

EVOLUTION: PSYCHIATRIC NOSOLOGY'S MISSING BIOLOGICAL FOUNDATION

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Summary

The new system of diagnostic criteria for emotional disorders has brought much needed reliability, but it neglects the most fundamental distinction in medical diagnosis, that between symptoms and the diseases that cause them. As a result, psychiatric diagnosis for emotional disorders remains where medical diagnosis was two centuries ago, classifying constellations of protective responses as if they were diseases. The result is emphasis on individual differences in tendencies to negative emotions, and neglect of other emotional disorders and the life situations that give rise to emotions. An evolutionary perspective can advance the diagnosis of emotional disorders in several ways. First, it emphasizes that disorders of both excess and deficiency are possible for every emotion, thus calling attention to neglected disorders with deficits of negative emotion or excesses of positive emotion. Second, it confirms that the normality of an emotion depends unavoidably on the context. Third, it notes that variations in brain mechanisms that make a person susceptible to anxiety or depression are only sometimes diseases; they may have the same causal significance as variations in brain mechanisms that make a person especially prone to cough or fever during a cold. Fourth, it notes that biologically normal responses may be aversive and even harmful to individuals. Finally, it suggests the importance of a detailed and evolutionarily informed analysis of the motivational structure of every patient's life. This understanding is the foundation for all treatment.

Key Words: Diagnosis – Nosology – DSM – Evolution – Psychiatry – Emotions – Defenses

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Hundreds of smart, sincere, sophisticated researchers and clinicians have collaborated for the past three decades to revise the diagnostic criteria for mental disorders (Wilson 1993). The products of their labors have many benefits, but they are nonetheless the source of widespread dissatisfaction and apparently irresolvable debates (Beutler and Malik 2002, Horwitz 2002). Clinicians often ignore the official diagnostic system, while researchers find themselves constrained by categories with no theoretical foundation and questionable reliability that include heterogeneous patients who show vast comorbidity. High prevalence rates lead nonprofessionals to join experts in asking whether there is a scientific foundation for the distinction between normal and pathological. Even the architects of the system suggest the need for fundamentally new perspectives.

Science strives for simplicity of explanation. Descriptive models tend to be piecemeal and complicated. We are at the epicycle stage of psychiatry where astronomy was before Copernicus and biology before Darwin. Our inelegant and complex current descriptive system will

undoubtedly be replaced by explanatory knowledge that ties together the loose ends. Disparate observations will crystallise into simpler, more elegant models that will enable us not only to understand psychiatric illness more fully but also to alleviate the suffering of our patients more effectively (Frances and Egger 2003).

Such extensive dissatisfaction after extended Herculean efforts suggests that persisting in the same path will not yield major advances. Instead, it appears either that we are looking at the problem from the wrong perspective or that the simplicity we seek does not match nature's patterns. This article argues that evolutionary behavioral biology offers a crucial but neglected scientific foundation for psychiatric nosology. By posing evolutionary questions about why we all are so vulnerable to negative emotions, it highlights a fundamental misunderstanding at the heart of the Diagnostic and Statistical Manual of Psychiatric Disorders (DSM) that has kept psychiatric diagnoses artificially different from those in the rest of medicine. By providing a biological foundation for understanding emotions, it reveals why the quest

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for simple criteria for emotional disorders is so frustrating and where we might look for solutions.

This article emphasizes the utility of evolutionary principles for classifying emotional disorders, but these principles are also useful for classifying other psychiatric disorders. For instance, behavioral disorders such as addiction or eating disorders make much more sense in an evolutionary framework. Personality disorders can be organized based on the strategies people use to influence other people. Even psychoses and neurological conditions are illuminated by evolutionary considerations of the selection forces that maintain the frequency of predisposing genes and how they interact with novel aspects of the modern environment. Here, however, the focus is on the emotions and the categories that describe their disorders.

Much has already been written, especially seminal work by Wakefield, about how an evolutionary approach can help distinguish pathological from nonpathological conditions (Wakefield 1992, McGuire and Troisi 1998, Clark 1999). His concept of "harmful dysfunction" brings a biological foundation to the philosophical question of where normal stops and pathology begins. The concept of dysfunction grounds diagnosis in the selective advantages of normally operating brain mechanisms. By also requiring a condition to be "harmful," his approach acknowledges that what is good for our genes is not necessarily good for our selves, and what is good in one culture may not be in another. This essay builds on Wakefield's work and attempts to extend it by emphasis on the simple error at the center of DSM classification of emotional disorders and what can be done about it. A similar perspective can be found in the article by Feerman in this issue, and in previous work by McGuire and Troisi (McGuire and Troisi 1998), who say in a recent review:

"The Darwinian concept of mental disorder builds from two basic ideas: (1) the capacity to achieve biological goals is the best single attribute that characterizes mental health; and (2), the assessment of functional capacities cannot be properly made without consideration of the environment in which the individual lives" (Troisi and McGuire 2002).

Before further considering such issues, however, it is essential to recognize the forces that shaped the DSM, and the enormous progress it represents compared to prior classification efforts. The history of mental illness taxonomy began with highly speculative informal categories originating thousands of years ago. At turns biological, phenomenological, and moral in orientation, such informal systems prevailed well into the 19th Century, when Emil Kraepelin took the first steps toward modern, systematic classification in collaboration with his colleague Allen R. Diefendorf (Kihlstrom 2002). In the United States, formal classification systems for mental disorders were first adopted not by clinicians, but by the federal government because of its need to track asylum populations accurately. This encouraged the American Medico-Psychological Organization (AMPA) to publish the first standardized psychiatric nosology, the Statistical Manual for the Use of Institutions for the Insane (Statistical Manual) in 1918. The absence of the word "diagnosis" in the title accurately represents the marginal utility of the manual to the era's mental health practitioners.

The Statistical Manual was revised for the last time in 1942 just as the United States entered World War II (Grob 1991, Houts 2000). Military practitioners found the statistical categories woefully inadequate to describe battlefield psychological casualties. Dr. George Raines, then head of the American Psychiatric Association (APA) Committee on Statistics and Nomenclature, noted in the introduction to the first edition of DSM that "only about 10% of the total cases seen [in WWII] fell into any of the categories ordinarily seen in public mental hospitals" (American Psychiatric Association 1952). Such dissatisfactions led the APA to replace the Statistical Manual with a new standardized nosology in 1952 – the DSM-I.

Although more useful to practitioner's, DSM-I and its revision, DSM-II, were unsatisfactory for research. Prior to the formulation of the Research Diagnostic Criteria and the publication of the Third Edition of the Diagnostic and Statistical Manual (DSM-III) (Association 1980), research reports were hard to compare because the subjects in one study of "schizophrenia" or "depression" might have quite different conditions from those in another study. Critics pointed to such inconsistencies to argue that psychiatry was unscientific or even that mental illnesses were not diseases at all (Szasz 1974). Examples of malingering were published in *Science* as evidence for the subjectivity of psychiatric diagnosis (Rosenhan 1973). At about the same time, the utility of psychotropic drugs was being widely recognized and insurance companies began paying only for the treatment of specific medical disorders. These several crises combined to create a consensus that psychiatry should become more like the rest of medicine (Houts 2002, Jackson unpublished). Operationalizing diagnostic criteria was the obvious place to start.

The committee charged with creating the DSM-III quickly found there would be no agreement on a theoretical foundation for psychiatric nomenclature (Wilson 1993). Psychoanalysts remained powerful, and their views of mental disorders were fundamentally at odds with "biological" psychiatrists who emphasized the brain origins of mental disorders. To get past this impasse, the group created diagnostic categories avowedly without theoretical foundation. The goal was a system derived empirically, that started from clinical observations of observable signs, symptoms and the disease course. Building on criteria from the International Coding Diagnoses and the Research Diagnostic Criteria group at Washington University (Feighner et al. 1972), the DSM-III and DSM-IV attempted to create categories defined by observable data (American Psychiatric Association 1994).

The inauguration of operationalized diagnoses transformed psychiatry (Guze 1992, Wilson 1993, Jackson unpublished). Indeed, the history of medicine contains few transitions so sudden and complete (Shorter 1997). Prior to the DSM-III, psychiatrist's diagnostic categories were theoretically-based and used to complement more valued narrative explanations for how an individual came to have his or her particular constellation of symptoms. Clinicians crafted highly idiographic explanations for problems in a particular individual in much the same way that historians explain the origins of a civil war or economic collapse in a particular country. Nomothetic (universally applica-

ble) principles were incorporated into such explanations, but different clinicians used different principles. For instance, psychoanalysts emphasized the ubiquitous importance of defenses against Oedipal wishes, while behaviorists emphasized the reinforcement history. Arriving at such diagnostic formulations was an occasion for deep thought, sophisticated discussion, theoretical battles and frequent flights of fancy. Two diagnosticians often arrived at plausible formulations with little in common and no way to decide between them. Reliability was low. Such diagnoses were nearly worthless for categorizing subjects into groups for study.

Progress and its price

Current criteria are nearly the polar opposite of their predecessors. The old emphasis on individualized explanations for constellations of symptoms has been replaced by diagnostic categories defined by the presence or absence of specific signs and symptoms. For instance, a diagnosis of major depression applies to anyone who has had at least five of nine symptoms for at least two weeks, at least one of which is depressed mood or lack of pleasure (American Psychiatric Association 1994). Precipitating events are not taken into account, with the exception of bereavement in the past two months. Whether symptoms arise during what should be a relaxing vacation, or a stay in intensive care, is irrelevant for making a diagnosis. Such exclusion of life context seems mindless, until you consider the problem of how to measure the kind and severity of precipitants. If criteria for depression required assessing the severity of recent life events, complexity would increase and diagnostic reliability would plummet.

The quest for criteria that yield reliable diagnoses is well-justified. If different clinicians examining a patient arrive at different diagnoses, the system is not all that useful (Goodwin et al. 1979). Explicit criteria made possible standardized interviews that further enhanced reliability (Spitzer et al. 1992). Versions useable by lay interviewers have made extensive epidemiology possible for the first time, not just in the USA, but in over 39 countries, where the same questions are administered using the same instrument translated into different languages (Kessler and Ustun 2004). This is real progress, and the data is useful for public health planning as well as research.

In short, the DSM has been essential for most recent progress in psychiatry. Treatment trials now target groups of well-defined patients and the results can be applied to other similarly defined groups. Research studies can now measure genes, neurotransmitters or brain structures in well-characterized groups of patients as compared to controls. Reliable diagnostic criteria have advanced psychiatric research more than any individual research project could.

Given such dramatic progress, why is there such dissatisfaction with the DSM and its approach to diagnosis (Beutler and Malik 2002, Watson 2005)? Many objections are based on the tangible factors outlined above—high prevalence rates, high comorbidity, heterogeneity within groups, and questionable reliability (Phillips et al. 2003). However larger issues are even

more important.

First, the distinction between normal and abnormal remains fundamentally arbitrary.

For cancer, pneumonia, rheumatoid arthritis and pinworms, you either have the condition or you don't. A zone of rarity separates the condition from normal (Kendell 1975). Most emotional disorders offer no such clean demarcation, leading some to suggest that diagnoses be dimensional instead of categorical. However, to communicate, humans insist on using words that refer to categories. People demand to know the boundary that separates pathology from normality. Dimensions are not diagnoses. Even high blood pressure is defined by a specific cut off.

Second, the DSM diagnoses are products of clinical observation unconnected to any theory of human behavior. This is not quite correct. Because explicit theories are excluded, the DSM criteria tacitly foster thinking about mental disorders as if they are diseases. This makes them fit easily into neuroscience models that seek to identify brain abnormalities correlated with each disorder.

Third, is the problem of how to incorporate context (Faust and Miner 1986). The DSM approach relegates much of what we know about the effects of life events to "stress," as if stress hormones mediated all adverse effects of social experience. Clinicians understand the far more complex relationships between life events and psychological structures (Brown and Harris 1978, Monroe et al. 2001). Many see the need to adjust the diagnostic threshold depending on the situation, lowering it for apparently unprovoked symptoms, increasing it in extreme life situations, however the criteria are unyielding (Wakefield and First 2003). For instance, panic disorder is the diagnosis for everyone who has problems for a month after recurrent unexpected episodes that include four out of ten possible panic symptoms, whether the patient had the onset in a grocery store, or in a prison camp. The reason for this rigidity is that attempts to include context would require difficult to define objective criteria for levels of provocation.

At the same time, most everyone recognizes the need to consider the circumstances in order to judge whether an emotion is normal or not. Following the debut of the multi-axial diagnostic system in DSM-III, it appeared that the editors of DSM had at least partially recognized the need to integrate life circumstances and context. Severity of psychosocial stress (Axis IV) and level of adaptive functioning (Axis V) were added to enrich the clinical context of the individual (Klerman 1984). However, the inclusion of environmental circumstances in separate axes excludes important contextual information from their important role in making Axis I diagnoses.

The DSM gives us categories for emotional disorders, but says nothing about what these disorders are. Are they diseases? Are some merely responses to life circumstances? Is the cause located in individual vulnerability differences, in exposure to environmental events, or in cognitive interpretations? Most everyone pays lip service to the bio-psycho-social model, but few are willing to get into the complexity of how individual differences interact with situations, events and cognitions to give rise to symptoms that have evolu-

tionary significance (Gilbert 1989).

Thinking about patients as DSM diagnoses instead of persons impoverishes clinical understanding (Faust and Miner 1986). For instance, a resident recently concluded a case presentation by saying, "The diagnosis is major depression so I prescribed an SSRI". When asked why this person was depressed now, the resident replied "Well, we think depression is caused mostly by genetic factors, but also by stress," omitting any mention of why this particular patient was depressed now. When pressed to do so, he explained that there was a family history of depression and the patient had been abused in childhood, was in a bad marriage and had recently lost his job after a DWUIL conviction. The resident clearly imagined that his job was to place his patient in the category "major depression" and to prescribe a treatment that was usually effective for someone in that category. He had not even tried to figure out whether the person had had a previous satisfactory and stable life adaptation, whether the alcohol use initiated the marital problems or came later, and whether the person was capable of maintaining good relationships. In short, like many young clinicians, the resident viewed DSM criteria as if they described specific diseases with specific consistent causes. He assumed that the diagnosis contained all that he needed to know to arrive at a treatment plan.

The same physician would never undertake such a crude approach to diagnosing and treating cough or pain. If a patient presented with a severe cough and fever, he would not be satisfied with a diagnosis of "cough disorder", he would instead consider all the possible causes of cough, and would not prescribe treatment until arriving at the best possible understanding of why this person had this cough now. Is it COPD, or pneumonia, or congestive heart failure, or COPD and congestive heart failure complicated by pneumonia? The physician would find out whether the individual was especially vulnerable because of immunosuppression or steroid use, whether there were exposures to infectious agents, and whether the person had allergies. General physicians recognize that cough is not a disease, it is a response to a disease. Likewise, while pain can be abnormal, physicians recognize that pain is usually a response to pathology, not a disorder in itself. Psychiatrists sometimes think of anxiety as a potentially useful response to a danger, but other emotions such as depression and jealousy are usually thought of as abnormalities instead of being recognized as potentially useful responses to untoward situations.

The Basic Fault

The flaw in the DSM approach to emotional disorders is fundamental; the DSM fails to distinguish protective responses from diseases. This flaw is by no means new; the DSM merely extends the Kraepelinian tradition. Kraepelin excluded etiology and anatomic considerations from mental disorder classification because reliable information was not accessible except in the case of obvious injuries and post-mortem neural lesions (Kihlstrom 2002). In his 1904 textbook, Kraepelin recognized the limits of a nosology based on

symptoms, but he also noted that diagnostic systems based on a comprehensive knowledge of symptoms or pathological anatomy or etiology should provide "uniform and standard classifications" that mapped well onto one another, no matter what the starting point (Kraepelin and Dierdorf 1907).

It is a short leap from this to equating the outcome of exhaustive identification of symptomatology with the exhaustive identification of etiology; if all nosologies carve up the pie identically, then any one system should work as well as any other. This explanation is especially appealing if some systems are inaccessible, as pathology and etiology were in the late 1800's. However, assuming that symptomatic categories will match etiological categories comes at the high price of blurring the directional relationship between cause and effect, leading to two kinds of errors. First, categories based on symptom constellations may contain subgroups that arise from fundamentally different causes. Second, such categories fail to distinguish symptoms that arise from pathological causes from those that are aroused by normally functioning systems. The former is an error of failing to distinguish distinct disorders (e.g., yellow fever vs. spotted fever), while the latter fails to distinguish disorders from the symptoms of disorders (e.g., mistaking fever or cough for disorders, when they are actually protective responses to the disorder of pneumonia).

The rest of medicine long ago replaced symptomatic diagnoses such as "cough disorder" with etologically based diagnoses such as pneumonia or lung cancer. (Kihlstrom 2002). The rest of medicine recognizes cough, fever, pain, nausea, fatigue, diarrhea, vomiting, and inflammation as responses to diseases, not diseases themselves. These responses are aversive, and they can be dangerous, disabling, and even fatal. High fever can cause convulsions and diarrhea causes thousands of deaths each year. Nonetheless, the capacities for fever, diarrhea and other defenses are the body's adaptive responses to problems, not problems themselves. They give important clues to the diagnosis, but they are themselves diagnoses only in special circumstances.

One circumstance is when the cause cannot be found. For instance, "fever of unknown origin" is a stand-in for a diagnosis when no reason for a fever can be identified. The other circumstance is when the system that regulates the response is presumed to be abnormal, as is the case in many chronic pain syndromes. Chronic fatigue is likewise usually thought to arise from an abnormality of a regulation system. When every other possible cause has been eliminated, even fever or pain may be attributed to an abnormal regulation mechanism.

The error of failing to distinguish defenses from diseases needs a name. Most simply it can be called, "*The fallacy of mistaking defenses for diseases*". It could be called "*The DSM fallacy*" because the DSM so resolutely ignores the distinction. The DSM takes great pains to carefully define when symptoms are severe enough to justify a diagnosis, but it mostly ignores the functions of negative emotions and the situations that arouse them.

This argument is based on the supposition that negative emotions are protective reactions akin to pain

and fever. The next section reviews reasons to think this is correct. However, major differences between physical protective responses and emotional responses make the correspondence hard to see at first. The situations that arouse fever and cough are observable changes in specific tissues. Most arise from diseases or injuries. The situations that arouse negative emotions are also adverse, but few are specific diseases with identifiable tissue pathology. Many are injuries to social resources such as relationships or social status that are less tangible despite their importance to function and Darwinian fitness. Some situations, such as exclusion from a group, directly arouse negative emotion. Other connections between situations and emotions are far less direct, such as the anxiety that follows a subtle vocal inflection that suggests new distance in a previously close relationship.

Fever and cough indicate the presence of an infection or some other disadvantageous abnormal state. Anxiety and sadness arise from states that are disadvantageous, but generally not abnormal. This apparently major difference can be turned on its head by noting that the infections that arouse fever and cough are not exactly diseases, they are just conflicts with pathogens of the sort that our bodies manage constantly. The symptoms are aspects of the body's well-established plan for dealing with infections. Both physical and emotional responses are useful only in certain situations. For physical responses these situations are more tangible and more likely to be abnormalities. For emotional responses, the etiology is not usually a disease process. To avoid confronting the complex social situations that arouse negative emotions, psychiatry has defined extremes of negative emotions as disorders. The result is a major emphasis on individual differences in "vulnerability" to negative emotions, and a relative neglect of all the interesting structure in the life events and situations that we call "stress."

Evolution and Emotions

The proper foundation for understanding emotional disorders is an evolutionary understanding of why the emotions exist at all (Nesse 1990, Tooby and Cosmides 1990, Nesse 1998). The same logic is at the heart of pathophysiology. To understand the kidney, we first try to understand what it is for. Armed with this knowledge, we can understand how the nephron works and why it is the way it is. Such evolutionary functional understanding is so intrinsic to physiology that it is easy to overlook that it includes two separate kinds of knowledge, one an evolutionary explanation for why a trait exists at all, the other a proximate explanation for the details of the trait's structure and how it works (Tinbergen 1963).

It is tempting to posit functions for emotions that are just as straightforward as functions for abdominal organs, but this is a mistake. The abdominal organs are always present and constantly useful, while emotional states are aroused only in certain situations and they are useful only in those situations. Panic, for instance, may be life-saving when serious danger is present, otherwise, it is worse than useless. The correct way to analyze the utility of an emotional state is define the

situations in which it is useful and the adaptive challenges posed by those situations. In the face of life-threatening danger, rapid breathing oxygenates the blood, muscle tension increases strength and insulin allows glucose to flow into muscles. Emotions have utility for communication, motivation, and for adjusting physiology and behavior, but there is no need to consider which of these is primary. All are part of a special coordinated state that gives an advantage in a certain situation (Nesse 1990 Tooby and Cosmides 1990). For instance, sweating, rapid heartbeat, muscle tension and a wish to escape are all useful when confronted by danger that demands fight or flight, and they serve a variety of related functions. Emotions are like computer programs that adjust multiple aspects of the organism to cope with the exigencies of situations that have recurred over evolutionary time. Organisms with such abilities to adjust have an advantage over those that make no adjustments.

Emotions are positive or negative for the simple reason that special states are useful only in situations that pose opportunities or threats. Positive or negative subjective experience is but one aspect of an emotional state that includes changes in arousal, motivation, physiology, memory, and action tendencies (Plutchik 2003). Negative emotions are naturally associated with untoward situations, so it is easy to incorrectly conclude that they are themselves problems. This "Clinician's Illusion" is a serious impediment to understanding and treating emotional problems (Nesse 2005).

It would be grand if all who treat emotional disorders could take several courses about emotions or at least read one good text (Plutchik 2003), but some of the debates in emotions research would likely be more distracting than illuminating (Ekman and Davidson 1994). For instance, arguments continue about whether emotions are best viewed as dimensions or as a few distinct basic kinds with combinations. An evolutionary approach offers a possible resolution by tracing the phylogeny of various emotions over evolutionary time as they have been gradually but only partially differentiated from one another in order to cope with diverse kinds of situations (Nesse 2004).

This view has profound implications for psychiatric diagnosis and the comorbidity of the emotional disorders. For instance, instead of attempting to determine whether the various anxiety disorders are fundamentally the same or fundamentally different, it suggests that anxiety has been partially differentiated into subtypes shaped to cope with a variety of different kinds of dangers. We should, therefore, not expect to be able to sharply differentiate subtypes of anxiety (Marks and Nesse 1994). Similarly, the profound overlap between anxiety, sadness, low mood and depression arises because they are responses to related kinds of danger. Anxiety is aroused by situations that pose threats of possible future loss. Sadness is aroused by loss. Low mood is aroused by the expectation that one will be unable to reach an important goal. Depression arises if efforts persists despite the decreased motivation associated with ordinary low mood.

There is no room here for a detailed consideration of the full spectrum of emotions to say nothing of the extensive research and writing about them (Barlow 1991, Izard 1992, Oatley and Johnson-Laird 1995,

Lewis and Haviland-Jones 2000, Fessler and Haley 2003, Fessler in press). Instead, consider a list of some common situations and the emotions they arouse.

- Opportunity → Desire, excitement
- Success → Joy, happiness
- Failure → Disappointment
- Threat of damage → Fear
- Threat of social loss → Anxiety
- Loss → Sadness
- Failure to make progress towards an important goal → Low mood
- Inability to get or protect an essential resource → Despair
- Betrayal → Anger
- Contamination → Disgust

The list could be greatly extended, but the relationship among different emotions becomes more clear if they are organized into groups that correspond to the two main classes of situations individuals need to cope with (Nesse 1990, Nesse 2004). The first is goal pursuit and the problem of what to do when, and with how much effort and persistence. Living is a sequence of episodes in which organisms attempt to reach goals and avoid losses. **Table 1** summarizes the domain-general emotions that arise in the situations associated with goal pursuit. It presumes that a somewhat consistent set of brain mechanisms has regulated the pursuit of diverse goals in different organisms over hundreds of millions

of years. For any particular species, these global emotions gradually get somewhat specialized to cope with particular kinds of goals. For instance, when faced with the possibility of losing a mate most humans experience not just generic anxiety, but the complex emotion of jealousy. The regulation of these emotions is further specialized by life experience.

The other group contains emotions shaped to deal with the situations that repeatedly arise in managing social relationships (see articles by Beahrs, this issue). As most readers will know, evolutionists and economists often model the trading of favors as a Prisoner's Dilemma in which the maximum net outcome emerges from repeated mutual cooperation, but on any given move, a player who defects gets a big gain at the expense of the other player (Axelrod and Hamilton 1981). I and others have argued that these situations are so ubiquitous that they have shaped specific emotions, trust and friendship after repeated successful exchanges, suspicion and anger before and after the other defects, and anxiety and guilt before and after the self defects (Ketelaar and Clore 1997).

These tables are not intended to be exhaustive. For instance, surprise is a more general emotion aroused by situations that give rise to unexpected outcomes. Disgust, far more specific, is aroused by the presence of contaminated materials. All of the above emotions deserve extended explication that are available elsewhere (Plutchik 2003). They are summarized briefly as a prelude to addressing the question of emotional disorders.

Table 1

Emotions shaped to deal with the situations that arise during goal pursuit

Situation	Before	During	Obstacle	After success	After failure
Opportunity					
<i>Social</i>	Excitement	Engagement	Frustration	Joy	Disappointment
<i>Physical</i>	Desire	Flow Interest	Anger Despair	Happiness Pleasure	
Threat					
<i>Social</i>	Anxiety	Confidence	Dread	Relief	Sadness
<i>Physical</i>	Fear	Coping	Despair		Pain

Table 2

Emotions shaped to deal with the situations that arise in relationships

	Other cooperates	Other defects
You cooperate	Trust Friendship, love	Before: Suspicion After: Anger
You defect	Before: Anxiety After: Guilt	Rejection Disgust

Emotional Disorders

An evolutionary framework for understanding emotions has several general implications for a nosology of emotional disorders.

1. Emotional disorders should be distinguished as a special class of mental disorders (Watson 2005). In DSM-II they were better unified, but they have since each been pulled out as separate disorders.

2. Because emotions adjust the organism to cope with certain kinds of situations, the normality of an emotional state cannot be assessed without information about the current situation (except for certain extreme emotional states that will be abnormal in almost all situations).

3. The word “disorder” implies an abnormality of the mechanisms that regulate emotions. This must be carefully distinguished from emotions that arise from normal mechanisms but nonetheless cause distress or impaired function (Wakefield 1992).

4. Two distinct classes of abnormalities are possible for each emotion:

- a. Too much: too quickly aroused, too intense, too long, or aroused by too many cues
- b. Too little: too slowly aroused, too mild, too short, or aroused only by excessively specific cues.

Emotions theorists now know well that emotions arise not from directly apprehended cues, but from an appraisal of what the new information means for an individual's ability to reach personal goals (Ellsworth 1991), a perspective that encourages attention to the life of the individual

5. Negative emotions are just as useful as positive emotions. It is essential to avoid the Clinician's Illusion that makes negative emotions seem abnormal. No one comes to the clinic complaining of too little anxiety or an inability to feel sad, but this is just an artifact of our limited imagination and the absence of a scientific foundation for diagnosis of emotional disorders. People with these disorders exist, they just are not complaining or coming for treatment. Instead, they show up in the emergency room or jail or unemployment lines.

6. The mechanisms that regulate expression of emotions are governed by the smoke detector principle—inexpensive defenses are often subject to false alarms that are perfectly normal (Nesse 2005).

7. What is useful for our genes is not necessarily useful for our selves. Much normal emotion, especially negative emotion, may not be worthwhile for individu-

als at all, but only for their genes, and sometimes only for their genes in kin. It is also important to recognize that emotions were shaped to deal with Paleolithic problems, so normal emotions may now be useless or harmful.

8. The distinction between negative and positive emotions intersects the distinction between abnormalities of excess and deficit to define four broad classes of emotional disorders, two of which have been neglected because they do not lead to subjective complaints. See **Table 3** for details.

These and related principles provide a foundation for a scientific nosology for emotional disorders. An improved diagnostic system based on them will seem senseless to those who do not understand the behavioral biology of emotions.

This framework encourages systematic consideration of disorders of excess and deficiency for every emotion, not just anxiety and depression. The vast majority of treatment is for anxiety and depression, of course. They are usually called affects instead of emotions, to reflect their more enduring presence and the difficulty of connecting them to a very specific situation, but the conclusions are the same nonetheless. Both positive and negative affects and emotions can be disordered by being excessive or deficient. Jealousy and disgust can be deficient as well as excessive. Guilt can be excessive or deficient, depending on the situation.

An emphasis on the evolved utility of negative emotions should not lead to the conclusion that they are always useful, nor should it distract attention from the huge genetic variation in emotional predispositions. Some people rarely experience guilt while others feel constantly that they have somehow transgressed. Some people rarely worry, others worry constantly. Some have never experienced romantic love, others fall madly in love with remarkable regularity. This variation poses a major problem for any attempt to determine what emotional experiences are normal.

Part of the answer is in how natural selection shapes the systems that regulate behavior. About half of the variation among individuals in most emotional traits arises from genetic differences. Why hasn't natural selection shaped a much more narrow range of responsiveness that we can recognize as “normal?” It is because humans have evolved in varying physical and social environments and because the fitness effects of being high or low on a particular trait are not likely to have a consistent effect on fitness. The resulting variation in personality traits is so large as to sometimes

Table 3

Categories of emotional disorders

	Excess	Deficit
Positive emotions	Mania, erotomania	Lack of joy, love, interest
Negative emotions	The usual emotional disorders: Anxiety, depression, etc.	Deficits of anxiety, low mood, jealousy, etc.

make us wonder if we are even justified in talking about human nature.

We can now return to the DSM approach to diagnosis and the problem of taking context into account. The criteria for some disorders have built in exclusions that generally ensure that anyone who meets criteria does indeed have a disorder. For instance, the criteria for panic disorder refer to “unexpected attacks” which excludes panic in life threatening situations. Panic disorder is a reliably pathological condition in which the threshold for panic is so low that attacks emerge spontaneously. What an evolutionary perspective adds is recognition that panic is a normal response that is expressed too readily in panic disorder (Nesse 1987). This simple fact is useful in psychotherapy. Patients who have spent months fearing they have heart disease or a brain tumor often can be helped to recognize that their symptoms would indeed be useful in extreme danger and that they are experiencing mere false alarms.

Jealousy is a more complicated example. In the face of threats to a mate’s fidelity, jealousy is normal and its absence is abnormal (Buss 2000). However in many instances jealousy seems to be pathological. In many such cases, later evidence reveals the emotional response was an accurate indication of what was actually happening. In others, jealousy is aroused in someone who is depressed or who otherwise feels that his or her partner could do better with someone else. Then there is the psychoanalytic observation that jealousy can arise from projecting illicit desires onto an innocent partner. The important point here is that different cases of pathological jealousy may have different origins, but differentiating and understanding them requires knowing the situations in which the emotion is useful.

The overwhelmingly common disorder is, of course, depression. Increasingly, patients receive a diagnosis after a brief interview with a general physician who prescribes antidepressants and advises a return visit in a month. Such perfunctory treatment is often justified by noting that the patient has met criteria for a pathological condition, major depression, whose presumed etiology is a deficiency of brain neurotransmitters. Drug treatment seems indicated and has been proven somewhat effective, so why not get on with it? This sequence completely ignores any possible utility of low mood, to say nothing of possible causes of an individual’s depression.

An evolutionary approach recognizes that low mood is useful to disengage effort from enterprises that are failing (Price and Sloman 1987, McGuire et al. 1997, Nesse 2000, Wrosch et al. 2003, Nettle 2004). If the person persists in useless efforts, the low mood escalates to full depression. It sounds easy to recommend giving up a fruitless pursuit, until you realize that the goal may be getting a child off drugs, finding a job or ending an affair. Treating depression without a careful examination of a patient’s motivational structure is like treating a cough without first trying to find its cause (Nesse 2005).

Understanding Motivational Structure

The most useful contribution evolution makes to

psychotherapy may be the framework it offers for analyzing the motivational structure of an individual’s life. Like other organisms, humans must allocate three kinds of effort to get resources in six different areas. Somatic effort yields personal resources and material resources. Reproductive effort yields mates and offspring. Social effort yields allies and status. Many people seem to imagine that there is some normal way to live without compromises, but an evolutionary perspective reminds us all that every human action is an investment in getting one kind of resource at the expense of others. More time working out means less time working. More time impressing potential mates means less time for childcare. More time seeking status means less time for everything else.

The motivation regulation system seems to be designed, sensibly enough, to focus effort where it is most needed. That is, wherever it will yield the greatest payoffs of reproduction-limiting resources for the least investment. It would be so nice if our minds settled comfortably to a focus on what we have, but after any satisfaction the mind turns quickly to solving the next problem (Nesse 2004). Many tasks are simply enterprises that work well such as a job or a marriage. What then is a life problem? A life problem is difficulty in getting or keeping some important resource. People describe their problems in such diverse ways it is at first amazing to see how easily they all fit into a behavioral biological framework. The foundation for any therapy, especially psychotherapy, is a detailed understanding of what resources and sources of resources the person has, what he or she wants, how he or she is going about reaching these goals, and what the expectations are for success or failure. Many depressed people seem to have nothing major lacking in their lives. But as we get to know them, we find that they are striving to get love from a cold mother, sex from an uninterested spouse, or praise from a competitive boss. Or, they are trying to be truly good at all times, or to be the world’s best in some status competition, achievements that are rare, and always temporary.

Good therapists intuitively grasp motivational structures and the exigencies that give rise to an individual’s problems. An evolutionary perspective and knowledge about emotions can help nearly every therapist to do this even better. There is the risk, of course, of using such insights to make crude suggestions. We recall a patient who visited the ER with depression he attributed to his wife’s disinterest in sex. He was told, “Well, you will have to leave her or put up with it, those are your choices.” Better therapists know that people have good reasons for why they live in the way they do. They examine their patients to see if symptoms arise from bipolar disorder or some other distinctive condition, but they recognize that diagnoses are no substitute for a deep understanding of a person’s life.

Towards an evolutionary foundation for psychiatric nosology

Medical knowledge about particular syndromes, whether psychiatric or non-psychiatric, tends to progress through a series of increasingly valid stages. It begins with observing symptom patterns, progresses

by identifying structural anomalies and disorders of function and pathology, and finally arrives at a classification based on etiology within a functional system (Scadding 1990, Houts 2002). In the later 19th century, Kraepelin had to be content with a nosology based on signs, symptoms and course. Psychiatry and clinical psychology in the early 21st Century need not be so constrained. An array of research findings now illuminates the pathology and etiology of many psychiatric illnesses. The crucial missing ingredient for a truly medical nosology for emotional disorders is a functional understanding of the emotions and their regulation that is comparable to the functional understanding that physiology provides for the rest of medicine. Brain mechanisms are an essential part of this missing knowledge, but they are no more complete in themselves than the anatomy and mechanisms of the kidneys are for understanding renal pathology. Understanding the adaptive utility of a system is just as important for emotional as for physiological systems. Evolution provides the missing functional perspective for understanding the emotions and their disorders.

Many readers may agree with much of the above argument and yet find themselves asking, "Yes, the problems are large and clear, but how can we craft a DSM-V that avoids them?"

A straightforward approach is to classify emotional disorders in the same way medical disorders are classified, based on the etiological factors that give rise to them. As already noted, this path is problematic because medical symptoms are usually aroused by fairly specific tissue changing pathologies, while emotional symptoms are most often aroused by untoward social situations that are much less susceptible to neat classification. However, clinicians routinely classify disorders based on the life situation, so perhaps this option should be taken seriously, especially since detailed information about context is essential to assess the normality of the response.

Sometimes, as in bipolar disorder or panic disorder, nearly all the variance is in genetic individual differences. In most cases, however, the circumstances giving rise to the emotion also play a major role. Although such situations are diverse, they can be categorized nearly as neatly as the aversive emotions. Here are a few examples of some of the causal situations that clinicians recognize intuitively:

- Unrequited love (inability to give up a hopeless romantic goal)
- Unable to find an intimate partner
- Unable to leave an unsatisfactory intimate relationship
- Unable to find a job
- Unable to leave an unsatisfactory job
- Personality disorder that disrupts adaptation in multiple domains
- Being blackmailed
- A child is in trouble but it is hard to help.
- Health problems prevent functioning in crucial roles
- An affair that threatens major relationships
- Partner may be having an affair
- Partner is ill or disabled

If someone was shivering, we would not look to

the brain center that mediates shivering for an explanation, we would instead look at the temperature, clothing, possible infection, etc. There is variation, both innate and acquired, in how readily different people shiver, but this is only part of the picture. We don't know what proportion of patients in our clinics have disorders of emotion regulation, and what proportion have basically normal mechanisms interacting with untoward circumstances. We need to know. Axis IV calls attention to life events. But because these events are carved out from consideration in reaching an Axis I diagnosis, the DSM remains fundamentally different from diagnosis in the rest of medicine. Imagine if general physicians still used a diagnosis of "cough disorder" and they had a separate axis for the importance of environmental precipitants, instead of carving diagnoses based on different etiologies. DSM-V should incorporate life events and life situations into the main diagnostic categories, where their role as elicitors of emotions will be more clear.

Actually, even stating the problem as such a choice between emphasizing individual differences or environmental effects highlights the most common mistake and why this mistake is so hard to escape. Every emotional disorder arises from an interaction between an individual's emotional responsiveness to certain situations and his or her experiences and appraisals of the personal significance of current and future situations. The first variable is influenced by genes, early experiences, drugs and other direct influences on brain mechanisms. The cognitive appraisal is influenced by personal and cultural experiences as well as individual idiosyncrasies from many sources. The events that arouse emotions arise from complex socio-cultural contexts, but also from the social network that grows around an individual thanks to all the other variables. This is complicated. There is no getting around the complexity without excluding important factors or causal links.

We began with the supposition that continuing consternation about psychiatric nosology suggests either that we are missing something basic or that nice neat categories for emotional disorders do not exist. We have been missing something. We have been trying to categorize emotional disorders without a foundation of understanding of the emotions and their origins and functions. This foundation illuminates many of the problems encountered by nosologies for emotional disorders. Unfortunately, however, it does not offer a radical simplification. Instead, it shows that extreme emotional states arise not from a one source, but from interactions of individual differences with complex life circumstances interpreted by diverse cognitive appraisals and psychological defenses. The categories of cleanly differentiated well-defined emotional disorders that we have been seeking do not exist.

One could conclude from this that the DSM-IV approach to emotional disorders is about the best that can be done. This would be like 19th century physicians being satisfied with the diagnosis of "fever" because they can measure it reliably even though they don't know its causes. Instead, we need to proceed the same way general physicians approach symptoms. They consider all possible causes in a differential diagnosis, then they investigate to find the etiology in this particular case. For emotional disorders, we must investigate the

motivational structures of individuals in the same kind of detail that has been lavished on brain mechanisms. We must also describe the Person and the Situation and their interactions.

To escape from abstractions, consider four cases.

Case 1: This 35 year old woman has moderate depression and anxiety with intense anger and jealousy. Situation: She learned her spouse is having an affair and wants to leave him, but she has no income and would have to give up her friends and her art career. Person and vulnerability factors: Somewhat emotional in general, she has a slight tendency towards negative affect, but no enduring abnormal regulation of emotions in general. Etiology: The affair; her emotions are normal responses to this situation.

Case 2: This 35 year old woman has moderate depression and anxiety with intense anger and jealousy. Situation: She suspects her spouse is having an affair but has no evidence of this. He denies it and tries to reassure her. Person and vulnerability factors: She has always believed men will prefer other women and has been pathologically jealous in most of her relationships. She attributes this to her father leaving her mother when she was 5 years old. Etiology: Personality problem; intense jealousy and other emotions are secondary.

Case 3: This 35 year old woman has moderate depression and anxiety with intense anger and jealousy. Situation: She suspects her spouse is having an affair but has no evidence of this. He denies it and he tries to reassure her. Person and vulnerability factors: She recognizes that her jealousy is unwarranted and she has a good stable relationship with her husband, but she nonetheless experiences jealousy almost daily. Etiology: Abnormally low threshold for jealousy in a person who has good relationships.

Case 4: Symptoms: This 35 year old woman has moderate depression and anxiety with intense anger and jealousy.

Situation: She accuses her spouse of having affairs, but only after she has been up several days, often while drinking. Person and vulnerability factors: Bipolar disorder. Etiology: Bipolar disorder, complicated by alcoholism, relationship problems and extreme jealousy.

These cases illustrate what most clinicians know: the same emotions can arise from fundamentally different causes, therefore it makes no sense to view these emotions as a specific disorder or as several separate emotional disorders. In Case 1 the symptoms arise from a situation, in Case 2 from a personality disorder, in Case 3 from a disorder of emotional regulation, and in Case 4 from bipolar disorder. Every clinician will think of a more realistic and complex Case: a person with bipolar tendencies, chronic relationship difficulties, a low threshold for jealousy and anger, heavy drinking, and a husband who might be having an affair.

These conclusions should have major implications for shaping the DSM-V, but we are not sure exactly how they can best be used to shape a diagnostic system that is both practical and biologically based. An extended discussion by a DSM-V Committee on the topic might very well be able to make progress on the question. It is essential to recognize that an evolutionary

foundation is fully compatible with other biological or medical approaches. The DSM-IV has encouraged much useful work on the problem of why some people have tendencies to excessive anxiety and depression, and the brain mechanisms that mediate those affects. Evolution puts this useful work in perspective by emphasizing that these affects can be normal, their regulation mechanisms were shaped by natural selection, and there are likely good evolutionary reasons why these mechanisms are so vulnerable to failure. It also highlights the need to look for disorders of regulation for all emotions, especially the neglected disorders characterized by deficient negative or excessive positive affect.

Some people think that the utility of negative emotions means that they should not be treated. This is a serious mistake. Here, we have much to learn from general medicine, where the harm caused by responses such as pain and diarrhea is well recognized, and where relief of suffering by blocking defensive responses is a routine goal of medicine, whether or not the regulation mechanism is awry. Pharmaceutical companies have supported campaigns to convince the public and practitioners that depression and anxiety are brain diseases, and this has motivated much useful research and has decreased stigma. An evolutionary approach supports a more medical model in which clinicians recognize symptoms as manifestations of more primary causes, treat the causes when possible, and use medications to block the symptoms whenever that is appropriate. An evolutionary approach supports a more complete biological foundation for psychiatric diagnosis that distinguishes symptoms from the diseases and situations that cause them. One of those causes is an abnormality in the brain mechanisms that regulate protective responses, but evolutionarily sophisticated clinicians do not assume this conclusion, they arrive at it only in conjunction with considering all other possibilities and the multi-causal pathways that account for most diseases.

Can an evolutionary approach help psychiatric nosology towards a stronger foundation in biology and stronger links to medicine? There are major challenges, but the principles of basic behavioral biology offer our best current hope for making the treatment of emotional disorders more like the treatment of other medical symptoms. They also will prove useful for fine-tuning diagnostic categories for cognitive, behavioral and personality disorders, but emotional disorders is a good place to begin.

References

- American Psychiatric Association (1980). *Diagnostic and statistical manual of mental disorders*. American Psychiatric Association, Washington, D.C.
- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders: DSM-IV*. American Psychiatric Association, Washington, D.C.
- Axelrod R and WD Hamilton (1981). The evolution of cooperation. *Science* 211, 1390-1396.
- Barlow DH (1991). Disorders of emotions: Clarification, elaboration, and future directions. *Psychological Inquiry* 2, 1, 97-105.
- Beutler LE and ML Malik (2002). *Rethinking the DSM: a psy-*

- chological perspective. American Psychological Association, Washington, DC.
- Brown GW and T Harris (1978). *Social Origins of Depression*. The Free Press, New York.
- Buss DM (2000). *The dangerous passion: why jealousy is as necessary as love and sex*. Free Press, New York.
- Clark LA (1999). Special section on the concept of disorder. *Journal of Abnormal Psychology* 108, 371-472.
- Ekman P and RJ Davidson (1994). *The nature of emotion: fundamental questions*. Oxford University Press, New York.
- Ellsworth P (1991). Some implications for cognitive appraisal theories of emotion. *International Review of Studies of Emotion*, 143-161. K. T. Strongman. Wiley, Chichester.
- Faust D and R A Miner (1986). The empiricist and his new clothes: DSM-III in perspective. *American Journal of Psychiatry* 143, 8, 962-7.
- Feighner JP, E Robins et al. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry* 26, 1, 57-63.
- Fessler DM (in press). The Strategy of Affect: Emotions in Human Cooperation. *Genetic and Cultural Evolution of Cooperation*: Dahlem Workshop Report, 29. MIT Press, Cambridge, MA.
- Fessler DM and K Haley (2003). The Strategy of Affect: Emotions in Human Cooperation. *Genetic and Cultural Evolution of Cooperation*: Dahlem Workshop Report, 29. P. Hammerstein. MIT Press, Cambridge, MA.
- Frances AJF and HL Egger (2003). Whiter psychiatric diagnosis. *Australian and New Zealand Journal of Psychiatry* 33, 161-165.
- Gilbert P (1989). *Human Nature and Suffering*. Lawrence Erlbaum, Hove, UK.
- Goodwin DW, SB Guze et al. (1979). *Psychiatric diagnosis*. Oxford University Press, New York.
- Guze SB (1992). *Why psychiatry is a branch of medicine*. Oxford University Press, New York.
- Horwitz AV (2002). *Creating mental illness*. University of Chicago Press, Chicago.
- Houts AC (2002). Discovery, Invention, and the Expansion of the Modern Diagnostic and Statistical Manuals of Mental Disorders. In L Beutler and M Malik (Eds) *Rethinking the DSM: A Psychological Perspective*. American Psychological Association, Washington, D.C.
- Izard CE (1992). Basic emotions, relations among emotions, and emotion-cognition relations. *Psychological Review* 99, 3, 561-565.
- Jackson ED (unpublished). Organizing Madness: Psychiatric Nosology in Historical Perspective.
- Kendell RE (1975). The concept of disease and its implications for psychiatry. *British Journal of Psychiatry* 127, 305-315.
- Kessler RC and TB Ustun (2004). The World Mental Health (WMH) Survey Initiative Version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI). *International Journal of Methods in Psychiatric Research* 13, 2, 93-121.
- Ketelaar T and GL Clore (1997). Emotion and reason: distinguishing proximate effects and ultimate functions. In G Matthews (ed) *Personality, Emotion, and Cognitive Science*, pp. 355-396. Elsevier Science Publishers, Amsterdam.
- Lewis M and JM Haviland-Jones (2000). *Handbook of emotions*. Guilford Press, New York.
- Marks IM and RM Nesse (1994). Fear and fitness: An evolutionary analysis of anxiety disorders. *Ethology and Sociobiology* 15, 5-6, 247-261.
- McGuire MT and A Troisi (1998). *Darwinian Psychiatry*. Harvard University Press, Cambridge, MA.
- McGuire MT, A Troisi et al. (1997). Depression in evolutionary context. In S Baron-Cohen (Ed) *The Maladapted Mind*, pp. 255-282. Psychology Press, Erlbaum, East Sussex.
- Monroe SM, K Harkness et al. (2001). Life stress and the symptoms of major depression. *Journal of Nervous and Mental Disorders* 189, 3, 168-75.
- Nesse RM (1987) An evolutionary perspective on panic disorder and agoraphobia. *Ethology and Sociobiology* 8, 73S-83S.
- Nesse RM (1990). Evolutionary explanations of emotions. *Human Nature* 1, 3, 261-289.
- Nesse RM (1998). Emotional Disorders in Evolutionary Perspective. *British Journal of Medical Psychology* 71, 4, 397-416.
- Nesse RM (2000). Is depression an adaptation? *Archives of General Psychiatry* 57, 14-20.
- Nesse RM (2004). Natural selection and the elusiveness of happiness. *Philosophical transactions of the Royal Society of London. Series B, Biological sciences* 359, 1449, 1333-47.
- Nesse RM (2005). Natural Selection and the Regulation of Defenses: A Signal Detection Analysis of the Smoke Detector Principle. *Evolution and Human Behavior* 26, 88-105.
- Nettle D (2004). Evolutionary origins of depression: a review and reformulation. *Journal of Affective Disorders* 81, 2, 91-102.
- Oatley K and PN Johnson-Laird (1995). The communicative theory of emotions: Empirical tests, mental models, and implications for social action. In LL Martin and A Tesser (eds) *Goals and Affect*. Erlbaum, Hillsdale, NJ.
- Phillips KA, MB First et al. (2003). *Advancing DSM: dilemmas in psychiatric diagnosis*. American Psychiatric Association, Washington, D.C.
- Plutchik R (2003). *Emotions and life: perspectives from psychology, biology, and evolution*. American Psychological Association, Washington, DC.
- Price JS and L Sloman (1987). Depression as yielding behavior: An animal model based on Schyelderup-Ebbe's pecking order. *Ethology and Sociobiology* 8, 85s-98s.
- Shorter E (1997). *A history of psychiatry: from the era of the asylum to the age of Prozac*. John Wiley & Sons, New York.
- Spitzer RL, JB Williams, et al. (1992). The Structured Clinical Interview for DSM-III-R (SCID). I: History, rationale, and description. *Archives of General Psychiatry* 49, 8, 624-9.
- Szasz TS (1974). *The myth of mental illness: foundations of a theory of personal conduct*. Harper & Row, New York.
- Tooby J and L Cosmides (1990). The past explains the present: Emotional adaptations and the structure of ancestral environments. *Ethology and Sociobiology* 11, 4/5, 375-424.
- Troisi A and M McGuire (2002). Darwinian psychiatry and the concept of mental disorder. *Neuroendocrinology Letters* 23, Suppl 4, 31-38.
- Wakefield JC (1992). Disorder as harmful dysfunction: a conceptual critique of DSM-III-R's definition of mental disorder. *Psychological Review* 99, 2, 232-47.
- Wakefield JC (1992). Disorder as harmful dysfunction: A conceptual critique of DSM-III-R's definition of mental disorder. *Psychological Review* 99, 232-247.
- Wakefield JC and M First (2003). Clarifying the distinction between disorder and non-disorder: Confronting the overdiagnosis ("false positives") problem in DSM-V. In MBFKA Phillips & HA Pincus (eds) *Advancing DSM: Dilemmas in psychiatric diagnosis*, pp. 23-56. American Psychiatric Press, Washington, DC.
- Watson D (2005). Rethinking the Mood and Anxiety Disorders: A Quantitative Hierarchical Model for DSM-V. *Journal of Abnormal Psychology* 114, 4, 522-36.
- Wilson M (1993). DSM-III and the transformation of American psychiatry: a history. *American Journal of Psychiatry* 150, 3, 399-410.
- Wrosch C, MF Scheier, et al. (2003). Adaptive self-regulation of unattainable goals: Goal disengagement, goal reengagement, and subjective well-being. *Personality and Social Psychology Bulletin* 29, 12, 1494-1508.