viewed as maladaptive is perhaps surprising given that many clinicians see other sorts of severe emotional pain, such as grief, as serving a useful purpose. The SNH suggests that, like grief, it may be important to support a process of depression (see also Gut, 1989). The SNH makes new clinical suggestions about how to support mild and severe depression.

Three insights from the SNH may help therapists discover the cause of a depression. First, it is important to focus on social factors that *limit* the client's inclusive fitness, not just things that may influence it. Second, the contents of the depressive's ruminations may provide clues to the *current* social causes of depression. Third, the therapist should not only consider potentially causal dyad-specific problems, but also fitness-limiting social constraints with diffuse origins. If a crucial social constraint seems dyad-specific, the therapist might consider whether the point of the depression is to motivate the social network to help deal with the dyad-specific problem. Moreover, the SNH suggests that two complimentary forms of talking therapy can bring long-term relief to depressives, namely, neo-Darwinian forms of informative therapy and social problem solving therapy.

Depressives' estimates of the net benefits to be had from a bout of low mood will vary in accuracy. Informative therapy can help the depressive to better analyze his need and ability to improve his socio-economic life via depression. This approach requires thorough knowledge of the patient's social circumstances and analysis of the patient's opportunities and constraints.

SNH-based informative therapy would not focus

on persuading the patient that their depression is unwarranted. Therapies that manipulate mood without addressing underlying causes may increase the danger that a recurrence of depression will be more resistant to treatment due to subtle, possibly unconscious, lack of trust. Removal of the depressive from their social network (e.g., by hospitalization) may interfere with the depressive's information-gathering and handicap social rumination, resulting in prolongation and dysregulation of symptoms.

Informative therapy may lead to the conclusion that the depression is based on a sound cost–benefit analysis. If so, the SNH suggests therapists should serve as insightful social educators and tenacious advocates for their depressed patients to help them solve their social problems. The SNH's heavy systems orientation suggests that group and family therapy often will be necessary, and a multi-disciplinary team may be needed to provide the interpersonal mediation, skill training, and other help needed to solve the depressive's problems.

The SNH implies that anti-depressant medications risk handicapping the client's ability to navigate and control their social environment; this could, in the long run, hinder the depressive from making key improvements in quality of life. If the SNH is correct, then a therapeutic prime directive to reduce suffering per se may be an irresponsible approach. Even when a therapist can implement a helpful talking therapy, it may be best to let depression work its miserable yet potentially adaptive magic on the social network under protective supervision. The SNH suggests that drugs should not be given unless the causative social problems also are being addressed, and that drugs not be allowed to emasculate the ruminative and motivational functions of a potentially adaptive depression.

6.1. Case history illustration

We offer a case history analysis that we think yields a useful therapeutic hypothesis that, without the SNH, would be difficult to formulate. A depressed but otherwise healthy post-reproductive age woman has three sons and four daughters. One daughter is unwed. All have low socioeconomic status. The woman has good relations with her children, but she wishes they would pay more attention to her advice and opinions. The woman feels her depression is due to insistent ''movies'' in her head that replay incidents from her youth in which her father exhibited oppressive, non-investing behavior toward her. The woman states that her father's negative attitude prevented her from using her talent and ambition to gain access to education and a career. Now, however, she just wants the old memories and the depression she feels they cause to go away so she can be happy and contribute more to the care of her grandchildren. Her children need and desire these things too.

In SNH-based therapy, there would be a working assumption that depressive symptoms have utility. Since the mind is causing much of the depressive's energy to be diminished by clinical symptoms, and her cognitive resources are devoted to a specific set of ruminations, then the mind should have tailored these phenomena to attack problems that *limit* her inclusive fitness. Since this woman is post-reproductive, the only thing that influences her fitness is the welfare of her children and current and future grandchildren. What is perhaps limiting to her fitness, especially in the low SES social milieu, is the levels of parental investment enjoyed by her grandchildren.

One therapeutic hypothesis that follows is that the movies the woman is compelled to watch are causing her to review her father's characteristics, including his body language, how he spoke, maybe even how he smelled. These details could prime the woman emotionally and cognitively to help her daughters choose and manage their mates for the benefit of her grandchildren. Similarly, it may help her to motivate her sons to consider the long term ramifications of how they treat her grandchildren.

Depression imposes costs on the mother's children that draws their attention and primes them to digest her concerns about their mate choices and the care of her grandchildren. Under this therapeutic hypothesis, if the mother perceives that the daughters' mate choice criteria improve in ways that help ensure the quality of paternal investment flowing to the grandchildren, then amelioration of the depression should ensue. Increased generosity in her sons' child care behavior also may be therapeutic. It may help to gather the family so the woman can tell her childhood story in an emotionally compelling way, fol-

lowed by discussions about mate choice and child-rearing.

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References

Abed, R.T., 2000. Psychiatry and Darwinism: time to reconsider? Br. J. Psychiatry 177, 1–3.

Abramson, L.Y., Metalsky, G.I., Alloy, L.B., 1989. Hopelessness depression: a theory-based subtype of depression. Psychol. Rev. 96, 358–372.

Ackermann, R., DeRubeis, R.J., 1991. Is depressive realism real? Clin. Psychol. Rev. 11, 565–584.

Ahrens, A.H., Zeiss, A.M., Kanfer, R., 1988. Dysphoric deficits in interpersonal standards, self-efficacy, and social comparison. Cog. Ther. Res. 12, 53–67.

Akiskal, H.S., 2001. Dysthymia and cyclothymia in psychiatric practice a century after Kraepelin. J. Affect. Disord. 62, 17–31.

Alcock, J., 2001. Animal Behavior: An Evolutionary Approach, 7th Edition. Sinauer, Sunderland, MA.

Alexander, R.D., 1989. Evolution of the human psyche. In: Mellars, P., Stringer, C. (Eds.), The Human Revolution. University of Edinburgh Press, Edinburgh, pp. 455–513.

Alloy, L.B., Abramson, L.Y., Whitehouse, W.G., Hogan, M.E., Tashman, N.A., Steinberg, D.L., Rose, D.T., Donovan, P., 1999. Depressogenic cognitive styles: predictive validity, information processing and personality characteristics, and developmental origins. Behav. Res. Ther. 37, 503–531.

Alloy, L.B., Abramson, L.Y., 1979. Judgment of contingency in depressed and nondepressed students. Sadder but wiser? J. Exp. Psychol. Gen. 108, 441–485.

Alloy, L.B., Ahrens, A.H., 1987. Depression and pessimism for the future: biased use of statistically relevant information in predictions for self versus other. J. Pers. Soc. Psychol. 52, 366–378.

American Psychiatric Association, 1994. Diagnostic and Statistical Manual of Mental Disorders, 4th Edition. APA, Washington, DC.

Anderson, C.A., 1999. Attributional style, depression, and loneliness: a cross-cultural comparison of American and Chinese students. PSPB 25, 482–499.

Andrews, B., Brown, G.W., 1995. Stability and change in low self-esteem: the role of psychosocial factors. Psychol. Med. 25, 23–31.

Andrews, P.W., 2001. The psychology of social chess and the evolution of attribution mechanisms: explaining the fundamental attribution error. Evol. Hum. Behav. 22, 11–29.

Andrews, P.W., Gangestad, S.W., Matthews, D., 2001. Adaptationism—how to carry out an exaptationist program. Behav. Brain Sci., submitted for publication.

Baumeister, R.F., 1993. Suicide attempts. In: Costello, C.G. (Ed.), Symptoms of Depression. Wiley, New York, pp. 259–289.

Biglan, A., Hops, H., Sherman, L., Friedman, L.S., Arthur, J., Osteen, V., 1985. Problem-solving interactions of depressed women and their husbands. Behav. Ther. 16, 431–451.

Bostwick, J.M., Pankratz, V.S., 2000. Affective disorders and suicide risk: a reexamination. Am. J. Psychiatry 157, 1925–1932.

Brewerton, T.D., 1995. Toward a unified theory of serotonin dysregulation in eating and related disorders. Psychoneuroendocrinology 20, 561–590.

Brown, G.W., Adler, Z., Bifulco, A., 1988. Life events, difficulties and recovery from chronic depression. Br. J. Psychiatry 152, 487–498.

Brown, G.W., Harris, T.O., Hepworth, C., 1995. Humiliation and entrapment among women developing depression: A patient and nonpatient comparison. Psychol. Med. 25, 7–21.

Brown, J.D., 1986. Evaluations of self and others: self-enhancement biases in social judgments. Soc. Cognition 4, 353–376.

Brugha, T.S., Bebbington, P.E., Stretch, D.D., MacCarthy, B., Wykes, T., 1997. Predicting the short-term outcome of first episodes and recurrences of clinical depression: a prospective study of life events, difficulties, and social support networks. J. Clin. Psychiatry 58, 298–306.

Buhot, M.-C., 1997. Serotonin receptors in cognitive behaviors. Cur. Opin. Neurobiol. 7, 243–354.

Buss, D.M., 1999. The evolution of happiness. Am. Psychol. 55, 15–23.

Buss, D.M., 2000. The Dangerous Passion: Why Jealousy is as Necessary as Love and Sex. Free Press, New York.

Choi, I., Nisbett, R.E., Norenzayan, A., 1999. Causal attribution across cultures: variation and universality. Psychol. Bull. 125, 47–63.

Coyne, J.C., Downey, G., 1991. Social factors and psychopathology: stress, social support, and coping processes. Annu. Rev. Psychol. 42, 401–425.

Coyne, J.C., Whiffen, V.E., 1995. Issues in personality as diathesis for depression: the case of sociotropy-dependency and autonomy-self-criticism. Psychol. Bull. 118, 358–378.

deCatanzaro, D., 1981. Suicide and Self-Damaging Behavior: A Sociobiological Perspective. Academic Press, New York.

Gannon, K.M., Skowronski, J.J., Betz, A.L., 1994. Depressive diligence in social information-processing: Implications for order effects in impressions and for social memory. Soc. Cognition 12, 263–280.

Giesler, R.B., Josephs, R.A., Swann, W.B., 1996. Self-verification in clinical depression: the desire for negative evaluation. J. Abnorm. Psychol. 105, 358–368.

Gilbert, P., Allan, S., 1998. The role of defeat and entrapment (arrested flight) in depression: an exploration of an evolutionary view. Psych. Med. 28, 585–598.

Godfray, H.C.J., 1991. Signaling of need by offspring to their parents. Nature 352, 328–330.

Gut, E., 1989. Productive and Unproductive Depression. Basic Books, New York.

Haaga, D.A.F., Ernst, D., Dyck, M.J., 1991. Empirical status of cognitive theory of depression. Psychol. Bull. 110, 215–236.

Hagen, E.H., 1999. The functions of postpartum depression. Evol. Hum. Behav. 20, 325–359.

Hartlage, S., Alloy, L.B., Vãzquez, C., Dykman, B., 1993. Automatic and effortful processing in depression. Psychol. Bull. 113, 247–278.

Hawton, K., Cole, D., O'Grady, J., Osborn, M., 1982a. Motivational aspects of deliberate self-poisoning in adolescents. Br. J. Psychiatry 141, 286–291.

Hawton, K., Osborn, M., O'Grady, J., Cole, D., 1982b. Classification of adolescents who take overdoses. Br. J. Psychiatry 140, 124–131.

Howell, N., 1979. Demography of the Dobe !Kung. Academic Press, New York.

Humphrey, N.K., 1976. The social function of intellect. In: Bateson, P.P.G., Hinde, R.A. (Eds.), Growing Points in Ethology. Cambridge University Press, Cambridge, pp. 303–317.

Jacobs, B.L., Fornal, C.A., 1997. Serotonin and motor activity. Curr. Opin. Neurobiol. 7, 820–825.

Joiner, T.E., Schmidt, N.B., Singh, D., 1994. Waist-to-hip ratio and body dissatisfaction among college women and men: Moderating role of depressed symptoms and gender. Int. J. Eat. Disord. 16, 199–203.

Kessler, R.C., 1997. The effects of stressful life events on depression. Annu. Rev. Psychol. 48, 191–214.

Kessler, R.C., McGonagle, K.A., Swartz, M., Blazer, D.G., Nelson, C.B., 1993. Sex and depression in the national comorbidity survey. 1. Lifetime prevalence, chronicity and recurrence. J. Affect. Disord. 29, 85–96.

Kessler, R.C., McGonagle, K.A., Zhao, S., Nelson, C.B., Hughes, M., Eshleman, S., Wittchen, H.-U., Kendler, K.S., 1994. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: results from the national comorbidity survey. Arch. Gen. Psychiatry 57, 14–20.

Klinger, E., 1975. Consequences of commitment to and disengagement from incentives. Psychol. Rev. 82, 1–25.

Kreitman, N., 1977. Parasuicide. Wiley, London.

Kumar, A., Jin, Z., Bilker, W., Udupa, J., Gottlieb, G., 1998. Late-onset minor and major depression: early evidence for common neuroanatomical substrates detected by using MRI. Proc. Natl. Acad. Sci. USA 95, 7654–7658.

Lane, J.D., DePaulo, B.M., 1999. Completing Coyne's cycle: dysphorics' ability to detect deception. J. Res. Personality 33, 311–329.

Lemelin, S., Baruch, P., 1998. Clinical psychomotor retardation and attention in depression. J. Psychiatry Res. 32, 81–88.

Lemke, M.R., Puhl, P., Koethe, N., Winkler, T., 1999. Psychomotor retardation and anhedonia in depression. Acta Psychiatr. Scand. 99, 252–256.

Lewis, A.J., 1934. Melancholia: a clinical survey of depressive states. J. Mental Sci. 80, 1–43.

Marsh, K.L., Weary, G., 1994. Severity of depression and responsiveness to attributional information. J. Soc. Clin. Psychol. 13, 15–32.

NIMH, 1994. Depression Fact Sheet. NIH Publication No. 00-4501. NIH, Bethesda, MD.

Nesse, R.M., 2000. Is depression an adaptation? Arch. Gen. Psychiatry 57, 14–20.

Nesse, R.M., Williams, G.C., 1994. Why Do We Get Sick? the New Science of Darwinian Medicine. Times Books, New York.

Pagel, M.D., Erdly, W.W., Becker, J., 1987. Social networks: we get by with (and in spite of) a little help from our friends. J. Pers. Soc. Psychol. 53, 793–804.

Paykel, E.S., 1994. Life events, social support and depression. Acta Psychiatr. Scand. Suppl. 377, 50–58.

Payne, R.J.H., Pagel, M., 1996. When is false modesty a false economy? An optimality model of escalating signals. Proc. Roy. Soc. Lond. B 263, 1545–1550.

Price, J.S., Sloman, L., Gardner, R., Gilbert, P., Rohde, P., 1994. The social competition hypothesis of depression. Br. J. Psychiatry 164, 309–315.

Schmaling, K.B., Jacobson, N.S., 1990. Marital interaction and depression. J. Abnorm. Psychol. 99, 229–236.

Sedikides, C., 1993. Assessment, enhancement, and verification determinants of the self-evaluation process. J. Pers. Soc. Psychol. 65, 317–338.

Sedikides, C., Campbell, W.K., Reeder, G.D., Elliot, A.J., 1998. The self-serving bias in relational context. J. Pers. Soc. Psychol. 74, 378–386.

Segrin, C., Dillard, J.P., 1992. The interactional theory of depression: a meta-analysis of the research literature. J. Soc. Clin. Psychol. 11, 43–70.

Sheppard, L.C., Teasdale, J.D., 1996. Depressive thinking: changes in schematic mental models of self and world. Psychol. Med. 26, 1043–1051.

Stengel, E., 1974. Suicide and Attempted Suicide. Penguin, New York.

Swallow, S.R., Kuiper, N.A., 1993. Social-comparison information in dysphoria and nondysphoria: differences in target similarity and specificity. Cognit. Ther. Res. 17, 103–122.

Sweeney, P.D., Anderson, K., Bailey, S., 1986. Attributional style in depression: a meta-analytic review. J. Pers. Soc. Psychol. 50, 974–991.

Tabachnik, N., Crocker, J., Alloy, L.B., 1983. Depression, social-comparison, and the false-consensus effect. J. Pers. Soc. Psychol. 45, 688–699.

Tennen, H., Eberhardt, T.L., Affleck, G., 1999. Depression research methodologies at the social-clinical interface: still hazy after all these years. J. Soc. Clin. Psychol. 18, 121–159.

Thornhill, R., 1997. The concept of an evolved adaptation. In: Bock, Cardew G. (Eds.). Characterizing Human Psychological Adaptation. Ciba Foundation Symposium No. 208. Wiley, West Sussex, pp. 4–13.

Thornhill, R., Palmer, C.T., 2000. A Natural History of Rape: Biological Bases of Sexual Coercion. MIT Press, Cambridge, MA.

Thornhill, R., Thornhill, N.W., 1989. The evolution of psychological pain. In: Bell, R., Bell, N. (Eds.), Sociobiology and the Social Sciences. Texas Tech University, Lubbock, TX, pp. 73–103.

Vonk, R., 1998. The slime effect: suspicion and dislike of likeable behavior toward superiors. J. Pers. Soc. Psychol. 74, 849–864.

Weary, G., Marsh, K.L., McCormick, L., 1994. Depression and social-comparison motives. Eur. J. Soc. Psychol. 24, 117–129.

Weisse, C.S., 1992. Depression and immunocompetence: a review of the literature. Psychol. Bull. 111, 475–489.

Weissman, M.M., 1987. Advances in psychiatric epidemiology: rates and risks for depression. Am. J. Public Health 77, 445–451.

Williams, G.C., 1966. Adaptation and Natural Selection. Princeton University Press, Princeton, NJ.

Yost, J.H., Weary, G., 1996. Depression and the correspondent inference bias: evidence for more effortful cognitive processing. Pers. Soc. Psychol. Bull. 22, 192–200.

Zahavi, A., Zahavi, A., 1997. The Handicap Principle. Oxford University Press, New York.