

The Problem of Defining Psychopathology and Challenges to Evolutionary Psychology Theory

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One of the major domains of individual differences is the study of psychopathology—mental disorders. In addition to differing on personality traits and mental ability, individuals also differ to what degree they have or are susceptible to develop mental disorders and the form the mental disorder takes. Like psychological research in general, research on psychopathology suffers from the lack of an overarching evolutionary and functional approach that would “carve nature at its joints” (Buss, 1995). The lack of understanding of the nature of psychopathology has resulted in diagnostic manuals such as APA’s DSM and WHO’s ICD, both of which attempt to define categories based on descriptive research in order to free diagnosis from theoretical, historical baggage.

Clinical psychology and psychiatry are important applied disciplines that are responsible for the treatment of the many who suffer from mental disorder and psychological pain. Alas, the field of mental health care is often divided into camps; “mindless” biopsychiatry that reduces almost every symptom to a neurotransmitter or brain disorder, and “brainless” psychotherapy that attempts to keep pet theories and practices alive despite scant empirical support. There is a need for integrative, biopsychosocial, and empirically supported approaches that address both symptoms and the interpersonal, therapeutic relationship. One major problem is that the conditions that are treated are poorly understood. An evolutionary approach might be able to help integrate the many different levels of analysis (Gilbert, 1995,

1998; Kennair, 2003; Nesse, 2002, 2005) and might also help us understand the functions of certain phenotypes or the malfunction of others (Troisi & McGuire, 2002). Unfortunately, in general the broad set of theoretical approaches within current evolutionary psychopathology has been too diverse to assist cumulative theory building.

Evolutionary psychology has already shown a great integrative potential within psychology (Buss, 1995; Kennair, 2002). This integrative power is also evident in bridging biology, the social sciences, and the humanities (Buss, 2005). This is due to the multi-disciplinary relevance of evolutionary theory, the cross-disciplinary approach of evolutionary psychology, as well as the broad relevance of considering the psychological level of analysis (the cognitive mechanisms behind human universal information processing).

The present chapter considers how an evolutionary psychology approach may assist the development of a scientifically based, general definition of psychopathology, as well as help integrate the disparate field of evolutionary psychopathology. The latter goal will require changes to mainstream evolutionary psychological theory so that it will encompass the major phenomena and symptoms of psychopathology. These changes will therefore be discussed. In so doing, I will not address specific disorders *per se*, but rather focus on psychopathology in general and how an evolutionary psychology approach may provide the theoretical foundation for defining the nature of mental disorders. Throughout the chapter examples from different mental disorders will be used to illustrate general principles.

Evolutionary psychopathology has been subject to a most exciting surge in both original and fruitful theoretical and empirical work the last few decades (see Kennair, 2003, for a review). However, the different, competing approaches, hypotheses and theoretical foundations make this a heterogeneous field. Scientists with different theoretical orientations within evolutionary psychopathology argue for all the possible positions in discussions of the theoretical underpinnings of evolutionary psychology. Such include: whether adaptations are identified through formal analysis vs. assumed at the outset; whether the adaptations should be the focus of research vs. adaptiveness (i.e., behavior as maladaptive or functional in the current ecology); whether the mind is a set of specific mechanisms or modules vs. a general problem solver; whether selection has designed species-specific adaptations vs. comparative studies are fundamental; and whether human universals are the appropriate unit of analysis vs. individual differences are genetic and adaptive, etc. (see also Buss & Reeve, 2003; Gangestad & Simpson, 2007; Hagen, 2005).

To overcome the lack of cumulative research, theory building, and integration of findings within evolutionary psychopathology, an overarching meta-theory is needed. Evolutionary psychology is the research program of

choice, in part due to the focus on cognitive, functional adaptations (Cosmides & Tooby, 1999). The evolutionary psychology research program (Buss, 1995; Tooby & Cosmides, 2000) is therefore able to map both functions and dysfunctions (Baron-Cohen, 1997)—laying the foundation for a harmful dysfunction analysis of pathology (Wakefield, 1999).

WHAT IS PSYCHOPATHOLOGY?

Providing a general definition of mental disorder is not the only problem. There is neither full consensus on what behaviors, states, or syndromes are the best descriptions of specific mental disorders, nor on what disorders warrant inclusion. Each new edition of the diagnostic manuals may be considered progress as it reflects a continuous attempt at increasing the scientific foundation of diagnosis. However, an overarching understanding of how to carve the nature of psychopathology at its "joints" (i.e. delineate discrete functional units) (Buss, 1995) and what phenotypes are most relevant, is missing from modern mental health nosology. In the meantime, subjective, individual suffering (e.g., depression) is classed together with intersubjective assessment of nuisance (dissocial personality disorder). Also problematic are the many value systems that define the harmfulness of these states (including society, culture, individuals, and scientific theories of varying quality). Some disorders are therefore only reluctantly recognized (e.g., panic disorder), while others are only grudgingly removed from diagnostic manuals (e.g., the dissociative disorders or homosexuality). There is therefore an obvious need for an overarching understanding of the nature of psychopathology, which may be provided by evolutionary theory (e.g., Kennair, 2003; Nesse, 2005). But as Wakefield makes clear, we cannot escape the value factor.

Although there are many attempts to define psychopathology (also from an evolutionary perspective; Cosmides and Tooby, 1999; Troisi and McGuire, 2002), the most influential and debated contribution is Wakefield's (1999, 2007) concept of the *harmful dysfunction*, which he (2007, p. 149) calls a hybrid account:

According to the [Harmful Dysfunction] analysis, a disorder is a harmful dysfunction, where "harmful" is a value term, referring to conditions judged negative by sociocultural standards, and "dysfunction" is a scientific factual term, referring to failure of biologically designed functioning. In modern science, "dysfunction" is ultimately anchored in evolutionary biology and refers to failure of an internal mechanism to perform one of its naturally selected functions.

Mental disorder or psychopathology is thus defined as a harmful dysfunction. While critics Fulford and Thornton (2007) claim that values are the most important part of the definition of harmful dysfunction, many evolutionary psychologists might find the value focus problematic. Natural scientists often advocate that a definition of psychopathology ought to be entirely founded on objective (or value free) science. From this perspective one might claim that Wakefield's definition of dysfunction is all that is needed to define pathology, and that the subjective "harmfulness" criteria could be dropped altogether. This stance would suggest that dysfunctions may be detectable regardless of whether the subject or society experiences "harmfulness." Further, it suggests that all mechanisms not functioning as they evolved to do will be accepted as "pathological." But neither of these claims is defensible. I would argue that Wakefield is correct. We need to consider both factors: If we were able to discover such a non-harmful dysfunction, it would not be something we would wish to treat or call pathology. Our values define homosexuality, increased general intelligence of the population over time (that has us processing information in an evolutionary novel manner), the kind of individuality typical of modern Western culture, or lack of violent responses to threats and infidelity out of the domain of psychopathology. Even if to some degree mental mechanisms are not functioning as they evolved to function in the EEA (environment of evolutionary adaptedness, i.e., the past environment in which the adaptation was selected) we do not necessarily wish to class the resulting behavior as pathology. Therefore, we need to include a consideration of harmfulness as well as dysfunction.

However, the major problem is that the harmful dysfunction definition is dependent on future evolutionary mental health research in order to provide a nosology/taxonomy of functions in order to derive dysfunctions. Understanding psychopathology based on an analysis of harmful dysfunction will therefore not be relevant from a practical clinical perspective without an evolutionary psychology of normal functional psychological mechanisms as well as psychopathology—that is, a science of function is necessary to define dysfunction. Whether or not something has an evolved function is currently not a question that may be easily resolved—the mapping of our adaptations has only just begun (Tooby & Cosmides, 2000). Thus, despite Wakefield's definition having the greatest promise of providing mental health research with a theoretically and scientifically based overarching definition of psychopathology, it currently lacks the research needed to be able to categorize mental states (harmful dysfunction and non-harmful dysfunction, as well as harmful function and non-harmful function) and create a more valid nosology.

It is possible that the combination of Wakefield's approach to the definition of psychopathology and the necessary evolutionary psychology basic

research of mapping both functional as well as dysfunctional mental mechanisms could emerge as a major influence on future diagnostic systems. As such, work on individual differences and human universals involved in both normal and psychopathological mental states are potentially relevant and beneficial from an applied, clinical perspective. As Cosmides & Tooby (1999, p. 463) suggest:

In sum, trying to decide where a condition belongs in an evolutionary taxonomy is not a sterile exercise in categorization. Achieving a genuine understanding of the adaptations that comprise our bodies and minds, how they interact, and how they breakdown, can produce new and important insights into how to conceptualize—and, therefore, treat conditions that cause human suffering.

THREE TYPES OF "PSYCHOPATHOLOGY"

Adding to the complexity outlined above, "psychopathology" is not a clear and homogenous category. Many forms of disorder may not be defined due to statistical deviancy, suffering, or sequelae (Troisi & McGuire, 2002)—as they are not unusual, do not cause suffering, and there is no identified sequelae.

Kennair (2003, see Figure 1) suggests three types of phenotypes that clinicians define as psychopathology: adaptive psychological pain; socially undesirable effects of adaptations functioning as they were selected in the EEA; and mechanism failure. Of these, only mechanism failure would be considered true pathology (dysfunction) from a strictly biological, functional perspective. But if we were to apply Wakefield's (1999) definition, this would only be considered pathology if these mechanism failures are also considered harmful. Psychological pain and socially undesirable traits are considered harmful, but need not be due to dysfunctions. Thus, Wakefield's definition might assist clinicians redefine how they understand psychopathology, while at the same time highlighting the need for research on biological function.

Adaptive Psychological Pain

The few mainstream evolutionary psychologists who have looked into what clinicians currently define as mental disorder have usually concluded that the behaviors and mental states actually *are* adaptive even if they cause discomfort (e.g., Hagen, 1999; Watson & Andrews, 2002) rather than conclude that

the mental disorder is due to mechanism failure. Hagen (1999) suggests that postpartum depression is an evolved adaptive behavior that is elicited by the lack of investment from the father, causing him to have to invest more in their common offspring. The notion that something proximally maladaptive (e.g., depression) can be ultimately adaptive is controversial, even among evolutionary psychopathologists (e.g., Paul Gilbert or Randolph Nesse). Nesse & Williams (1996) point out that sometimes pain is an evolved defense. Nesse and Gilbert (e.g., Nesse, 2000; Sloman & Gilbert, 2000) generally suggest that mechanisms involved in depression may be adaptations (e.g., mechanisms to prevent evolutionary maladaptive behavior, by causing discomfort), but that Major Depressive Disorder probably never was evolutionarily adaptive.

Consider also Marks' (1988) explanation of blood phobia—where the patient faints at the sight of blood due to a sudden drop in blood pressure. The phobia-induced drop in blood pressure is atypical of phobias, and the fight or flight mechanism, which is typical of fear activation, but makes sense if the individual is in danger of losing blood. But as such, many states that cause psychological pain (e.g., Hagen's theory of postpartum depression or Mark's approach to blood phobia) may not be dysfunctions in the evolutionary sense of the word.

Adaptive psychological pain, including different types of anxiety (e.g., blood phobia) and some types of depression, may therefore not be psychopathology according to the harmful dysfunction definition. Despite not being defined as psychopathology, such phenotypes may be considered *treatable conditions* (Cosmides & Tooby, 1999). Whether such conditions ought to be treated would need to be decided based on an understanding of the function of the evolved defense and possible consequences of blocking this defense (Nesse & Williams, 1996).

Socially Undesirable Behavior

Some conditions listed in current diagnostic manuals may be considered pathology by society at large because they are undesirable or harmful, but are not considered pathology primarily by the subject experiencing them; they are ego-syntonic (congruent with the patient's value system or desires) or even pleasurable. Such disorders include both substance abuse as well as personality disorders. The previously discussed possibly functional states (i.e., depression, blood phobia) were considered harmful by the individual due to the psychological pain they caused. In contrast, for the present case, the possibly functional states are considered harmful by society.

An understanding of pathology needs to incorporate the idea that the context and understanding of the condition is often what defines a state as a disorder. Cosmides and Tooby (1999) call this "value-condition divergence." It might be behavior that is generated by a fully functioning evolved adaptation, but this behavior may be considered harmful or undesirable by society.

Diagnosing ego-syntonic dysfunction is challenging given our current lack of understanding of function—the mapping of the mind's normal psychological functions is still fairly limited. Also, many types of conditions may prove to not be treatable, at least not with current evidence-based psychotherapeutic, social, or medical interventions. Mealy (1995) suggests for example that dissocial personality disorder might have increased fitness, as such, despite a lack of empathy, people with dissocial personality are functioning from an evolutionary perspective. Despite this, society in general finds sociopathic or non-empathic behavior undesirable, and would like to provide treatment. So far such treatment is not available.

Thus an unhealthy, exaggerated intake of calories in subjects with no metabolic dysfunction, substance abuse, and several types of dependencies may all be due to fully functioning adaptations in a culture where the availability of calories, alcohol, and other ingestible substances is a consequence of the same adaptations that cause the problem behavior. The same may be the case for violent jealousy—it may have been adaptive in the past by increasing reproductive success, but is not acceptable in official Western culture. These examples would therefore not be pathologies according to the harmful dysfunction definition. Clinicians need to be able to differentiate between pathology and undesirable function (including treatable conditions).

Mechanism Failure

The most obvious and prototypical definition of disorder is that the evolved mental mechanism is malfunctioning—mechanism failure or dysfunction. An adaptation may be calibrated differently due to genes, development, or current or past environment. There is a limit, though, to how much such factors may adjust the reactivity of an adaptation before the mechanism can be considered to be malfunctioning. But malfunction is not as easy to identify as one might think. It is premature to conclude that every disorder in the diagnostic manuals is mainly explained by mechanism failure. There will therefore be many diagnoses that are not covered by the harmful dysfunction definition, and thus might be relevant for consideration as treatable conditions or functionally adaptive states.

There are few mental disorders that are clearly due to mechanism failure; I will discuss some candidates below. Whether the mechanism failure is caused by inadequate environmental stimulation, developmental disturbance, or genetic deficiencies is also difficult to ascertain. Despite this, mechanism failure is still the most typical explanation within mental health research. Pathology is often claimed to be due to brain disorder, as in the American National Institute for Mental Health's (NIMH) explanation of depression.

On the other hand, when evolutionary psychology attempts to offer competing theoretical explanations, it is not surprising given the adaptationist program that one typical hypothesis is that the disorder is not due to mechanism failure, but actually is adaptive or due to the workings of adaptations. There is potential in considering both adaptations (states that function, so that one may avoid iatrogenic conditions) as well as dysfunctions (in order to provide Wakefield's definition with an empirical foundation).

Also, most of the obvious brain malfunctions are grouped apart from mental disorders, and are assessed and handled by neuropsychologists rather than psychiatrists and clinical psychologists. One example of mechanism failure is the case history of the patient with neurological damage that caused a specific impairment of the cheater detection mechanism discovered by Cosmides (Stone et al., 2002). But this kind of explicit mechanism failure is not typically a mental health case.

Among mental disorders, some features of schizophrenia are more typical neuropsychological mechanism failures, or impairments. Further, the mood swings of bipolar disorder are maladaptive in modern society, and seem to be dysfunctional—but we do not know enough to conclude. In general, depression seems to reduce fitness. The anxiety disorders (fear of non-threatening stimuli) reduce function—although the basic emotion fear is adaptive when it protects us against real threats. Yet again, it is hard to conclude that there is mechanism failure. Also, anxiety disorders and obsessive-compulsive disorders respond rapidly to psychological treatment, and thus the mechanism failure model seems unlikely (it seems to be more a case of oversensitive calibration, rather than dysfunction). The personality disorders reduce social adaptation. But even suicidal behavior may increase inclusive fitness (de Catanzaro, 1995). The lack of empathy in psychopathy (Mealy, 1995) may be adaptive for the individual, in general as a social strategy and under specific ecological conditions (e.g., war, famine). Many dysfunctions may exist, but they need to be researched from a functional, adaptationist perspective before we may conclude that they really are dysfunctions. Thus the conclusion is that while we have a number of candidates for true mechanism failure, we are not currently able to conclude.

AN INTEGRATIVE REVISED EVOLUTIONARY PSYCHOLOGY FOR THE STUDY OF PSYCHOPATHOLOGY

In order to conceptualize individual differences in psychopathology, mainstream evolutionary psychology needs to integrate theory and findings from evolutionary developmental psychology, behavioral genetics, and research on the etiology of disorders, as well as modern cognitive behavioral therapy research as a basic science of proximate mechanisms. Evolutionary psychology's integrative potential is largely due to the fact that evolutionary approaches are aware of different levels of analysis, but also that the cognitive mechanism level of analysis makes for a generic theory in all approaches that consider systematic information processing. As such, other research programs within mental health science that focus on mental mechanisms and information processing, such as cognitive behavioral therapy research (Alford & Beck, 1997; Gilbert, 2002; Kennair, 2007), might be of special interest to evolutionary psychologists venturing into this field.

There are therefore two major tasks for an evolutionary psychology of psychopathology:

First, evolutionary psychology must be expanded to include mental disorders. This means continuing evolutionary psychology's task of predicting and mapping the functional, adapted mind. When considering psychopathological phenomena, this mapping would include describing syndromatic dysfunctions and providing good descriptions of the phenotypes and adaptations that need to be studied. A good understanding of the abnormal demands an understanding of normal phenomena and universals, as well as when and how these phenomena malfunction.

The second challenge is to provide the current disparate evolutionary approaches to psychopathology with an overarching meta-theory. Providing evolutionary psychopathology with a relevant integrative theory will demand theoretical developments to better conceptualize and predict individual differences. This will involve evolutionary psychology becoming fundamentally developmental, incorporating genetic individual differences at a conceptual level, and focusing on predicting environmental cues that may alter or harm the development of adaptations.

The model presented in Kennair (2003) may aid further evolutionary psychology research into psychopathology (see Figure 16.1). The model itself is a synthesis of different approaches within mainstream evolutionary psychology and general evolutionary psychopathology. I also recommend considering Buss and Greiling's (1990) and Nesse's (2005) lists of factors that cause individual differences. The Kennair (2003) model highlights a few of the important revisions of evolutionary psychology theory that might be

necessary when investigating phenomena that are not universal (e.g., either due to genetics or due to age). The following section elaborates on the levels of analysis and phenomena an evolutionary psychology approach will need to encompass based on Figure 16.1.

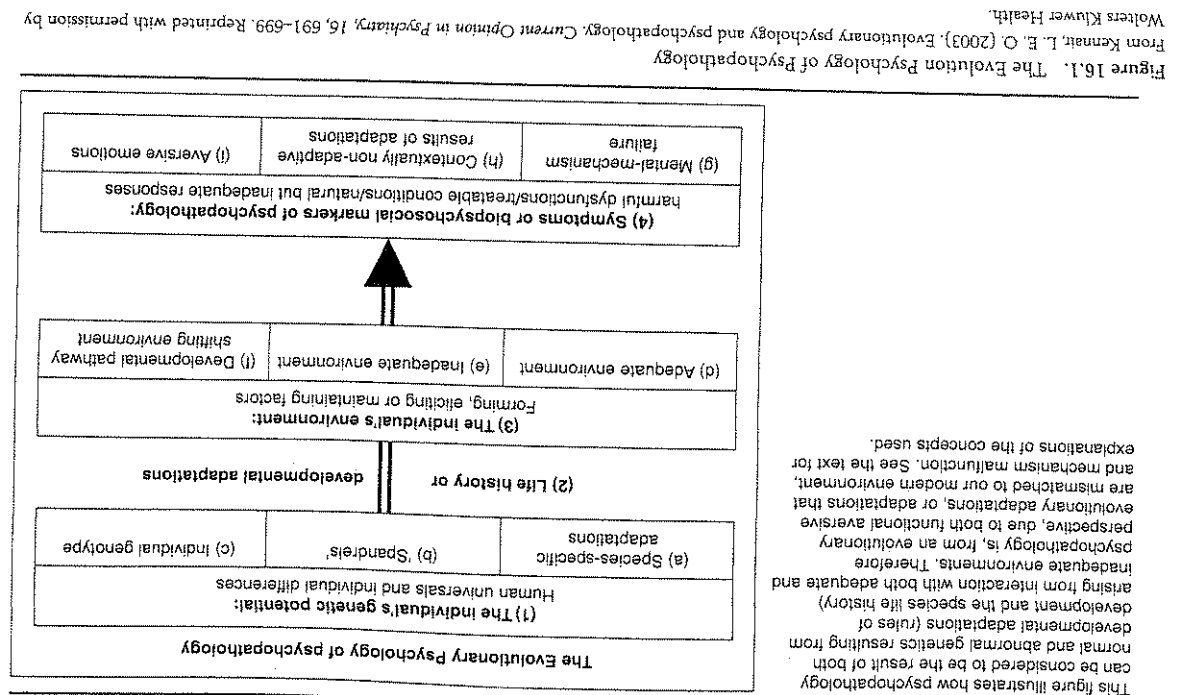
The Individual's Genetic Potential

The individual's genetic potential includes both the human universal adaptations and by-products of those adaptations (Tooby & Cosmides, 1992). Research into adaptive snake phobia (Mineka et al., 1984), the function of blood phobia (Marks, 1988), Watson and Andrews' (2002) approach to depression in general, Hagen's (1999) approach to postpartum depression, or de Catanzaro's (1995) suggestion that suicide may historically have increased fitness, are prime examples of investigations of adaptations that result in mental disorders. Much of the mainstream evolutionary psychology research would naturally focus on adaptations. But the analysis additionally needs to be expanded to cover other phenomena such as by-products (non-selected processes of the mind with no current or previous function, Buss et al., 1998).

Crow's (2000; Berlin et al., 2003) research on schizophrenia is an example of conceptualizing the disorder as a by-product rather than the selected trait. Crow's theory attempts to resolve the schizophrenia paradox: How and why a disorder that reduces fitness has not been selected out of the human gene pool, but is equally frequent in almost all ethnic groups. Crow suggests that the speciation event that provided humans with a capacity for language and brain lateralization is the reason why schizophrenia exists. Crow's approach may be called a by-product approach to mental disorder. The human universal is the capacity for language—the accidental by-product is schizophrenia for a subset of the population due to genes that are universal in the species' gene pool.

Another possible by-product disorder is panic disorder. Panic disorder is maintained by a misinterpretation of bodily or cognitive sensations (anxiety symptoms). The patient typically believes these are signs that he or she is going to faint, have a heart attack, choke, or go mad. As these are not real threats, it would seem that our capacity to incorrectly interpret our bodily sensations causes the disorder. The treatment of panic disorder is relatively simple—the patient is helped through behavioral experiments to discover that the imagined catastrophes cannot come true (Clark et al., 1999; Kennair, 2007).

This level must also include an analysis of individual genetic differences. Some of these differences may be noise or pathogen defense (Tooby &



Cosmides, 1990); others may be recent harmful mutations that are part of the explanation of subpopulations of specific disorder patterns (Ozaki et al., 2002). And some of these may be genetic differences that result in different adaptive morphs within the population (e.g., Caspi et al., 2003), due to either frequency dependent selection (Tooby & Cosmides, 1990) or assortative mating (Rowe, 2002).

Findings from behavioral genetics also need to be integrated with evolutionary psychology research (Segal, 2005; Segal & Hill, 2005). Such integration is necessary for evolutionary psychology to become updated on modern developmental research. Behavioral genetics has provided new understanding of developmental processes, and the effects of genes as well as non-shared environment. Phenotypic plasticity may be an important explanation when considering individual differences—and environmental cues may thus cause individual differences (Buss & Greiling, 1999; Tooby & Cosmides, 1990; West-Eberhard, 2003). At the same time, it is necessary to also consider the possible consequences of genetic differences when one refocuses from human universals to an in-depth and integrative approach to individual differences (see Wilson, 1994). Behavioral genetic research also shows that environmental factors are important.

Sex differences are crucial—and are a specific level of analysis when considering human universals and individual differences. Evolutionary psychologists expect to find sex differences only when males and female have recurrently faced different adaptive problems over human evolutionary history (Buss, 1995)—in domains such as mate choice, parental investment, paternal certainty (e.g., in jealousy, Buss & Haselton, 2005, or mate preferences, Buss et al., 1990). The fact that there are robust cross-cultural sex differences in the susceptibility to develop mental disorders (e.g., Gater et al., 1998) is important theoretically. Most of the relevant genes are shared between the sexes, and both genes *and* environments cause, e.g., depression (Sullivan, Neale & Kendler, 2000). Evolutionary psychology is already able to handle sex differences and hypothesize about effects of environmental factors. An evolutionary psychology that is more aware of individual genetic differences and development might be a fruitful approach to address, explain, and predict sex differences in mental disorders.

Developmental Adaptations

Mainstream evolutionary psychology has focused primarily on the adult human mind. The reproductive focus of evolutionary theory has made the sexually mature adult the relevant subject of study for most evolutionary researchers. Bjorklund (2003) and the field of developmental evolutionary

psychology are critical to this focus. The human animal's life history is unique: We are born so early relative to our level of maturation at birth that we need more caretaking for a longer period of time than other species. Our complex social systems also demand years of socialization and maturation to navigate (e.g., Flinn, 2005; Flinn & Ward, 2005). Thus, survival at every point of the human development is important for the individual to reach sexual maturity and reproduce. Also, human females live longer in a non-reproductive state than other mammals.

Developmental psychopathology (e.g., Rutter, 2006) is a foundational approach to the study of mental disorder. Most types of pathology—from schizophrenia to social phobia—have developmental precursors or developmental or life history events (e.g., first pregnancy) characterized by large hormonal changes (i.e., puberty) accompanied by a large reorganization of the brain that seem to increase likelihood of disorder onset. This developmental reality suggests that different adaptations may be coming online in theoretically predictable patterns.

Evolutionary developmental psychology (Burgess & MacDonald, 2005; Ellis & Bjorklund, 2005) has experienced a boost of interest the last few years. This work is filling a conceptual hole and a lacking level of analysis within mainstream evolutionary psychology. One of the sets of human universals is our developmental adaptations and species' life history. There are both differences and similarities between members of our species due to maturational and developmental processes—not only environmental input or genome. Considering these factors may aid the merger of developmental psychology, behavioral genetics, and evolutionary psychology (Kennair, 2005).

Evolutionary developmental psychopathology (e.g., Pitchford, 2001) is therefore a necessary level of analysis to add to any investigation of mental disorder. And while the trend within evolutionary psychology is toward a greater interest in developmental psychology, there has been little empirical research (although consider e.g., Ellis, 2003; Ellis & Essex, 2007; Hawley & Little, 1999). The current model suggests that both normal evolutionary psychology and evolutionary psychological investigation of psychopathology need to consider developmental aspects closely—including life history theory (Kaplan & Gangestad, 2005).

Certain disorders appear at different times through development and are considered differently depending on the age and context of the individual. The same behavior in a five-year-old child might be age typical learning of social rules and rituals, but be considered pathological OCD in a teenager. Some disorders seem to "burn out" with age (e.g., borderline personality disorder), while others may become more pronounced (e.g., depressions). Disorders are often considered more chronic today (e.g., depressions often

return), and we are more aware that shy children often turn into socially phobic or avoidant adults. As with the normal range of human personality, many of the same questions about stability and change are relevant. Many disorders (e.g., generalized anxiety disorder) are present throughout life, and hence suggest stability. Moreover, different individuals may have different developmental courses of their disorder. And the same disorder may manifest itself differently at different ages, much as personality traits do.

Etiological research from a behavioral genetics perspective is also an area that one needs to consider. The fact that many disorders have a genetic component is accepted by most researchers today. The surprising findings (Pinker, 2002; Turkheimer, 2000) are the actual effects of environmental factors: Family environments do not shape personalities as our theories have suggested; where family members are similar due to genetics, they are different due to non-shared environment.

Environmental Influences

Evolutionary psychology has traditionally explained most phenotypic variance through environmental variance—an evolved universal human nature differentially stimulated by differing contexts (Tooby & Cosmides, 1990). Thus, Buss and Greiling (1999) suggest that the Belsky-Steinberger-Draper hypothesis may be an important developmental and environmental explanation of how an individual's developmental pathway may be shifted due to specific environmental cues and contexts in interaction with a universal human nature. The Belsky-Draper hypothesis (Belsky, 2000; Belsky, Steinberger & Draper, 1991; Draper & Harpending, 1982; Quinlan, 2003) suggests that girls raised without an investing father mature sexually, partake in sexual activity, and become pregnant earlier than girls raised by an investing father. This is due to the early experience of a father's investment calibrating the specific adaptations evolved to assess the likelihood of paternal investment in the relevant ecology and generating coordinated psychophysiological behavior and development that solves the statistically likely adaptive problem. Rowe (2000) criticized this research for focusing too much on environmental causes, pointing out that the traits being studied are heritable. Ellis et al. (2003) found further support for the Belsky-Steinberger-Draper hypothesis, while Mendle et al. (2006) provide research that tested both evolutionary and behavioral genetic hypotheses where genetics seemed to explain most of the variance (but see also Tither & Ellis, 2008).

It would seem that an assortative mating model best explains the findings, as both environmental as well as genetic factors explain variance (see Rowe,

2002). One has to combine two perspectives: The importance of heritability, not merely environmental input (Wilson, 1994), and the importance of having a developmental perspective (Kennair, 2005; Pitchford, 2002). The idea that different environments can elicit different sets of adaptations due to specific ecological cues attains importance from both life history and evolutionary developmental perspectives.

Within behavioral genetic research on personality traits, the most surprising finding is the effect of the environment. The fact that genes explain a moderate amount of variance was an important empirical finding, although probably as expected from a genetic perspective. Turkheimer (2000; see also Pinker, 2002) sums up the effects of the environment thus: Genes explain more of the variance than growing up in the same family, but environmental effects—other than families (shared environment)—explain most of the variance. At the group level of analysis, genes make siblings similar, but the environment makes them different. Without resorting to group selection explanations, this does make some sense from an evolutionary perspective. There is limited genetic variance in our species, compared to similar species (Cosmides, Tooby & Kurzban, 2003). Parents and their children, or siblings, are 50% genetically similar—and in many cases in the EEA with small groups (Dunbar, 1993), and thus mating among closer relatives than what is typical today, this number may have been larger. If genes made us similar—which they necessarily would—and parents and siblings made us even more similar, as most theories outside of behavioral genetics suggest, then there would be very little phenotypic variance. If phenotypic variance was beneficial, selection would have increased the likelihood of being influenced by environmental forces other than those one was most genetically similar to, reducing effects of shared environment over lifespan. See also Harris (1995) for an alternative model that invokes the evolutionary importance of peer influences.

These perspectives are relevant for psychopathology. Genes influence the development of disorders. Poulton and Menzies (2002) and Kendler, Prescott and Myers (2002) challenge the mainstream ideas of the etiology of phobias specifically—and psychopathology in general. Poulton and Menzies (2002) challenge the idea that association is the major explanation of how phobias are acquired (see Rachman, 1977). Summing up years of longitudinal research (e.g., Poulton et al., 1998, 1999), they conclude that it would seem that non-associative explanations better explain how phobias develop. Kendler, Myers, and Prescott (2002), moreover, challenged the stress-diathesis model of the etiology of phobia. They conclude that there is little support for the idea that disorders are caused by the interplay between genetic vulnerability and the severity of the traumatic experience or stressor. For many years most clinicians have considered the anxiety disorders to be caused by conditioning experiences—and that these disorders thus had the most obvious

pathways. While non-shared environmental factors explain most of the variance of who develops anxiety disorders, we now know that we do not really know what specific factors in the environment explain specific disorders. This is true of all disorders. Evolutionary psychologists interested in how individual differences develop, and how differences in mental mechanisms are maintained, may be able to predict under what circumstances the development of normal mental adaptations will be harmed or changed.

Theories about effects of shared environment lack strong empirical support and the general findings within behavioral genetics challenge most of the existing consensus. Thus mainstream evolutionary psychology's approach to phenotypic plasticity and effects of environmental cues on developmental pathway shifting mechanisms (e.g., Buss & Greiling, 1999; Ellis et al., 2003) or phenotypic variance—might be an important approach for the study of etiology. Incorporating insights from genetics and developmental studies with evolutionary analysis increases the sophistication of the models and empirical tests. Despite the importance of genes, non-shared environmental factors account for most of the variance (Turkheimer, 2000). And sometimes common genes, but different environments, present as different phenotypes or psychopathological syndromes (Kendler, 2004).

It is further important to consider possible mismatch (e.g., Nesse & Williams, 1996; Nesse, 2005) between the modern or current environment and the evolved mental adaptations that evolved to function under different environmental conditions. This means that the adaptations may not be receiving the expected and thus adequate environmental input and stimulation, causing them to process information in a non-adaptive manner in the current environment. Alternatively, the adaptations' development may be disturbed (Buss & Greiling, 1999; Kennair, 2003; Nesse, 2005). There is little that decisively concludes that the modern environment has caused an increase in mental disorder (Nesse, 2005), although there is some evidence that depression is rising (Compton et al., 2006; Klerman & Weissman, 1989), especially among young women. Any rapid change of prevalence suggests that recent changes in western societies may cause increased mismatch, although what these changes are we do not know. Sandseter and Kennair (submitted) suggest that limiting risky play among kindergarten children, a recent environmental change, may cause an increase of anxiety due to less possibility for habituation or learning to cope with one's environment and normal interaction with one's ecology.

Social cognition, social development, and social selection have been important for human mental evolution—our species' most important selection forces have been other hominins. As such we have replicated and conserved our most psychologically relevant environmental features as these consist of the social and relational behavior generated by the human nature

(i.e., the behavior) of other people. It is therefore reasonable to assume that most of our relational and social context has been relatively stable since our species evolved and migrated out of Africa. And, as Buss (1996) suggested, when considering the evolution of personality traits, other people's traits laid the basis for some of our most important and stable adaptive problems, and also through cooperation some of the most available solutions to our adaptive problems. Consider Flinn's recent approach (2005; Flinn & Ward, 2005) to the evolved developing social mind; it suggests that the very recent breakdown of intergenerational families might have detrimental effects on mental health.

The environment is more than psychosocial; it is also biological and chemical. The environment must be conceptualized as the biopsychosocial context at all levels from genes and chemicals and nutrients to social and political factors. One must consider how these contexts influence the development of the individual's adaptations. Evolutionary psychology has generally been sensitive to environmental influences—especially social factors. Biochemical factors may need to be given more attention (e.g., Myerud & Poleszynski, 2003). One may further note how mismatch will not necessarily only have negative effects. Despite the ready availability of sugar in our modern environment causing dental caries, pathological obesity, and diabetes, an increased availability of calories throughout development may cause the population-wide increase in brain development manifesting in an increase in a population's intelligence (the Flynn effect). As such, it would be interesting to investigate the effects of the evolutionary and culturally novel child-rearing practice of letting infants sleep on their own. Does it increase the likelihood of disorder? Does it cause a greater individuality—and thereby better function in Western society? Mismatch that is too detrimental would be selected against (e.g., the child rearing practices of orphanages in Ceaușescu's Romania; Rutter, 2006).

LIMITATIONS AND FOUNDATIONS OF THE INTEGRATIVE REVISED MODEL

This model is an attempt to describe a synthesis of recent work within both general evolutionary psychopathology and mainstream evolutionary psychology. The model itself does not make any specific predictions. It aims to make explicit several of the important biopsychosocial factors and levels of analysis that are involved in the study of psychopathology. Human nature and individual genetic differences, age and life history, environmental factors and how one hypothesizes these factors to influence the state or expression of the disorder; and whether the disorder is or was adaptive or due to adaptation

malfunction. For Wakefield's definition to be relevant, it is crucial that there exists work that can illuminate the conditions and phenotypes that may be classed as "dysfunctions."

The mainstream evolutionary psychology research methodology is central to generating predictions based on strategic analysis of the EEA and the computational, functional analysis of information-processing modules of the mind, based on the application of middle level evolutionary theories to generate predictions about the evolved architecture of the modern mind. One must be able to identify function in order to assess dysfunction. It is also based on evolutionary psychology's modular model of the mind, with a focus on adaptations at all levels of the model; in the investigation of spandrels (as the adaptation is the testable hypothesis) as well as in mechanism malfunction or developmental analysis. What is needed is a focus on the relevant phenotypes within psychopathology, and an increased focus on individual differences, development, and genetics, all while maintaining the focus on multiple adaptations and the effects of environments on adaptive shifts in development or causing phenotypic variance.

INTEGRATING EVOLUTIONARY PSYCHOLOGY WITH EVOLUTIONARY PSYCHOPATHOLOGY

Above I suggested possible revisions to evolutionary psychology theory and methodology for the study of the nature of psychopathology. The main message is the double claim that the general field of evolutionary psychopathology needs an integrative and rigorous metatheory and research program and that the cognitive psychological science of evolutionary psychology is the best candidate. Moreover, mainstream evolutionary psychology must be somewhat modified to become more focused towards individual differences and developmental issues; which is why this volume is important.

Changes through the last few years within mainstream evolutionary psychology have already laid some of the groundwork for a more relevant theory for psychopathology. Much of the work by David Buss and his students on sex differences and individual differences (including personality traits, e.g., Buss, 1996; Buss & Greiling, 1999) have moderated the universal human nature approach. Differences due to age have also become more central (see Burgess & MacDonald, 2005; Ellis & Bjorklund, 2005; Kennair, 2005).

Evolutionary psychopathology needs an integrative metatheory in order to provide cumulative theory building and an overarching research program. The alternative to evolutionary psychology becoming the integrative metatheory for the study of the nature of mental disorders would seem to be to not have any overarching model and research program.

What are the major hindrances that prevent evolutionary psychology from already being an integrative metatheory? To a large degree, opposition to evolutionary psychology may be due to academic traditions and borders between different areas of research. Clinical psychology and psychiatry have their own strong traditions and models of human nature. Within these disciplines the influence of human ethology is greater than that of evolutionary psychology (e.g., through the work of John Bowlby; Ainsworth & Bowlby, 1991). The divide between social and cognitive research psychology and academic clinical psychology has been great, and there has been remarkably little transmission of information and models between these disciplines. An integrative approach such as evolutionary psychology might be able to improve this situation.

The developmental pathways of psychopathology—the etiology of mental disorders—may be understood better from an evolutionary life history perspective (Kaplan & Gangestad, 2005). Developmental issues and the question of why some people develop disorders are among the most interesting and least understood mysteries of mental health care. For years these quandaries were considered answered by Freudian theory or by mainstream developmental psychology. Today we are aware that even the whys and hows of simple phobias are not as obvious as we once thought (e.g., Kendler, Myers & Prescott, 2002; Poulton & Menzies, 2002). We actually know less about development than we thought we did. Many studies are comparisons of children and parents. Similarities between parents and children usually are more due to common genes than environment (Plomin et al., 2000; Turkheimer, 2000). Thus most developmental research has probably studied effects of heritability more than the effects of environmental factors. We are therefore not aware of many environmental factors that explain the etiology of psychopathology. The evolutionary approach might be able to generate predictions that help identify influential environmental factors.

There are several fruitful consequences to including the study of psychopathology into the mainstream normal psychology research program of evolutionary psychology. Baron-Cohen (1997) pointed out how the malfunction of the mind may inform us of how the mind works. Stone et al. (2002) illustrate how the lack of a specific mental ability in a patient with brain injury is part of the evidence of the existence of a specific module in the fully functioning brain. Considering psychopathology as something apart from normal function is a limited perspective. Mental disorder is to a large degree part of universal human nature. Although most people do not have mental disorders, the percentage of reproductive age population that does is so large that mainstream evolutionary inquiry into how and why is warranted.

Many clinicians within evolutionary psychopathology are skeptical about the contributions of non-clinicians (e.g., the work from non-clinicians on the benefits of depression; Hagen, 1999, 2002, 2003; Watson & Andrews, 2002). This may be due to different causes: A protectionism among clinicians of their own status and position, the clinicians' preconceptions of the phenomena, or a lack of insight on the part of the non-clinicians on the specific clinical phenomenon due to a lack of training. Increased research on psychopathology within evolutionary psychology will necessarily mean that more non-clinicians will focus on clinical questions.

EVOLUTIONARY INFORMED TREATMENTS

While some authors (e.g., Sloman & Atkinson, 2000; Troisi & McGuire, 2002) already have suggested interventions based on evolutionary insights, it is important to note that from a scientific perspective, a treatment must be proven effective through clinical trials before one can recommend interventions to patients. Although it is possible to imagine several theoretically predictable interventions, it is far from certain that these work or address the relevant maintaining factors of the disorder. One needs to compare interventions to no-treatment control groups, and compare them with other evidence-based therapies. It is important to not jump to conclusions based on science in such a manner that the applied intervention is not scientific: Science-based interventions must also be evidence-based.

Currently there are few therapies based on evolutionary theory. Even fewer of these are documented through clinical trials. For example, even though Ilardi (2009) has developed a treatment package that combines a broad set of insights from evolutionary studies with cognitive behavior elements, as of this writing this approach still needs to document efficacy.

Additionally, Gilbert and co-workers (Gilbert & Irons, 2005; Gilbert & Mayhew, 2008; Gilbert & Procter, 2006) have developed a treatment method based on insights from ethological work such as Price (1967). Gilbert's treatment, called Compassionate Mind Training, may be considered an ethological cognitive behavior therapy in that it focuses on the reduction of internalized hierarchical mindsets such as low self esteem, shame, and self criticism, which are generic features of mental disorder. Despite promising results, this approach still needs further work to prove its efficacy with major diagnostic categories.

These beginnings are promising, as is an increased interest in the evolved nature of psychopathology. This might provide us with a broader set of interventions in our clinical work.

CONCLUSIONS

Thus far, evolutionary psychopathology has largely been a theoretical, rather than empirical, endeavor, by focusing on how one may theoretically understand and explain the evolution of psychopathology or the nature of mental disorders. As with evolutionary personality psychology (Buss, 1991), theory in evolutionary psychopathology is ahead of the data (see Figueredo et al., 2005). This is probably true of all science, but the gap needs filling. If evolutionary personality psychology shall continue to be relevant, there will need to be a surge of empirical research in the next few years. The same is true for evolutionary psychopathology.

On the other hand, empirical research needs an overarching meta-theory to guide investigation. Accumulation of knowledge is hard to accomplish without such a framework. Empirical investigations will under such circumstances tend toward fragmented data; dustbowl empiricism. Also, without an integrative meta-theory there will be an unfruitful discussion between researchers within the field over theoretical foundations, methods, and whether the research is *truly* evolutionary or how to best synthesize findings.

The evolutionary psychology research program offers a set of meta-theoretical rigorous principles: To focus on mental adaptations rather than merely on phenotypes or on adaptiveness; to attempt to predict what adaptations may exist based on an analysis of the past environment; and to use established evolutionary middle-level theories in an attempt to formulate hypotheses about modern human nature. A specific meta-theory and research program for the study of the evolution of mental disorders may prove fruitful and integrative, causing greater interest in the study of clinical topics among empirical research psychologists.

The current integrative, synthetic approach might help to bridge the gap between mainstream evolutionary psychology and clinicians and personality psychologists. Mainstream evolutionary psychology is first and foremost the study of human universals (Tooby & Cosmides, 1990)—but as soon as one considers important topics such as sex differences or personality traits due to stable selection pressures, one needs to expand the meta-theory. An integrative meta-theory for all of psychology needs to consider both human universals and individual differences (Scarr, 1995). Topics such as personality or psychopathology demand meta-theory and models that can handle individual differences.

A broad, integrative biopsychosocial approach may also convince clinicians and theoreticians outside of evolutionary approaches of what an evolutionary perspective has to offer. In conclusion, the present chapter argues

that mental mechanisms—which are the major integrative feature of evolutionary psychology—need to be the focus of future evolutionary psychopathology. At the same time, evolutionary psychology needs to better understand how these mental mechanisms develop, are influenced by different environments and genetic differences, and break down or cause social or emotional maladjustment.

Evolutionary psychology is—after two decades—still a rather young approach, and is still maturing theoretically and expanding into new research areas. Evolutionary psychology's forte is its widespread meta-theory and well-defined research program within psychology. Thus, it offers a foundation for a more rigorous evolutionary study of human nature and concomitant functional mental mechanisms. Evolutionary psychopathology, more generally speaking, is an older approach, including theorists such as Freud, Bowlby, Meyer and Price. Researchers using different evolutionary approaches to psychopathology cover almost the entire field of mental disorders, while neither having an integrative research program nor an integrative theory or model of human nature that collects larger groups of researchers. Thus there is little accumulative power within the field, and not enough focus on mental mechanisms. Furthermore, within general psychiatry and clinical psychology, there is no general understanding or agreement of what constitutes mental disorder. The scientific progress of the field depends in large part on defining these phenotypes and phenomena.

Being convinced that the human universal adaptations are predictable and fundamental aspects of our minds, I would claim that the evolutionary psychology research program is the most relevant first approach when studying individual differences in the form of psychopathology. We need a predictive, evolutionary mechanism-focused science of function as well as dysfunction.

As argued here, the most promising general definition of psychopathology is Wakefield's concept of the harmful dysfunction. Despite clinicians treating treatable conditions (i.e. clinicians in general have disparate theoretical definitions of pathology, and in general do not consider function or dysfunction, merely theoretically defined treatable conditions), the harmful dysfunction might be the best definition of pathology. The practical application of this new definition is dependent upon the development of an evolutionary psychology of psychopathology that focuses on the mental mechanism, and that can provide us with an objective nosology of mechanism failure that enables clinicians to recognize true dysfunctions. This restructuring has potential novel clinical value. The future definition and understanding of mental disorder may be decided by researchers that work under the empirical and theoretical auspices of evolutionary psychology.

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