

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]).

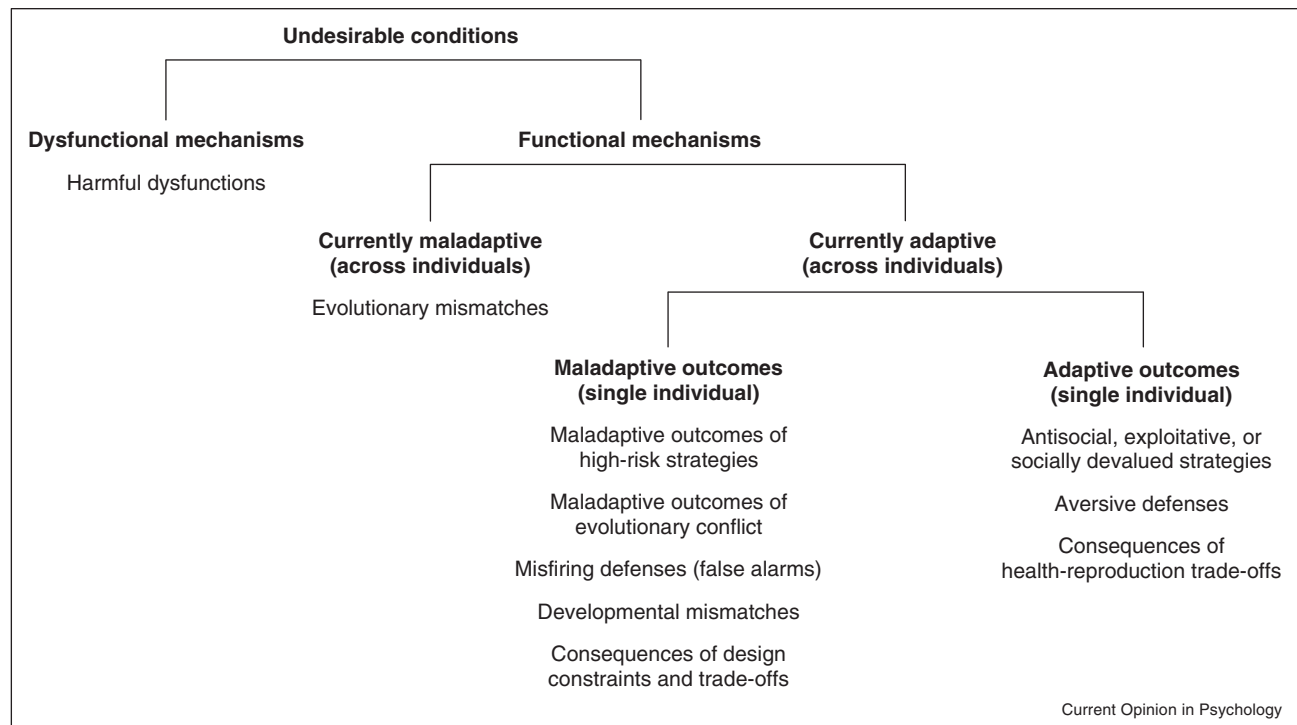
Here I focus on another emerging approach, that of *evolutionary psychopathology*. This approach draws on biological models and concepts to understand the functions of the neural and psychological processes involved in mental disorders and how they have been shaped by selection during our evolutionary 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 *mentalist 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

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