

# Darwinian psychiatry and the concept of mental disorder

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## **Abstract**

In this paper, we discuss the concept of mental disorder from the perspective of Darwinian psychiatry. Using this perspective does not resolve all of the quandaries which philosophers of medicine face when trying to provide a general definition of disease. However, it does take an important step toward clarifying why current methods of psychiatric diagnosis are criticizable and how clinicians can improve the identification of true mental disorders. According to Darwinian psychiatry, the validity of the conventional criteria of psychiatric morbidity is dependent on their association with functional impairment. Suffering, statistical deviance, and physical lesion are frequent correlates of mental disorders but, in absence of dysfunctional consequences, none of these criteria is sufficient for considering a psychological or behavioral condition as a psychiatric disorder. 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. These two ideas reflect a concept of mental disorder that is both functional and ecological. A correct application of evolutionary knowledge should not necessarily lead to the conclusion that therapeutic intervention should be limited to conditions that jeopardize biological adaptation. Because one of the basic aims of medicine is to alleviate human suffering, an understanding of the evolutionary foundations of the concept of mental disorder should translate into more effective ways for promoting individual and social well-being, not into the search for natural laws determining what is therapeutically right or wrong.

## Introduction

During the past 2 decades, psychiatric epidemiological studies have contributed a rapidly growing body of empirical knowledge on the prevalence data for mental disorders. Two large community surveys conducted in the United States, the National Institute of Mental Health Epidemiologic Catchment Area Program (ECA) [1] and the National Comorbidity Survey (NCS) [2], showed overall 1-year mental and addictive disorder prevalence rates approaching 30% and lifetime rates approaching 50%. This means that, according to current diagnostic criteria, one out of every two persons will suffer from a mental disorder during his or her lifetime. The findings of these epidemiological surveys are even more striking considering that several disorders such as most personality disorders, adjustment disorders and impulse control disorders were not included in the ECA and NCS studies.

These implausibly high prevalence rates have led to concerns about the validity of the current methods of psychiatric diagnosis and have reinvigorated the debate about the concept and definition of mental disorder. According to many, the most basic problem with current criteria of psychiatric diagnosis is that they fail to distinguish mental disorders from “problems in living”, that is the vast array of problematic but nondisordered human conditions which reflect “the aches and pains of normal life” [3, p. 119]. Because there is no accepted way to define the boundary between mental disorder and psychological health, current methods of psychiatric diagnosis are overinclusive and produce a high number of false positive [4].

In this paper, we discuss the concept of mental disorder from the perspective of Darwinian psychiatry [5, 6]. Using this perspective does not resolve all of the quandaries which philosophers of medicine face when trying to provide a general definition of disease [7, 8]. However, it does take an important step toward clarifying why current methods of psychiatric diagnosis are criticizable and how clinicians can improve the identification of true mental disorders.

We begin with a brief review of the Darwinian model of mental health and normal functioning. This section is necessarily the first step of a line of reasoning aimed at redefining the concept of mental disorder from an evolutionary perspective. To identify what has gone wrong with the individual’s mental and behavioral functioning, one should have a detailed idea of how the individual functions or would function when nothing is going wrong. This section introduces the evolutionary concepts that will be used in the next section of the paper, which consists of a critical analysis of the common criteria of psychiatric morbidity. The limits of suffering, statistical deviance, and organic lesion as criteria for validating psychiatric diagnosis will be discussed. Next we will focus on the concept of functional impairment arguing that (1) it is the best single criterion for distinguishing true mental disorders from pseudo-psychopathologies; (2) the Darwinian concept of functional impairment differs in important ways from other views

of this construct that have been previously advanced in the psychiatric literature. A discussion of the clinical and social issues related to the Darwinian concept of mental disorder is the final section of the paper.

## Mental health

The evolutionary concept of mental health 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. These two ideas reflect a concept of mental health that is both functional and ecological [9].

During the course of evolutionary history, natural selection favored those psychological and behavioral traits that served a specific function more efficiently than available alternative traits did. Therefore, an evolutionary account of the human mind and behavior is an account of how psychological and behavioral traits function as adaptations and how they vary across persons. In an evolutionary context, individuals can be viewed as a mosaic of evolved traits employing a variety of strategies to achieve biological goals. The term “strategy” refers to a cluster of coevolved anatomical, physiological, psychological, and behavioral traits designed by natural selection to enhance inclusive fitness. When we say that a trait is functional or adaptive, we mean that it enhances the inclusive fitness of the individual. Human beings, like all other organisms, have been designed by natural selection to strive for the achievement of specific short-term goals or experiences, such as acquiring resources, making friends, developing social support networks, having high status, attracting a mate, and establishing intimate relationships. In the ancestral environment, the achievement of these short-term goals correlated consistently with a gene-transmitting advantage, the ultimate goal of any evolved strategy. In many respects, human beings no longer live in the environment for which they were adapted. Because the modern world is so different from the ancestral environment, the ancestral-fitness consequences of evolved strategies may no longer be realized. Nevertheless, the capacity to achieve short-term biological goals remains a valid measure of mental health because it is an indication that the individual possesses those optimal functional capacities that, in the ancestral environment, promoted biological adaptation.

The study of interactions between individuals and their environments (the ecological perspective) is essential to evaluate the efficiency of functional capacities. Optimal functional capacities are sets of coevolved traits that are (or were) best suited to increasing inclusive fitness in specific environments. No trait is adaptive in all environments. The same trait can be highly adaptive in one environment and minimally adaptive in another.

The preceding discussion underscores two important points. (1) The evolutionary concept of mental health is consequence-oriented: what makes a condi-

tion pathological are its consequences, not its causes or correlates. And (2), the degree of efficiency of functional capacities is dependent on features of the environment. Adverse environments can compromise the efficiency of optimal capacities, just as favorable environments can offset or mitigate the inefficiency of suboptimal capacities. In conclusion, the Darwinian view of mental health places functional capacities and biological adaptation at the core of attempts to define mental disorder. As a result, issues of psychological suffering, statistical deviance, and organic lesion are de-emphasized and the issue of functional impairment moves to the center stage.

### Criteria for defining morbidity

#### *Suffering*

People seek medical advice because they feel ill. The apparent triviality of this statement hides an idea that is deeply rooted in common sense: nociceptive experiences, ranging from discomfort and malaise to severe pain, are a crucial feature of disease [10]. In many European languages, the etymological roots of the word "disease" are terms referring to suffering: *maladie* in French and *malattia* in Italian derive from the Latin *male habitum*, "in a bad state"; *pathos* in Greek means suffering, and *boljezn'* in Russian is derived from *bol'* (i.e., pain); and *disease* in English originally meant disease [11]. The importance of psychological suffering as a criterion for diagnosing the presence of a psychiatric disorder is also emphasized in the definition of mental disorder adopted by the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) of the American Psychiatric Association. According to the DSM-IV, a mental disorder is "a clinically significant behavioral or psychological syndrome or pattern that occurs in an individual and that is associated with present distress (e.g., a painful symptom) or disability (i.e., impairment in one or more important areas of functioning) or with a significantly increased risk of suffering death, pain, disability, or an important loss of freedom." [12, p. xxi].

Even though most psychiatric disorders involve unpleasant or painful symptoms at some stage of their course, there are obvious problems with the idea that psychological suffering is an essential criterion for diagnosing mental disorder. First, there are psychiatric disorders in which patients do not complain of psychological suffering: the negative syndrome of schizophrenia, schizoid personality disorder and some forms of anti-social personality disorder are examples. Second, people often experience painful emotions (e.g., anxiety and depression) even though they have no psychiatric diagnosis. Loss of a loved one, poverty, and social ostracism may induce a psychological suffering comparable to that of some psychiatric disorders. Third, some neuropsychiatric disorders produce pleasurable feelings instead of mental distress: ecstatic states in some forms of epilepsy, grandiose delusions in some psychoses, hypomania, and intoxication induced by alcohol and drugs are examples. In other words, the association

between psychological suffering and mental disorder is complex and includes several possible combinations (mental disorder with suffering, mental disorder without suffering, suffering without mental disorder, mental disorder with pleasure). Such a complexity not only questions the validity of suffering as an essential criterion of psychiatric morbidity but also requires an explanation. An evolutionary analysis of the design features of both the nociceptive warning system and the brain reward system may help clarify the complex relationships between mental disorders and mental pain and pleasure.

When examined from an evolutionary perspective, the fact that mental disorder is often associated with psychological suffering can be easily reconciled with the fact that many forms of psychological suffering do not qualify as mental disorders. Suffering is an evolved response to maladaptation. Under minimally adaptive circumstances, individuals experience mental pain that functions in part as a warning system that one's goal seeking efforts are failing and in part as a social signal to others to elicit their assistance in achieving goals [13]. Because true mental disorders are, by definition, maladaptive patterns of behavior, it is usual for psychiatric disorders to be associated with mental distress.

However, in a person with intact functional capacities, psychological suffering often correlates with the implementation of alternative strategies. Focusing the individual's attention on impending or actual obstacles to the achievement of biological goals, negative emotions may stimulate adaptive reactions. If the alternative strategies are effective, a negative cost-benefit situation may be offset, and the negative mood may dissipate. Thus, emotions function as a source of information about short-term goal achievement. Mood regulates the allocation of effort and resources toward strategies likely to give a high payoff and away from unprofitable enterprises and times when efforts will likely be wasted or dangerous [14]. Negative affect has evolved as an emotional indicator that biological goals have not been or are not being achieved, that is, that one's fitness has been or is being compromised.

Sometimes, the functional significance of negative emotions is evident only if the context of occurrence is evaluated from an evolutionary perspective [15, 16]. For this reason, current methods of psychiatric assessment can occasionally mistake adaptive psychological distress for pathological response. Commenting on their implausibly high prevalence rates, Regier *et al.* [17] have acknowledged such a possibility: "it is reasonable to assume that some syndromes in the community represent transient homeostatic responses to internal or external stimuli that do not represent true psychopathologic disorders... many people with currently defined mental syndromes (in particular among the affective and anxiety disorders)... may be having appropriate homeostatic responses that are neither pathologic nor in need of treatment." (p. 114).

Also the fact that some neuropsychiatric disorders are associated with pleasurable feelings is better understood when examined from the perspective of Darwin-

ian psychiatry. The achievement of adaptive goals often depends on a long chain of events and requires the implementation of complex behavioral strategies. In species characterized by behavioral plasticity and flexibility, natural selection has favored the evolution of brain reward systems as a means to guide behavioral choices and to address the individual toward the achievement of biological goals. The neuroscientist Jaak Panksepp has succinctly expressed this concept with these words: "Pleasure is nature's way of telling the brain that it is experiencing stimuli that are useful." [18].

In humans, the brain reward system and the positive emotions originating from its stimulation play a central role in controlling the correct execution of adaptive behaviors. The capacity to experience mental pleasure helps the individual to pursue goals relevant to biological adaptation [19]. In addition, pleasant emotions associated with adaptive behavior increase the probability that one will engage in similar behavior in the future. In this context, it is clear why mental pleasure and adaptive behavior are so strictly associated. Then, how is it possible that maladaptive conditions such as certain mental disorders are associated with pleasurable feelings?

The conventional explanation derives from the biomedical model of psychiatric disorders and postulates the existence of a structural or physiological dysfunction in the neural mechanisms that modulate mental pleasure. This is certainly the case in some forms of epilepsy and bipolar disorder. However, the association between pleasurable feelings and maladaptive patterns of behavior may also result from the artificial and voluntary manipulation of the brain reward system, as in the case of drug abuse.

Under natural conditions, the brain reward system was activated when (and only when) the individual was pursuing or achieving a goal relevant to biological adaptation. In contrast, drugs of abuse directly interact with specific receptors in the brain that normally help mediate feelings of satisfaction and pleasure associated with the execution of adaptive behaviors. Direct chemical stimulation of these receptors creates a signal in the brain that indicates, falsely, the achievement of biological goals [20]. The individual who uses drugs such as opiates or psychostimulants no longer needs the presence of natural rewards to experience the positive emotions that, under normal conditions, are the psychic reflections of doing the "right thing" biologically. The behavior of some drug addicts resembles that of rats implanted with electrodes located in the septal area and allowed to self-stimulate through lever pressing [21]. These animals stop drinking, eating, and mounting estrous females because they continuously engage in bar pressing to receive electrical stimulation in the brain reward system. When viewed from an evolutionary perspective, drug use and abuse qualify as psychiatric disorders because of their disruptive impact on different aspects of adaptive behavior [22]. In accord with such a view, clinical reports have described the adverse effects of drug abuse on the capacity to search for and

enjoy natural rewards associated with a variety of adaptive behaviors.

### *Statistical deviance*

Sir Henry Cohen's [23] definition of disease as "a quantitative deviation from the normal" (in which by normal he meant the statistical norm) exemplifies the statistical approach to the problem of defining mental disorder. Traditional critiques to such an approach have discussed a number of problems that complicate the use of statistical deviance as a valid criterion of morbidity [24, 25]. There are diseases which are extremely frequent or normal in the statistical sense, yet abnormal for an individual from a clinical viewpoint. There are many behavioral profiles that are statistically deviant and undesirable but are not disorders (e.g., criminality or extreme shyness). And there are some socially valued traits such as excellence in intelligence, artistic talent or physical strength that are statistically deviant but are not disorders. An evolutionary analysis goes beyond these objections and helps to better understand the complexity of the relationship between statistical deviance and mental disorder.

Individual variability is important to evolutionary processes. Natural selection occurs only when differences in some phenotypic characteristic result in consistent differences in rates of survival and reproduction. There are different modes of selection, however, and they produce different types of interindividual variation [26]. Continuous variation in physical and mental traits is the most common situation in nature. If intermediate phenotypes are most fit, selection is stabilizing and the resulting variation is continuous. In continuous variation, the individuals do not fall into sharp classes but are almost imperceptibly graded between wide extremes. Because the phenotypes that deviate in either direction from an optimal value are selected against, the extremes constitute only a small percentage of the total population with the far larger percentage clustering around the middle. Thus, if a trait is subject to stabilizing selection, it is not surprising that health (to the extent that it depends on the optimal functioning of that specific trait) coincides with the statistical norm [9].

However, not all psychological or behavioral traits are distributed on a continuum. Variability between individuals can be discontinuous: if two or more phenotypes have high fitness, but intermediates between them have low fitness, selection is diversifying; that is it acts in favor of two or more modal phenotypes and against those intermediate between them. Diversifying selection produces discontinuous variation which divides the individuals of a population into two or more sharply distinct forms. Not only may this occur at the anatomical and physiological levels, but also at the behavioral level.

Ethological studies have documented the existence of alternative strategies in many animal populations. Alternative strategies are the product of diversifying selection. Even though statistically deviant, these behavioral strategies are employed to compete success-



fully with rivals and reflect the normal operation of adaptive mechanisms. Primate studies offer nice examples of alternative strategies in species phylogenetically close to humans.

In mandrills, there are two morphological and behavioral variants of adult males that differ in terms of secondary sexual adornments and reproductive strategies [27]. "Fatted" males have highly developed sex skin coloration, large testes, high plasma testosterone levels, and fat rumps, whereas "nonfatted" males have paler sex skin, smaller testes, lower plasma testosterone, and slimmer rumps. While "fatted" males mate-guard fertile females, less developed males remain in the periphery of the group and mate sneakily with females. Similar intermale differences have been observed in orangutans [28]. In the presence of many dominant males, adolescent male orangutans undergo a developmental arrest: They become fertile but do not develop fully adult secondary sexual features, such as cheek flanges, laryngeal sac, beard and mustaches, large body size, and a musky odor. Developmental arrest is associated with a distinct hormonal profile [29]. Arrested males lack levels of luteinizing hormone (LH), testosterone, and dihydrotestosterone (DHT) necessary for development of secondary sexual traits. However, they have sufficient testicular steroids, LH, and follicle stimulating hormone (FSH) to fully develop primary sexual function and fertility. Like in mandrills, the two morphological variants of male orangutans use different mating strategies [30]. Developed males are frequently involved in male-male aggression, are attractive to females and typically consort with them, and may sire many offspring over a relatively short period of time. In contrast, being inconspicuous and less attractive to females, arrested males adopt a low-cost, low-benefit reproductive strategy based on sneaky matings and forced copulations.

In the short period, the reproductive success of "nonfatted" male mandrills and arrested male orangutans is lower than that of fully developed males. However, there are advantages associated with the use of the "sneak and rape" mating strategy. While the "combat and consort" strategy imposes costs on dominant males in terms of metabolic energy and exposure to intermale aggression, the suppression of secondary sexual traits allows subordinate males to minimize aggression and injury from dominant, fully mature males, while still being able to sire. Both the strategies are maintained by natural selection because the disadvantages associated with each strategy are balanced by advantages in a different context. In both mandrills and orangutans, subordinate males can rapidly switch over to the "combat and consort" strategy if the density of dominant males decreases. Arrested male orangutans develop into flanged males if a more favorable reproductive situation occurs, and subordinate male mandrills develop secondary sexual traits when they become dominant. Such a flexibility is a further indication that the "sneak and rape" strategy is an adaptive alternative strategy based on a continuous assess-

ment of reproductive opportunity and risk of intermale aggression.

How do these findings relate to the concept of mental disorder? Darwinian psychiatry suggests that some statistically-deviant behavioral profiles currently classified as psychiatric disorders may be in fact evolved alternative strategies. This hypothesis has been tentatively advanced to explain the interpersonal behavior of individuals with antisocial personality disorder [31] and chronic somatization [32]. More compelling evidence has been gathered on different patterns of attachment. According to attachment theory, individual differences in the organization of the attachment system emerge from caregiving interactions with attachment figures and subsequently have numerous influences on personality and social behavior. The traditional perspective views "secure" attachment as the healthy pattern and the insecure patterns as psychological maladaptations [33]. In contrast, Belsky [34] has advanced the argument that the principal evolutionary function of early social experience is to provide children with diagnostic information about the kinds of social and physical environments they are most likely to encounter during their lifetime. This information would permit individuals to facultatively adopt an appropriate reproductive strategy in future environments. Data on the relationships between social experience in childhood and sexual and parental behavior in adulthood support the interpretation of insecure patterns of attachment as alternative strategies.

### *Lesion*

Anyone familiar with the evolution of the concept of disease in medicine would take it for granted the intrinsic "weakness" of the criteria of suffering and statistical deviance and would probably consider as scarcely original the evolutionary critique delineated in the previous sections. In fact, since the 18th century, in medicine the "strong" criterion of morbidity is the presence of organic lesion.

To most of the schools of medicine of the ancient world, symptoms and signs were themselves diseases. In the 17th century, the English physician Thomas Sydenham introduced the view that symptoms must be distinguished from one another, that common clusters of symptoms (i.e. syndromes) must be recognized, and that the natural course of these clusters should be appreciated. Even though the syndromal approach contributed greatly to the advance of medical nosology in the two centuries that followed, linking symptoms together does not provide the physician with any causal explanation of the disease process.

The next advance in the definition of disease owed a great deal to the work of Giovanni Battista Morgagni, an eighteenth-century Italian physician who introduced the method of clinical-pathological correlation which consists in tracking down in the organs and tissues the clinical manifestations of disease. The view that "a disease entity is an altered part of the body" [35] came to dominate medical thinking because its apparent objectivity and strict relationship with causal

explanations of the patient's symptoms. During the last fifty years the development of new techniques of investigation has expanded the concept of lesion to include physiological, biochemical, and molecular abnormalities, without relinquishing the basic assumption that illness necessarily involves a demonstrable physical abnormality of some sort [24].

Until recently, the Virchowian revolution had not invested psychiatry because of the methodological problems in unveiling the neurobiological bases of mental disorders. However, the recent years have witnessed major advances in the study of neuroscience which have increased our understanding of the relationship between cerebral processes and behavioral, cognitive and emotional disorders. The search for neurobiological explanations for psychiatric disorders is in full flow, spurred on by the same reductionist philosophy and technological advances that are proving so dominant in the study of human psychology and behavior. As a consequence, the old idea [36, 37, 38] that the findings emerging from neurobiological research will ultimately solve the problem of distinguishing between sanity and insanity has been recently reformulated in more sophisticated terms by many representatives of mental health disciplines who seem to embrace the Virchowian view that organic lesion is the essential criterion of psychiatric morbidity [39, 40, 41].

Evolutionary reasoning suggests that such a distinction cannot be based on the demonstration of organic pathology because there is no definitive criterion for sorting out altered brain functioning from normal inter- and intraindividual variability, unless we judge a condition in terms of functional consequences. Ascertaining the pathological nature of a somatic change is relatively straightforward as long as one is concerned with a departure from a recognized and standard pattern. The problem is that it is not always apparent where normal variation ends and pathology begins. Thus, the objectivity of the criterion of lesion is only apparent. Defining lesion is as difficult as defining disease, and the risk of circular reasoning is always present. For example, the 15th edition of the *Encyclopedia Britannica* defines lesion as "a structural or biochemical change in an organ or tissue produced by disease processes". In logical terms, this means that the definition of lesion is dependent on the definition of disease, not vice versa. Starting with the search for somatic changes in absence of an agreed upon diagnosis of morbidity can yield confusing results, as shown by the following examples selected from recent neurobiological studies.

Studies using positron emission tomography (PET) have demonstrated that cerebral blood flow in orbital frontal areas is higher in patients with obsessive-compulsive disorder (OCD) than in controls [42]. However, blood flow increase in orbital frontal regions has been reported to occur also in healthy subjects during self-induced dysphoria [43]. Several studies have reported that, in homosexual compared to heterosexual men, three brain structures are different: one hypothalamic nucleus, INAH 3, is smaller, while another, the SNC, is

larger, as is one of the connecting pathways between the cerebral hemispheres, the anterior commissure [44]. Subjects who are in the early romantic phase of a love relationship are not different from OCD patients in terms of density of the platelet 5-HT transporter, which proves to be significantly lower than in the normal controls [45]. Using PET during the continuous performance task, murderers have been found to have significantly lower glucose metabolism in both lateral and medial prefrontal cortex relative to controls [46].

Are these data sufficient to conclude that transient dysphoria, homosexuality, romantic love, and murder are psychiatric disorders? Are the brains of individuals with these conditions "lesioned"? Are these neurobiological findings likely to influence the judgment about disorder attribution to the same extent for each of these conditions? The complexity of these questions and the fact that different clinical psychiatrists are likely to give different answers show that the concept of lesion is all but objective and that its use as criterion of psychiatric morbidity requires the reference to other factual elements.

### **How Darwinian psychiatry conceptualizes functional impairment**

The preceding discussion shows that the validity of the conventional criteria of psychiatric morbidity is dependent on their association with functional impairment. Suffering, statistical deviance, and physical lesion are frequent correlates of mental disorders but, in absence of dysfunctional consequences, none of these criteria is sufficient for considering a psychological or behavioral condition as a psychiatric disorder.

At this point, a brief clarification of how Darwinian psychiatry conceptualizes the criterion of functional impairment is needed. Evolutionary definitions of mental disorder based on the functional approach have been suggested by a number of authors [7, 24, 47, 48]. For example: (1) A disease is composed of phenomena which are not only abnormal by their species norm, but also place the living organisms displaying the disease at a biological disadvantage. (2) A person is healthy if his body functions with at least species-typical efficiency. And (3), disorders are the results of things that have gone wrong with evolved structures that allow for adequate functioning. These definitions have attracted many criticisms, such as the concept of biological disadvantage is too vague and that it is seldom possible to demonstrate that a difference in behavior affects fertility or mortality. Critics emphasize that defining as abnormal anything that does not function according to its design is useful in instances of somatic illness; however, it is less helpful in behavior disorders because the function of many behavior pattern is unknown.

These criticisms, which reflect the fact that the problem of measuring adaptation in the study of human behavior has proven to be difficult, may be addressed in the following ways: (1) The achievement of short-term biological goals is a valid measure of behavioral adaptation; and (2), the assessment of functional capacities is

the best predictor of future goal achievement [5, 9]. The focus on functional capacities and short-term biological goals allows clinicians to make cross-sectional functional assessments of individual patients which would be impossible to make by applying the lifetime criteria of fertility and mortality. In addition, it excludes from the category of mental disorders those conditions that involve the failure to achieve ultimate biological goals even if a person's functional capacities are intact (e.g., infertility due to rational use of birth-control measures).

Being based on the most recent theoretical advances of evolutionary psychology with its emphasis on evolved psychological mechanisms rather than on longevity and fertility, Wakefield's definition of mental disorder as "harmful dysfunction" is extremely similar to the one proposed here. However, there is an important difference. Wakefield [25] argues that "disorder cannot be simply identified with the scientific concept of inability of an internal mechanism to perform a naturally selected function. Only dysfunctions that are socially disvalued are disorders." (p. 384). We disagree because the reference to social values reintroduces the risk of cultural relativism in the definition of mental disorder, a long-standing problem in the history of psychiatry. In our opinion, the important question raised by Wakefield (i.e., that clinicians should be interested only in those functions that people care about and need within the current social environment) can be adequately addressed by distinguishing between diagnosis of mental disorder and need for treatment, as explained in the next section.

### Clinical and social issues

We wish to conclude with a note of clarification about the clinical and social implications of the ideas expressed in this article. A major problem with the application of evolutionary theory to human behavior is the risk of misinterpretation. In the field of ethics and morality, the term naturalistic fallacy has been used to denote the erroneous translation of evolutionary explanations of human behavior into normative or prescriptive terms. The naturalistic fallacy consists in offering some supposedly neutral descriptive statement about what is allegedly natural, as if it could by itself entail some conclusion about what is in some way commendable. The risk of committing the naturalistic fallacy is high when the Darwinian concept of mental disorder is used to decide what conditions should be treated.

A correct application of evolutionary knowledge should not necessarily lead to the conclusion that therapeutic intervention should be limited to mental and behavioral conditions that jeopardize biological adaptation. In contemporary medicine, many therapeutic interventions address problems that are not diseases but that are associated with subjective suffering or undesirable consequences. Cosmetic surgery and anti-aging therapies are just two examples. It is unrealistic to think that psychiatry will remain extraneous to this process that is changing cultural expectations toward

medical therapies. Because one of the basic aims of medicine is to alleviate human suffering, an understanding of the evolutionary foundations of the concept of mental disorder should translate into more effective ways for promoting individual and social well-being, not into the search for natural laws determining what is therapeutically right or wrong.

However, Darwinian psychiatry dictates some ethical rules that the reflecting clinician should follow to minimize the risk of inappropriate interventions. The patient should take the decision to be treated on the basis of detailed information concerning the possible adaptive significance of his/her symptoms and their evolutionary origin. In addition, patients who seek treatment for undesirable psychological or behavioral traits that are not pathological should be warned of not confusing medical intervention with medical diagnosis (similarly to what happens today to persons who seek surgical correction for undesirable physical traits). It is not a contradiction that a clinical psychiatrist embracing the Darwinian view of mental disorder informs the patient that his/her condition is not a true mental disorder and, at the same time, accepts to help the patient with the therapeutic means that are used to treat true psychopathologies. Redefining an undesirable condition as a normal variant is more than an academic exercise. Labeling a psychological or behavioral condition as sick may have serious individual and social consequences, including self-reproach and social stigmatization. Since a major contribution of Darwinian psychiatry is the insight that diversity and individual differences are core evolutionary features of any animal species, including *Homo sapiens*, the clinician should share such an information with the patient, not underestimating its reassuring and emancipating potential.

### REFERENCES

- 1 Regier DA, Myers JK, Kramer M, Robins LN, Blazer DG, Hough RL *et al.* The NIMH Epidemiologic Catchment Area program: historical context, major objectives, and study population characteristics. *Arch Gen Psychiatry* 1984; **41**:934-941.
- 2 Kessler RC, McGonagle KA, Zhao S, Nelson CB, Hughes M, Eshleman S *et al.* Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. *Arch Gen Psychiatry* 1994; **51**:8-19.
- 3 Frances A. Problems in defining clinical significance in epidemiological studies. *Arch Gen Psychiatry* 1998; **55**:119.
- 4 Spitzer RL. Diagnosis and need for treatment are not the same. *Arch Gen Psychiatry* 1998; **55**:120.
- 5 McGuire MT, Troisi A. Darwinian psychiatry. New York: Oxford University Press; 1998.
- 6 McGuire MT, Troisi A. Evolutionary biology and psychiatry. In: BJ Sadock, VA Sadock, editors. *Kaplan & Sadock's comprehensive textbook of psychiatry*, VII ed., vol. 1. Philadelphia: Lippincott Williams & Wilkins; 2000. pp. 484-492.
- 7 Boorse C. On the distinction between disease and illness. *Philosophy and Public Affairs* 1975; **5**:49-68.
- 8 Hesslow G. Do we need a concept of disease? *Theor Med* 1993; **14**:1-14.
- 9 Troisi A, McGuire MT. Evolution and mental health. In HS Friedman, editor. *Encyclopedia of mental health*, vol. 2. San Diego: Academic Press; 1998. pp. 173-181.



- 10 Taylor FK. The medical model of the disease concept. *Br J Psychiatry* 1976; **128**:588–594.
- 11 Wulff HR, Andur Pedersen S, Rosenberg R. *Philosophy of medicine: An introduction*. Oxford: Blackwell; 1986.
- 12 American Psychiatric Association. *Diagnostic and statistical manual of mental disorders, Fourth Edition (DSM-IV)*. Washington, DC: American Psychiatric Association, 1994.
- 13 McGuire MT, Troisi A, Raleigh MM. Depression in evolutionary context. In S Baron-Cohen, editor. *The maladapted mind: Classic readings in evolutionary psychopathology*. Hove, UK: Psychology Press; 1997. pp. 255–282.
- 14 Nesse RM. Is depression an adaptation? *Arch Gen Psychiatry* 2000; **57**:14–20.
- 15 Troisi A, McGuire MT. Evolutionary biology and life events research. *Arch Gen Psychiatry* 1992; **49**:501–502.
- 16 Troisi A. Gender differences in vulnerability to social stress: A Darwinian perspective. *Physiol Behav* 2001; **73**:443–449.
- 17 Regier DA, Kaelber CT, Rae DS, Farmer ME, Knauper B, Kessler RC *et al.* Limitations of diagnostic criteria and assessment instruments for mental disorders: Implications for research and policy. *Arch Gen Psychiatry* 1998; **55**:109–115.
- 18 Panksepp J. *Affective neuroscience. The foundations of human and animal emotions*. New York: Oxford University Press; 1998.
- 19 Nesse RM. Evolutionary explanations of emotions. *Hum Nature* 1990; **1**:261–289.
- 20 Nesse RM, Berridge KC. Psychoactive drug use in evolutionary perspective. *Science* 1997; **278**:63–66.
- 21 Olds J, Milner P. Positive reinforcement produced by electrical stimulation of the septal area and other regions of rat brain. *J Comp Physiol Psychol* 1954; **47**:419–427.
- 22 Troisi A. Harmful effects of substance abuse: a Darwinian perspective. *Funct Neurol* 2001; **16**:237–243.
- 23 Cohen H. The evolution of the concept of disease. In AL Caplan, HT Engelhardt, Jr., JJ McCartney, editors. *Concepts of health and disease: Interdisciplinary perspectives*. Reading, MA: Addison-Wesley; 1981. pp. 209–220.
- 24 Kendell RE. *The role of diagnosis in psychiatry*. Oxford: Blackwell; 1975.
- 25 Wakefield JC. The concept of mental disorder: On the boundary between biological facts and social values. *Am Psychologist* 1992; **47**:373–388.
- 27 Setchell JM, Dixon AF. Arrested development of secondary sexual adornments in subordinate adult male mandrills (*Mandrillus sphinx*). *Am J Phys Anthropol* 2001; **115**:245–252.
- 26 Futuyma DJ. *Evolutionary Biology*, 2nd edn. Sunderland, MA: Sinauer Associates, Inc.; 1986.
- 28 Maggioncalda AN, Czekala NM, Sapolsky RM. Growth hormone and thyroid stimulating hormone concentrations in captive male orangutans: implications for understanding developmental arrest. *Am J Primatol* 2000; **50**:67–76.
- 29 Maggioncalda AN, Sapolsky RM, Czekala NM. Reproductive hormone profiles in captive male orangutans: implications for understanding developmental arrest. *Am J Phys Anthropol* 1999; **109**:19–32.
- 30 Galdikas B. Adult male sociality and reproductive tactics among orangutans at Tanjung Puting. *Folia Primatol* 1985; **45**:9–24.
- 31 Mealey L. The sociobiology of sociopathy: An integrated evolutionary model. *Behav Brain Sci* 1995; **18**:523–599.
- 32 Troisi A, McGuire MT. Deception and somatizing disorders. In CN Stefanis, AD Rabavilas, CR Soldatos, editors. *Psychiatry: A world perspective*, vol. 3. Amsterdam: Excerpta Medica; 1991. pp. 973–978.
- 33 Sroufe LA. The role of infant-caregiver attachment in development. In J Belsky, T Nezworski, editors. *Clinical implications of attachment*. Hillsdale, NJ: Erlbaum; 1988. pp. 18–38.
- 34 Belsky J. Modern evolutionary theory and patterns of attachment. In J Cassidy, PR Shaver. *Handbook of attachment: Theory, research, and clinical applications*. New York: The Guilford Press; 1999. pp. 141–161.
- 35 Virchow R. *Hundert Jahre Allgemeiner Pathologie*. Berlin: August Hirschwald; 1895.
- 36 Schneider K. *Klinische Psychopathologie (3rd edn)*. Stuttgart: Thieme; 1950.
- 37 Szasz TS. *The myth of mental illness. Foundations of a theory of personal conduct (rev. ed.)*. New York: Harper & Row; 1974.
- 38 Ludwig AM. The psychiatrist as physician. *J Am Med Ass* 1975; **234**:603–604.
- 39 Guze SB. *Why psychiatry is a branch of medicine*. New York: Oxford University Press; 1992.
- 40 Andreasen NC. The validation of psychiatric diagnosis: New models and approaches. *Am J Psychiatry* 1995; **152**:161–162.
- 41 Detre T, McDonald MC. Managed care and the future of psychiatry. *Arch Gen Psychiatry* 1997; **54**:201–204.
- 42 Schwartz JM, Stoessel PW, Baxter LR, Martin KM, Phelps ME. Systematic changes in cerebral glucose metabolic rate after successful behavior modification treatment of obsessive-compulsive disorder. *Arch Gen Psychiatry* 1996; **53**:109–113.
- 43 Pardo JV, Pardo Pj, Raichle ME. Neural correlates of self-induced dysphoria. *Am J Psychiatry* 1993; **150**:713–719.
- 44 Harrison PJ, Everall IP, Catalan J. Is homosexual behaviour hard-wired? Sexual orientation and brain structure. *Psychol Med* 1994; **24**:811–816.
- 45 Marazziti D, Akiskal HS, Rossi A, Cassano GB. Alteration of the platelet serotonin transporter in romantic love. *Psychol Med* 1999; **29**:741–745.
- 46 Raine A, Buchsbaum MS, Stanley J, Lottenberg S, Abel L, Stoddard J. Selective reductions in prefrontal glucose metabolism in murderers. *Biol Psychiatry* 1994; **36**:365–373.
- 47 Scadding JG. Diagnosis: the clinician and the computer. *Lancet* 1967; **2**:877–882.
- 48 Klein DF. A proposed definition of mental illness. In RL Spitzer, DF Klein, editors. *Critical issues in psychiatric diagnosis*. New York: Raven Press; 1978. pp. 41–71.