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## EVOLUTIONARY PSYCHIATRY AND NOSOLOGY: PROSPECTS AND LIMITATIONS

**ABSTRACT:** In this paper, I explain why evolutionary psychiatry is not where the next revolution in psychiatry will come from. I will proceed as follows. Firstly, I will review some of the problems commonly attributed to current nosologies, more specifically to the DSM. One of these problems is the lack of a clear and consensual definition of mental disorder; I will then examine specific attempts to spell out such a definition that use the evolutionary framework. One definition that deserves particular attention (for a number of reasons that I will mention later), is one put forward by Jerome Wakefield. Despite my sympathy for his position, I must indicate a few reasons why I think his attempt might not be able to resolve the problems related to current nosologies. I suggest that it might be wiser for an evolutionary psychiatrist to adopt the more integrative framework of “treatable conditions” (Cosmides & Tooby 1999). As it is thought that an evolutionary approach can contribute to transforming the way we look at mental disorders, 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 is thought by some to be the crucial backdrop to identifying specific mental disorders and distinguishing them from normal conditions. I will also provide two examples of how evolutionary thinking is supposed to change our thinking

about some disorders. Using the case of depression, I will then show what kind of problems evolutionary explanations of particular psychopathologies encounter. In conclusion, I will evaluate where evolutionary thinking leaves us in regard to what I identify as the main problems of our current nosologies. I'll then argue that the prospects of evolutionary psychiatry are not good.

### 1. INTRODUCTION

This paper was originally a chapter prepared for a volume which unfortunately never got to be published. When I began writing this paper, I was a post-doctoral student working under Stephen Stich, and was attending one of his seminars about the philosophy of psychopathologies. Given that I had just finished a thesis about the philosophical implications of evolutionary psychology, I was naturally drawn to evolutionary psychiatry.<sup>1</sup> This rather new discipline (which has old roots, for instance in the likes of Maudsley, James, and Freud<sup>2</sup>) proposed to add to the focus on proximal (psychological) mechanisms of current psychiatry, the consideration of evolutionary origins of those mechanisms. I was interested in evolutionary psychiatry not only because I saw it as a natural extension of evolutionary psychology, but also because it solved a problem which was dear to me at the time: the problem of the unification of disciplines, especially the problem of the unification of psychology. While teaching a philosophy of psychology class, I became acquainted with the debate between partisans of unification of psychology (Staats 1987a,b, 1989, 1991) and partisans of the disunification of psychology (Ermer et al. 2007b; Koch 1981; Kendler 1987). The debate never echoed much in philosophy of psychology, but I thought the question of the importance of unification for psychology was interesting and worth pondering. One incarnation of this problem that seemed to me of crucial importance—because, among other things, of its practical consequences—was that which was encountered in psychiatry. Psychiatry, as many experts saw it, was badly in need of unification. Solutions to the unification problem had been tried before, but according to many the answer that had been adopted had failed. Indeed, tired of internal wars between various theoretical factions (psychoanalysis, behaviorism, humanism, phenomenology, etc.), the architects of the DSM had been looking for ways to increase

unity within their field. The solution they hit upon was to attempt to produce a nosology independent of particular etiological theories that divided the field. As Wiggins and Schwartz put it, the problem was the following:

Because no particular orientation or limited subgroup of schools has established its credentials as the sole scientific approach, there remains no scientific criterion for officially adopting one orientation over the others (Schwartz and Wiggins, 1988). Thus the field of psychiatry must somehow accommodate all of the divergent schools and yet arrive at a single classification scheme that all agree to use. How then to reach agreement amid such unyielding disagreement? (1994, p. 91)

In brief (and to be elaborated on later in this article), the architects of the DSM adopted what can be called the “vacuum strategy of unification”, trying to separate psychiatric observation from psychiatric theories: “The common classification scheme would consist of categories whose meaning could be defined as far as possible through direct observation” (idem, 91). The use of observation was thought to protect categories from “infection” from theories and from its effects on unification of the discipline. But, as many observed at the time, such classification is in essence shallow, it evacuates “theoretical entities” as well as etiological explanations by relying only on clinical phenomenology. I thought that the solution to the problem of unification was elsewhere—namely in the adoption of an evolutionary point of view. At the time, I thought that the adoption of an evolutionary psychology point of view would provide the much-needed meta-theoretical framework to achieve the unification of psychiatry.

Ten years later, my views of evolutionary psychology and its potential for the unification of psychiatry have changed. I now see a number of problems with this view (some of which I detail in recent and forthcoming publications, for instance Faucher & Blanchette 2011; Faucher in preparation a,i) and my hope in its potential for unification has waned. In this paper, I will explain why evolutionary psychiatry is no longer where I think the next revolution in psychiatry will come from.

I will proceed as follows. Firstly, I will review some of the problems commonly attributed to current nosologies, more specifically to the DSM. One of these problems is the lack of a clear and consensual definition of mental disorder; I will then examine attempts to spell out such a definition. One definition that deserves particular attention (for a number of reasons that I will mention later), is one put forward by Jerome Wakefield. Despite my sympathy for his position, I must indicate a few reasons why I think his attempt might not be enough to resolve the problems related to current nosologies. I suggest that it might be better to place accounts like Wakefield’s into the larger framework of “treatable conditions” (Cosmides & Tooby 1999). As it is thought that an evolutionary approach can contribute to transforming the way we look at mental disorders, 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 is thought by some to be the crucial backdrop to identifying specific mental disorders and distinguishing them from normal conditions. I will also provide two examples of how evolutionary thinking is supposed to change our thinking about some disorders. In conclusion, I will evaluate where evolutionary thinking leaves us in regard to what I identify as the main problems of our current nosologies.

## 2. THE DSM AND ITS DISCONTENTS

The *Diagnostic and Statistical Manual of Mental Disorders*, better known as the DSM, is one of the main classification manuals for mental disorders used by clinicians around the world.<sup>3</sup> Having achieved an iconic status, it is often referred to as the “bible of psychiatry”. The third edition of the DSM (DSM-III, 1974) is considered as something of a paradigm shift in psychiatric classification with its emphasis on descriptive diagnosis and provision of explicit criteria sets (Jensen et al. 1997, p. 236). Further editions have stayed within the limits of the paradigm set by the editors of the third edition. Many researchers, some of whom are even members of the task force responsible for the new edition of the DSM (for instance, Hyman 2011), think that DSM-V will not be a significant departure from the vision embodied by the previous editions.

Despite its practical success, the DSM has come under the fire numerous times for what are thought to be its conceptual flaws (see for instance: Cooper 2005; Galatzer-Levy & Galatzer-Levy 2007; Hyman 2011; McHugh 2005; Spitzer & Wakefield 1999; Wakefield 1996, 1997a,b). Geoffrey Miller nicely summarized the current situation by saying that because of tools like the DSM “psychiatry is a mess” (2011, vi); as he sees it, “evolutionary psychiatry promised to bring order in this chaos” (idem).

Let’s start by identifying some of the problems with current nosologies that the evolutionary approach might be able to solve (note that this list isn’t intended to be exhaustive); presented below are four principle problems currently discussed in the literature:

- (1) First is an acknowledged lack of a clear and widely accepted definition of “mental disorder”. As Widiger and Sankis noted 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; for a similar judgment, see Jensen et al. 1997, p. 232). A few years later, in their *A Research Agenda for the DSM-V*, Kupfer and his colleagues echoed Widiger and Sankis’ claim stating that “[d]espite the difficulties involved, it is desirable that DSM-V should, if at all possible, include a definition of mental disorder that can be used as a criterion for assessing potential candidates for inclusion in the classification, and deletions from it” (2000, p. 3). Allan Frances joins the proverbial choir, writing “When it comes to defining the term mental disorder or figuring out which conditions qualify, we enter [a] world of shifting, ambiguous and idiosyncratic word usages. This is a fundamental weakness of our field” (2010, p. 5). More recently, Kendler and colleagues (“Issues for DSM-V: DSM-V Should Include a Conceptual Issues Work Group”, 2008) call for the formation of a work group devoted to conceptual questions, primary among them, the question of the definition of mental disorder. As Kupfer and colleagues remarked, the question is important because a definition of mental illness is instrumental to determining which conditions should be considered disorders and which are normal conditions. Cooper

provides the example of hypomania which she thinks has been wrongly listed as a condition by DSM’s own standards. She argues, “[h]ypomanic episodes are characterized by a mood that is “unusually good, cheerful, or high ... the expansive quality of the mood disturbance is characterized by enthusiasm for social, interpersonal, or occupational interactions” (APA, 1994, 336). Hypomanic episodes are distinguished from manic episodes in that there is no, or little, impairment in the person’s social or occupational functioning, and there are no psychotic features. Quite simply a hypomaniac episode is generally a great thing to experience. [...] I suggest that hypomania in and of itself should not be considered to be a disease because it is not a bad thing to have” (2002, p. 8–9).

- (2) A related problem relates to the objectivity of mental illness. In a paper where they tried to provide a definition for the DSM, Spitzer and Endicott wrote that “[t]he initial impetus [for their definition] grew out of the controversy as to whether or not homosexuality, per se, should be deleted from the psychiatric nomenclature” (1978, p. 15). This concern is still alive as Kupfer and colleagues note that providing a definition for mental disorder “is important because of rising public concern about what is sometimes seen as the progressive medicalization of all problem behaviors and relationships” (2002, p. 3).

It is no secret that part of the motive for providing an adequate definition of mental disorder is to counter the claims of the strong anti-psychiatry movement that emerged in the 60’s (this is made clear in Kendell where he argues against the idea that ‘what psychiatrists regard as mental illness are not illnesses at all’ [1975 305; see also Boorse 1975]). For instance, 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 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, use of illegal drugs), then they are not diseases” (1994, p. 35). According to Szasz, the concept of mental illness is a metaphor that hides under its allure of objectivity, a nor-

mative 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 an agenda. Similarly, some argue that the same is true of disorders like ADHD (Timimi & Taylor 2004); depression (Healy 1997) and post-traumatic stress disorder (Young 1997). The fact that DSM-V is or has considered including binge eating, Internet addiction (Pies 2009), or hypersexuality to their catalogue of disorders does not help to dissipate doubts.

Is Szasz’s diagnostic about mental illness correct? Should we really abandon the notion of mental illness? Is it impossible to introduce some form of objectivity into the concept? It goes without saying that this question is paramount, 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. As Galitzer-Levy and Galitzer-Levy put it: “Most medicine describes disorders as deviations from normality. Psychiatry is unique in approaching pathology without a firm concept of normality and health” (2007, p. 171). Yet, such an image of normal functioning is crucial to 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).<sup>4</sup> The result in this case is over-inclusiveness (or false positives): some cases that shouldn’t be considered depression are treated as such (see Horwitz & Wakefield (2007) for a detailed argument about the over-inclusiveness of the DSM concerning depression).

The problem of the lack of an image of normal functioning 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 psychiatrists in their treatment of patients, but would also provide them with an objective standard by which to judge what is normal and what is deviant.

- (4) A fourth problem—probably the most often discussed—is identified by Poland, von Eckardt and Spaulding who maintain that DSM “constitutes a faulty conceptualization of the domain of psychopathology and [that it] interferes with optimal pursuit of clinical and scientific purposes” (1994, p. 236; a similar point is made by Coltheart & Langdon 1998) According to Poland et al., the 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 structure [...]” (idem, p. 240-1).<sup>5</sup> But as Kendell and Jablensky put it: “... the surface phenomena of psychiatric illness (i.e. the clustering of symptoms, signs, course and outcome) provide no secure basis for deciding whether a diagnostic class or rubric is valid in the sense of delineating a specific, necessary and sufficient biological mechanism” (2003, p. 7).

The problem that architects of the DSM-III had tried to solve was the following: prior to the DSM-III, subjectivity in categorization was the rule.<sup>6</sup> Depending on who you were seeing (a psychoanalyst or a humanist) and where geographically you were seeing them, you could end up with a different diagnosis (Bentall 2006, p. 222); there was very little consistency. To solve this problem, “Spitzer asked expert clinicians and investigators what features they used to identify, rather than explain, the disorder they studied. He then picked those features that were observable, such as hallucinations and delusions, and insisted they be so defined that any psychiatrist could confidently recognize them. Finally, he tested delineated features as diagnostic criteria in field trials to uphold those criteria that helped psychiatrists make replicable diagnostic decisions” (McHugh 2005, p. 2526). In a nutshell,

the solution to the problem of subjectivity was thus to jettison a priori theories (like psychoanalysis) and “replace clinical impressions with operationalized indicators” (Nesse & Stein 2012, p.1).

McHugh illustrates the result of this “revolution in diagnostic” with the following comparison: “Being appearance driven, it is similar to a naturalist’s field guide with the advantages and disadvantages of such. Just as Roger Tory Peterson’s *A Field Guide to the Birds* distinguishes a prothonotary from a yellow- or blue-winged warbler by the bird’s coloring, voice, and range, the DSM distinguishes and then arranges mental disorders by their appearance—on their shared phenomenological features” (2005, p. 2526).

The use of phenomenological features for diagnostic purposes can be compared to determining the problems with a TV set using only observable manifestations (Murphy & Stich 2000). Many things can be responsible for the fact that nothing appears 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 directly observable symptoms at the phenomenological level.

The problem is magnified by what has been called the “Chinese menu” method of diagnosis (Galatzer-Levy & Galatzer-Levy 2007, p. 163). The DSM-III’s authors listed common clusters of findings, and stipulated that an individual suffers from a condition when a certain number of findings from each cluster were present. For instance, “the DSM-IV diagnosis of major depression requires that a patient have at least five of nine possible symptoms. In this scenario, it is possible for two patients to receive the same diagnosis with only one symptom in common” (Miller 2010, p. 1437). Given the presence of polythetic criteria it comes as no surprise that people diagnosed with the same disorder are not similar at a biological level (I will return to this idea shortly). Moreover, it appears that there is a high level of comorbidity among DSM-IV diagnoses (individuals who qualify for one disorder also qualify for other disorders), which suggests problems

with the current classification. As Hyman puts it: “The open question is whether different manifestations of a basic pathological process have been divided into multiple silos, creating artificial comorbidity in certain circumstances” (2011, p.7).

In short, 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” (Poland et al. 1994, p. 250).

Poland and colleagues (1994) suggest that an alternative nosology should be based on more intimate relationships with basic sciences like the cognitive sciences, neuroscience, molecular biology, etc; their view has found echoes in psychiatry, as most psychiatrists nowadays believe that categories should be built on stronger ‘etiological’ foundations. For instance, Andreasen (1995) argues in favor of new validation models (that Schaffner 2002 labeled “etiopathogenic validity”<sup>7</sup>). She asserts: “New models of validation probe beneath such surface features and seek to identify actual neural and genetic mechanisms. Because it draws closer to actual causes, the second structural program of validation can give mental illnesses a powerful credibility” (162; for similar statements, see McHugh 2005, p. 2527).<sup>8</sup>

Some have argued that evolutionary psychology could also contribute positively to the elaboration of a new and more accurate nosology (Abed 2000; Baron-Cohen 1997; Gangestad & Yeo 1997; Keller 2008a,b; Keller & Miller 2006; Kennair 2003; McGuire & Troisi 1998; Nesse 2009; Nesse & Jackson 2011, 2006; Nesse & Stein 2012; Stein 2006; Stevens & Price 2000). The above-mentioned are generally not claiming that evolutionary psychology will do the job by itself, as other disciplines will doubtless play an important role in the elaboration of a more accurate nosology. It is usually suggested 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. To sum up Cosmides and Tooby, the Darwinian approach to mental disorders should transform the way we conceptualize, investigate, and classify disorders (1999).

In the following, I will evaluate how a psychiatry—informed by evolutionary theories—could fare with our four major problems.

### 3. A CONCEPT OF MENTAL DISORDER

The potential for an evolutionary perspective to provide solutions to problems (1) and (2) has been recognized early on in the debate with anti-psychiatry. In the mid-seventies, two important contributions to the debate made use of an evolutionary perspective, trying to spell out a definition of mental disorder that would provide an objective basis for the attribution of disorder. Both presented themselves as trying to capture the professional medical use of the concept (they weren't interested in capturing folk uses). Kendell (1975), following Scadding, proposed understanding disorders in terms abnormal phenomena displayed by members of a species which put them at 'biological disadvantage' compared to those who do not exhibit that phenomena, where biological disadvantage is to be understood in terms of reduction of fertility or augmentation of mortality. Given that reduction of fertility or augmentation of mortality is something that can be measured objectively, it is possible to establish on a factual basis who is and who isn't afflicted by a disorder. Hence, Kendell can confidently conclude his paper by saying that "we have adequate evidence that schizophrenia and manic-depressive illness, and also some sexual disorders [homosexuality] and some forms of drug dependence, carry with them an intrinsic biological disadvantage, and on these grounds are justifiably regarded as illness" (1975, 314).

Similarly, Boorse (1975; 1976; 1977) defined disorder (he refers to 'disease' in his work, but this makes no difference in the present context) in terms of its biological consequences:

A disease is a type of internal state of the organism which:

- (1) Interferes with the performance of some natural function—i.e., some species-typical contribution to survival and reproduction—characteristic of the organism's age; and
- (2) Is not simply in the nature of the species, i.e., is either atypical of the species or, if typical, mainly due to environmental causes. (1976, 62-3)

To the extent that the typical functional organization of a species is a biological fact, and that the goals of this organization are survival and reproduction (1976, 84), establishing that an individual of a given species suffers from a disorder is in principle something that can be established objectively. As Boorse put it, "if certain types of mental processes perform standard functions in human behaviour, it is hard to see any obstacle to calling unnatural obstructions of these functions mental diseases, exactly as in the physiological case" (1976, 64). The problem for psychiatry then becomes, getting a clear view of normal psychological faculties: "[W]e cannot call it [an individual neurosis] unhealthy until we know that the mind is not supposed to work that way [...] any stronger vindication of current clinical categories would require a detailed and well-confirmed theory of the functions of a normal human mind" (1976, 71 and 76).

As we will later see, it is exactly this well-confirmed theory of the function of a normal human mind that evolutionary psychology seeks to provide. But before arriving there, let's simply say that despite its references to evolutionary biology, most people haven't been satisfied with these definitions. One problem probably comes from the fact that this view doesn't seem to exclude conditions that we (socially) do not want to consider as disorders, such as homosexuality. It is also considered too sensitive to context (though this wasn't seen as a problem by advocates of this view), as the capacity to meet biological goals depends crucially on the environment where the organism is. Another problem stems from the fact that these accounts seem to rest on an inadequate conception of biological function. For some people, the kind of function used by psychiatry has to be defined in terms of natural selection, not in terms of the actual capacity of an organism to meet its biological goals.

A proposal that has attracted more attention than the previous two belongs to Jerome Wakefield. Spitzer, one architect of the DSM-III, describes Wakefield's analysis of the concept of mental disorder as "easily the most often quoted and most provocative analysis of the concept of mental disorder that exists today, defining simultaneously the concept as legitimate and providing a framework for ... the criticism of the actual diagnostic standards as being too liberal" (2007, p. viii). Elsewhere, he continues, adding that the "HD [harmful dysfunction]

concept is a considerable advance over the DSM definition of mental disorder [...] [and it should therefore] be adopted by the DSM-V and the DSM-IV diagnostic criteria revised after a careful HD analysis" (1999, 432). So, what is this analysis which both legitimates DSM's concept and provides grounds for criticism?

In a series of papers, Jerome Wakefield (1992a; 1992b; 1999; 2000; 2007a) proposed a definition of the concept of "mental disorder" that he hopes will provide psychiatry with an *objective* criterion for declaring a mental condition a pathology. His definition presents an explicitation of the intuitive concept of disorder used not only by health professionals, but also by the general public. He believes 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<sup>9</sup> 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 little to say about the "value" component of his definition;<sup>10</sup> 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, according to Wakefield, only the evolutionary theory can analyze these in causal and scientific terms. Wakefield proposes understanding the previous uses of function as cases of what he calls "blackbox essentialism". This theory is an extension 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, 39). However, the process responsible for the phenomena was not known until the advent of the Darwinian theory.

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 organisms' ancestors, the effects of which had been beneficial enough for the organisms' ancestors to preserve their species through natural selection. The function for which a trait (or a mechanism) had been selected is what has been called in philosophical literature the "normal function"<sup>11</sup> 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, contrary to Kendell and Boorse, 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 maladaptive 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 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 have been found to have been selected and that they were adaptive in certain cases in the history of our species. If such were the case, we would have to judge the mechanisms responsible for these behaviors as being in good working order, even if we abhor and disvalue the behaviors they produce. The objectivity of the concept of function would protect us from the abusive use of the determination of mental illness denounced by Szasz.

#### 4. THE USEFULNESS AND RELIABILITY OF CONCEPTUAL ANALYSIS

Many criticisms have been voiced against Wakefield's definition of mental disorder (see commentaries about Wakefield's theory in *Abnormal Psychology*, 1999 and *World Psychiatry*, 2007). The criticism focused on here concerns the fact that there is no *a priori* reason to suppose that our folk concept of "disorder" is worth keeping.

As I see it, there are two problems with the folk concept of disorder.

The first one is that it is not clear that the analysis of the folk concept that Wakefield uses is the one that scientists are using, or that they should be using for their purposes. The second, which was suggested to me by Stephen Stich in a personal communication, is that it is not clear that people are unwilling to categorize as “disorder” those mechanisms that no longer produce adaptive behaviors or mechanisms that are otherwise in perfect working order, but that give rise to behaviors that are not socially acceptable.<sup>12</sup> Stich goes so far as to propose the elimination of the concept of disorder because of conflicting intuitions concerning what it applies to.

Regarding the first point, Roe & Murphy (2011) have argued that “the *systemic capacity view* of biological function and dysfunction seems better suited than the *selectionist view* to capture what bio-medical scientists take themselves to be doing” (217). In their view, when medicine (as well as disciplines interested in the functional organization of the mind, such as cognitive neurosciences) considers the function of a trait, it is usually not concerned with its evolutionary history, but rather with its contribution to the overall functioning of the organism. For instance, researchers interested in our capacity to obey norms will posit that two cerebral structures are involved: the dopaminergic system and some lateral structures of the prefrontal cortex (Barbey & Grafman 2010; Montague et al. 2004). This research may try to explain how each structure contributes to the general capacity, but nowhere is there speculation about evolutionary origins. Likewise, when attempting to explain problems (disorders) with obeying norms, such work might refer to the fact that one or the other structure (or both) is (are) not functioning properly, but again, evolutionary considerations play no essential role in the explanation.

In terms of the second point, let me say a word about Wakefield’s method of conceptual analysis. According to Wakefield: “[The] conceptual enterprise [conceptual analysis] is also an empirical enterprise aimed at discovering a certain fact about the world, namely what conceptual criterion or definition in the heads of people in our linguistic community ultimately determines and explains their judgments about whatever conditions are mental disorders” (1997a, 257). Indeed, conceptual analysis contends that it can discover which definition people entertain by proposing cases that are seen as falling under a version of

a concept which counters one’s own intuitions about what should or should not fall under said concept. If a version of the concept accords with our intuition, it is then proposed that it is the concept that we entertain. Wakefield can thus maintain that: “the process of conceptual analysis does not look empirical because one generally uses one’s own intuitions about the clear cases rather than going out and collecting data. However, this oddity results from *the presupposition that one is dealing with a culturally shared concept, and the confidence that one’s clear intuitions about the application of the term are likely to be shared*” (idem; my emphasis).

But what are the grounds of that presupposition? Why should we think that our intuitions are representative of other people’s intuitions? What are the grounds for supposing that people share a unique concept? These questions call for a more rigorous kind of empirical investigation of the concepts. Luckily, in recent years we have seen the emergence of an alternative methodology to study folk intuitions: experimental philosophy. As Knobe, one of the main proponents of this method, puts it: “Experimental philosophy focuses on many of the same types of intuitions that have long been at the center of philosophical study, but it examines those intuitions using the methods associated with contemporary cognitive science—systematic experimentation and statistical analysis” (2007, 81). Oddly enough, though authors of the *Research Agenda for DSM-V* do not think that this will settle which concept of mental disorder should be used, they nonetheless propose it as part of the research agenda to “[c]onduct surveys ... to elucidate the concepts of disease or of mental illness or disorder used, explicitly or implicitly, by psychiatrists, other physicians, clinical psychologists, research workers, patients, health care providers, and members of different social and ethnic groups. This could be done either by exploring the meaning they attribute to such terms or by asking them to decide which of a list of contentious conditions they themselves regarded as disease or mental disorders ...” (2002, 7).

Despite his professed use of conceptual analysis, Wakefield also uses experimental philosophy methods (see Kirk et al. 1999; Wakefield et al. 2002). I will not comment about his results here (see Faucher in preparation b), but I will make two suggestions. The first consists of the need to use the method of experimental philosophy not only to

test lay people, but also different groups of health professionals, and to perform these tests in different cultures. The following is an example of the kind of test that could be used. We could ask subjects to read the following statement “In ancestral environments, it was adaptive for some children to move a lot and to have a very short span of attention in order to be able to respond to ever-changing environmental conditions. These children are biologically identical to children presently labeled as suffering from ADHD”. We would then ask them: “In the present time, do you consider these children to be suffering from a mental disorder?” We could also propose stories in which ADHD is swapped out for pedophilia, depression or schizophrenia. If a group of people see these adaptative conditions as mental disorders, this would go against HD analysis, as per Wakefield: “The HD analysis implies that such claims that disorders are naturally selected are not merely false but incoherent. A disorder is a failure of function and thus cannot itself be a function of a naturally selected trait, according to the HD analysis” (2005, 895). We could also test people with statements that would test the necessity of the history of selection for a functional trait, or the impact of particular environments on disorder judgments. For instance, De Block (2008) proposes the following: “SAD [Seasonal Affective Disorder] is an adaptative pattern of responses that contributes to the individual’s reproductive success in higher latitude regions (Davis and Levitan, 2005). Let’s imagine a girl born in Sweden. Mild SAD was part of her ancestors’ phenotype, but, due to minor mutations, here parents lacked this ‘capacity’. Yet, new mutations have provided the girl with mild SAD”. Then, we can ask the subjects: “Is her SAD functional?” or “One winter, her SAD mechanism stops working. Is she suffering from a disorder?” or “Before her new mutation, she moves to Africa where she doesn’t need SAD. Does she have a disorder in Africa?”. I don’t want to prejudge how different groups would reply to questions like these, but it is possible to think that they will reply differently (or perhaps in ways incompatible with HD analysis). The question then becomes what should we do with HD? Should we still retain it as the best way to explain mental disorder, even if it is not a concept shared by all?

My second suggestion is also methodological. It follows a proposal made by Colombo et al. (2003) in their “Evaluating the Influence of

Implicit Models of Mental Disorder on Processes of Shared Decision Making Within Community-Based Multidisciplinary Teams”. Their idea is to try to tap into people’s implicit models of disorder, the one, they hypothesize, which is more explanatory of people’s actions and decisions. As they suggest: “Asking people, whether professionals or users, directly about mental disorders will elicit, mainly, their explicit views. [...] if the linguistic-analytic insight [this is their view of concept attribution, a view inspired by Wittgenstein according to which meaning is use] is right, on the other hand, if such concepts use is a surer guide to meaning than explicit definition, then [...] how they actually respond to [...] mental disorders, will be driven by their implicit models of disorder” (Fulford & Colombo 2004, 136). To achieve their goal, they supplied subjects with a vignette describing someone who is suffering from schizophrenia. Instead of asking forced choices (“Is he suffering from a mental disorder?”), they asked the subject about possible etiologies, responsibility for actions, potential treatments, etc. Then they code the answers according to six models of pathologies they constructed (which are: the medical model, the social model, the cognitive-behavioral model, the psycho-therapeutic model, the family model and the conspiratorial model). This technique is supposed to reveal the “implicit theory” of the subjects. What they found is that psychiatrists and community nurses shared an implicit medical model (where disorders are the result of brain dysfunction), but that social workers are more likely to entertain the social model (where the causes of the disorder are social). What their data reveals is that even in groups where a model of disorder dominates, some members of the same group are attracted to different models. Even worse, a more recent study from Harland and colleagues (2009) shows that judgment concerning adequate models of explanation for a disorder varies from disorder to disorder (they asked subjects about four conditions: schizophrenia, major depressive disorder, antisocial personality disorder and generalized anxiety disorder).

In a nutshell, not only should we test the intuitions of various groups of professional and laypeople (and different groups of laypeople, including patients, family care-givers, etc.), but we should also be aware of the possibility of a split between our implicit and explicit conceptions of mental disorder. We should therefore use methods to tap

into our implicit conceptions (for a survey of potential methodological tools, see [Nosek et al. 2011](#)).

I do not want to prejudge the results of the studies of our intuitions that I have proposed, but I do think Stich might be quite right in regards to the state of our intuitions about mental illness. I wonder though, if we should accept his conclusion. I agree that our concept of mental disorder might be different from that which Wakefield proposes. Instead of elimination, I would propose a split (similar to the one proposed by Boorse in his papers) between the lay concept and the professional concept of disorder. Following Rachel Cooper (2005), I agree that our folk concept is a bit like the “weed” concept, i.e. a concept structured by our practical interests. We also need a professional concept, one that researchers would use, a concept that might be different from the lay concept and that would study conditions that result from the dysfunctions of psychological mechanisms, independently of the fact that they generate distress or demand a “call for action” (as Spitzer & Endicott (1978) put it). For practical purposes, I think that what we need is to develop a clearer picture of what can go wrong with the mind and what can cause the distress that leads people to seek treatment or assistance from psychiatrists. In other words, I think we should not focus our attention exclusively on the notion of mental disorder as proposed by Wakefield.<sup>13</sup> For that reason, I would rather propose (following Cosmides & Tooby 1999), considering the notion of disorder within a larger framework of *treatable conditions*, i.e. conditions that are judged harmful enough that they require treatment. I propose that it would be good to abandon the lay concept of mental disorder for one involving treatable conditions. Being told that your distress is the product of a normally functioning mechanism provides little comfort—similar to someone saying that your headache is the result of a normal physiological mechanism. We should not deny treatment to people who desire it, or forgo research into conditions that are thought to be “normal”. Therefore, we should be prepared to break the link between medical treatment and the presence of disorder. As Nettle puts it: “To accept these conditions as medical is to acknowledge that psychiatric diagnosis is in fact based on values, such as the need to reduce human suffering, rather than only on natural kinds of mental functions or dysfunctions. We can’t clearly tell when people’s mood

systems are disordered, but we can clearly tell when they are having life difficulties as a result of their mood” (2011, 212). Another reason to adopt the label “treatable condition” will get clearer in section 9. At the present moment, and maybe forever in certain cases, we have no idea of the evolutionary history of some traits or mechanisms. What should we do in the mean time? Note that, according to Wakefield himself, ignorance of the evolutionary origins of a mechanism forces one to rely on intuitions about its evolutionary functions. But, as he would be the first to acknowledge, our inclinations have proved in the past to be a poor guide to dysfunction, as the cases of masturbation or lack of female orgasm illustrate. As he notes, the ignorance of facts about the origins of the traits leaves the door open to norms and values in the attribution of mental disorders. The label “treatable condition” has the advantage of dissociating the question of factual knowledge of evolutionary origins from the pressing needs of the individual or society.

In the remainder of this paper, I want to demonstrate why evolutionary psychiatry seems to be in a perfect position to explain treatable conditions, and to study dysfunctions. In Sections 6 and 7, I will review some forms of treatable conditions (for more exhaustive reviews see Cosmides & Tooby 1999; Nesse 1999). Prior to that, I want to say a brief word about the link between psychiatry and evolutionary psychology.

##### 5. EVOLUTIONARY PSYCHOLOGY AND THE IMAGE OF THE NORMALLY FUNCTIONING MIND

As Boorse before him (see also Kennair 2003), Wakefield thinks it is critical for the advancement of the field of mental health “to understand the nature of mental mechanisms” (1992a, 385). For Wakefield, only evolutionary psychology will reveal our “human species-typical biological design” (Horwitz & Wakefield 2007, 38). Therefore, “... in the long run, the DSM must be replaced by a more theoretical explanation of mental mechanisms. Thus, the destiny of the professions of mental health in regard to theoretical and scientific process in the comprehension of the etiology, the diagnostic and the treatment of mental disorder might depend in a large part on progress in evolutionary psychology”

(2005, 900).

Let me briefly summarize what Wakefield (and others) has in mind when he speaks of ‘evolutionary psychology’. Evolutionary psychology, or EP for short, results from the meeting of evolutionary biology and the cognitive sciences. Evolutionary psychologists see the mind as “an information-processing description of a subset of physical activity of the brain that implements organized computational activity” (Cosmides & Tooby 1999, 454). They see this computational activity as its evolved function, that is, the mind’s function is “to regulate behavior and the body adaptatively in response to informational input” (Tooby & Cosmides 2005, 5). However this doesn’t distinguish EP from other kinds of psychology influenced by the theory of evolution (such as Human Behavioral Ecology [“HBE”], Winterhalder & Smith 2000). What distinguishes evolutionary psychology are 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”.<sup>14</sup> The concept of modules used by EP is not as strict as the one used by Fodor, who treats them as more or less natural kinds (that is, he postulates they have seven properties that more or less always co-occur; see Ermer et al. 2007a, p. 153). For evolutionary psychologists, modularity refers first and foremost to “functional specializations” or “evolved specializations” (Baron-Cohen 2007; Barrett & Kurzban 2006; Pinker 2005; Tooby et al. 2005). Contrary to Fodor, the properties of modules cannot (and should not) be stipulated a priori, but should be discovered through empirical work, because the properties that modules will instantiate depend on the problem they are to solve (see Sperber & Caton 1996, 170).

Again, 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), advocates of EP posit what has been called a “massive modularity hypothesis”, according to which the mind is made of hundreds and thousands of modules, some of which are central modules (Cosmides & Tooby 1995; see also Pinker who is not in favor of a very massive modularity and says he’s content with “two dozen emotions and reasoning faculties” (2005, 16)). As mentioned earlier, one

should beware of the fact that the modules that are postulated are not Fodorian, but rather functional boxes specialized in the resolution of evolutionary problems.

*Thesis 2: Adaptationism.* The process of natural selection has shaped cognitive architecture. As Tooby and Cosmides express, mental modules have been “invented by natural selection during the species’ evolutionary history to produce adaptative ends in the species’ natural environment” (Tooby & Cosmides 2005, 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 or even new uses of old structures (i.e., exaptations). 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: Environment of Evolutionary Adaptedness.* Modules have been shaped to solve particular problems of our Environment of Evolutionary Adaptedness. (EEA). As Tooby and Cosmides describe it, EEA for a given adaptation is “the statistical composite of the enduring selection pressures” (2005, 22). This environment also contains clues that can both help develop the adaptation and be used to solve problems. For this reason, EEA “refers jointly to the problems hunter-gatherers had to solve and the conditions under which they solved them (including their developmental environment)” (idem). Note that “[b]ecause adaptations evolved and assumed their modern form at different times and because different aspects of the environment were relevant to the design of each, the EEA for one adaptation may be somewhat different from the EEA for another.” (idem). For instance, the EEA for trichromatic vision that reappeared in some non-human primates (Mollon 2000) is different from the EEA for a form of advanced cooperation that is found only in certain groups of humans (Dubreuil 2010).

These pressures or problems and the cues used to solve them can be regrouped into specific domains (reproduction, predatory behavior, social interaction, poison avoidance, incest avoidance, etc.) each with specific properties (a good acquaintance does not necessarily 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, these modules exploit cues that co-vary (often enough) with properties in the

EEA in order to produce adaptative behavior.

The picture of the mind offered by evolutionary psychology provides the backdrop against which the notion of mental disorder can be understood. Since the goal of evolutionary psychology is to discover the structure of the normal mind, it becomes the reverse of evolutionary psychiatry. Evolutionary psychology produces an image of what proper or normal functions of the mechanisms are that constitute the mind. These mechanisms are the same as those that go astray in mental disorder, and that are the object of psychiatry.

Evolutionary psychology, as presented in this section, has a rather narrow definition: “evolutionary psychology” refers to what is sometimes called the Santa Barbara school of evolutionary psychology. Yet the term ‘evolutionary psychology’ can be used more widely to capture other forms of psychology informed by an evolutionary perspective (for instance human behavioral ecology, gene-culture co-evolution, or memetics) and these forms could also contribute precious tools to psychiatry. Human behavioral ecology, with its focus on “fitness maximization”<sup>15</sup> and the effects of ecological and social factors on behavioral variability could be better used with a definition of mental illness in terms of failure of adaptative behavior, such as is seen in Kendell and Boorse. Gene-culture co-evolution (Richerson & Boyd 2004) with its focus on the role of culture and social learning biases in human adaptation, and on the concept of niche construction (Laland et al. 2001), could be useful in explaining how certain non-adaptative ideas fixate in individuals (in certain crucial periods of development, ideas concerning personal worth or the acceptable level of violence or a legitimate object of sexual fixation could be acquired and be the source of problematic behaviours; for the latter, see Aronsson 2011) or how new human-created conditions (for instance schooling and the lack of play-time it imposes on children) are causing mental disorder (for instance, ADHD, Panksepp 2007). Finally, memetics with its focus on the spread of certain ideas in a population could be useful in explaining certain mental disorder epidemics (such as multiple personality disorders or hysterical fugue; Hacking 1995 and 1998).

It is thus possible to imagine an evolutionary psychiatry that does not rest on an evolutionary psychology understood narrowly (Gerrans 2007). At the moment, however, the four forms of evolutionary psy-

chology should most likely be used together, to the extent that they are not exclusive one to the other, to provide testable explanations of mental disorders. In what follows, I will focus exclusively on evolutionary psychology understood narrowly, since it is the form of evolutionary psychology most referred to by evolutionary psychiatrists.

## 6. TREATABLE CONDITIONS THAT ARE DYSFUNCTIONS

As mentioned earlier, many psychiatrists that are in favor of adopting an evolutionary point of view find that it would be preferable to talk in terms of treatable conditions. In this section and the next, two kinds of treatable conditions will be discussed: some that are the result of dysfunctions and some that are the result of functioning mechanisms in proper order.

The first group of treatable conditions is the one resulting from cognitive mechanism dysfunctions.<sup>16</sup>

**(1) Simple Breakdown:** The simplest kind of dysfunction is the breakdown of a module. Cognitive neuropsychiatry has provided multiple examples of such breakdowns. One example is given by Frith (1992): in his model, certain schizophrenia symptoms (control delusions and “voices”) are a result of the failure of a monitor mechanism in charge of distinguishing our actions from those of others. This mechanism’s breakdown leaves the patients without knowledge that they are the source of a movement or a thought leading them to think that someone else is controlling them, or that they are hearing voices.

Another case of breakdown involves the severing of a link between modules, which can be used to explain Capgras’ delusion (Young 1994, 1996). Individuals suffering from Capgras’ syndrome have no problem recognizing people with whom they are familiar in terms of their physical attributes, however there is a feeling that something is wrong—familiar people appear to be somehow changed. The sufferer may confabulate, believing that the people are not *really* their friends, family members, or acquaintances, but exact replicas (Young 1994, 1996). According to current theories, this condition relies on the fact that there are two pathways to the visual system, one affective (feeling of familiarity) and the other cognitive (a template-matching system). The cognitive system is working properly, so there is recogni-

tion, while there is a breakdown of the affective system, so recognition is not accompanied by the normal feeling of familiarity.

A prediction that is dependent on assumptions made about the architecture of the mind specific to evolutionary psychology, is the one concerning cheaters. Cosmides and Tooby (Cosmides 1989; Cosmides & Tooby 1992; Fiddick et al. 2000) have argued for years that we have a module dedicated to detecting cheaters in social interactions, and that this module is different (i.e., uses different cues) than the one involved in precautionary reasoning. In a paper written with Valerie Stone (Stone et al. 2002; for an extensive review on the subject see also Cosmides & Tooby 2005a), they study a patient case that purportedly demonstrates that social exchange reasoning can be dissociated from reasoning about other domains. Such a case goes against assumptions made by most theories of reasoning, according to which both forms of reasoning are accomplished by a general-purpose mechanism.

In principle, evidence such as that found for social reasoning could be looked for in mechanisms in domains as diverse as predator avoidance, mating, responses to landscape, kin recognition, parental investment, group living, etc. Evolutionary psychology could truly play an important heuristic role in the discovery of new conditions by going beyond intuition and instinct-blindness (Cosmides & Tooby 1995). Note that because people are in principle “blind” to instincts that allow humans to solve adaptative problems, and that these instincts are numerous, there is the possibility of the existence of many more disorders than what we find now in the DSM.

Moreover, evolutionary psychology has its own concept of disorder: if a mechanism does not perform its evolved function, it decreases the organism’s fitness and therefore harms it (there is no place for value in this concept, as you might notice)—though the harm may not be visible from the organism’s perspective (or from the point of view of its pairs) or in the short duration of its life. In the long run (over generations), organisms with such mechanisms would decrease in frequency in the population. The fact that the fitness of an individual with a mechanism  $x$  is decreased by .02 might have dire effects in the long run, it might be non-functional in a sense, but I doubt that this is the sense which is of interest for actual patients or psychiatrists.

**(2) Over or Under-responsiveness:** Another kind of dysfunction is

when the module is working, but not computing according to the criterion that constitutes its evolved function. Examples of such cases are frequent in emotional disorders; 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, i.e. that emotions are adaptative responses to stimuli that represent an adaptative problem in the EEA,<sup>17</sup> a lack of response to fearful objects should be as detrimental for the organism as, for example, a lack of pain. It might be that cases such as these remained unnoticed because they are not distressing per se, but if functional theory is correct, they must impose a cost on those individuals who are subject to them. As Nesse and Jackson put it: “no one comes to the clinic complaining of too little anxiety or an inability to feel sad, but this is just an artifact of our limited imagination and the absence of a scientific foundation for the diagnosis of emotional disorders” (2011, 187).

One thing that these cases make clear is that some reference to a theory concerning the normal function(s) of particular emotions and to the context in which an emotion is aroused seems necessary to establish what is normal and what is abnormal. Indeed, an emotional state should be classified as a disorder only if it is in excess or in deficit in relation to the situation that triggered it. A useful comparison is pain, a protective reaction to stimuli that are potentially damaging for the organism. Before making a diagnosis of chronic pain, one would usually try to look for environmental factors (including physical conditions) for which the pain response would be responsible. If no factors can be found, one would conclude that there is a malfunctioning of the pain mechanism. According to some researchers, current nosologies—DSM included—do not pay enough attention to what constitutes a normal reaction to environmental factors. For instance, Horwitz & Wakefield (2007) criticize the DSM’s definition of major depression because of its sole focus on the presence of symptoms, which does not consider that sadness is a normal reaction in certain cases (except for bereavement, yet even this exclusion is hotly contested now, see for instance the debate between Kendler et al. 2008 and Wakefield & First 2012).

**(3) Balancing Selection and 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 resist selection pressures and remain in the gene pool? This situation has been referred to as the ‘evolutionary paradox of severe mental disorders’ (Keller 2008a, 396; Keller & Miller 2006).

There are many explanations of the paradox, some of which make reference to variation in the cumulative effects of mutation: “Mutations that degrade the brain’s performance differentiate everyone on a panoply of behavioral dimensions, making some people slow at learning, others bad at remembering, others too anxious or not anxious enough, and so forth. But some people inherit an especially high ‘load’ of mutations ... that disrupt particular neurodevelopmental pathways, increasing the risk of aberrant behaviors and psychiatric categorization” (Keller 2008a, 397; such departures from design are called “developmental instability”, Gangestad & Yeo 1997, 104)). Another cause of the presence of mental disorders is the coevolution between pathogens and their hosts. As Gangestad and Yeo put it: “Perhaps the most important changes introducing maladaptation are those caused by the evolution of organisms that we are in conflict with. Parasites and other pathogens continually, and rapidly, evolve to be better adapted to their hosts” (1997, 104). Studies have shown that pathogens are known risk factors for mental disorders: for instance, “several studies have found that childhood Streptococcal infections are weakly associated with adult obsessive-compulsive disorder” (Keller 2008a, 398). Similar suggestions have been made concerning prenatal exposure to infection and schizophrenia (Brown et al. 2004). If mental disorder risk is affected by either pathogens or an evolved defense against them in the host, the continuous co-evolution between the two can maintain genetic variations in mental disorder risks, and therefore escape the elimination of bad allele by natural selection.

Some other factors explaining the persistence of risk alleles in the population are balancing selection and pleiotropy (the latter a type of balancing selection). As defined by Keller, ‘balancing selection’ “... occurs when natural selection actively maintains two or more equally fit alleles at a gene ... Heterozygote advantage—in which individuals who are heterozygote (*Aa*) at a gene have higher fitness than those with either homozygote (*AA* or *aa*)—is a special case of this process” (Keller 2008a, 397). For instance, the same genes that cause sickle-cell

anemia in certain individuals also protect others from malaria. But as Keller himself notes, evidence in favor of such an explanation for mental disorders is at the moment scarce. Another type of evidence consistent with the balancing-selection hypothesis would be finding that relatives of those with mental disorders have some sort of fitness advantage. This might suggest that low doses of risk alleles (typically found in relatives) have positive effects that counterbalance their high-dose negative effects. The most intriguing support for this notion comes from studies demonstrating that schizotypy (a personality dimension, whose extreme form may constitute schizophrenia) is higher among highly creative individuals (Nettle & Clegg 2006). One interpretation is that low doses of schizophrenia risk alleles increased creativity and fitness in ancestral environments” (Keller 2008a, 398).

“Pleiotropy’ refers to the phenomenon in which a single locus affects two or more apparently unrelated phenotypic traits, and is often identified as a single mutation that affects two or more wild-type traits” (Stearns 2010, 767).<sup>18</sup> One explanation makes reference to the fact that a gene or set of genes can control more than one phenotypic trait, especially if one of the non-disordered phenotypes is highly adaptive. McGuire et al. (1997a) 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). Further, ‘antagonistic pleiotropy’ might be useful in explaining late-developing mental illness. That antagonistic pleiotropy may be useful in this regard has been suggested by Williams (1957) and refers to the fact “... that genes with antagonistic effects at different life stages could contribute to aging in a way that natural selection could not alter. That is, genes with beneficial effects prior to reproduction but negative effects after reproduction would be favored by natural selection over those that increased longevity but were less favorable to reproduction and survival to reproductive age” (Stearns 2010, 769). Conditions related to senescence, like Alzheimers dementia, might also be explained with reference to such a process.

**(4) The Extreme Variant Phenomena:** The distribution of a single trait in a population goes from functional to dysfunctional; the very same genes that lead an individual to be dysfunctional in one domain

might lead another to exhibit superior functioning in a different domain. Recently, Baron-Cohen has used this explanation in his account of some autism symptoms. Autistics are known to have impaired mind-reading capacity (impaired folk psychology), but also pay exaggerated attention to details, have strong obsessions, and islets of ability. Apparently, this impaired folk psychology goes together with superior “folk physics” capacities (i.e. they perform better at the embedded figure test, which consists of 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 occurrence of high-functioning autism is biased towards males in a proportion of 9:1. In view of these studies, Baron-Cohen has hypothesized that autism might be a form of “extreme male brain”. In his view: “... 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” (Baron-Cohen 2000, p. 1254).

**(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 what Segal called “diachronic modules” (1996). In order to function organisms must first construct an “implemental adaptation” or “synchronic module” that characterizes the adult cognitive make-up, and then it must calibrate it. Dysfunction can result from the absence of the 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 output from that mechanism results in the lack of development of a ToMM that depends on that output for its development. Dysfunctions can also result from a miscalibration of a mechanism due to its exposure to an atypical environment. Cosmides and Tooby offer an example of such a 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; for the idea of periconceptual, fetal and infant programming, see also Stein 2006, 769 and Meaney & Szyf 2005 for a spectacular example in rats).

#### 7. TREATABLE CONDITIONS THAT ARE NOT THE RESULT OF A DYSFUNCTION

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

**(1) Evolved defense:** ‘Evolved defenses cases’ are sometimes confused with dysfunctions because they may cause pain or discomfort. However 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 sometimes seek 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 return to this notion in Section 8.2, using "normal depression" as another possible 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 that in which it has been selected to work (especially in new environments where the cues that once indicated fitness benefits no longer indicate them). Nesse and Berridge refer to drug abuse as examples of such cases: "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 & Berridge 1997a). If drug abuse is a case of environmental mismatch, individual variations in susceptibility to drug addiction are better understood as quirks instead of as defects because they probably had no deleterious effects in ancestral environments, they only appear in modern environments where drugs are more easily accessible.

## 8. HOW THE EVOLUTIONARY APPROACH MODIFIES THE WAY TO CONCEIVE OF SOME TREATABLE CONDITIONS

In this section, I would like to examine two examples of conditions that are generally thought of as the result of a single kind of etiology. I will suggest that the evolutionary approach provides 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 will discuss are also useful in that they further our list of conditions that arise 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, and named this mechanism "Violence Inhibitor Mechanism" (VIM). Blair explained that the mechanism plays a crucial role in the explanation of psychopathy. Despite what has been hypothesized before, he postulates that the problem of psychopaths has nothing to do with their capacity to read another's 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 (1995) has shown that despite the fact that autistics have enormous difficulty reading other's minds, they still develop moral emotions and the so-called moral/conventional distinction (a distinction that adults and children are argued to spontaneously make between different kinds of transgressions) that psychopaths are unable to develop. For that reason, Blair believes that the core symptoms of psychopathy (absence of guilt and remorse, lack of empathy, no inhibition of violent action) should be explainable by the breakdown of the VIM (in addition to some impairments in executive functioning. This clause would allow for undetected psychopaths who don't end up committing violent crimes, but who might nonetheless behave unusually. This subgroup would be constituted of psychopaths suffering from a VIM breakdown, but without impairments in executive functioning).

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. Yet 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 cognitive architecture is the same in every normal human being. This group proposes instead the image of a *polymorphic mind*, i.e. the idea that there exists more than one "normal" cognitive architecture. In this context, they ask if psychopathy could not be considered an adaptation rather than a disorder.

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 with very low frequency in a population of discriminative reciprocal altruists, because continually checking for cheaters is too costly. Ergo there would be a niche for that kind of individual (a psychopathic individual), which would explain the maintenance of the trait across generations. If this is right, the trait would thus be *frequency-dependent* (a type of balancing selection we talked about earlier, Keller 2008b, 3); psychopathy would be the result of a normally functioning mind. Mealey does not stop there; she posits that if a subgroup of those whom we call psy-

chopaths 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 environmental cues lead them to pursue a life strategy similar to those who are “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 diagnosed with psychopathy (secondary psychopaths can react to cues of distress for instance).

### 8.2. *The Case of Depressive Disorders*

As Kennair (2003) noted in a review of the field of evolutionary psychiatry, “The disorder that has received most attention recently from an evolutionary perspective is depression: most of the key researchers within EPP [evolutionary psychopathology] are involved in the study of this disorder. Within the review period covered here, papers on depression stand out as most ground-breaking and probably provocative ...” (693). Though seven years have passed since this statement, I believe it is still accurate. In the last several years there has been a flurry of papers about depression from some of the main advocates of evolutionary psychiatry (Allen & Badcock 2006; Andrews & Thomson Jr 2009; Gilbert 2006; Keller & Miller 2006; Nesse 2009; Nettle 2004; Sloman 2008; Stein 2006). In what follows, I will focus my attention on two recent papers: Nesse (2009) “Explaining Depression: Neuroscience is not Enough, Evolution is Essential” and Andrews & Thomson Jr (2009) “The Bright Side of Being Blue: Depression as an Adaptation for Analysing Complex Problems”. Despite sharing the common framework of evolutionary theory, evolutionary psychiatrists who try to explain depression can be divided by the positions they take as to the adaptative character of depression, and the evolved domain of mechanisms involved in depression. More precisely, the evolutionary explanation of depression can be divided in two camps: one camp holds that depression is dysfunctional and that its domain is non-essentially social; the other one holds that depression is functional and that its

domain is social. Though each camp has many representatives, for the sake of space, I’ll focus on only two of them.

In his “Explaining Depression: Neuroscience is not enough, evolution is essential” (2009), Nesse argues that “... serious depression is not an adaptation shaped by natural selection. It has no evolutionary explanation. However, we do need an evolutionary explanation for why natural selection left us so vulnerable to a disease as common and devastating as depression. Some abnormal depression is related to normal low mood, so explaining the origins and functions of mood is an essential foundation for understanding depression” (21). Thus, an evolutionary perspective does not commit one to supposing that depression is an adaptation, but it highlights the necessity of explaining why we are vulnerable to it. It also grounds its explanation in the dysfunction of an otherwise functional mechanism, a mechanism in charge of what Nesse calls “low mood”. Since low mood is crucial in the explanation of depression, let’s say a few words about it.

Nesse’s theory of mood is based on a functional theory of moods and emotions. Nesse asserts that (for a statement of his position, see Nesse 1990 and more recently Nesse 2009) emotions and moods are organized adaptative responses to recurrent problems of our ancestral environment. As he puts it: “Mood regulates patterns of resource investment as a function of propitiousness” (2009, 24). Negative emotions and moods are responses to situations of threat, loss, or situations where costs and risks are greater than benefits. More precisely, low mood would be elicited by cues indicating a loss of resources of adaptative significance: “The losses that cause sadness are losses of reproductive resources... A loss signals that you may have been doing something maladaptive” (Nesse & Berridge 1997a, p. 9). The type of losses that cause low mood can be “somatic resources (personal health, attractiveness and ability, and material resources), reproductive resources (a mate and offspring), and social resources (allies and status” (Nesse 2009, p. 27). So, low mood can be triggered by the sudden loss of your pension fund, parental death, the loss of a loved one after a departure or a breakup, the loss of friend after a fight, the loss of social status, etc. The pattern of behavior characteristic of low mood reinforces the idea that it has an adaptative function. Following Klinger’s (1975) seminal work, Nesse proposes that low mood

functions in two steps: “When efforts to reach a goal are failing, low mood motivates pulling back to conserve resources and reconsider options. If conditions do not improve and no other strategy is viable, low mood disengages motivation from the unreachable goal so efforts can be turned to more productive activities. If an individual persists in pursuing an unreachable goal, ordinary negative affect can escalate into pathological depression” (2009, 23).

Note that in this theory, depression is not caused by stress or anxiety, but rather by the inability to disengage from an unreachable goal.<sup>19</sup> I will return to this later, but Nesse posits that stress or anxiety is caused by the fact that one is not able to reach a specific goal in the current environment (for instance, not finding happiness in a relationship).

Recently, Nesse (Keller & Nesse 2005; Keller & Miller 2006; see also Keller et al. 2007) has suggested that selection might have shaped different subtypes of depression to deal with different types of problems. This prediction resulted from the “situation-symptom congruence hypothesis” according to which symptoms should be adapted to deal with adaptative challenges characteristic of the different type of situations. According to the studies he conducted, bereavement and romantic breakups are associated with different symptoms than chronic stress and failures (sadness, anhedonia, appetite loss and guilt in the first and fatigue and hypersomnia in the last).<sup>20</sup>

As mentioned earlier, the adoption of evolutionary perspectives is motivated not only by new testable hypotheses that one can derive from it, but also by the possibility of explaining general as well as individual vulnerability. At the present time, there is no accepted explanation of general vulnerability: Nesse mentions the possibility that we might live in a depressogenic world (where goals are often times unrealistic), or that new physical factors like artificial light, changes in exercise or diet might influence brain mechanisms responsible for depression. Other explanations might make reference to the fact that traits such as low mood tend to have a high variance between individuals, so much so that some might be at the pathological extreme of the spectrum. As for individual vulnerability, as stated earlier, there is a genetic polymorphism on the 5-HTT gene that increases the risk of depression. Though there is no current hypothesis concerning the possible benefits of having the S 5-HTT, an evolutionary perspective

suggests that there might be benefits linked to certain circumstances, which might motivate research in this direction. Nesse also mentions the fact that exposure to repeated episodes of stress might lower the threshold of low mood until it becomes pathological.

In “The Bright Side of Feeling Blue” (2009), Andrews and Thomson propose what they call a “social navigation hypothesis of depression”. Their hypothesis belongs to a family of models that put forward the role of depression in social relationships as well as asserting its functional nature (I am not arguing that all social theories of depression advocate for an adaptative view of depression; see for instance Allen & Badcock 2006). Before turning to their model, I present quickly some of the other models belonging to that family.

The first model is called the “social competition” or “social rank” theory of depression. Price and colleagues (1997) advocated this position, suggesting that depression is an “involuntary subordinate strategy” (sometimes also called “involuntary defeat strategy” or IDS, Sloman (2008)) that evolved from mechanisms mediating ranking behavior. Price and colleagues state that depression has three functions: (1) preventing an individual from making a costly comeback when their defeat in a hierarchical struggle is inevitable; (2) sending a “no threat signal” to dominant individuals; (3) placing the individual in a state of yielding which encourages acceptance of the outcome. As Sloman puts it: “It is exquisitely designed to influence the individual to give up certain aspirations such as winning the affection of a possible mate, or to end a confrontation. It can lead to submission, the development of more realistic goals and a redirection of energy towards more productive pursuits.” (2008, 221).<sup>21</sup>

Note that, for the authors, depression is not always adaptative (one might wonder if these researchers shouldn’t have distinguished low mood from clinical depression). As Sloman recently put it: “In general, depression and anxiety are adaptative when they are switched off early before they become too intense. Because a mechanism that is proving ineffective in coping with agonistic conflict tends to become more entrenched which makes it more difficult to switch it off and the continued action of the mechanism may lead to a maladaptative cycle of escalating depression or anxiety.” (2008, 222).

A second model is the “depression as bargaining model” proposed

by Hagen (1999; 2002). In short, depression is seen as a sort of strike, i.e. it is a way for an individual to say that they are not accepting the terms of relationship anymore, and that they are demanding a better treatment. As Hagen puts it: “When simple defection from a costly cooperative venture is socially constrained because, for example, each participant has a monopoly on essential resources or can impose costs on defection, individuals suffering net costs from their participation may benefit by withholding the benefits they are providing until better terms are offered, that is, they may benefit by bargaining or ‘going on strike’” (2002, 324). Hagen has tested his theory using postpartum depression as a model for depression in general. This decision allows one to make a number of predictions. Among those: (1) individuals with no other children and few future chances to invest in offspring (those who have everything to lose) should have lower level of PPD; (2) individuals who for social reasons (social norms on abortion, for instance) are forced to have unwanted children should experience higher level of PPD (new costs are imposed on the individual who may want to renegotiate his or her current arrangement); (3) PPD in one spouse should be associated with increased parental investment by the other spouse. All these predictions were confirmed according to Hagen.

Andrews and Thomson’s theory has a family resemblance with Hagen’s; like him, they view depression as a form of strategy to extort increased investment from others. Their theory also tries to explain the cognitive features of depression, which Hagen’s theory leaves unexplained (Watson & Andrews 2002, 3). Using both Andrews and Thomson’s recent paper, and Watson and Andrews (2002) earlier position statement, I’ll present their explanation of these cognitive features (on which they put much more emphasis in their recent paper), after which I’ll return to the social motivation features of depression.

According to Andrews and Thomson, depression is “an evolved stress response mechanism” (2009, 621). More precisely, its function is to deal with two classes of problems: social dilemmas and avoidable stressors.<sup>22</sup> The authors state that these problems are complex and should be addressed in an analytical way, in that they must be broken down into smaller pieces to be resolved.<sup>23</sup> Thus, if depression is designed to help resolve these kinds of problems, it must “promote an analytical reasoning style in which greater attention is paid to detail

and information is processed more slowly, methodically, thoroughly, and in smaller chunks” (idem, p. 622). This is exactly what most features of depression can be thought of as doing.

Andrews and Thomson state that the central designed feature of depression is rumination, which can be seen as an analytical and methodological way of considering complex problems whose goal is to generate and evaluate solutions to these problems. This is consistent with studies that show that depressive thinking is more analytical in nature, and is focused on “regretful thoughts”, i.e., focused on understanding why an episode happened and what could have been done to avoid it (Andrews and Thomson call this ‘upward counterfactual thinking’). Other features of depression might also be considered as adaptive, to name a few:

- Depressive individuals tend to attribute more of their failures to their lack of ability and more of their success to chance while non-depressives show the inverse pattern. Due to this, some refer to a ‘depressive attributional style’ (2009, 636). This style makes them less prone to the fundamental attribution error.
- Negative mood also seems to lead to more accurate decisions related to complex situations, but also to conservative implementation strategies for these decisions than those in positive moods.
- In certain complex situations, depressed individuals are better than non-depressed individuals at estimating the control they exert on a situation (2009, 639).
- “[...] depressed people are more sensitive to costs of cooperating than nondepressed people and are more likely to defect when it is costly to cooperate” (2009, 634).

The other features generally associated with depression, such as anhedonia and psychomotor changes, sleeping and eating dysfunctions, would be mechanisms that contribute to ensuring undisturbed rumination. For instance, anhedonia would assist rumination by making the individual indifferent to pleasures that could distract them from problem solving. Preference for solitude (a psychomotor change) would also help the depressive individual by promoting avoidance of social

contact that can be cognitively demanding. This idea predicts a relationship between rumination and anhedonia so that need for more rumination should produce more intense anhedonia. In the case of psychomotor changes, it predicts that if an environment is conducive to rumination, lethargy will work to keep the individual in that environment, but if the environment is not conducive to rumination, it will motivate them to look for superior environments (and lead to agitation).

What makes their “analytical rumination hypothesis” interesting (ARH; idem, p. 623) is the idea that since most cognitive resources are devoted to solving the complex problems that triggered depression, there are none left for other non-related tasks. This would explain the poor results of depressive individuals on laboratory tasks. Indeed, when distracted from thinking about their problems, depressive individuals’ performances on memory or executive control tasks are similar to non-depressive individuals, while they are impaired otherwise. Contrary to what has been traditionally proposed on the basis of laboratory task results, depressive cognition is not dysfunctional, rather it is perfectly tailored to solving a specific kind of problem.<sup>24</sup> For instance, analyzing problems requires using working memory (WM). Due to the gravity of the problem considered by the depressive individual, all resources should be devoted to the problem; therefore irrelevant tasks that tap WM will show poorer results. These poorer results are not explained by a dysfunctional WM, but rather by the fact that this structure has limited resources and is impervious to disruptive conditions; in other words, it is distraction-resistant (this may be achieved via attention control structures, as suggested by increased activity in the left VLPFC<sup>25</sup>).

ARH makes four claims:

- (1) Complex problems trigger depressed affect;
- (2) Depression coordinates changes in body systems that promote sustained analysis of the triggering problem;
- (3) Depressive rumination often helps people solve the triggering problem;
- (4) Depression reduces performance on laboratory tasks because depressive rumination takes up limited processing resources.

Now a few words about the motivational aspect of depression. Previously, Sloman, Gilbert and McGuire and others think that the function of depression is to send a “no threat” message to social dominants. This would function to reduce aggression towards the depressive individual. Andrews and Thomson makes a different claim. In their view—and consistent with Hagen’s position—depression is used as a means to gather social support either by honestly signaling need (in this framework, suicidality can be seen as adaptative: “As an honest signal, the risk of death associated with a suicidal attempt could inform partners about the attempter’s level of need” (9)) or by motivating fitness extortion, that is, in showing that one is ready to inflict costs onto themselves and others, in order to acquire more support or a new social role. A prediction that follows from this model is that depression should end when support is gathered.<sup>26</sup> It also predicts that depression should generate more support from closer social partners than distant ones. Finally, it predicts that depression should increase when one is taken away from their social milieu (for instance, by being hospitalized).

So because depression is conceived as an adaptation to solve a specific kind of problem, “... performance on the triggering problem [should be considered] as a crucial metric for evaluative depressive cognition. [...] the conclusion that depression impairs social skills depends on accepting the notion that some behaviors, such as friendliness and cooperation, are always better for social problem solving, regardless of the situation or context. A more direct definition of social competence is simply the ability to achieve social goals, especially in situations of social conflict.” (637). In other words, what appears as cognitive and social malfunctions might indeed be functional (but disvalued) ways to achieved adaptative goals.

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As we have seen in this section:

- (1) Evolutionary hypotheses about the same disorder can diverge.
- (2) Specifically concerning depression: some hypotheses might explain depression as a disorder resulting from vulnerabilities (i.e. Nesse’s); while others might defend what Murphy labeled as a “persistence explanation” according to which putative disorders

are indeed adaptive in current environments (i.e. Andrews and Thompson's); while finally others revert to mismatch explanation (as in the case of SAD).

- (3) One has to distinguish between evolved defense and disorder. The symptoms of depression function similarly to pain, fever or cough: they are normal symptoms that have a function of defending the body against some harmful stimuli or infection. They might sometimes be dysfunctional, but in order to determine this, one needs to know if the stimuli or virus are present, and if the response is proportional to the danger (i.e. in the case of chronic pain, it appears that the pain response is unrelated to anything in the environment). To distinguish between evolved defense and disorder in the case of depression, one needs to know if an individual's response is disproportionate to its cause. Here the "cause" is not an objective feature of the environment, but a construct that depends in large measure on the subject's motivational structure (what are their goals, how they assess the situation, etc.). Depression could be normal response to a life event, therefore it would be a mistake to establish a diagnosis based only on the presence of symptoms.<sup>27</sup>
- (4) One interesting proposal made by Nesse is that depression is a generic term which encompasses different types of responses to loss. Each type of response is tailored to the problem it tries to solve (loss of romantic partner, loss of social position, etc.). Accordingly, Andrews and Thompson's view could be seen as describing one subtype of depression.<sup>28</sup>
- (5) Being aware of the "ecological function" of the symptoms of depression (or low mood) helps in the design of "ecologically valid" experiments to test their adaptativity.

#### 9. PROBLEMS WITH THE EVOLUTIONARY APPROACH TO SPECIFIC MENTAL DISORDERS

In this section, I will briefly present two problems for the evolutionary approach to psychiatry (for more see [Murphy & Stich 2000](#); [Murphy 2006](#); and [Faucher in preparation a](#); [in preparation b](#); [Faucher &](#)

[Blanchette 2011](#)). These problems do not affect the general framework, but rather particular hypotheses about specific mental disorders.

As stated earlier, for evolutionists, psychiatry's destiny is linked with progress in evolutionary psychology. The first problem relates to some of evolutionary psychology's limits. Evolutionary psychology's central theoretical commitment, which allows the use of both of its methods (i.e. adaptive thinking and reverse engineering), is to the existence of a strong relationship between biological form and adaptive forces. Without such a relationship, there would be no reason to expect that isolating adaptive problems will be of any help in discovering the architecture of the mind, or that starting with known mechanisms will lead to the reliable discovery of adaptive pressures that have acted on them in the past. As [Griffiths \(1996\)](#) observed, "... adaptive generalizations ... cannot explain form except in conjunction with a rich set of historical initial conditions" (515). In short, to say that a trait or a mechanism is an adaptation is to make an historical claim to the effect that the presence of the trait in actual population is explained by the fact that it was more adaptive than others in a past environment. More precisely, an adaptation is relative to:

- 1) Traits that were present at the same time at the moment of selection;
- 2) A particular selective regime (environmental pressures).

Adaptations are usually identified using the comparative method, which consists of comparing a trait to the ones from phylogenetic ancestors, and to prevalent environmental conditions. By using this method, it is shown that

- 1) A trait had not given an adaptive advantage to its bearer over others who didn't have it, therefore it can't be an adaptation;
- 2) 2) If the trait appears in the phylogenetic ancestors of the actual bearers before, or independent of being exposed to the new selective regime, it can't be an adaptation to this particular regime.

What this reveals is that to be able to use the comparative method, information is required about a number of things: (1) traits that were

present at the same time at the moment of selection; (2) traits possessed by the immediate ancestors of the bearer of the studied trait; (3) the particular selective regime under which selection has taken place.

Sometimes we do have the information needed to establish that a human trait (the one that psychiatry is interested by) is an adaptation: for instance, we have access to traits that were present in our non-human primate ancestors or to traits which vary according to certain features of the evolutionary environment of adaptation (as in the case of malaria resistance, AIDS resistance, skin color or lactose tolerance). The problem is, for many specifically universal human adaptations, there is not the evidence necessary to establish that a trait is an adaptation. As Kaplan put it: "I argue that such evidence is rarely available in the case of purported 'universal' human psychological adaptations. The very limited information available on the environments in which key aspects of human evolution took place makes optimization techniques difficult to apply here. Further, while in some cases phylogenetic information about *Hominidea* may provide evidence relevant to adaptative hypotheses in humans, nature and history have 'conspired' to make the task more difficult with humans than it is in many other species" (2002, 297).<sup>29</sup>

What this means for evolutionary psychiatry is that it will be possible to establish the evolutionary functional criteria for some mechanisms that evolved before the *Homo* genus. For instance, if Price and colleagues are right about depression,<sup>30</sup> we should be able to use the comparative method and establish that the mechanism is an adaptation. Similarly, in principle it should be possible to establish the adaptative character of traits that vary inside the human population. The case of psychopathy is an example of a trait where the comparative method could be used (even if it seems that there are some obstacles to its application, as noted by Murphy 2005, 759). For some mechanisms (the number of which has to be empirically determined<sup>31</sup>), we might just never know the facts necessary to establish that they are adaptations (for similar claims, see Richardson 2007, 38).<sup>32</sup> Therefore, judgments about their dysfunction will be based on hunches about what is normal or abnormal; as the past amply demonstrates, (in cases of masturbation, homosexuality, or female orgasm), hunches are unreliable, as they are especially open to the influence of values and norms.

The second problem involves the fact that, as Murphy puts it, evolutionary psychiatry is "unfaithful to the psychology [it tries] to explain" (2005, 746). In short, evolutionary psychiatry sometimes explains an aspect of a disorder or a particular symptom, leaving aside other aspects or symptoms. Murphy highlights this particular failure when he writes: "One of the besetting sins of evolutionary psychology is the tendency to take some crude characterization of a human capacity and try to explain that" (idem, 762). Take the case of depression from the previous section: Nesse as well as Andrews and Thompson are trying to account for a number of aspects of depression or low mood. But they leave other aspects unexplained. I'll consider a few of them in the following section.

Kendler's work (Kendler et al. 2006, 2009) suggests that there are at least three, maybe four, major pathways that lead to depression (internalizing symptoms, externalizing symptoms, adversity and interpersonal difficulty as well as one linked with vascular disease in late age onset patients). Many of these pathways (except the last one) include events that took place in early childhood. Given this, one might be tempted to say there might be different subtypes of depression. Among these subtypes, some might be adaptative, some not (because they are vestige, like SAD, or because they are the result of non-adaptative random genetic mutations), some might be the result of the cognitive malfunction of adaptative mechanisms specialized to deal with some problems (due to social factors, like poor parenting or abuse), while others might be the result of brain structure breakdowns that affect mood. Keller and Nesse's (2005; 2006) proposition heads in the right direction by identifying two subtypes of depression. The problem is that they focus exclusively on types of depression linked with adversity or interpersonal difficulties, which according to Kendler, represent only a subtype of depression; they owe an explanation of other forms of depression. By focusing only on the resolution of complex social problems, Andrews and Thomson's theory can be seen as the explanation of only one subtype of depression, so it leaves unexplained other types of depression, as well as depressions resulting from other pathways.

Murphy (2006) remarks that many symptoms of major depression are left unexplained by Nesse. Why exactly does the breakdown of the low mood mechanism generate loss of sleep or inability to make deci-

sions or concentrate? Further, why is the disengagement mechanism unaccompanied by a positive affect or motivational structure of some sort that would cause the behavior to change?

This idea has precedence in the literature. For instance, animals in conditions featuring severe food restrictions will increase, not decrease their energy expenditure and increase their risk-taking behavior. Indeed, in a recent paper [Nettle \(2009\)](#) uses optimal-foraging models which suggest that Nesse is right: “when things are going quite badly, it is not time to take risks, but as things improve, greater experimentation is warranted” (3). However, the model also predicts that “... there comes a dire point beyond which it is maladaptative to avoid risks and conserve energy: the situation is already too dangerous for that. Instead, the individual should be highly motivated to take risks and try new solutions; to do anything that has any chance of returning her to the acceptable range of states” (3). Nettle notes that this state might be found in patients classified as depressive because of their negative affective tone, but whose symptoms include locomotor acceleration and restlessness, and feelings of racing thoughts, as well as the desire to follow risky pleasurable impulses (this might be thought of as a form of “dysphoric mania”). What Nettle proposes is a further refinement of functional theories of the kind defended by Nesse. Adaptative responses in the case of loss of resources will be different as a function of the evaluation of the severity of the condition in which the individual finds himself or herself. As Nesse puts it, “[t]he mood responses to different types of situations will show different suites of design features that represent adaptative strategies in that context ... Thus, a mood representing a response to dire circumstances could involve simultaneous activation of negative emotion systems ... and behavior approach systems. Such a mood state would be like depression, in its negativity, but also like positive mood, in its energy and risk-proneness” (4). Here, it seems, it is still possible to salvage Nesse’s theory.

Andrews and Thomson’s theory fared slightly better in the sense that it incorporates all features associated with depression. Although I have not presented it here, it also explains why (and predicts in which situations, 2009, p. 645) people will attempt to escape pain generated by depression (a note: if individuals continued avoiding thinking about the situation, that would be an argument against their theory).

Their idea that features of rumination might be adaptative, and that the allocation of cognitive resources to social problems might impede non-relevant laboratory tasks is worth exploring. One problem with their account is the fact that, as Nettle pointed out, “all normal human beings have the capacity to feel physical pain [...] However, there is no evidence that all individuals have the capacity to become clinically depressed. Rather, it seems likely that most depression is the result of an inherited diathesis borne by a minority of the population” (2004, 93).<sup>33</sup>

Another problem with their proposal is the fact that they assume that the triggers of depression are social or predominantly social in nature. Here, one might wonder about the direction of causality. Is depression caused by social problems or are social problems caused by depression? Depression can cause marital problems, lack of social support, or defection of social partners, all of which are also identified as factors in depression. If depression is caused by social problems, how does it fair in getting additional support or new deals with cooperative partners? Hagen has provided data for PPD, but no such data are available for depression in general. For the time being, it does not fit well with the demobilization and the social withdrawal that characterize depression ([Allen & Badcock 2006](#), 818).

If the mechanism underlying depression is adaptative and is designed to solve social problems, why is it that, as per Keller et al. (2007), 70% of people who suffer from bouts of major depression will have at least another episode and 20% will develop a chronic condition (rate of continuous freedom from illness is very low, around 11% over 25 years; [Nettle 2004](#), 95)? What this suggests is, as Murphy notes, “if depression is an adaptation designed to make them [the depressive] function better in society, it is not working” (295). Indeed, once depression has achieved its function, shouldn’t it disappear? Why is it becoming chronic in 20% of cases? As [Nettle \(2004\)](#) and [Nesse \(2000a\)](#) observe, as depressive episodes continue (for third and subsequent episodes of endogenous depression), the triggers that are required to produce depression become smaller and less related to life events.

Andrews and Thomson also pretend that depressive individuals have cognitive features that facilitate the resolution of social problems. Yet

as Nettle (2004) points out, they also have cognitive features that might handicap them for this task: depressive individuals are slower and less accurate than control subjects when reading non-verbal social cues, they show impaired social skills, seem to be more realistic than others only when the normal population is unrealistically positive (and depressive individuals are unrealistic when the normal population is reasonably accurate) and "... depressives perform worse than controls on tasks designed to tap inter-personal problem solving skills" (96).<sup>34</sup>

Finally, Andrews and Thomson are not able to explain the comorbidity of anxiety and depression,<sup>35</sup> nor for that matter, the comorbidity of depression and hypomania. Nesse fares slightly better on this account because he at least tries to explain it, even if his account is not very convincing. He states that "like pain and fever, anxiety and depression are nonspecific symptoms that can be aroused by many different problems, so comorbidity and heterogeneity are to be expected" (2012, 5). What would be needed for this to be convincing are more precise predictions in regards to when such comorbidity is to be expected.

All in all, each explanation of depression explains some aspects of depression, but leaves others unexplained. One could defend evolutionary psychiatry by saying that the science is young and that one has to start somewhere in the explanation of mental disorders such as depression; with time, other symptoms of depression will eventually be explained. I believe that this is possible and I would not want to foreclose such a possibility. At this time however, we have to keep in mind that evolutionary psychiatry's conclusions are very provisional—more so than in other sectors of science—they are explanations in the making, and we should not draw too much from them for therapeutic purposes.

Compounded with the first obstacle, the second obstacle should make it obvious that evolutionary psychiatry is not ready to deliver the proverbial goods yet. At present, it can't offer well-confirmed theories, and it may never be able to produce such theories in certain cases, but it can play a heuristic function by changing the focus of current brain sciences, and by questioning traditional positions in this field (by introducing the idea of functional low mood that appears similar to depression, etc).

## 10. CONCLUSION

I will conclude this paper by recapitulating how well an evolution inspired psychiatry would fare in providing solutions to the four problems we identified in section 2.

Concerning our first problem, we have seen that there have been many attempts to provide a definition of mental disorder that would ground a psychiatric classification by using an evolutionary approach. Concerning the definition of mental disorder, we have explained the reasons why some have rejected Kendell's and Boorse's accounts (mainly because it was concerned with actual adaptiveness of mechanisms). We then turned to Wakefield's definition. We saw that there were problems with both the idea that the concept to be analysed is a concept shared by everyone (lay people as well as professionals) and with the proposed method of studying that concept (conceptual analysis vs empirical method). For a number of reasons presented in the paper, I think that it would be better to adopt a different attitude (a somewhat revisionary attitude) towards the definition of mental disorder. This attitude is in line with Boorse's proposal. I argued that for practical purposes we should use the notion of "treatable conditions" to refer to the conditions that are the object of psychiatric concern. For scientific purposes, we might want to use a notion of function inspired by evolutionary theory to single out psychological mechanisms that are the source of psychiatric problems. But one has to be aware that psychology did not and does not need such theory to single out the mechanisms it studies. Therefore, one will have to provide an argument concerning the advantage of adopting an evolutionary perspective (I think that there is at least a heuristic advantage to doing so).

How does the evolutionary approach answers Szasz' concern about psychiatry? Well, first 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 dysfunction or disorder of computational devices or mental organs that constitute 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.<sup>36</sup> What the evolutionary perspective to disorder suggests, however, is that we cannot merely identify disorders by looking at the brain. Rather, we need to know what the (normal)

functions the brain is supposed to carry out are before being able to make such an identification. And as we have seen, minds meeting their evolutionary criteria of functioning are not always producing behaviors that we value (as the case of primary sociopathy illustrates). The problem, as I have shown in section 9, is that evolutionary psychiatry might never be able to provide a theory of normal function for a certain (limited) set of traits. If such is the case, the door is wide open to a predominant role for values and norms in diagnosis and for the unfortunate consequences that Szasz was preoccupied with.

As for the third question, it seems clear to many 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). As we saw, this image of the normal mind has the particularity of taking into account the features of the environment that provoke certain reactions that are the object of psychiatric preoccupation (for example, a depressive reaction or a hallucination). Symptoms of depression can thus be elements of a normal reaction in certain contexts, just as pain might be a normal reaction in response to certain nociceptive stimuli. And as it is not by looking at the brain mechanisms underlying pain reactions that one knows if it is or is not normal, it is not by looking at the brain mechanisms underlying the depressive symptoms that one will know if it is a normal reaction or a pathological one.

If evolutionary psychology’s picture of the mind is accurate, it is possible that the image of the architecture of the normal mind might be more complicated than has been first thought. First, as we saw in section 7, evolutionary psychology claims that it can remove the blinders of our imagination when the time comes to postulate psychological mechanisms. Second, 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 work of evolutionists makes it clear that we are dealing with a polymorphic mind (see for instance Kimura (2000)).<sup>37</sup> But again, this revolutionary potential of evolutionary depends ultimately on its capacity to deliver the goods, that is, its capacity to validate the

existence of the mechanisms it postulates and their functions. And as section 9 showed, this is not only difficult, it might also be impossible in certain cases.

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, homogeneous (for instance, primary and secondary sociopathy or depression caused by romantic breakup and by the death of a loved one). 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 et al. 1997a, 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 et al. 1997a, 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, 35). But if evolutionary psychiatry is to play such a role, it has to be true to the psychology it tries to explain. And as Murphy claims, and as I tried to show, this is not always the case.

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A few years ago, 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, but as this paper has made clear, it is not at all clear that, or how, these renegotiations should also include the insights provided by the evolutionary approach.

### Notes

<sup>1</sup> Evolutionary psychiatry is sometimes also called “evolutionary psychopathology” (Kennair 2003), and “darwinian psychiatry” (McGuire & Troisi 1998).

<sup>2</sup> See Adriaens & De Block (2010) who refer to these older versions of evolutionary psychiatry as “psychiatric darwinism”. Their intent is to distinguish a tradition which used Darwin’s idea, but was imbued by non-darwinian ideas, like Lamarckism or degeneration theories, from the more recent enterprise that has its roots in contemporary biology and cognitive sciences. For a well worked-out example of the influence of evolutionary theory on psychiatry, see Sulloway 1992.

<sup>3</sup> The other classification system of mental disorders is the chapter 8 of the *International Classification of Diseases* or ICD. Many of the problems affecting the DSM are also affecting the ICD. For the sake of space, I will focus my attention on the DSM only.

<sup>4</sup> 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).

<sup>5</sup> 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.” (1978, p. 92)

<sup>6</sup> As Nesse and Stein illustrate this point by using the following telling example: “... the DSM-II definition of Depressive Neurosis was: ‘An excessive reaction of depression due to an internal conflict or to an identifiable event such as the loss of a love object or cherished possession’. Is depression after the loss of a favorite cat ‘excessive’? One diagnostician would say, ‘Yes’, another, ‘Obviously not!’ Such unreliability made research impossible, and psychiatry’s scientific aspirations laughable” (2012, p. 1)

<sup>7</sup> According to Schaffner, “this approach typically involves a search for neuroanatomical, neurophysiological or molecular genetic factors, and thus tends to be reductive or reductionistic” (2002, p. 222).

<sup>8</sup> The hope to isolate discrete entities that correspond to the DSM’s diagnostic categories starts to wane as research moves forward. As Hyman puts it: “Insights emerging from genetics and, increasingly, from neuroscience suggest that the exclusive use of categorical diagnoses and the predominant ‘splitting’ approach of the DSM-III and DSM-IV represent obstacles to the near-term development of a more scientifically and clinically satisfactory classification” (2011, 3). For an example of results coming from genetics and neurosciences that suggest problems with current classificatory assumptions, see Happé et al. 2006.

<sup>9</sup> Recently, Wakefield defined a bit more precisely ‘mental’ in ‘mental disorder’: “Mental dysfunctions are not specific mental states but rather dysfunctions in the brain mechanisms designed to produce or regulate mental states, and the dysfunction emerges in irregularities in the production and the regulation of mental states” (2007b, 127).

<sup>10</sup> 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 or the society in general?

<sup>11</sup> Neander (1995) notes rightly that this notion of normativity is neither evaluative nor 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).

<sup>12</sup> 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 is possible that we are using “function” to talk about the “current adaptivity” of behaviors, that we are using the probable future selective success rather than past historical success as a way of establishing functionality.

<sup>13</sup>A similar move is proposed, for different reasons, by Bentall who suggests to “[abandon] psychiatric diagnoses altogether. Instead of attempting to explain mythical diagnostic entities, we should try and explain the actual complaints that patients bring to the clinic, such as hallucinations, delusions, disordered communication and mania” (2006, 224). Bentall’s proposal is not a reaction to conflicting intuitions concerning mental disorders but rather to the rejection of the belief in disorders as “discrete entities”. He thinks that psychiatry should be concerned by cognitive endophenotypes rather than disorders.

<sup>14</sup> “[T]he brain is composed of many different programs, many (or all) of which will be specialized for solving their own corresponding adaptive problems. That is, the evolutionary process will not produce a predominantly general-purpose equipotential, domain-general architecture” (Tooby & Cosmides 2005, 17).

<sup>15</sup> “Human behavioral ecology applies the theoretical perspective of animal behavioral ecology to human populations, examining the degree to which behavior is adaptively adjusted to environmental (including social) conditions, emphasizing conditional strategies of the form “in situation X, maximize fitness payoffs by doing a; in situation Y, do b”.” (Smith et al. 2001, 128)

<sup>16</sup> Notice here that the use of evolutionary psychology leads psychiatrists to adopt a version of ‘cognitive neuropsychiatry’ or CNP (Halligan & David 2001). CNP “attempts to bridge this gap [between cognition and neuroscience] by first, establishing the functional organization of psychiatric disorders within a framework of human cognitive neuropsychology, and second, linking this framework to relevant brain structures and their pathology” (Halligan & David 2001, 209; see also Hohwy & Rosenberg 2005). Like cognitive neuropsychology (Shallice 1988), CNP starts with a picture of the mind as constituted by specialized information processing mechanisms (like a belief-formation mechanism, a face recognition mechanism, a control of action mechanism, etc.) and postulates that it is these mechanisms that break in particular disorders. The picture of the mind is informed both by our lay-theory of mental functions and by retroactions from psychiatry that sometimes force modification of our lay-theory. Evolutionary psychiatry is a version of CPN in that rather than starting with a lay-theory of mental functions, it starts with a picture of mental functions informed by the evolutionary theory (for instance by the kinds of problems that the mind has been designed to solve).

<sup>17</sup> According to a rather dominant evolutionary view about emotions, they “... are modes of functioning, shaped by natural selection, that coordinate physiological, cognitive, motivational, behavioral, and subjective responses in patterns that increase the ability to meet the adaptive challenges of situations that have recurred over evolutionary time. They are adaptations that are useful only in certain situations” (Nesse 2009, 129; see also Cosmides & Tooby 2005b).

<sup>18</sup> Pleiotropy might be one important key to the explanation of comorbidity as “... a few genes have extensive pleiotropic effects, but most genes are more limited in their effects on the phenotype. However, nearly all genes have some degree of pleiotropy. ... [certain studies show that in animal models, like *c. elegans* or the fruit fly] each genes ... affects on average four or five proteins ... [in other studies it has been found that] the number of traits affected per gene was about six or seven” (Stearns 2010, 770-771).

<sup>19</sup> Leahy’s (1997) sunk costs model suggests “that depression occurs when people persevere too long with behaviours resulting in low or diminishing rewards” (Allen & Badcock 2006, 816).

<sup>20</sup> Other subtypes might include seasonal affective disorder (SAD) which is a recurrent

depression associated with winter and which is characterized by fatigue, increased appetite, sleeping and carbohydrate craving. Nesse (2000a) make 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.

<sup>21</sup> This hypothesis has found support in studies made by Raleigh and McGuire who found that in vervet monkeys the highest ranking males (alpha) had levels of serotonin twice as high as other males (as Murphy (2006, 292) notes, this shift in serotonin levels associated with change of social ranks is also found in lobsters and crayfish). 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 humans (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 gave some antidepressants to a male randomly chosen, that individual becomes in every instance the alpha male (see also, McGuire et al. 1997a).

<sup>22</sup> Note that this is a move from Andrews’ previous theory where he stated that “[t]he functional domain of depression may be social complexity” (Watson & Andrews 2002, 4), in that depression is now not only exclusively devoted to solving social problems. Still, in their more recent paper, they suggest that “complex social problems may be the primary evolutionarily relevant trigger of depression in human beings” (626).

<sup>23</sup> The authors propose that their position implies the existence of a mechanism that distinguishes simple from complex problems (2009, 625).

<sup>24</sup> Indeed, the fact that depression seems so exquisitely designed for solving an adaptive problem suggests that it is an adaptation. As they put it : “ ... depression evolved by natural selection because there is a neurological orderliness that appears to specifically and proficiently promote analysis in depressive rumination and is not likely to have evolved by chance.” (2009, 623)

<sup>25</sup> Because sustained firing of neurons augments the chance of neuronal apoptosis (programmed cell death), Andrews and Thomson affirm that there should be a mechanism that reduces it. According to them, this mechanism involved an augmentation of the production of 5 HT. The idea that depression could be characterized by an increased production of 5 HT goes against common wisdom on depression, but the authors claimed to have evidence that it is the case. I won’t try to evaluate their claim here (but they would need to explain why another structure like the hippocampus that has a lot of 5 HT receptors is getting smaller).

<sup>26</sup> “Recovery from depression is hastened by improvements in social relationships and strong social support.” (Watson & Andrews 2002, 4)

<sup>27</sup> “In psychiatry, emotions sufficient in duration and intensity are categorized as disorders irrespective of the situation. This encourages treatment without investigating possible causes, on the assumption that anxiety and depression are abnormal” (Nesse & Stein 2012, 4).

<sup>28</sup> See Kennair (2003) for a similar idea: “... it may be important to consider these different explanations as different taxonomic types of functional depression, rather than as competing theories for all forms of depression” (694).

<sup>29</sup> The problems are of two kinds. The first kind concerns the variation present at the

moment of selection: "... we have no extant relatives which are suspected of sharing similar selective regimes and that can therefore be used to test the fitness consequences of the supposed adaptations. If all or most of the estimated dozen or so extinct hominid species ... still existed, phylogenetic studies would certainly be easier, and might well be useful for distinguishing between competing hypotheses about the spread and maintenance of phenotypic traits of interest. Unfortunately for testing adaptive hypotheses in humans, all the other hominids are extinct, and so comparisons between the groups, with special attention to the fitness consequences of differences in key traits, are impossible. The second one has to do with the selective regimes: "... we do not know where (in the world) the various key evolutionary innovations that led to *Homo Sapiens* took place nor when these innovations took place; given this, even if we knew what the environment was like at each place and in each time (which we don't), we still would not have sufficient information to use the comparative method" (Kaplan 2002, 297)

<sup>30</sup>If "those hen-pecked animals [Hamadryas baboons and long-tailed macaques] could talk and were allowed access, they would be queuing up at the Emergency Clinic to complain of anxiety, depression and widespread aches and pains" (2002, 532; quoted by Adriaens & De Block 2010)

<sup>31</sup>Some, like Downes (2009), have argued that evolutionary psychology has exaggerated the importance of the Pleistocene as the period where human adaptations have been shaped. As he claims, "... human behavior is a result of evolutionary processes both much older and more recent than the Pleistocene" (244). If such is the case, the prospects of evolutionary psychiatry are quite good.

<sup>32</sup>We might think here of schizophrenia. Indeed, Crespi and colleagues have shown that some of the genes that mediate the liability to schizophrenia have been selected in a recent past. But when (it appears that they have not been selected at the same time) and in response to which selective pressures were those traits selected, this is unknown and it might well stay unknown as we might never have the relevant information to establish it.

<sup>33</sup>Nettle (2004) also notes that depression is not like pain. While there are individuals who do not have pain (congenital anesthesia) and have reduced life expectancy, there is a bunch of people who do not have depression and who do not suffer a reduction of their life expectancy. Indeed, it is rather those who suffer from depression that have "impaired psychosocial functioning, excess of mortality and poorer physical health than those who do not ..." (97)

<sup>34</sup>As Allen & Badcock (2006) observe: "... although some recent studies have shown that mild depressed states facilitate both social reasoning and performance on theory of mind tasks, other studies using the same assessment procedures have found that in clinical populations, these advantages are absent or even reversed" (822). So the jury is still out on this.

<sup>35</sup>Nesse (2009) explains the comorbidity in saying that the problems that trigger depression sometimes also demand greater vigilance and thus trigger also the threat systems (the question is thus, why do these two systems break so often together?).

<sup>36</sup>Though it might not be a lesion, like Szasz wanted. Many advocates of evolutionary psychiatry think that psychiatric problems can be like glitches in a program, that is, something that you can't see by looking at the hardware, but that affect the performance of the computer (Wakefield 2007b).

<sup>37</sup>It is possible to think of conflicts between different kinds of minds or variants of

the mind arising from the fact that they do not have the same (adaptive) 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.

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