PUBLISHED IN THE JOURNAL OF SCIENCE AND HEALTH POLICY

DEPRESSION: DISEASE OR BEHAVIORAL SHUTDOWN MECHANISM?

Gregg Henriques, Ph.D.

University of Pennsylvania

How depression is conceptualized is a major public health issue. The prevailing model in psychiatry is that Major Depressive Disorder (MDD) is a disease of the brain. However, recent developments in evolutionary theory suggest that negative emotions and depression are likely evolved strategies that facilitated behavioral solutions to problems in the ancestral environment. A Behavioral Shutdown Model (BSM) of depression is offered and explored. The model proposes that depressive reactions are passive, avoidant behavioral strategies that have been fashioned by evolution and are activated in response to situations that are chronically dangerous, humiliating, or repeatedly result in failure to achieve one's goals. The BSM challenges the disease model because it suggests that many instances of MDD do not involve biological dysfunctions. Instead, this analysis suggests that Major Depression is conceptually more akin to pain than to a disease. The BSM concept and implications for health policy are discussed.

onsider a television commercial that begins with an attractive woman isolating herself at a party. Everyone else appears to be having a good time, yet she stands in the background, ostensibly gripped in the throes of a seemingly inexplicable sadness. The cultural milieu is of upper middle class suburbia. A soft voice inquires and informs, "Have you experienced periods of depressed mood? Have you lost interest in things you used to enjoy? Do you feel tired, guilty, ineffective or hopeless? Depression is an illness. Ask your doctor about new antidepressant treatments available." The implicit message of the commercial is clear. When people are suffering from depression, something has gone wrong with the physiology of the brain.

Now imagine a different commercial. This one begins with an impoverished woman getting slapped by her husband. Her three children are having difficulties in school. Her husband controls her, and

Psychiatry, University of Pennsylvania, Philadelphia, PA 19104-2648, E-email address:

henri@landru.cpr.upenn.edu.

¹ This article was generated in part due to a fruitful exchange on the Evolutionary Psychology Internet-Group. I would like to thank those individuals that contributed to the discussion on depression as an adaptation. I would like to thank Aaron T. Beck, M.D., Gregory K. Brown, Ph.D., Ivan Cohen, M.D., and Stephen Miles Sacks, Ph.D. for their helpful comments on earlier drafts of this work. Correspondence regarding this article should be sent to: Gregg Henriques, Ph.D., 3600 Market Street, The Science Center Room 754, Department of

she has little in the way of social support. She recently immigrated to the United States and cannot get a job because she only speaks a little English. She frequently faces prejudice and racism. The voice overlay asks, "Have you been feeling down or depressed, guilty or hopeless? Have you lost interest in things you usually enjoy? Depression is an illness. Ask your doctor about new antidepressant treatments available." Somehow the "depression as disease" message in this commercial is less convincing.

As these two vignettes illustrate, different portrayals can lead to radically different notions regarding the nature of depression. Yet, how depression is conceptualized is critically important. Depression is a major public health issue, and the theoretical paradigms that guide our understanding of the condition influence public opinion, health policies, treatment strategies, and research. A Major Depressive Episode (MDE) is defined by the *Diagnostic and Statistical Manual-IV* (American Psychiatric Association, 1994) as the presence of 5 out of 9 nine psychological and behavioral symptoms (depressed mood, anhedonia, agitation or retardation, fatigue or low energy, feelings of worthlessness or guilt, thoughts of death, change in appetite/weight, sleeping difficulties, and diminished ability to concentrate) present most everyday for a period of two weeks.

Epidemiological studies have documented that depressive disorders are common. Estimates of the lifetime prevalence of Major Depressive Disorder (MDD) and dysthymia (a more chronic, but less intense depressive condition) are 15% for men and 24% for women and appear to be on the rise (Hirschfeld, Keller, et. al., 1997). The 1-year prevalence of MDD and dysthymia among community residents is estimated to be approximately 10%, with an additional 11% of the population having significant subclinical symptoms (Zhang, Rost, & Fortney, 1999). These estimates suggest that in the United States alone, approximately 27 million people suffered from a depressive disorder and another 29 million suffered from substantial depressive symptoms in the past year.

Research has demonstrated that depressive disorders are associated with difficulties in both physical and psychosocial functioning. MDD is



Dr. Gregg Henriques is a Research Fellow in the Department of

Psychiatry at the University of Pennsylvania Health System. He received his Ph.D. in Clinical Psychology from the University of Vermont and his MA degree in Clinical/Community Psychology from the University of North Carolina-Charlotte. He is the Project Director for a randomized controlled clinical trial examining the efficacy and effectiveness of a brief cognitive therapy intervention for individuals who have recently made a suicide attempt.

associated with suicide and higher mortality rates in general (Harris & Barraclough, 1998), missed work (Kessler, Barber, et al, 1999), cognitive processing difficulties (Merriam, Thase, Haas, Keshavan, & Sweeney, 1999), and difficulties in social functioning (Reinherz, Giaconia, Hauf, Wasserman, & Silverman, 1999). The direct and indirect costs associated with MDD are estimated to be over \$36 billion dollars annually, similar to costs associated with coronary heart disease (Hirschfeld, et al., 1997). Thus, depression is a major public health issue.

It is important not to make a correlation-causation error when interpreting these findings, however. A diagnosis of MDD is made on the presence of psychological and behavioral symptoms alone, not on etiology. The correct interpretation of the above findings is that depressive symptoms are correlated with difficulties in functioning. To infer that "depression" is the causal process underlying these difficulties requires one to make assumptions that are, at the very least, debatable. As is anecdotally illustrated by the second imaginary commercial, there are reasons to believe that depressive symptoms are often a reaction to difficulties in functioning. As will be argued below, it seems likely that depressive symptoms are both a cause and a consequence of difficulties in functioning in modern society.

DISEASES AND DEFENSIVE STRATEGIES

The prevailing model in psychiatry is that MDD is a disease. In a commentary in the <u>Archives of General Psychiatry</u>, Judd (1997) states a "fundamental paradigmatic shift" is occurring among the general public, health experts, and the practicing psychiatric community. The shift is that most people, including the large majority of practicing psychiatrists, now view MDD as a disease of the brain.

There are, of course, good reasons for this position. In addition to the fact that depressive disorders are associated with difficulties in psychosocial functioning and higher mortality rates, neuroimaging studies have shown differences in the activity of the prefrontal cortex, the basal ganglia, the amygdala-hippocampus complex, and the thalamus in the brains of depressed individuals (e.g., Drevets, 1998; Soares & Mann, 1997). Differences in the neuro-endocrine systems of depressed individuals have also been well documented (e.g., Arborelius, Owens, Plotsky, & Nemeroff, 1999). Additionally, Selective Serotonin Reuptake Inhibitors (SSRIs) are some of the most effective treatments for reducing depressive symptoms (e.g., Gorman & Kent, 1999). Taken together, such findings form an impressive body of knowledge demonstrating that depressive disorders are associated with difficulties in functioning and that there are differences in the brain activity and brain chemistry of depressed individuals.

Despite these important findings, neurophysiological causal models of depression remain elusive (Doris, Ebmeier, & Shajahan, 1999). One

possible reason for this failure is how depression is being conceptualized. When viewed as a disease, the psychological and behavioral symptoms that result in a diagnosis of MDD are generally assumed to be the product of neurophysiological dysfunctions. As such, differences in brain chemistry and/or brain activity between depressed individuals and controls are generally taken as evidence of brain pathology.

The assumption of biological dysfunction may not be valid, however. Recent developments in evolutionary applications in psychology have suggested that depression may be a defensive strategy that evolved because it facilitated solutions to adaptive problems in the ancestral environment (e.g., Beck, 1999; Nesse, 2000). If depression is an evolved defensive strategy, however, then one would expect marked differences in the brain activity and brain chemistry of depressed individuals. As such, the differences would not be biological dysfunctions, but instead would reflect the evolved architecture of the mind.

The Behavioral Shutdown Model (BSM) explored here suggests an alternative interpretation of the genesis of MDD and depression in general. Rather than viewing MDD as the consequence of a neurophysiological dysfunction or genetic illness, the BSM suggests that depression is actually an evolved defensive strategy. The distinction between biological dysfunction and a defense strategy is an important one (Nesse & Williams, 1994). A biological dysfunction is the failure of an organ or system to function in accord with evolved its design (Wakefield, 1992). A heart attack is an example of a dysfunction because the heart was fashioned via evolutionary processes to circulate blood throughout the body. Broken bones, cancers, and strokes are also examples of dysfunctions. Jerome Wakefield has convincingly argued that the concept of disease in medicine invariably involves the deviations or breakdowns in the functioning of evolved mechanisms (Henriques, 2000; Spitzer, 1999; Wakefield, 1999a; 1999b).

A defensive strategy, on the other hand, is an evolved method for signaling and/or reacting to a problem (Nesse & Williams, 1994). When the influenza virus infects the human body, a number of different things happen, some of which are manifestations of defects and some of which are defenses. Internally, the virus infects and transforms the human cells for its own reproductive benefit, causing clear defects. Symptoms include fever, coughing, and feeling achy, tired and run down. It used to be believed the coughing and fever were caused by the virus and were manifestations of cellular defects. Yet, the fever and coughing are in fact evolved defenses. The increase in body temperature, for example, hinders the speed and effectiveness with which the virus can reproduce. Identifying a symptom as a defense strategy rather than a manifestation of a defect is important because it leads to a different intervention strategy. For example, medications

given to reduce fever (once presumed to be part of the defect) actually prolong the duration of the flu virus in the body (Nesse & Williams, 1994).

EMOTIONS AS EVOLVED STRATEGIES

But how might depression be a defensive strategy? On the surface, the depressed mood, decreased energy, loss of interest in pleasurable activities, and change in sleep patterns associated with depression appear quite dysfunctional. To understand how depression might be functional in an evolutionary sense, it is useful to first consider the evolutionary significance of pain. To effectively solve problems in its environment, an organism must have mechanisms that allow it to approach situations that are beneficial and avoid situations that are harmful. Pleasure can be thought of as the signal to approach and pain the signal to avoid. Although pain is almost always unwanted, the capacity to experience physical pain is immensely important. Physical pain signals something is wrong with the structural integrity of the body. Broken bones, lacerations, torn ligaments, ulcers etc., put the organism at risk or hinder its capacity to function and pain signals the presence of the problem. Pain also motivates the organism to avoid whatever is causing the difficulty and helps the organism to learn to avoid it in the future.

Evolutionarily informed theorists now recognize that emotional pain serves a very important function, similar to that of physical pain (e.g., Johnson, 1999). Whereas physical pain signals problems with the structural integrity of the body, emotional pain signals problems with how the individual is interacting with some aspect of his or her environment, usually the social environment (Price, 1998). We feel emotional pain when we fail to achieve, when a loved one dies, or when we are criticized, rejected or controlled because these types of events involve loss of important resources in the social environment such as status, solidarity, or autonomy (Nesse, 1998).

There are many different types of emotional pain because there are different types of problems in the social environment that one must avoid. Disappointment, sadness, and grief signal one has incurred losses or failures (e.g., Archer, 1999). Fear and anxiety signal emotional or physical pain might occur in the future (Seligman, 1971). Shame signals loss of status and functions to avoid conflicts and submit to more powerful others (Gilbert & Andrews, 1998). Anger is activated to defend oneself from others' control or, conversely, to punish others for insubordination or betrayal (Trivers, 1971). Guilt involves making reparations for selfish behavior to avoid the problem of retaliation (Trivers, 1971). In short, negative emotions are evolved strategies that allow for the identification and avoidance of potential problems, particularly in the social domain. As such, the presence of intense negative emotion is not necessarily indicative of a biological

dysfunction. This is important because, as evidenced by the symptom list in the DSM-IV, there is significant overlap between intense negative affect for a period of two weeks or more and a diagnosis of MDD.

DEPRESSION AS AN EVOLVED BEHAVIORAL SHUTDOWN MECHANISM

To explore the possibility that depression is an evolved defensive strategy, it is useful to consider behavior in terms of evolutionary theory. From a Darwinian perspective, behavior can be thought of as the process of expending energy or working in order to control and structure the environment in a way that allows for survival and reproductive success (Beck, 1999; Geary, 1998). Control of larger territories, access to better food, higher social status, etc. is obviously advantageous. However, the behavioral investment needed to acquire and maintain these resources is expensive. It costs energy both in terms of basic calories and in terms of increasing risk of injury and loss. Resources are frequently not available or cannot be acquired which means behavioral investments are fruitless (Nesse, 2000). Additionally, competition over valuable resources can be fierce, often resulting in injury (Gilbert, 1992). This analysis gives rise to a cost to benefit ratio of behavioral investment, a ratio much like that in economics.

But what does this model have to do with depression? The cost to benefit ratio suggests that organisms can maximize the ratio by increasing benefits or by decreasing costs. Increasing benefits is associated with actively acquiring some resource (food, sex, status) in the environment via behavioral investment. The individual's state of actively working to increase benefit can be described as desire. Decreasing behavioral investment can also be a way in which organisms deal with the cost to benefit ratio. There are many examples of behavioral shutdown mechanisms in nature, such as sleep, hibernation and exhaustion, that function to decrease behavioral expenditure and conserve energy.

Broadly speaking, behavioral shutdown should result if an organism is getting a poor return (i.e., high costs, little benefit) from its behavioral investment (Beck, 1999). That is, if an organism is expending 8 behavioral units and only getting back 4 units, that is a bad ratio. If it tries everything in its behavioral repertoire, yet the ratio remains the same, a "best in a bad situation" solution is to decrease the amount of the behavioral investment in an effort to reduce net loss. It is better to expend 2 and get back 1 over the same period of time than the 8:4 ratio previously obtained. This understanding gives rise to the Behavioral Shutdown Model (BSM) which suggests that depression may represent an evolved tendency to decrease behavioral expenditure in response to chronic danger, stress, or consistent failure to achieve

one's goals (see also Beck, 1992, 1999; Gilbert, 1992, 1998; McGuire, Troisi, & Raleigh, 1997; Nesse, 2000).

The BSM offers a potential explanation for many features of depression. For example, it strongly predicts that depression should be more likely to occur in situations that are chronically dangerous, humiliating, or repeatedly result in failure to achieve one's goals. These are circumstances in which the cost to benefit ratio is the worst and therefore the most effective strategy is to reduce costs. Consistent with this prediction, situations in which the individual feels chronically trapped or humiliated are most likely to produce symptoms of depression (Gilbert & Allan, 1998). To give just one example, almost 50% of battered women are depressed (Golding, 1999). There is also strong evidence that the onset of many Major Depressive Episodes are preceded by major stressful life events (e.g., Kendler, Karkowski, & Prescott, 1999). Also consistent with the BSM, rates of MDD vary with socioeconomic status. Those individuals in the lowest quartile of socioeconomic status are almost twice as likely to be depressed compared with those in the highest quartile (Yu & Williams, 1999).

In addition to offering an explanation as to why certain situations are more likely to result in depression, the BSM also explains many of the symptoms of depression. The model explains why emotional pain is such a prominent feature of depression, as the pain is a signal that things are not going well. Additionally, behavioral shutdown is the antithesis of active behavioral investment and thus the BSM explains why anhedonia is such a fundamental characteristic of depressive conditions. It also directly accounts for why low energy is such a prominent complaint. The model also explains why negative cognitions are so prominent in depression. Cognitive theorists have clearly documented how depressed individuals are hypersensitive to any indications of loss, failure, or rejection (Beck, 1967; Clark, Beck, & Alford, 1999). In direct accordance with the BSM, recent cognitive models have conceptualized depressed individuals as investors with few resources who take risk-aversive strategies to avoid loss (Leahy, 1997). In short, the BSM offers a potential explanation for many of the symptoms of depression.

The BSM also provides explanations for findings that are difficult to explain from a disease model perspective. Because so many different things can result in difficulties in solving important problems, the BSM model accounts for why so many different causal pathways result in depression (Winokur, 1997). Behavioral shutdown should be a matter of degree, thus the BSM also accounts for why symptoms of depression exist on a continuum that range from chronic, severe depressions to minor depressions to adjustment disorders to low mood (Nesse, 2000). Since the model suggests depression should be associated with difficulties in functioning, the BSM explains why depressive symptoms evidence such a high comorbidity with other mental disorders (Kessler

& Walters, 1998). Finally, because it is an evolutionary model, the BSM also readily accounts for the fact that there is a substantial genetic component associated with depression (e.g., Kendler, Walters, et al., 1994).

The BSM is valuable in that it links the causes (triggers) with the effects (symptoms) of depression in a logical sequence. It also bears the hallmarks of a good hypothesis because it is parsimonious, consistent across disciplines (from physics to the human social sciences), and makes clear predictions (Wilson, 1998). To give just a few examples, the model predicts that because depressed individuals are focused on avoiding further loss, they should perceive more negative and pessimistic outcomes. Depressed individuals should also be risk aversive and tend to avoid potentially threatening stimuli. Likewise, depressed individuals should be hypersensitive to loss, failure or rejection. Because depressed individuals should be inclined to give up when faced with difficulty, such individuals should demonstrate a very low tolerance for frustration. Also, depressed individuals should exhibit diminished curiosity and explorative tendencies and should shun uncertainty, novelty and sensation seeking. They should be very averse to conflict, particularly with others who are of equal or higher status. They should also engage in less social exchange. Depressed individuals should also demonstrate a decrease in behavioral activity. In short, the BSM makes many clear, easily testable predictions about both the triggers and symptoms associated with depressive condition. If these predictions were not born out by empirical data, then the model would be wrong.

CLARIFYING THE EVOLUTIONARY APPROACH AND THE BSM

Many are unfamiliar with evolutionary approaches to psychology and there is often much controversy and misinterpretations by both adherents and opponents. As such, it is important to address some potential misconceptions that might result from the current proposal. Although a detailed examination of the nature-nurture debate is beyond the scope of this article, some may question whether the BSM suggests that depression is due to social learning or biology (usually meaning physiology and genetics). Indeed, some may be quite surprised that the BSM is a biological approach that strongly emphasizes environmental variables. The wonderful thing about the Darwinian perspective is that it allows one to conceptualize how organisms exist in relationship to their environment (Pinker, 1997). Organisms are viewed as complex problem solvers and the environment is the field in which organisms must solve those problems. As such, behavior is always thought of in organism-environment relationships. The environment relationship is thought of both in terms of the history of the species (phylogeny) and the history of the individual (ontogeny).

The false nature-nurture dichotomy splits "organism" in to one group of causal forces (genetics/physiology/nature) and "environment" in to another group of causal forces (experience/learning/nurture). The conceptual problem this creates can be easily illustrated by drawing analogies with how physicists describe the behavior of objects. Imagine if, when attempting to answer why an object fell (behaved), some physicists vehemently argued that it was the mass of an object (characteristics of the organism) that caused it to fall. They note that objects do fall differently in different environments, but believe the key causal force is the mass (nature) of the object. Other physicists disagree. They note the mass may be important, but the field in which the object exists (environment) is the key to understanding how it behaves. If an object is on a table, it does not fall; but if the table is removed (experience), the object falls.

One does not need to be a physicist to realize that such dichotomizing of causal forces is nonsensical. The behavior of objects can only be legitimately conceptualized as change in object-field relationships. Likewise, behavior of organisms must be thought of as change in organism-environment relationships. In short, the Darwinian perspective does not split causal forces into social learning and physiology/genetics. Instead, the approach conceptualizes how organism-environment relationships unfold over time, both for the history of the species and for the history of the individual.

The evolutionary perspective taken here is a population level analysis that attempts to explain why the tendency to become depressed is present in humans in general. This is a very different level of analysis than the more common approaches in psychology and psychiatry, which usually attempt to explain how and why individuals differ in their tendencies to become depressed. This difference in focus might lead to some confusion. For example, those more familiar with adopting an individual differences perspective on depression will likely point out that there is tremendous variation among people in their tendencies to become depressed. Some people become depressed following minor failures or rejections, whereas others do not become depressed even when faced with the most difficult of circumstances. Does the BSM allow for such marked individual variation? Yes. Although the BSM suggests that the capacity to become depressed is present in everyone, this does not mean that the tendency to become depressed is the same in everyone, nor does it mean that the exact same events should trigger depression in everyone. Virtually all complicated behavioral predispositions, such as dominance, extraversion, or agreeableness, vary significantly throughout the population. Likewise, one would expect tendencies toward depression to vary as well. Indeed, personality theorists have identified a dimension in temperament referred to as neuroticism which is closely related to tendencies toward anxiety and depression (Roberts & Kendler, 1999). The fact that there

is marked individual variation in tendencies to become depressed does not directly challenge the BSM.

Some clinicians or physicians may also note that occasionally patients will experience substantial depressive symptoms without any obvious stressful circumstances. Such patients may report that they should be happy, or that do not know why they are depressed. Do the presence of such patients invalidate the BSM? No. First, the BSM does not purport to account for every instance of depression. There are clear examples of biological defects that can result in depressive symptoms (e.g., hypothyroidism, vascular accidents). That depression may be the activation of an evolved behavioral shutdown mechanism in many, perhaps even the majority of cases, does not mean that depressive syndromes are never the consequence of neurophysiological defects. Second, as any psychodynamic clinician worth her salt will tell you, people are not always aware of why they feel the way they do. Many individuals work very hard at pretending their lives are fine, when in fact they are experiencing significant distress. Other individuals simply have very poor insight into their emotional reactions and thus fail to make connections between important relational conflicts or failures and depressive symptoms.

It will also likely be noted by many clinicians that depression contributes to many difficulties in psychosocial functioning, and thus many will be highly suspect of the notion that depression might be functional at some level. There are several key issues that are necessary to be understood here. First, what is adaptive or maladaptive in an evolutionary sense is very different than what is adaptive or maladaptive in a clinical sense. The yardstick for measuring what is adaptive in an evolutionary sense is the number of genetic copies that are left behind in the subsequent generation. The reference for what is adaptive or functional in a clinical sense is generally considered along the lines of quality of life. Quality of life and number of genes left behind are obviously two very different reference points.

Due to this difference, that which is maladaptive from an evolutionary perspective can be quite different from that which is maladaptive from a clinical perspective. For example, if a patient states that she does not want to have children, this is generally taken as neutral from a clinical standpoint. The individual could be very high functioning and such a choice per se obviously does not represent pathology. From an evolutionary standpoint, however, such a decision is almost the equivalent of dying². Or consider an opposite example. Some one tells

-

Note that this terminology is simply used to illustrate and compare the differences between clinical and evolutionary reference points. I am not suggesting that evolution has the goal of building organisms that reproduce effectively or that evolution "cares" whether an organism lives or dies. This is obviously not accurate.

you he is breaking into a sperm bank and replacing all the other sperm with his own. Such behavior can be considered quite clinically dysfunctional, yet can be considered evolutionarily advantageous in the sense that it would increase his reproductive success. As these examples clearly demonstrate, clinical perspectives and evolutionary perspectives are very different in what is considered adaptive or advantageous. Because of these differences, the depression as defense model does not mean that depression is necessarily good or valuable in the individual or societal sense (see Cosmides & Tooby, 1999). Further, as will be discussed below, this analysis also does not mean that MDD or depression in general should not be treated, either pharmacologically or psychologically.

Another issue regarding functionality is that depression might not be beneficial in an evolutionary sense in modern environments (Beck, 1999; Glantz & Pearce, 1989). Indeed, the BSM strongly suggests that Major Depression can be thought of in terms of a mismatch between modern and ancestral environments. Although many of the characteristics of the ancestral hunter-gatherer environment remain open to speculation, one can be quite certain that our hominid ancestors used to be much more vulnerable and much less in control of their environment than we currently are. Famine, warfare, drought, disease and infection, extreme temperature fluctuations, high predation, authoritarian leaders etc. were likely frequent problems that individuals had little or no control over.

As such, one can be quite certain that behavioral activity was much more dangerous then than now. It is easy to overlook the fact that a laceration that might require 10 stitches in modern times and almost no mortality concerns, would have surely been a serious injury, often leading to infection and perhaps not uncommonly, death. Because of the lesser control and greater danger, the propensity to shut down in unpropitious situations may very well have been adaptive in ancestral environments. However, because the environment is less physically dangerous, injuries are less life threatening, and the presence of powerful drugs and guns make suicide a significant possibility, tendencies toward depression may well be quite disadvantageous in modern cultures (Beck, 1999).

There is also good reason to believe the mismatch between past and present environments is particularly strong in Western societies because of the emphasis on achievement, competition and productivity. In this socio-cultural environment, the passive, avoidant behaviors of a depressed individual are evaluated as particularly dysfunctional. As such, one can easily hypothesize that a vicious cycle is created. Individuals with passive, avoidant tendencies have more difficulty in achievement and competition. The inability to effectively solve important problems in living activates a defensive, depressive response, which in turn makes the situation worse because the passive and

avoidance based behaviors are devalued. Such a model accounts for why depression appears dysfunctional from a clinical standpoint (also see Pattern, 1999).

In sum, the BSM seeks to answer why humans in general become depressed. When one looks broadly at the way in which depression is distributed throughout the population, the types of circumstances that are typically associated with depression, the manner in which the depressive symptoms cluster together, and the psychological characteristics of depressed individuals, the BSM appears to offer a logical, parsimonious explanation that can account for a tremendous amount of information. However, such an approach might generate misconceptions because it contrasts with the more common individual differences approach taken in psychology and psychiatry. The fact that there is substantial individual variation in tendencies to become depressed does not invalidate the BSM. Further, depressive syndromes may occasionally be caused by biological defects. Finally, the BSM makes no claims that tendencies toward depression are currently advantageous in either a clinical or evolutionary sense.

HEALTH POLICY IMPLICATIONS OF THE BSM

The Behavioral Shutdown Model highlights how it is possible to view depression as an evolved defense and, in so doing, challenges the dominant medical model. The BSM suggests that the brain disease model is not valid because many instances of MDD do not involve biological dysfunctions. Instead, the BSM suggests depressive tendencies are passive, avoidance based behavioral strategies that have been fashioned via evolution because such strategies facilitated how organisms dealt with difficult situations in ancestral environments.

The BSM has significant health policy implications. First, the BSM argues for a fairly radical shift in how depression is conceptualized by clinicians, theorists, and researchers. The BSM suggests that depression may be to mental health what pain is to physical health. One could imagine the conceptual confusion if the medical profession at large viewed pain as a disease. Although sometimes this might be a valid conception (as in some cases of chronic pain), for the most part viewing pain as a disease as opposed to a symptom would be highly problematic. Such a paradigm would lead to significant confusion about cause and effect. This would be particularly true if no assessment of structural damage to the body was made. Yet, the BSM suggests just such a paradigm is operating in the case of MDD. Diagnoses are made based solely on symptoms which are, according to this analysis, clearly akin to physical pain. Additionally, although emotional pain is a signal of difficulty interacting with the environment, diagnoses of MDD are generally made regardless of environmental and psychosocial

stressors³. In short, the BSM suggests there may be significant conceptual problems that underlie the medical model approach to depression.

Second, it is well recognized that depressive disorders are grossly under treated (Hirschfeld, et al, 1997). Patients often fail to recognize symptoms or are afraid to talk to professionals. Physicians also often fail to see signs of depression and frequently receive poor professional school education about depression. The BSM suggests that part of the reason that depressive conditions are so under treated is because the disease model fails to provide a readily understandable framework that captures all of the immensely complex personality and social factors that influence the condition. Because of this limiting framework, it is hard to teach both physicians and the lay public about the nature of the condition. In contrast, the BSM offers a readily understandable framework for the nature of depressive conditions. The framework can integrate physiological, psychological and sociological variables into a model that can easily be relayed to the public and professionals alike. Such a model can have tremendous pedagogic value, which in turn could have significant value to society.

The BSM also suggests the "depression as brain disease" message may be problematic in regard to treatment. The disease interpretation provides patients with an alternative explanation for their negative feelings that can ultimately result in dismissal of what their negative feelings are telling them. It makes a big difference if you attribute your unhappiness to your job, to your relationship with your spouse, to your underlying beliefs about the world, or to a brain disease. While the brain disease attribution can alleviate feelings of responsibility, it can also justify turning attention away from socio-emotional problems that may not be genuinely fixed with SSRIs. This possibility is particularly worrisome as more and more primary care physicians are treating depressive conditions simply via prescription (Majeroni & Hess, 1998).

The BSM also makes clear that treatments of depressive conditions with antidepressants alone are useful because they eliminate symptoms. Symptom relief is obviously greatly desirable for many conditions, and the new class of antidepressants should be hailed as a significant advance in the treatment approaches to depression. Nonetheless, the

_

³ Interestingly, there is one exception to this rule. If the depressive symptoms appear in conjunction with the death of a loved one within the past two months, the individual should be considered bereaved. Yet, death of a loved one is the only such exemption. Further, there is no empirical or theoretical justification given for why death of a loved one results in an exemption whereas other intense stressors do not. This raises an important question. If the justification for exempting those individuals who recently experienced the death of a loved one is because such a depressive reaction is deemed normative, then what about individuals who have experienced job loss, spousal infidelities or abuse, divorce, lack of social support, financial problems, or death of a loved one three months ago?

BSM strongly suggests that, as a society, we should be very aware that we are treating symptoms not causes when prescriptions are given for depressive conditions. Although SSRIs clearly appear to benefit individuals, it is unclear at this time if wide spread use of SSRIs will have a negative or positive impact on society at large. On the one hand, it is very possible that the SSRIs are simply suppressing an archaic behavioral shutdown response that leads to dysfunctional behavior in modern environments. As such, these treatments may be purely beneficial. On the other hand, simply providing anesthetics for pain without treating the cause of the pain provides a clear analogy for how excessive reliance on SSRIs for depressive symptoms may have significant hidden costs for individuals and society at large. On the whole, the BSM strongly suggests that psychological and social factors must be addressed if depressive conditions are to be fully treated.

In conclusion, negative emotions and depressive symptoms are the signals that things are not well, that one is having problems, and that one is not getting one's needs met. Although depressive reactions often cause problems, the notion that depression is a manifestation of disease fails to appropriately capture both the nature of disease and the nature of the human experience. Instead, the Behavioral Shutdown Model offers an alternative explanation for depressive conditions that carries with it important implications for public health policy.

REFERENCES

- American Psychiatric Association. 1994. Diagnostic and Statistical Manual of Mental Disorders (4th ed.). Washington, DC: American Psychiatric Association
- Arborelius, L., M. J. Owens, P. M. Plotsky and C. B. Nemeroff. 1999. "The Role of Corticotropin-releasing factor in Depression and Anxiety Disorders" Journal of Endocrinology. Vol. 160: 1-12.
- Archer, John. 1999. The Nature of Grief. New York, NY: Oxford University Press
- Beck, Aaron T. 1967. Depression: clinical, experimental, and theoretical Aspects. New York: Harper & Row
- Beck, Aaron T. 1992. "The descent of Man: An evolutionary Perspective on Major Depression" Newsletter of the Society for Research in Psychopathology. Vol. 3: 3-6.
- Beck, Aaron T. 1999. Cognitive Aspects of Personality Disorders and their relation to Syndromal Disorders: A Psychoevolutionary Approach. In Cloninger, C. R. ed., Personality and Psychopathology. Washington, DC: American Psychiatric Press
- Clark, David. A., Aaron T. Beck and Brad A. Alford. 1999. Scientific Foundations of Cognitive Theory and Therapy of Depression. New York, NY: John Wiley & Sons
- Cosmides, Leda and John Tooby. 1999. "Toward an evolutionary Taxonomy of treatable Conditions" Journal of Abnormal Psychology. Vol. 108: 453-464.
- Doris, A., K. Ebmeier and P. Shajahan. 1999. "Depressive Illness" Lancet. Vol 354: 1369-1375.
- Drevets, W. C. 1998. "Functional Neuroimaging Studies of Depression: The anatomy of Melancholia" Annual Review of Medicine. Vol. 49: 341-361.
- Geary, David. 1998. Male/Female: The Evolution of Human Sex Differences. Washington, DC: American Psychological Association
- Glantz, Klerman and John Pearce. 1989. Exiles from Eden: Psychotherapy from an evolutionary perspective. New York: Norton

- Gilbert, Paul. 1992. Depression: The Evolution of Powerlessness. Hillsdale, NJ; Lawrence Erlbaum Associates
- Gilbert, Paul. 1998. "The Evolved Basis and Adaptive Functions of Cognitive Distortions" British Journal of Medical Psychology Vol. 71: 447-463.
- Gilbert, Paul and S. Allan. 1998. "The Role of Defeat and Entrapment (arrested flight) in Depression: An Exploration of an Evolutionary View" Psychological Medicine Vol. 28: 585-598.
- Gilbert, Paul and B. Andrews. 1998. Shame: Interpersonal Behavior, Psychopathology and Culture. New York, NY: Oxford University Press
- Golding, J. M. 1999. "Intimate Partner Violence as a risk factor for Mental Disorders: A Meta-analysis" Journal of Family Violence Vol. 14: 99-132.
- Gorman, Jack M. and Justine Kent. 1999. "SSRIs and SNRIs: Broad Spectrum of Efficacy beyond Major Depression" Journal of Clinical Psychiatry Vol. 60: 33-39.
- Harris, E. C., and B. Barraclough. 1998. "Excess Mortality of Mental Disorder". British Journal of Psychiatry Vol. 173: 11-53.
- Henriques, Gregg R. 2000. The Harmful Dysfunction Analysis and the Differentiation between Mental Disorder and Disease. Manuscript submitted for publication.
- Hirschfeld, Robert M., Martin B. Keller, Susan Panico, Bernard S. Arons, David Barlow, Frank Davidoff, Jean Endicott, Jack Froom, Michael Goldstein, Jack M. Gorman, Don Guthrie, Richard G. Marek, Theodore A. Maurer, Roger Meyer, Katharine Phillips, Jerilyn Ross, Thomas L. Schwenk, Steven S. Sharfstein, Michael Thase, and Richard J. Wyatt. 1997. "The national depressive and manic-depressive association consensus statement on the undertreatment of depression" The Journal of the American Medical Association Vol. 277: 333-340.
- Johnston, Victor S. 1999. Why We Feel: The Science of Human Emotions. Reading, Mass: Perseus Books
- Judd, Lewis L. 1997. "The clinical Course of unipolar Major Depressive Disorders" Archives of General Psychiatry Vol. 54: 989-991.
- Kendler, Kenneth S., Laura M. Karkowski, and Carol Prescott. 1999. "Causal Relationship between stressful Life Events and the Onset of Major Depression" The American Journal of Psychiatry Vol. 156: 837-841.
- Kendler, K. S., E. E. Walters, K. R. Truett, A.C. Heath, M. C. Neale, N. G. Martin and L. J. Eaves. 1994. "Sources of Individual Differences in depressive Symptoms: Analysis of Two Samples of Twins and their Families" American Journal of Psychiatry Vol. 151: 1605-1614.
- Kessler, R. C., C. Barber, H. G. Birnbaum, R. G. Frank, P. E. Greenberg, R. M. Rose, G. E. Simon, and P. Wang. 1999. "Depression in the Workplace: Effects of short-term Disability" Health Affairs Vol. 18: 163-171.
- Kessler, R. C. and E. E. Walters. 1998. "Epidemiology of DSM-III-R Major Depression and Minor Depression among Adolescents and young Adults in the National Comorbidity Survey" Depression & Anxiety Vol. 7: 3-14.
- Leahy, Robert L. 1997. "An Investment Model of Depressive Resistance" Journal of Cognitive Psychotherapy Vol. 11: 3-19.
- Majeroni, B. A. and A. Hess. 1998. "The Pharmacologic Treatment of Depression" Journal of the American Board of Family Practice Vol. 11: 127-139.
- McGuire, Mark T., and A. Troisi and M. M. Raliegh. 1997. Depression in evolutionary context. In: Baron-Cohen S., ed. The Maladapted Mind. Hillsdale, NJ: Lawrence A Erlbaum Associates
- Merriam, E.P., Michael E. Thase, G. L. Haas, M. S. Keshavan and J. A Sweeney. 1999.
 "Prefrontal cortical Dysfunction in Depression Determined by Wisconsin Card Sorting Test performance" American Journal of Psychiatry Vol. 156: 780-782.

- Nesse, Randolf M. 2000. "Is Depression an Adaptation?" Archives of General Psychiatry, 57, 14-20.
- Nesse, Randolf M. 1998. "Emotional Disorders in evolutionary perspective" British Journal of Medical Psychology Vol. 71; 397-415.
- Nesse, Randolf M. and George C. Williams. 1994. Why We Get Sick: The new Science of Darwinian Medicine. New York, NY: Vintage Books
- Pattern, S. B. 1999. "Depressive Symptoms and Disorders, Levels of Functioning and psychosocial Distress: an Integrative Hypothesis" Medical Hypotheses Vol. 53: 210-216.
- Pinker, Steven, 1997. How the Mind Works. New York: Norton
- Price, John. 1998. "The adaptive Function of Mood Change" British Journal of Medical Psychology Vol. 71: 465-477.
- Reinherz, H. Z., R. M. Giaconia, A. M. Hauf, M. S. Wasserman and A. B. Silverman. 1999. "Major Depression in the Transition to Adulthood: Risks and Impairments" Journal of Abnormal Psychology Vol. 108: 500-510.
- Roberts, Seth B. and Kenneth Kendler. 1999. "Neuroticism and Self-esteem as Indices of the Vulnerability to Major Depression in Women" Psychological Medicine Vol. 29: 1101-1109.
- Seligman, Martin. 1971. "Phobias and preparedness" Behavior Therapy, 2, 307-320.
- Soares, J. C. and J. J. Mann. 1997. "The Anatomy of Mood Disorders--Review of Structural Neuroimaging Studies" Biological Psychiatry Vol. 41: 86-106.
- Spitzer, Robert L. 1999. Harmful dysfunction and the DSM definition of mental disorder. Journal of Abnormal Psychology, 108, 430-432.
- Trivers, Robert 1971. "Evolution of Reciprocal Altruism" Quarterly Review of Biology, 46, 35-57.
- Wakefield, Jerome C. 1992. "Disorder as harmful Dysfunction: A conceptual Critique of the DSM-III-R's definition of Mental Disorder" Psychological Review, 99, 232-247.
- Wakefield, Jerome C. 1999a. "Evolutionary versus Prototype Analyses of the Concept of Disorder" Journal of Abnormal Psychology Vol. 108: 374-399.
- Wakefield, Jerome C. 1999b. "Mental Disorder as a black Box essentialist Concept" Journal of Abnormal Psychology Vol. 108: 465-472.
- Wilson, Edward O. 1998. Consilience: The unity of Knowledge. New York: Alfred A. Knoff. Inc.
- Winokur, G. 1997. "All Roads Lead to Depression: Clinically homogeneous, etiologically heterogeneous" Journal of Affective Disorders Vol. 45: 97-108.
- Yu, Yan and David R. Williams. 1999. Socioeconomic Status and Mental Health. In Aneshensel, C. S., & Phelan, J. C. (Eds), Handbook of the Sociology of Mental Health. New York, NY: Plenum Publishers
- Zhang, Mingliang, Kathryn Rost and John C. Fortney. 1999. "Earnings changes for depressed individuals treated by mental health specialists" The American Journal of Psychiatry Vol. 156: 108-11.