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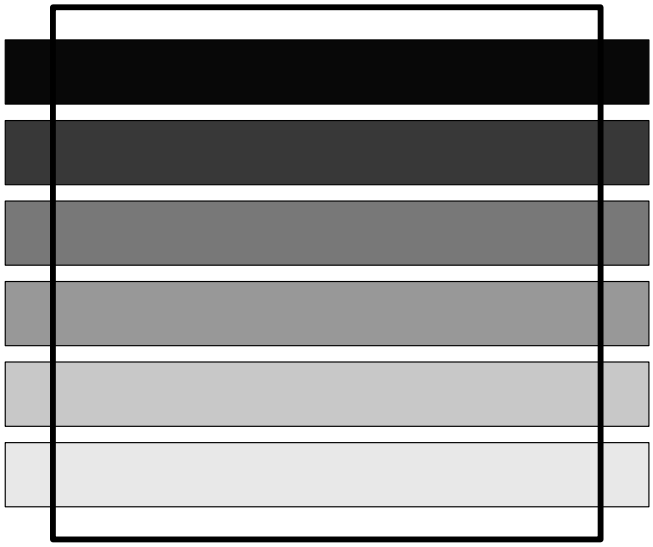

EVOLUTIONARY PSYCHIATRY AND NOSOLOGY

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EVOLUTIONARY PSYCHIATRY AND NOSOLOGY

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1) Introduction

In the recent years, we have witnessed what Williams and Nesse (1991; 1996) have called the "dawn of the Darwinian medicine". It is argued that the Darwinian approach to medicine would not only transform the way we conceptualize, investigate and classify diseases, but also how we treat them. Here is an example, offered by Cosmides and Tooby (1999), of the possibilities of the evolutionary approach. In the past iron supplements were given to those diagnosed with anemia. However, evidence indicates that many infectious bacteria are rate-limited by their access to bioavailable iron. From an evolutionary perspective a decrease in available iron, as produced by anemia, should rather be understood as an evolved defense against infection rather than a dysfunction. Treating this evolved defense would just reinstate the problem that it was designed to help solve. This case shows how an evolutionary approach to pathology introduces a distinction previously neglected between evolved defense and disorder resulting from a dysfunction exemplifies one of the ways to re-conceptualized the domain of the conditions treated by medicine (see Nesse and Williams 1999).

For a number of reasons internal and external to psychiatry[1], the use of evolutionary theories in abnormal psychology has been at first slow, but now is catching up as more and more

1. Randolph Nesse (1991) pointed to a few factors that might have slowed down the integration of the evolutionary view to psychiatry. He writes: "Why has psychiatry been so slow to incorporate the advances of evolutionary psychobiology? Conceptual and historical issues both appear to be responsible. The first conceptual difficulty is that evolution offers insights mainly about adaptation, while psychiatry's concern with pathology seems, at first glance, to be quite different. Second, by trying to understand the functions of traits, evolutionary psychobiologists understake an enterprise that is viewed with suspicion by those unaware of advances in basic biology (Mayr 1988) and especially by those who believe psychiatry should disassociate itself from all by the "hardest" sciences. Third, the research methods of evolutionary psychobiologists lack specificity and elegance, and firm findings are few.
Historical and political factors also offer important explanations. Sociobiology has arisen at the very moment when its insights are least welcome in psychiatry. Just as psychiatrists are returning to their medical identities and searching for the physical causes of diseases, sociobiology suggests that many psychiatric disorders my not be diseases at all. Just as psychiatry has gained the capacity to relate various aspects of brain function to mental disorders, sociobiology suggests

psychiatrists integrate the evolutionary framework into their studies (Baron-Cohen 1995; McGuire and Troisi 1998; Nesse 2000; Stevens and Price 1996; Wenegrat 1990). In what follows, I wish to look at the implications of these recent works with respect to the way we conceive and classify disorders.

My game plan in this paper will be the following. First, I will review some of the problems commonly attributed to current nosologies, including the DSM. Second, because I think that an evolutionary approach can contribute to transforming the way we look at mental illnesses, I will provide the reader with a brief sketch of the basic tenets of evolutionary psychology. The picture of the architecture of the human mind that emerges from evolutionary psychology will serve as a background for defining mental illness. Third the definition put forward by Jerome Wakefield is discussed. Despite my sympathy for his project, I must indicate one reason why I think his attempt might not be enough to resolve the problems of current nosologies. I will suggest that it is better to place Wakefield's account of mental illness in the larger framework of "treatable conditions" (Cosmides and Tooby, 1999). Finally, I will give two examples of the way evolutionary thinking might change our way of thinking about some disorders. In conclusion, I will evaluate where evolutionary thinking leaves us in regard to what we identify as the main problems of our current nosologies.

2) Some problems with current nosologies

Let's start by identifying some of the problems with current nosologies that the evolutionary approach might be able to solve (I don't pretend to be exhaustive here). Presented are the four major problems currently discussed in the literature:

(1) First is a lack of a clear and widely accepted definition of "mental disorder". As Widiger and Sankis noted recently in their review of the issues and problems affecting adult psychopathology: "An ongoing concern that is fundamental to the science of psychopathology is the absence of an established definition of the construct of mental disorder." (2000, p. 377).

(2) A related problem is the one regarding the objectivity of mental illness. Thomas Szasz is notorious for having proposed that the notion of "mental illness" is, as he put it, a "myth" (1960). What he had in mind was the following: "If mental illnesses are diseases of the central

that brain abnormalities may be unrelated to the etiology of many psychiatric disorders. [...]" (p. 38-39). We might want to add to these factors the fact that psychiatry just recently got out of the grip of psychoanalysis and is very reluctant to replace it by any other "theory" (see note 2).

nervous system (for example, paresis), then they are diseases of the brain, not the mind; and if they are the names of (mis) behaviors (for example, using illegal drugs), then they are not diseases" (1994, p. 35). According to him, the concept of mental illness is a metaphor that hides, under its allure of objectivity, a normative judgment about some kind of behavior: "The norm from which deviation is measured whenever one speaks of a mental illness is a psychosocial and ethical one." (1960, p. 114). It is hard not to side with Szasz when considering the history of psychiatry. Many mental "diseases" like "drapetomania", "hysteria", and "homosexuality", were obviously social constructs hiding a social agenda. But is Szasz's diagnostic about mental illness correct? Should we really abandon the notion of mental illness? Is it not possible to introduce some form of objectivity into the concept? This question, needless to say, is a pressing one since it concerns the foundation and legitimacy of the psychiatric enterprise.

(3) A third problem is the lack of an explicit (and scientific) image of what constitutes the normal functioning of the mind or what constitutes normality in our current nosologies. Such an image is crucial for the establishment of diagnostics. For instance, Widiger and Sankis note that "... the DSM-IV criteria set for major depressive disorder (APA 1994) excludes uncomplicated bereavement, presumably because depressive reactions to the loss of a loved one are normal (non pathological). However, DSM-IV makes no exclusion for comparably uncomplicated reactions of sadness to other major stressors, such as a terminal illness, divorce, or loss of job" (2000, p. 378).[2] This problem relates to the problem of the alleged lack of objectivity of our nosologies. A scientific image of the normal functioning of the mind would not only guide the psychiatrists in their treatment of patients, but would also provide them with an objective standard to judge what is normal and what is deviant.

(4) A fourth problem is the one identified by Poland, von Eckardt and Spaulding who maintained that DSM "constitutes a faulty conceptualization of the domain of psychopathology and [that it] interferes with optimal pursuit of clinical and scientific purposes" (1995, p. 236; a similar point is made by Coltheart and Langdon 1998). According to Poland *et al.*, an assumption behind the DSM's categorization is that "it is possible to individuate psychopathological conditions on the basis of directly observable clinical manifestations [...] the operationally defined categories within the DSM system are supposed to be *natural kinds* with a characteristic causal

2. Nesse makes a similar remark, saying that: "The psychiatrist does not know the normal functions of the systems disrupted by mental disorders, except in the most general terms. For example, when a patient presents with depression, the psychiatrist does not know the normal functions of the capacity for mood and therefore has difficulty in distinguishing between normal and pathological sadness. When a patient presents with extreme jealousy, few psychiatrists understand its evolutionary origins and functions" (1991, p. 24).

structure [...]" (idem, p. 240-1).[3] This can be compared to determining the problems with a TV set using only observable manifestations (Murphy and Stich, in press). Many things can be responsible for the fact that nothing comes on the screen: the bulb might be burned-out, the TV might be disconnected, etc. As in the case of the TV, there is no reason to believe that it is possible to identify the *natural kinds* of psychopathology only by taking into account the symptoms directly observable at the phenomenological level. In other words, the problem with the DSM approach is that it "... provides no representation of the underlying biological, psychological, or environmental processes that constitute the pathology of a given mental disorder... [as a consequence it] will very likely continue to classify within the same category individuals who exhibit superficial similarities but differ significantly on underlying process." (Idem, p. 250)

Poland and colleagues (1994) suggest that an alternative nosology should be based on more intimate relationships with basic sciences like cognitive sciences, neurosciences, molecular biology, etc. I want to argue that evolutionary psychology could join these sciences and contribute positively to the elaboration of a new and more accurate nosology. I'm not claiming that evolutionary psychology will do the job by itself. Other disciplines will no doubt play an important role in the elaboration of a more accurate nosology. This said, I suggest that one of the major contributions of the evolutionary framework to this enterprise is the introduction of new ways of understanding conditions that go beyond the simple disorder/non-disorder dichotomy.

In the following, I will try to evaluate how a form of psychiatry, informed by evolutionary theories, could fair with our four major problems.

3. One reason for this state of affairs is that the conceptors of the DSM have tried to produce a "theory-free" nosology. Spitzer, who worked on revising the DSM, explained the reason why psychiatric diagnosis has culminated in categories based on observation and induction, rather than theory. According to him, it is "[b]ecause no particular orientation or limited subgroup of schools has established its credentials as the sole *scientific* approach, [and, for that reason,] there remains no *scientific* criterion for officially adopting one orientation over the others. Thus the field of psychiatry must somehow accommodate all the divergent schools and yet arrive at a single classified scheme that all agree to use. How then to reach agreement amid such unyielding disagreement?
The authors of DMS-III sought to achieve this agreement by separating psychiatric observation from psychiatric theory. The common classification scheme would consist of categories whose meanings could be defined as far as possible through direct observation. In this way the adherents of different schools could nonetheless agree on basic terminology because disputes regarding definitions could be settled by appeal to what all could observe and could no reasonably deny. ... Agreement over terminology requires, then, that the definitions of the terms remain operational and atheoretical." (1995, p. 92)

3) Evolutionary Psychology

Before going farther, let me briefly say what is evolutionary psychology. The content of evolutionary psychology can be captured by the following three theses:

Thesis 1: *Massive modularity of the mind*. The first thesis concerns the architecture of the mind. According to evolutionary psychologists, the cognitive architecture of the mind is composed to a large extent of what Chomsky has called "mental organs" or "modules". Following Jerry Fodor: "One can conceptualize a module as a special-purpose computer with a proprietary database" (1990, pp. 200-201). Baron-Cohen (1997) proposed that the modules have the following properties. They are:

(1) Domain specific;

(2) Mandatory;

(3) Rapid;

(4) Have characteristic ontogenesis;

(5) Dedicated neural architecture;

(6) Have characteristic pattern of breakdown.

Contrary to Fodor who postulates the existence of a few modules (six or seven input systems and as many output systems with no central modularity), the advocates of evolutionary psychology are positing what has been called a "massive modularity hypothesis", according to which the mind is made of hundreds and thousands of modules.

Thesis 2: *Adaptationism*. The process of natural selection has shaped the cognitive architecture. As Tooby and Cosmides put it, mental modules have been "invented by natural selection during the species' evolutionary history to produce adaptative ends in the species' natural environment" (1995, xiii). This does not mean that *all* aspects of our actual cognitive architecture can be explained by the fact that they have been selected for the accomplishment of a biological function (some traits can be by-products or vestiges). The main thesis is that the explanation of the presence of complex and well-adapted mechanisms in an organism must invoke natural selection as a major factor.

Thesis 3: *Evolutionary Adaptative Environment*. Modules have been shaped to answer particular problems of our Evolutionary Adaptative Environment (EAE). These problems are regrouped in specific domains (reproduction, predatory behavior, social interaction, etc.) each having specific properties (a good mate does not necessary have the same properties as a good meal or a good friend). Since many of the properties that organisms need to access are not "visible" to them, the

modules exploit cues that co-vary (often enough) with these properties in order to produce adaptative behavior in the environment in which they evolved.

The picture of the mind offered by evolutionary psychology provides the background against which the notion of mental disorder can be understood.

4) A Concept of Mental Disorder

Jerome Wakefield (1992a 1992b, 1999, 2000) has proposed a definition of the concept of "mental disorder" that he hopes will provide psychiatry with an *objective* criterion for declaring something a pathology. He thinks that we can analyze the intuitive concept of "*mental disorder*" underlying the field of abnormal psychology by saying that it is a "*dysfunction*" of a psychological mechanism that is judged "*harmful*". This definition is a hybrid account of disorder for it has both a purely scientific and factual component (the notion of *dysfunction*) and a value component (the notion of *harm*). According to Wakefield, both of these components are jointly necessary to capture our intuitive concept of mental illness (1992a, p. 374). Wakefield has not much to say about the "value" component of his definition.[4] He is far more interested in the notion of dysfunction that he expects will provide psychiatry the objective foundations it needs.

Although the notions of "function" and "dysfunction" or "malfunction" have been used in medicine and psychiatry for a long time only the evolutionary theory can analyze these in causal and scientific terms. Wakefield proposes to understand the previous usage of function as cases of what he calls "blackbox essentialism". This theory is a spin-off of Putnam's theory of reference that asserts that we use concepts on the basis of prototypes before the underlying essence of what we refer to is scientifically discovered (e.g. the concept of "water" existed long before we finally discovered its underlying essence). Wakefield's idea is that the notion of function (and malfunction) used by Aristotle, Harvey and others has been based on certain prototypical instances of "non-accidentally beneficial effects like sight [in the case of the eyes] and on the idea that some common underlying process must be responsible for such remarkable phenomena" (2000, p. 39). But the process responsible for the phenomena was not known until the advent of the Darwinian theory.

4. That surely does not mean that it is without problems. For instance, it is not clear for whom the dysfunction has to be harmful to be judged as a fullfledged "harmful dysfunction". Does it have to be harmful for an individual, his genes, his family, the society in general?

According to the evolutionary theory, the presence of certain traits (including psychological mechanisms responsible for behaviors) is explained by the fact that these traits (or mechanisms) performed certain functions in the ancestors of the organisms, the effects of which had been beneficial enough for ancestors of the organisms to lead to their conservation through the process of natural selection. The function for which a trait (or a mechanism) had been selected is what has been called in the philosophical literature the "normal function"[5] or "proper function" of that trait (or mechanism). In other words, the normal or proper function of mechanism X is to do what it has been designed to do by natural selection. It follows that there is a dysfunction or a malfunction when a trait (or a mechanism) is not able to accomplish its normal function. It must be noted that the notion of "normal function" is independent of the current adaptivity of the trait (or the mechanism). Thus, the fact that a trait (or mechanism) is maladaptative in a current environment is not a sign of a dysfunction. For instance, according to Wakefield (1999), the fact that we are not capable of breathing under water is not an indication of a malfunction of the lungs, but of the fact that they can't perform on their functions in certain environments for which they have not been designed. It should also be noted that the notion of function is independent of our values. For instance, imagine that rape or infanticide has been found to have been selected and that they were adaptative in certain cases in the past history of our species. If such was the case, we would have to judge the mechanisms responsible for these behaviors as being in perfectly good working order, even if we abhor and disvalue the behaviors they produce. The objectivity of the concept of function would protect us against the abusive use of mental illness denounced by Szasz.

5) The usefulness of a conceptual analysis

Many criticisms have been voiced against Wakefield's definition of mental disorder (see for instance the commentaries on Wakefiled's theory in *Abnormal Psychology* 1999). One concerns the fact that there is no *a priori* reason to suppose that our folk concept of "mental disorder" is worth

5. Neander (1995) notes rightly that this notion of normativity is neither evaluative or statistical. As she writes: "Teenage fertility is biologically normal, but it does not follow that teenage fertility is a good thing; on the contrary, if we could induce (temporary and reversible) infertility in all girls under the age of twenty, that would probably be better [Boorse, 1975]. Judging that something is functioning properly is not the same as judging that its functioning is good. Nor is the judgement that something is functioning properly just a statistical abstraction, as epidemic and pandemic diseases testify. If we were all struck blind it would still be the function of our eyes to see. Sight, not blindness, would remain biologically normal proper functioning, and blindness, not sight, would remain dysfunctional. Not suprisingly, we can't cure diseases just by spreading them around" (p. 111).

keeping. It is generally agreed that it is unlikely to be referring to a "natural kind". Stich (personal communication) as gone so far as to propose the elimination of the concept because of the conflicting intuitions concerning to what it applies. According to him, it is not clear that people are not willing to call "disorder" those mechanisms that no longer produce adaptative behaviors or mechanisms that are in perfect working order but that give rise to behaviors that are not socially acceptable.[6]

I agree with this statement by Stich concerning the state of our intuitions about mental illness, but I disagree with his proposed remedy. I think we need to get a clearer picture of what can go wrong with the mind and what can cause the distress that leads people to seek treatment or help from psychiatrists. In other words, I think that we should not focus our attention exclusively on the notion of mental disorder as proposed by Wakefield. For that reason, I would rather propose, following Cosmides and Tooby (1999), considering the notion of disorder within a larger framework of *treatable conditions*, i.e. the conditions that are judged harmful enough by people to seek a treatment for them. This larger picture should help us to make up our mind about what we want to call "mental illness" and might lead to a revision of the concept of "mental illness" (the extent of that revision is an empirical matter). In the next two sections I will review some forms of treatable conditions (for more exhaustive reviews see Cosmides and Tooby 1999; Nesse 1999).

6) Treatable conditions that are dysfunctions

The first group of treatable conditions is the one that results from dysfunctions of cognitive mechanisms.

(1) **Simple breakdown**: The simplest kind of dysfunction is the mere breakdown of a module. One example is given by Frith (1992). In his model, certain symptoms of schizophrenia (the control delusions and the "voices") are a result of the failure of a monitor mechanism in charge of distinguishing our actions from that of others. The breakdown of this mechanism leaves the patient without knowledge that he/she is the source of a movement or a thought leading him/her to think that someone else is controlling him/her or that he/she is hearing voices.

6. It is possible that those intuitions come from the fact that we consider the environment as part of the organism (a sort of "extended phenotype" view) or that we use another notion of function than the one suggested by Wakefield. It

Another case of breakdown, involving the severing of a link between modules, ca be used to explain the Capgras' delusion (Young 1994, 1996). Persons suffering from Capgras' syndrome have no problem recognizing the people with whom they are familiar with respect of their physical attributes, however there is a feeling that something is wrong (the familiar people look somewhat changed). The inflicted may confabulate indicating that the people are not really their friends, family members or acquaintances, but exact replicas (Young 1994, 1996). This condition, according to current theories, depends on the fact that there are two pathways to visual system one affective (feeling of familiarity) and the other cognitive (a template-matching system). The cognitive system is working properly (so there can be recognition) while there is a breakdown of the affective system (so recognition is not accompanied by the normal feeling of familiarity).

(2) **Over or under-responsiveness**: Another kind of dysfunction is when the module is working but is not computing according to the criterion that constitutes its evolved function. For instance, it is possible that some cases of chronic anxiety might result from an *over-active* response to danger.

It is also possible to think of cases of *under-responsiveness*, for instance hypophobia. If the functional theory of emotion is true such that emotions are adaptative responses to stimuli in the environment, the lack of response to fearful objects should be as detrimental for the organism as, for example, is the lack of pain. It might be that cases such as theses unnoticed because they are not distressing.

(3) **Pleiotropy**: There are situations where it is hard to understand how a dysfunction that seems so detrimental to individuals can still be so widespread. How can it resists the selection pressures and stay in the gene pool? One explanation makes references to the fact that a gene or set of genes can control more than one phenotype, especially if one of the non-disorder phenotypes is highly adaptative: for instance, the same genes that are causing sickle-cell anemia in certain individuals are also protecting others from malaria. McGuire, Troisi and Raleigh (1997) consider pleiotropy as the explanation for manic depressive illness, which, has some periods of dysfunctionality, but "is often associated with superior intelligence and/or creative capacities" (p. 265; see also Nesse 1999, p. 264).

(4) **The extreme variant phenomena**: The distribution of a single trait is able to go from functional to dysfunctional, so that the very same genes that lead an individual to be dysfunctional in one domain might lead another to superior functioning in a different domain. Baron-Cohen recently uses this explanation in his account of some symptoms of autism.

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is possible that we are using "function" to talk about the "current adaptivity" of behaviors, that we are using the

Autistics are known to have an impaired mind-reading capacity but also pay exaggerate attention to details, to have strong obsessions and islets of ability. Apparently, this impaired folk psychology goes together with superior folk physics capacities (e.g. they are better at embedded figure tests—tests that consist in finding a hidden figure in a more complex one). Family studies show that students in math/physics/engineering are more likely to have a relative with autism than students in social sciences, and epidemiological studies indicate that the ratio of high-functioning autistic is biased towards males in a proportion of 9:1. In view of these studies, Baron-Cohen has made the hypothesis that autism might be a form of "extreme male brain". According to him: "... if the male brain involves this combination of impaired folk psychology and superior folk physics in a mild degree, in autism spectrum disorders this combination occurs to a more marked extent" (2000, p. 1254).[7]

(5) **Modules fail to develop**: The first task of an organism is to assemble itself. For that reason, some postulate the existence of what Cosmides and Tooby call "developmental adaptations" (1999) or Segal called "diachronic modules" (1996). In order to function they must first construct an "implemental adaptation" or "synchronic module" that characterizes the adult cognitive make-up and then calibrate it. Dysfunction can result from the absence of development of a synchronic module *per se*, as in the case of autism where it is postulated that the absence of a Theory of Mind Module (ToMM) is caused by severe deficits in *joint attention* skills. Such skills include pointing gestures, gaze-monitoring and showing gestures. Simon Baron-Cohen (1997) attributes this lack of joint attention skills to the breakdown of a part of the Mind-Reading System, more precisely, of the "Share Attention Mechanism". The absence of outputs from that mechanism results in the lack of development of a ToMM that depends on those very outputs for its development. Dysfunctions can also result from a miscalibration of a mechanism due to the exposure to an a-typical environment. Cosmides and Tooby offer an example of such of phenomena: "[...] violent treatment in childhood increases the likelihood that a person has been born into a social environment where violence is an important avenue of social instrumentality. Therefore, the threshold of activation of one's mental organs should be lowered, so one is prepared to act in and cope with such a world. The observation that abused children are disproportionately aggressive when they become adults may be accounted for by a mechanism of this kind." (1999, 461).

probable future selective success rather than past historical success as way of establishing functionality.
7. It is not clear how Baron-Cohen reconciliate this view on autism with his developmental explanation.

7) Treatable conditions that are not the result of a dysfunction

There are also conditions that are thought to require treatment even if they do not result from any dysfunctions. They are usually considered for treatment because they either are interfering with the well being of the individual or because they produce socially unacceptable behaviors.

(1) **Evolved defense**: We mention in the introduction the case of iron-withholding as an example of an evolved defense. Cases of evolved defenses are sometimes confused with dysfunctions because they might cause pain or discomfort. But pain and discomfort are not good cues of what is dysfunctional and what is not. Cosmides and Tooby mention the case of *excessive sexual jealousy* as a case of an evolved defense for which people are sometimes seeking help:

> "Jealousy mechanisms often cause the mates that bear them enormous suffering, and often motivate coercive, violent, or even deadly actions toward women ... Yet jealousy is solely for the 'benefit' or fitness-enhancement of the genes underlying the jealousy mechanism, not the individual who bears them, and its function is to cause patterned behaviors that spread those genes and retard the spread of competitive alleles. ... Using intuitive notions of well-being as the standard, many therapists regard jealousy as a pathology (by which they mean it is disvalued and potentially treatable condition), but to call this a disorder is to confuse the values of the patients involved (or psychiatrists) with the functional integrity of the cognitive adaptations that generate jealousy." (1999, p. 458)

I'll come back to this in section 8.2 with "normal depression" as another example of an evolved defense.

(2) **Environmental mismatch**: Conditions might emerge in cases where a cognitive mechanism, in otherwise perfect working order, has to perform its function in an environment that is completely different from the one in which it has been selected to work (especially in new environments where the cues that used to indicate fitness benefits do not indicate them anymore). Nesse and Berrige mentioned the case of drug abuse as an example of such a case: "Drugs of abuse create a signal in the brain that indicates, falsely, the arrival of a huge fitness benefit. ... We are vulnerable to such fitness-decreasing incentives because our brains are not designed to cope with ready access to pure drugs..." (Nesse and Berrige, 1997). If drug abuse is a case of environmental mismatch, individual variations in susceptibility in drug addiction are better understood as quirks than defects because they probably had no deleterious effects in ancestral environments. They only appear in modern environment where the drugs are more easily available.

8) How the evolutionary approach modifies the way to conceive some treatable conditions

In this section, I would like to consider two examples of conditions that are usually thought to be the result of single kind of etiology. The evolutionary approach, I will suggest, provide reasons to think that these conditions, while displaying similarity at the clinical phenomenology level, are indeed the result of multiple causes. The two examples I chose are also useful in that they further our list of conditions arising from non-dysfunctional mechanisms.

8.1 The case of psychopathy

John Blair (1995) suggested that humans possess a mechanism that mediates the suppression of aggression in the context of distress cues. He called that mechanism "Violence Inhibitor Mechanism" (VIM). According to him, that mechanism plays a crucial role in the explanation of psychopathy. Despite what have been thought before, he postulates that the problem of psychopaths has nothing to do with their capacity to read other mind: their problem is not that they don't perceive the pain their behaviors are producing in others. The fact that they are so good at manipulating others is anecdotal evidence against their mind-blindness. Blair (1996) has shown that despite the fact that autistics have enormous difficulty to read other minds, they still develop moral emotions and the so-called moral/conventional distinction that psychopaths are unable to draw (this is a distinction that adult and children make between moral and conventional transgressions). For that reason, Blair thinks that the core symptoms of psychopathy (absence of guilt and remorse, lack of empathy, no inhibition of violent action) should be explain by the breakdown of the VIM (plus, maybe, some impairments in executive functioning. This clause would allow for undetected psychopaths who don't end up committing violent crimes, but who might nonetheless act in unusual ways).

Blair's account suggests that psychopathy is the harmful (for others at least) result of the malfunction of a mechanism, thus it is a disorder by Wakefield's criteria. But this account is contested by some researchers who by the same token react against the idea of a *monomorphic mind*, i.e. the idea that the cognitive architecture is the same in every normal human being. To that image of the mind, they oppose the image of a *polymorphic mind*, i.e. the idea that there exist more than one "normal" cognitive architecture. In this context, they ask themselves if psychopathy could not be considered as an adaptation rather than a disorder.

For instance, Linda Mealey (1995) has suggested that psychopaths are the "product of evolutionary pressures which ... lead some individuals to pursue a life strategy of manipulative

and predatory social interactions" (p. 135). The existence of such individuals is inferred from game theoretic models which predict that this strategy is to be expected in a really low frequency in a population of discriminative reciprocal altruists because checking all the time for cheaters is too costly. So there would be a niche for that kind of individuals and that would explain the maintenance of the trait across generation. If this story is right, the trait would thus be *frequency-dependent*. Psychopathy would be the result of a normally functioning mind.

Mealey does not stop there. She thinks that if a subgroup of those whom we call psychopaths are what they are as a result of their genetic makeup, another subgroup of psychopaths are what they are in response to certain environmental conditions during their development. These cues from their environment lead them to pursue a life strategy similar to the one of the "born" psychopaths. She therefore introduces a distinction between *primary psychopaths* and *secondary psychopaths*. In a nutshell, her hypothesis is that there are many different causal pathways that might lead to a similar phenotype (psychopathy) and that the consideration and knowledge of these particular pathways is of a crucial importance in determining the type of action to be taken to help those individuals diagnosed with psychopathy (secondary psychopaths can react to cues of distress for instance).

8.2 The case of depressive disorders

One trend in recent evolutionary psychiatry research is to consider that some forms of depression are adaptative (in other words, they might be considered as "evolved defense"). Nesse (2000) call these adaptative forms "*low mood*" to distinguish them from "*chronic depression*", that is a dysfunction that might be due to different factors like trait-variation or a disrupted maturation of the mechanisms in charge of processing information.

The adaptative form of depression would be elicited by cues indicating a loss of adaptative significance: "The losses that cause sadness are losses of reproductive resources... A loss signals that you may have been doing something maladaptative" (Nesse and Williams 1997, p. 9). The losses that can trigger depression are thus, death of parents, loss of a love one after a departure or a breakup, loss of friend after a fight, loss of social status, etc. The pattern of behavior characteristic of depression reinforces the idea that it has an adaptative function. Among other things, it makes the subject stop his current activities, the same one that might had lead to the depressive situation. It also seems that in a depressive state, subjects are able to give more accurate estimate of their capacities (this going against our natural tendency for optimism and over-evaluation of our causal role in success and under-evaluation of our role in failure) which is

obviously good for reassessing goals and strategies. At the same time, it keeps us from impulsively give up project in which we invested a lot. But if difficulties persist, it also helps to disengage from the situation (quitting a mate because you finally realized that he or she is the cause of your depression).

This way of considering "low mood" as an adaptative strategy caused by a loss of reproductive resource is compatible, though more inclusive, than what has been called the "social competition hypothesis of depression". Price et al. (1997), who are advocating that position, suggest that depression is an "involuntary subordinate strategy" that evolved out of mechanisms mediating ranking behavior. According to them, depression would have three functions: (1) preventing the individual of attempting to make a come-back; (2) sending a no threat signal to dominant individuals; (3) putting the individual in a giving up state which encourages acceptance of the outcome. This hypothesis has found support from works by Raleigh and McGuire who found that in vervet monkeys highest ranking males (alpha) had level of serotonin twice as high as other males. When an alpha male lost his position, his serotonin levels fell immediately and he huddled and rocked, refusing food, which are behavior characteristic of depression in human (thus making us think that it is what they experienced). They also found that if they removed the alpha male from the rest of the group and give some antidepressants to a male randomly choosen, that individual becomes in every instance the alpha male (see also, McGuire et al. 1997).

Before closing that section, let me just mention two other types of depression that throw a light on another type of conditions not resulting from a malfunction, but from the fact that the mechanisms are not performing an adaptative function in recent environment anymore. In other words, these mechanisms are vestiges, the equivalent of wisdom tooth. These types of depression might had been adaptative in the environment of our ancestors but lost their adaptativeness in nowadays environment. The first type is what has been labeled the *Seasonal Affective Disorder* (or SAD). It had been noted that there is an increase of depression when the amount of daylight decreases in the fall (what is also sometimes called "*winter blues*"). Some (Nesse 2000) had made the hypothesis that low mood might be a variant or remnant of a hibernation response in some remote ancestor. It would make sense, apparently, to slow down your activities in a period of the year where resources are scarce. But in the kind of environment in which we live now where seasons are playing a minor role in the food acquisition process, such a mechanism has no function anymore.

Another quite speculative hypothesis is that in ancestral environment *postpartum depression* "was an adaptative response which led women to limit their investment in the new child when ... a

major investment would be likely to reduce the total number of offspring produced by that woman during her lifetime who would reach reproductive age and reproduce successfully." (Murphy and Stich, ms). This behavior would be triggered by a variety of cues signaling problems with resources or potential loss of resources in an offspring with low chance of survival: cues would be problems with pregnancy, insufficient father investment, harsh winter, famine conditions. As in the case of seasonal affective disorder, this form of depression might *not be as adaptative* in modern world, specially where resources are available all year round and where two parents are not necessary for the survival of the offspring.

9) Conclusion

I will conclude with a few brief remarks concerning the perspective of solving the problems we identify at the beginning of this paper.

Concerning our first problem, I think that Wakefield's analysis of the concept of mental illness in terms of harmful dysfunction is on the right track. However, there are still questions to be answered concerning his analysis of the term "function". Should we accept the historical view on function he proposed (the one that fix the function of a trait by reference to its evolutionary history) or should we rather adopt a "propensity view" of function (i.e. one that fix the function of a trait by reference to its probable future reproductive outcomes)? Both account can provide us with an objective basis for analyzing function and dysfunction, both are also evolutionary based, but the adoption of one or the other view modifies considerably what will be considered as mental illness. Woolfolk (1999) notes a few of the differences between the two views:

> "Because propensity function are [...] environment-relative, unlike etiologically-defined malfunctions, their malfunction can be direct result of a mismatch between a current environment and evolutionary design, rather than a simple failure of a mechanism to function according to its design. [...] Because propensity functions involve the conferring of advantage relative to a specified environment, either actual or potential, categories predicated upon propensity concepts are rather malleable. Almost any trait, in some environment, might enhance the chances of survival or procreation either of individuals or kinship groups to which they belong. [...] One consequence of employing propensity functions [...] is that, to the degree that adherence to cultural values enhances fitness, proper functioning becomes intertwined with values. It is indeed likely that what would enhance fitness in recent and future human environments is not independent of culture and its values" (1999, p. 664).

I won't develop this idea further here, but I think that psychiatry has been using the notion of "propensity function" to isolate the class of unwanted behaviors. One problem with that notion,

as Woolfolk made it clear, is that it is extremely liberal in that it treat a lot of conditions as mental disorder and its use might had lead to some excess in the past. The notion of function used by Wakefield has the advantage of being less liberal and not as intertwined with values as the propensity account. Because of the abuses of psychiatry in the past, we might be tempted to opt for this more stringent notion of function. As I made clear, the adoption of that notion of function will force the revision of our intuitions concerning mental illness.

How does the evolutionary approach answers Szasz' concern about psychiatry? Well, it shows that after all, mental illness is literally, and not metaphorically as Szasz claimed it was, a physical illness. A mental illness has to do with the harmful dysfunctional disorder of computational devices or mental organs that constitutes our mind. Since these devices are thought to supervene on the brain, it is expected that a dysfunction produced by a disordered psychological mechanism will have a physical base. What the evolutionary perspective to disorder suggests, however, is that we cannot merely identify disorders by looking at the brain. We first need to know what are the (normal) functions supposed to be carried out by the brain before being able to make such an identification. But that hardly makes mental illness different from physical illness.

As for the third question, it seems clear that evolutionary psychology can provide us with a scientific image of the normal functioning of the mind. As Nesse puts it: "Evolutionary biology offers psychiatry the conceptual tools needed to construct a framework for understanding normal mental function akin to that which physiology provides for understanding the normal functions of other bodily systems." (1991, p. 24-25). However, it is possible that this image might be more complicated than we first thought. There are, as we saw in section 8.1, reasons to think that Mother Nature has settled for more than one kind of mind. Indeed, the works of evolutionists makes it clear that we are dealing with a polymorphic mind (see for instance Kimura 2000). It is possible to think of conflicts between different kind of minds or variants of the mind arising from the fact that they do not have the same (adaptative) interests or values (for instance, between reciprocal altruists and psychopaths, but also between men and women, or between children and adults). The pursuit of these different values or interests might lead sometimes to suffering, even if the root of the problem is not a dysfunction. The evolutionary approach is well-equipped to identify and understand the sources of those conflicts that cause pain. The effects of that knowledge are not negligible. As David Buss was putting it recently, the understanding of the competitive mechanisms and of the different mindsets, "[t]he selective processes that designed them, their evolved functions, and the contexts governing their

activation—offers the best hope for holding some evolved mechanisms in check and selectively activating others to produce an overall increment in human happiness." (2000, p. 15).

The answer to the fourth question is probably the most straightforward. The lesson that can be drawn from the two examples of section 8 is that the evolutionary way of thinking is likely to lead to a fragmentation of conditions that seem, at the phenomenological level, homogenous. The idea that what has been classified under current psychopathological concepts is indeed a multitude of different conditions would explain the fact that in cases like depression, for example, "... some instances [...] remit spontaneously; some respond to one type of anti-depression medication but not to another; some do not respond to any type of medication but response to electroconvulsive treatment [...]" (McGuire, Troisi and Raleigh 1997, p. 257). The complicating factor here has to do with what have been called the "*common final pathway phenomena*", i.e. the idea that "multiple causes can lead to similar phenotypes because of constraints on phenotypic expression" (McGuire, Troisi and Raleigh, 1997, p. 257). Since similar expressions might have different etiologies, some can be core adaptations (low mood), while some others can be just maladaptative (chronic depression). As we have seen, the evolutionary approach to psychiatry acts as a prophylactic against the temptation to posit a "unitary adaptative explanation" for each mental condition (see also Nesse, 1991, p. 35).

*

Recently, Ian Hacking made the following remark concerning mental illness and psychiatry:

> "We have objective difficulties, at present, in grappling with the idea of real mental illnesses. This is not because we are in general prone to confusion about reality, but because psychiatry is in a transitional stage in the development of treatments for, and diagnoses of, mental illnesses. We think the problem is about reality when in fact the difficulty lies in the rapid progress of psychiatry itself.
>
> "[...] We have the feeling that there is some fixed, super thing about mental illness, a reality that divides the real illnesses from the fakes. I believe that our conceptions of real illnesses are of necessity being, as Putnam puts it, renegotiated at present. This is because of rapid changes in biological and chemical psychiatry" (1998, p. 92-95).

I believe that Hacking is right in thinking that we are currently renegotiating our conceptions of (real) mental illness, and this paper has make it clear that these renegotiations should also include the insights provided by the evolutionary approach.

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