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The evolutionary future of psychopathology

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Evolutionary approaches to psychopathology have made considerable progress over the last years. In this paper, I review recent advances in the field focusing on three core themes: the role of trade-offs and conflicts in the origins mental disorders, the evolution of developmental mechanisms, and the emergence of alternative classification systems based on life history theory. I situate these advances in the context of current research in psychopathology, and highlight their connections with other innovative approaches such as developmental psychopathology and computational psychiatry. In total, I argue that evolutionary psychopathology offers an integrative framework for the study of mental disorders, and allows complementary approaches to connect and cross-fertilize.

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Introduction

The study of mental disorders is at an exciting juncture. Since the turn of the century, a number of innovative trends have been picking up speed and are now reaching critical mass. One of these trends is the growing influence of *developmental psychopathology*. This approach centers on the interplay of personal and environmental factors in the origin of mental disorders, including genotype-environment interactions, epigenetic encoding of life events (e.g., prenatal stress, early neglect or abuse), and their role in the development of neurobiological systems [1,2]. An even more recent trend is the rise of *computational psychiatry*, which employs mathematical models of cognitive and neural processes (e.g., decision making, synaptic excitation-inhibition) to identify the mechanisms involved in mental disorders [3,4•,5]. This approach resonates with the Research Domain Criteria (RDoC) promoted by the National Institute of Mental Health [6], which aim to identify dysfunctions in specific neural systems, breaking away from the standard diagnostic categories (e.g., depressive disorders, schizophrenia) of the Diagnostic and Statistical Manual of mental disorders (DSM [7]).

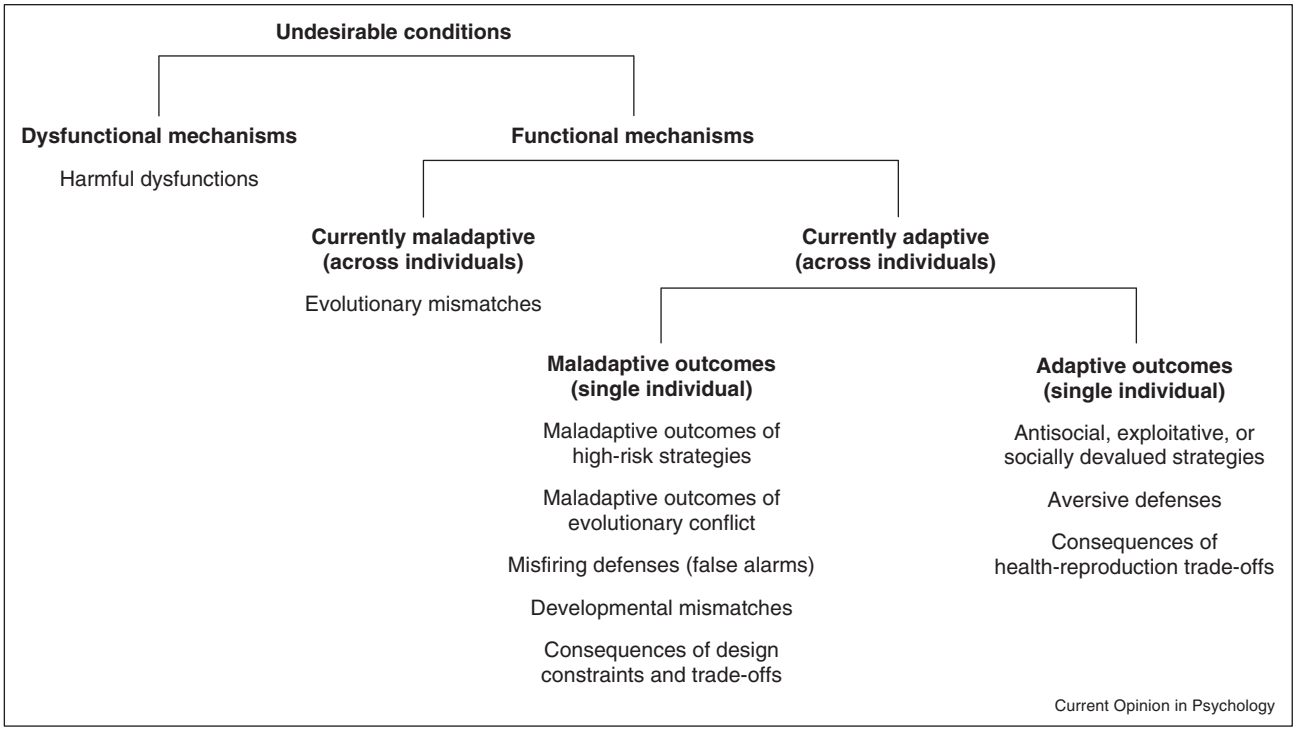
Here I focus on another emerging approach, that of *evolutionary psychopathology*. This approach draws on biological models and concepts to understand the functions of the neural and psychological processes involved in mental disorders and how they have been shaped by selection during our evolutionary history. A key feature of the evolutionary approach is that it does not automatically regards mental disorders as dysfunctions, and considers a broader range of alternative explanations — including the possibility that some conditions may reflect adaptive processes designed to promote an individual's biological fitness (for example by increasing his/her reproductive success) at the expense of well-being or social adjustment (Figure 1). The evolutionary program is not an alternative to the other approaches described here but rather complements and extends them [8–11]. A limitation of current approaches is that they tend to ignore the evolutionary level of analysis [12] (Figure 2). In the field of developmental psychopathology, for example, behaviors that decrease well-being are usually regarded as 'maladaptive' by default, without consideration of their potential fitness benefits. Computational psychiatry deals with the proximate functions of neurocognitive mechanisms — mostly domain-general processes such as reinforcement learning — but fails to consider their role in managing specific adaptive tasks (e.g., choosing mates, avoiding pathogens).

In this paper I review recent advances in the field of evolutionary psychopathology, highlighting their connections with other approaches and their implications for the future of the discipline. First I consider how biological conflicts and trade-offs can shed light on the origins of mental disorders. I then review some important evolutionary contributions to understanding the developmental processes that lead to psychopathology. Finally, I present a novel evolutionary framework for the classification of mental disorders.

Conflicts, trade-offs, and the origins of psychopathology

As shown in Figure 1 mental disorders have many possible causes (see [10••,11–14]). Two common reasons for the evolution of vulnerability to pathology are trade-offs between competing traits or functions, and biological conflicts of interest between individuals (and/or their genes). The heuristic power of trade-off and conflict thinking is illustrated by the *diametrical model* of autism and psychosis advanced by Crespi and Badcock [15].

Figure 1



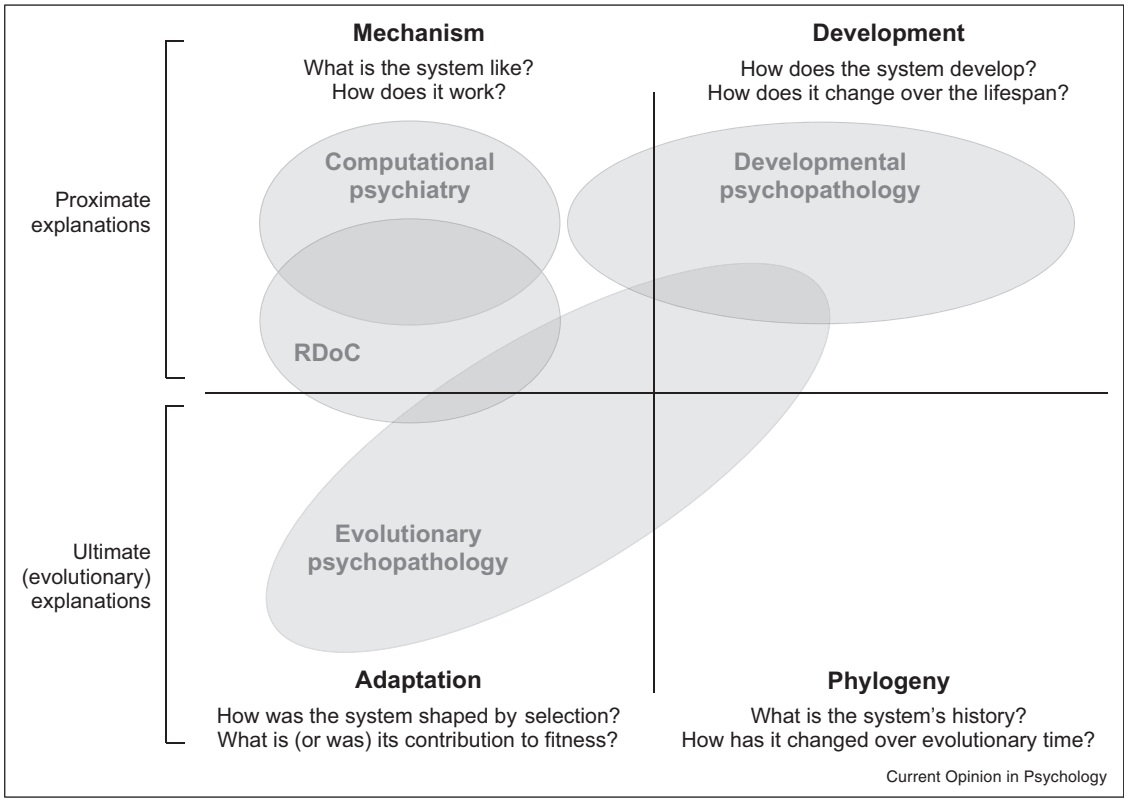
Possible explanations of mental disorders from an evolutionary perspective. Psychopathological conditions may arise from dysfunctional mechanisms, or from functional mechanisms that produce maladaptive outcomes because the present environment is different from the one in which they evolved (mismatch). Other conditions are the occasional maladaptive outcomes of generally adaptive mechanisms. Finally, some conditions may represent biologically adaptive but undesirable behavioral strategies (see [11]).
Reproduced from [11].

According to the model, autism spectrum disorders (ASDs) and psychotic disorders (including schizophrenia and bipolar disorder) are pathological extremes of a continuum of individual variation. ASDs are characterized by hyper-developed *mechanistic cognition* (e.g., systemizing, visuospatial skills) and under-developed *mentalistic cognition* (e.g., empathy, theory of mind), whereas psychosis shows the opposite profile. A trade-off seems to exist between these two aspects of cognition, which may require different information-processing styles [16]. The model also maintains that ASDs are associated with over-expression of genes inherited from the father and/or under-expression of genes inherited from the mother (with the opposite pattern in psychosis), so that evolutionary conflicts between maternal and paternal genes [17,18] contribute to the risk of pathology. The diametrical model has been empirically successful — for example, a recent study found that ASDs and psychosis show diametrical associations with birth weight (higher in ASDs, lower in psychosis), consistent with the hypothesized genetic effects [19•]. Also, the model may help explain the largely opposite effects of oxytocin and testosterone on social behavior, as well as their involvement in ASDs and psychosis [20••,21]. However, the relations between mechanistic and mentalistic cognition have not yet been formalized in a computational model of the relevant cognitive processes — a potentially fruitful goal for future research.

My colleagues and I have extended the diametrical model by framing individual variation in autistic-like and psychotic-like traits in the context of a specific evolved domain, that of mating and reproduction [22]. The hypothesized trade-off is between short-term mating with multiple partners (favored by traits associated with psychosis risk, e.g., creativity, mentalistic skills, and impulsivity) and investment in long-term romantic relationships (favored by traits associated with the autism spectrum, e.g., technical skills, reduced sex drive, and preference for routines). Sexual selection in the context of short-term and long-term relationships could help explain the maintenance of autistic-like and psychotic-like traits in human populations. The sexual selection hypothesis has received promising empirical support; for example, autistic-like and psychotic-like traits in non-clinical samples show the predicted diametrical associations with sexual behavior, investment in long-term relationships, and impulsivity [22,23].

Another area of recent progress is the evolution of mood. Long-lasting mood states — including depression and

Figure 2



How emerging approaches to psychopathology relate to the four types of explanation in biology (see [11,12,57]) and to one another. RDoC = Research Domain Criteria.

anxiety — have been modeled as solutions to the trade-offs involved in pursuing rewards and avoiding punishments [24,25]. For example, depressed mood involves a high threshold for detecting and/or responding to potential rewards, which may reflect adaptive adjustment rather than maladaptive bias [24]. Evolutionary approaches to mood share much conceptual ground with computational models of depression [5,26]. The latter offer a sophisticated analysis of what can go wrong in valuation and decision processes, but concentrate on the short-term utility of behavior; the risk is to misconstrue adaptive responses that have high short-term costs but maximize fitness in the long run. Evolutionary research has identified several scenarios in which depressed mood may represent a costly but adaptive strategy — for example involuntary status loss, disengagement from unattainable goals, and social problems requiring sustained analytical thinking [27•]; these insight could inform computational analysis, facilitating the task of distinguishing between normal and pathological responses.

The evolution of developmental mechanisms

An important evolutionary contribution to the field of developmental psychopathology is the concept of *differential susceptibility* [28]. In a nutshell, individuals can be more or less sensitive to the effects of experience owing to a combination of genetic and early developmental factors, so that those who are more susceptible to adverse conditions are also more responsive to safe, supportive ones [29–31]. There are a number of plausible explanations for differential susceptibility [29,32,33], including the hypothesis that individual differences in plasticity are an adaptive response to unpredictable fluctuations in the environment [34•]. This line of research has generated a host of clinically relevant findings, including promising meta-analytic evidence that variation in serotonin transporter genotype contributes to generalized susceptibility [35] and that variation in serotonin-related and dopamine-related genes moderates the effect of behavioral interventions [36•].

More broadly, evolutionary research is beginning to change established ideas about what constitutes a risk factor and how early stress affects later development. A key tenet of this approach is that early adversity may not impair development so much as adaptively shape it, following evolved programs designed to maximize survival and reproduction in dangerous/unpredictable

contexts [37]. While this perspective does not negate the existence of costs and negative side effects [38], it shifts the focus of attention to the potential adaptive role of 'negative' outcomes such as impulsivity, anxiety, and cognitive biases. At the physiological level, it challenges the assumption that chronic stress exposure causes long-term dysregulation of neural processes. Although dysregulation can arise as a side effect, chronic stress also provides crucial information about the local environment, and contributes to entrain alternative developmental trajectories via the ubiquitous regulatory effects of stress hormones on growth, cognition, and behavior [39,40].

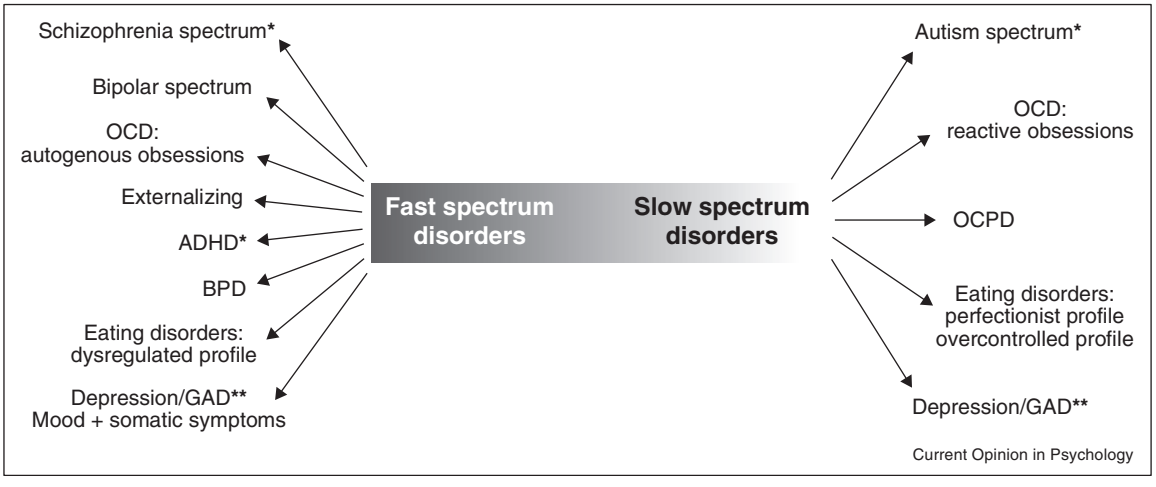
Finally, an evolutionary perspective affords unique insights into the development of sex differences in the prevalence of mental disorders. Martel [41] employed sexual selection theory to explain the higher prevalence of childhood-onset externalizing disorders in boys versus adolescent-onset internalizing disorders in girls, as well as clarify the developmental pathways that link prenatal and pubertal exposure to sex hormones to the regulation of serotonergic and dopaminergic activity in childhood and adolescence.

Life history theory and the classification of mental disorders

The classification of mental disorders is a central topic of psychopathological research. The DSM approach faces two main problems, that of *comorbidity* between disorders (supposedly distinct disorders tend to co-occur at high rates in the same individuals) and that of *heterogeneity* within disorders (diagnostic categories often include subgroups with different pathogenic mechanisms, risk factors, and so on). Researchers in developmental psychopathology have responded by advancing empirically grounded taxonomies of mental disorders. These taxonomies center on the distinction between *externalizing disorders* marked by antisocial and rule-breaking behaviors and *internalizing disorders* characterized by anxiety, fear, and distress. In addition, recent studies have identified a general *p factor* reflecting generalized susceptibility to psychopathology [42•,43,44]. On the computational side, there have been promising attempts to identify homogeneous subgroups of patients based on neurocognitive profiles [3,4•].

A novel development in evolutionary psychopathology is the proposal that *life history theory* may provide a biologically informed framework for the classification of mental disorders. Life history theory deals with the way organisms allocate time and energy to the activities that comprise their life cycle [45]. Life history models provide crucial insights into the evolution of biological trade-offs (e.g., early vs. late reproduction, investment in mating vs. parenting) and the way they shape developmental processes and schedules. Life history *strategies* are suites of morphological, physiological, and behavioral traits that implement life history allocations at the individual and species level. At the broadest level of description, life history-related traits tend to covary along a *fast–slow*

Figure 3



The current version of the life history taxonomy of mental disorders. Correlates of fast spectrum disorders include social antagonism, precocious/promiscuous sexuality, risk-taking, and impulsivity; low conscientiousness and agreeableness; early, fast maturation; and early exposure to stress and adversity. Slow spectrum correlates include social compliance, delayed/restrained sexuality, and risk aversion; high conscientiousness and agreeableness; late, slow sexual maturation; and relatively low exposure to ecological stressors [10••,51,52•]. One asterisk (*) indicates heterogeneous categories (e.g., ADHD is likely to include a smaller subgroup of slow spectrum conditions [52•]). Two asterisks (**) indicate non-specific conditions. OCD = obsessive-compulsive disorder. OCPD = obsessive–compulsive personality disorder. ADHD = attention deficit-hyperactivity disorder. BPD = borderline personality disorder. GAD = generalized anxiety disorder.Adapted from [10••].

continuum. In humans, dangerous and unpredictable environments tend to favor faster strategies characterized by early maturation and reproduction, sexual promiscuity, unstable relationships, impulsivity, risk taking, aggression, and exploitative tendencies, whereas safe and predictable environments tend to entrain slower strategies characterized by late maturation and reproduction, stable relationships, high self-control, aversion to risk, and prosociality. All these life history-related traits reflect the joint contribution of genetic and environmental factors [45,46].

Evolutionary researchers have long argued that some mental disorders — particularly those in the externalizing spectrum — can be interpreted as adaptive manifestations or maladaptive side-effects of fast strategies (e.g. [47–50]). More recently, I proposed that a unifying framework for psychopathology can be built on the distinction between *fast spectrum* and *slow spectrum* conditions ([10••,51]; Figure 3). In principle, the fast–slow distinction can help make sense of large-scale patterns of comorbidity, while providing a guide to detect functional heterogeneity within diagnostic categories. At present, the fast–slow distinction is mainly operationalized in terms of broad behavioral and personality traits (Figure 3); an important next step will be to connect those broad constructs to specific neurobiological, genetic, and computational profiles (including for example specific patterns of decision making and reward sensitivity).

Even at this initial stage, the framework has generated some promising results. In a simulation study, I showed that a model of mental disorders based on the fast–slow continuum can faithfully reproduce the observed structure of psychiatric disorders, including the internalizing–externalizing distinction and the emergence of a general p factor [52•]. In addition, simulations showed that the p factor may not be a unitary construct, and may arise as a combination of two largely independent dimensions of fast life history and reduced neurological integrity. It remains to be seen how the fast–slow distinction relates to other broad-band taxonomies, such as the distinction between disorders of *impulsivity* and disorders of *compulsivity* [53,54].

Conclusion

The future of psychopathology is taking shape at a rapid pace. Evolutionary psychopathology offers a framework for the study of mental disorders and allows complementary approaches to connect and cross-fertilize, as is beginning to happen in the medical sciences at large with the rise of evolutionary medicine [55,56].

Conflict of interest statement

None declared.

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