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(Article begins on next page)
An Evolutionary Life History Framework for Psychopathology

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Abstract

In this paper, I outline a general framework for the evolutionary analysis of mental disorders based on the concepts of life history theory. I synthesize and extend a large body of work showing that individual differences in life history strategy set the stage for the development of psychopathology. My analysis centers on the novel distinction between fast spectrum and slow spectrum disorders. I describe four main causal pathways from life history strategies to psychopathology, argue that psychopathology can arise at both ends of the fast-slow continuum of life history variation, and provide heuristic criteria for classifying disorders as fast or slow spectrum pathologies. I then apply the fast-slow distinction to a diverse sample of common mental disorders: externalizing disorders, schizophrenia and autism spectrum disorders, obsessive-compulsive disorders, eating disorders, and depression. The framework integrates previously disconnected models of psychopathology within a common frame of reference, and has far-reaching implications for the classification of mental disorders.

Keywords

Evolutionary psychopathology; fast-slow continuum; individual differences; life history strategies; life history theory; mental disorders.
Introduction

Evolutionary psychopathology is the application of evolutionary theory to the study of mental disorders, including their etiology, development, and classification. Evolutionary approaches are gaining momentum in both psychology and medicine (Buss, 2005, 2011; Dunbar & Barrett, 2007; Gluckman, Beedle, & Hanson, 2009; Stearns & Koella, 2008; Stearns, Nesse, Govindaraju, & Ellison, 2010), making psychopathology a natural candidate for theoretical integration. Evolutionary-minded researchers call for a revision of psychopathological theory and research, informed by a renewed understanding of the evolved functions of mental processes and their neurobiological substrates (Abed 2000; Brüne et al. 2012; Cosmides & Tooby 1999; Kennair, 2003; Nesse, 2001a, 2004a; Nesse & Jackson, 2006, 2011; Nesse & Stein, 2012; Troisi & McGuire, 2002).

Many competing evolutionary hypotheses on the origins and etiology of individual disorders have been advanced, and their predictions are being tested in an expanding empirical literature (see Adriaens & De Block, 2011; Brüne, 2008; McGuire & Troisi, 1998). However, the field as a whole is still highly fragmented (Kennair, 2003, 2011) and lacks organizing principles capable of explaining how disorders relate to one another and to the non-clinical range of individual differences in personality and behavior.

In this paper, I outline a framework for the evolutionary analysis of mental disorders based on the concepts of life history theory (Charnov, 1993; Kaplan & Gangestad, 2005; Stearns, 1992). For more than two decades, life history concepts have been applied to psychopathology, yielding insight into a broad range of conditions including psychopathy (Barr & Quinsey, 2004; Figueredo & Jacobs, 2010; Harris et al., 2007; Jonason et al., 2009; Jones & Paulhus, 2011; Lalumière, Mishra, & Harris, 2008; Mealey, 1995; Mishra & Lalumière, 2008); autism and schizophrenia (Del Giudice, Angeleri, Brizio, & Elena, 2010); borderline personality disorder (Brüne, Ghiassi, & Ribbert, 2010); attention deficit-hyperactivity symptoms (Frederick, 2012); internalizing and externalizing symptoms (Belsky, Steinberg, & Draper, 1991; Del Giudice, Ellis, & Shirtcliff, 2011); and eating disorders (Salmon, Figueredo, & Woodburn, 2009). The framework I present synthesizes and extends this body of work, based on the idea that individual differences in life history strategy—and specifically along the fast-slow continuum of life history variation—set the stage for the development of psychological symptoms and mental disorders.

Central to my analysis is the novel distinction between fast spectrum and slow spectrum psychopathology. As I will show, this distinction is a powerful tool for analyzing and classifying disorders based on deep functional principles rather than symptom similarity. The goal of the framework is not to explain mental disorders exclusively in relation to life history strategies, nor to replace other functional explanations of mental disorders. While the fast-slow continuum represents a fundamental dimension of individual differences, any satisfactory explanation of a mental disorder must involve multiple levels of explanation, from general functional principles to specific neurobiological mechanisms. The present framework aims to capture the broadest and most general level of this explanatory hierarchy. Accordingly, its goal is not to replace existing explanations but rather refine them, connect them to one another, and ultimately integrate them within a common frame of reference.
Overview of the Paper

I begin by introducing the basic concepts of life history theory in non-mathematical terms, and presenting an overview of the fast-slow continuum of life history variation as an organizing principle of individual differences. I then review the growing empirical literature on life history strategies and individual differences in motivation, self-regulation, and personality in humans. In the next section, I build on these ideas and findings to outline a general life history framework for psychopathology. I begin by describing four main causal pathways from life history strategies to mental disorders. I then argue that psychopathology can arise at both ends of the fast-slow continuum, and provide heuristic criteria for classifying disorders as fast or slow spectrum pathologies. Next, I apply the framework to a diverse set of mental disorders: externalizing disorders, schizophrenia spectrum disorders, autism spectrum disorders, obsessive-compulsive spectrum disorders, eating disorders, and depression. I conclude by exploring some implications of the framework for the classification of psychopathological conditions. In particular, I argue that the fast-slow distinction is both more inclusive and more accurate than the standard distinction between internalizing and externalizing disorders.

Terminological Notes

In evolutionary biology, the terms adaptive and maladaptive denote the effects of traits and behaviors on fitness—the differential replication of genes in subsequent generations. From the standpoint of an individual organism, adaptive traits enhance inclusive fitness, a function of the individual’s contributions to its own reproductive success and that of related individuals (see Grafen, 1985; West, Griffin, & Gardner, 2007). The biological notions of adaptation and maladaptation contrast sharply with how the same terms are usually employed in psychology and psychiatry. In these disciplines, the term “adaptive” refers to traits and behaviors that promote health, subjective well-being, and mutually rewarding social relations; socially undesirable, distressing, or health-damaging traits are viewed as maladaptive. Since natural selection promotes reproductive success rather than happiness or health (Cosmides & Tooby, 1999; Gluckman, Low, Buklijas, Hanson, & Beedle, 2011; Nesse, 2001a, 2004a), biologically adaptive traits may or may not be socially desirable or conducive to health and well-being. In this paper, I always employ the terms “adaptation” and “adaptive” in their biological sense.

Mental disorders are the main topic of this paper, yet the concept of disorder has no straightforward biological definition (Nesse, 2001a). In an influential paper, Wakefield (1992) advanced a biological analysis of disorders as harmful dysfunctions. A condition is a harmful dysfunction if (a) it is caused by the failure of a biological mechanism to perform its evolved function, and (b) it inflicts some harm or damage on the affected person, as judged by sociocultural standards (see also Wakefield, 1999, 2011). Current diagnostic systems in psychopathology emphasize harm over biological dysfunction; as a result, diagnosable mental “disorders” are likely to include harmful dysfunctions but also various other types of undesirable conditions. While many of those conditions may be clearly maladaptive, others may represent the outcomes of adaptive biological processes even if they have undesirable consequences (see Cosmides & Tooby, 1999; Gluckman et al., 2011; Nesse & Jackson, 2006). For the sake of simplicity as well as consistency with current diagnostic systems, in this paper I employ the term “disorder” in its conventional sense. Thus, for the present purposes, a condition may be labeled as
a disorder regardless of whether or not it reflects a harmful dysfunction, and—more generally—whether it reflects biologically adaptive or maladaptive processes.

Life History Theory and the Fast-Slow Continuum

Life history theory is a branch of evolutionary biology dealing with the way organisms allocate time and energy to the various activities that comprise their life cycle (see Charnov, 1993; Ellis, Figueredo, Brumbach, & Schlomer, 2009; Hill, 1993; Hill & Kaplan, 1999; Kaplan & Gangestad, 2005; McNamara & Houston, 1996; Stearns, 1992). All organisms live in a world of limited resources; for example, the energy that can be extracted from the environment in a given amount of time is intrinsically limited. Time itself is a limited good; the time spent by an organism looking for mates cannot be used to search for food or care for extant offspring. Since all these activities contribute to an organism’s evolutionary fitness, devoting time and energy to one will typically involve both benefits and costs, engendering trade-offs between different fitness components (Gadgil & Bossert, 1970; Williams, 1966). For example, there is a trade-off between bodily growth and reproduction because both require substantial energetic investment, and thus producing offspring reduces somatic growth. Natural selection favors organisms that schedule developmental tasks and activities so as to optimize resource allocation. Different allocation decision result in different life history strategies.

Life History Strategies

Life history strategies\(^1\) are adaptive solutions to fitness trade-offs within the constraints imposed by physical laws, phylogenetic history, and developmental mechanisms (Braendle, Heyland, & Flatt, 2011). At the most basic level, the resources of an organism must be distributed between somatic effort and reproductive effort. Somatic effort can be further subdivided into growth, survival and body maintenance, and developmental activity (Geary, 2002). Developmental activity includes play, learning, exercise, and other activities that contribute to building and accumulating embodied capital—strength, coordination, skills, knowledge, and so forth (Hill & Kaplan, 1999; Kaplan & Gangestad, 2005; Kaplan, Hill, Lancaster, & Hurtado, 2000). Reproductive effort can be subdivided into mating effort (finding and attracting mates, conceiving offspring), parenting effort (investing resources in already conceived offspring), and nepotistic effort (investing in other relatives).

The critical decisions involved in a life history strategy can be summarized by the fundamental trade-offs between current and future reproduction, between quality and quantity of offspring, and—in sexual species—between mating and parenting effort (see Ellis et al., 2009; Hill, 1993; Kaplan & Gangestad, 2005). By delaying reproduction, an organism can accumulate resources and/or embodied capital, thus increasing the quality and fitness of future offspring; however, the risk of dying before reproducing increases concomitantly. When reproduction occurs, the choice is between many offspring of lower quality and fewer offspring of higher 

\(^1\) The term “strategy” denote an organism’s realized phenotype among a set of possible phenotypes. Following what has become standard usage, I make no further distinctions between “strategies” and “tactics”. Adoption of a given strategy can depend on both environmental and genetic factors. It is important to stress that the term does not imply conscious planning, deliberation, or even awareness; an organism’s “choice” between alternative strategies can be implemented by low-level physiological means, such as a hormonal switch or a change in genetic expression.
An Evolutionary Life History Framework for Psychopathology

Quality. While intensive parental investment is a powerful way to increase the embodied capital (and long-term prospects) of one’s descendants, the fitness gains accrued through parenting must be weighed against the corresponding reduction in mating opportunities. Different life history strategies solve these problems in different ways by determining how organisms allocate effort among fitness-relevant traits. The same basic framework can be used to describe differences between species and between individuals of the same species (Réale et al., 2010; Sæther, 1987, 1988).

Life history strategies as organizers of physiology and behavior. The traits modeled in classical life history theory include growth rates, age and size at maturity, number and size of offspring, age-specific mortality rates, length of lifespan, and so forth (Stearns, 1992). However, life history strategies have a much broader range of correlates in an organism’s physiology and behavior. Indeed, life history strategies are best thought of as functionally complex phenotypes, resulting from the integration of a suite of morphological, physiological, and behavioral traits (e.g., Braendle et al., 2011).

In order to be adaptive, life history strategies must be functionally self-consistent. Imagine for example an animal whose life history strategy entails early reproduction and high mating effort in an ecological context of elevated mortality. To succeed in finding mates and reproducing, it needs to develop the morphological (e.g., size, muscle mass, fighting weapons) and behavioral traits (e.g., aggression, risk-taking) required to successfully challenge and outcompete its same-sex conspecifics. In the context of this strategy, investing in body maintenance at the expense of mating-related traits would be a waste of resources, given the low probability of long-term survival.

Life history strategies organize individual differences across domains, from physical growth and sexual maturation to social, sexual, and parental behavior. This requires physiological mechanisms capable of coordinating the development of life history-related traits in an integrated, adaptive fashion—often through endocrine signaling pathways (Braendle et al., 2011; Finch & Rose, 1995; Ricklefs & Wikelski, 2002). Sex hormones are crucially involved in the management of life history trade-offs, both in humans and in nonhuman animals (e.g., Bribescas, Ellis, & Gray, 2012; Gettler, McDade, & Kuzawa, 2011; Hau, Ricklefs, Wikelski, Lee, & Brawn, 2010; Worthman & Brown, 2005). Another vital role is played by the stress response system, which participates in the regulation of most life history-related traits, including growth and maturation, fertility, immune function, risk-taking, pair-bonding, and so forth (reviewed in Del Giudice et al., 2011; Worthman, 2009; Worthman & Kuzara, 2005). There is extensive cross-talk between the stress response system and the hypothalamic-pituitary-gonadal axis, and both interact bidirectionally with the major neuromodulator systems—including dopaminergic, serotonergic, and oxytocinergic pathways (see Alexander et al., 2011; Beauchaine, Neuhaus, Zalewski, Crowell, & Potapova, 2011; Ellis, 2004; Flinn, Nepomnaschy, Muehlenbein, & Ponzi, 2011; Korte, Koolhaas, Wingfield, & McEwen, 2005; Porter, Gallagher, Watson, & Young, 2004; van Goozen, Fairchild, Snoek, & Harold, 2007).

Sex differences in life history trade-offs. The asymmetries introduced by sexual reproduction have important implications for the life histories of males and females. For example, in most species males tend to engage in higher mating effort and lower parental effort than
females (Geary, 2002; Kokko & Jennions, 2008; Trivers, 1972). In addition, males usually undergo stronger sexual selection, that is, their reproductive success is more variable than that of females; they also tend to mature more slowly, in order to gain the competitive abilities and qualities needed for successful competition for mates. Sexual asymmetries in life history strategies can be attenuated in species with monogamous mating systems and when both parents contribute to offspring care. Compared with other mammals, humans show an unusually high degree of paternal investment; we are clearly adapted for the possibility of monogamous, long-term relationships. However, human paternal care is also highly variable and facultative (e.g., Geary, 2005; Quinlan, 2008), and strict monogamy is rarely, if ever, found (Marlowe, 2000, 2003). The reproductive success of men is more variable than that of women, especially in societies characterized by polygyny or serial monogamy (Brown, Laland, & Borgerhoff Mulder, 2009). Overall, human mating is best characterized as strategically flexible (Gangestad & Simpson, 2000), with a widely documented tendency for men to engage in higher mating effort than women (e.g., Schmitt, 2005).

As a result, the trade-off between current and future reproduction is more pressing for women than for men: women’s reproductive rate is limited by the long duration of gestation and the considerable energetic investment of pregnancy and lactation, and their window for successful reproduction necessarily ends with menopause. In contrast, men can potentially sire many offspring in a very short time, as well as for a more extensive period of their lives. Men’s crucial trade-off is the one between mating and parenting: the payoffs of high mating effort are potentially much larger for males, who can benefit directly from having access to a large number of partners; women can usually have only one child at a time, and thus benefit comparatively less from mating with multiple partners (see Bribescas et al., 2012).

The Fast-Slow Continuum

Because life history trade-offs are not functionally independent of one another, differences in life history strategies between and within species show a general pattern of trait covariation. Slow growth and late reproduction correlate with long lifespan, high parental investment, fewer offspring of higher quality, and low juvenile mortality. Conversely, fast growth and early reproduction correlate with high juvenile mortality, short lifespan, larger numbers of offspring and reduced parental investment in each. This is commonly referred to as the fast-slow continuum of life history variation (Sæther 1987, 1988; see Ellis et al., 2009; Jeschke & Kokko, 2009). Despite some exceptions and caveats (see Jeschke & Kokko, 2009; Réale et al., 2010), the same general pattern holds both across and within species.

The fast-slow continuum has profound implications for the organization of behavior. A short lifespan, higher mortality, and early reproduction make it optimal to discount future rewards and favor short-term gains over long-term benefits; future-oriented behavior is only beneficial in the context of slow strategies. Furthermore, organisms betting on future reproduction must maximize their chances of surviving and remaining healthy. This is best obtained through risk aversion—that is, avoidance of variable rewards in favor of surer outcomes, even at the price of a lower average payoff. Wolf and colleagues (Wolf, van Doorn, Leimar, & Weissing, 2007) formally showed that individual differences in present- vs. future-oriented strategies should result in consistent individual differences in risk-related traits, such as boldness, exploration, and
aggression (see also Hill, 1993; Hill, Ross, & Low, 1997; Stamps, 2007). More generally, the fast-slow continuum is emerging as an integrative concept for understanding coordinated bundles of metabolic, hormonal, immunity, and behavioral/personality traits in nonhuman animals (Réale et al., 2010; see also Wolf & McNamara, 2012).

In species with complex social lives, the implications of the fast-slow continuum extend beyond risk-related traits to include cooperation, reciprocity, and pair-bonding. The benefits of cooperation and reciprocity are usually reaped in the long term, and may require forgoing immediate gains, whereas antagonistic and exploitative behaviors have short-term benefits but carry the possibility of long-term damage. In species with biparental care, stable pair-bonding promotes intensive investment by both parents, and often involves trading present reproductive opportunities for enhanced reproductive success in the future. Slow strategies should then be associated with increased cooperation, the disposition to enter reciprocal relationships, and the formation of stable mating pairs.

**Determinants of individual life history variation.** Life history traits and strategies tend not to be genetically fixed, but rather show adaptive developmental plasticity (see Belsky et al., 1991; DeWitt & Scheiner, 2004; Kuzawa & Bragg, 2012; Pigliucci, 2001; West-Eberhard, 2003). Adaptive plasticity in life history strategies means that developing organisms assess their local environment (based on contextual cues) and adjust their allocation decisions accordingly, following evolved rules that maximize expected fitness in different ecological conditions (McNamara & Houston, 1996).

The key dimensions of the environment that affect the development of life history strategies are resource availability, extrinsic morbidity-mortality, and unpredictability, as signaled by observable cues (see Ellis et al., 2009; Kuzawa & Bragg, 2012). Energetic conditions—caloric intake, energy expenditures, and related health conditions—set a baseline for many developmental processes, including development of life history strategies. Evolutionary biologists and psychologists (e.g., Ellison, 2001; MacDonald, 1997, 1999; Surbey, 1998) have argued that energetic stress causes the developing person to shift toward a slower life history strategy. This translates into development of a more energy-sparing phenotype, including slower growth, delayed sexual maturation, and low fecundity.

Development of fast strategies depends on adequate bioenergetic resources to support growth and development. Once this energetic threshold is crossed, other environmental conditions become salient determinants of life history strategy (Ellis et al., 2009). Extrinsic morbidity-mortality constitutes external sources of disability and death that are relatively insensitive to the adaptive decisions of the organism. Environmental cues indicating high levels of extrinsic morbidity-mortality cause individuals to develop faster life history strategies (Belsky et al., 1991; Chisholm, 1993, 1999a; Pennington & Harpending, 1988; Placek & Quinlan, 2012; Quinlan, 2007). Faster strategies in this context—a context that devalues future reproduction—function to reduce the risk of disability or death prior to reproduction. Accordingly, exposure to environmental cues indicating extrinsic morbidity-mortality (i.e., observable cues that reliably covaried with morbidity-mortality risks during our evolutionary history, such as exposures to violence, dangerous ecological conditions, or harsh childrearing practices) can be expected to shift life history strategies toward current reproduction by anticipating maturation and onset of
sexual activity (Belsky et al., 1991). Moreover, high extrinsic morbidity-mortality means that investing in parental care has quickly diminishing returns, which favors reduced parental investment and offspring quantity over quality. While adult and juvenile mortality rates have somewhat different implications for life history development, they tend to be highly correlated in humans, making the distinction less relevant for our species (Ellis et al., 2009; Jones, 2011).

In addition to average levels of extrinsic morbidity-mortality, unpredictable variation in extrinsic morbidity-mortality over time and space—environmental unpredictability—also regulates life history development. The effects of unpredictability are more complex and nuanced than those of morbidity-mortality per se (Ellis et al., 2009). Unpredictable environments can lead organisms to invest in behavioral flexibility and adaptability; this has probably been a factor in the evolution of human traits such as a large brain, protracted development, and an extended learning period (see Chiappe & MacDonald, 2005; Jones, 2011; Potts, 1998). On the timescale of human development, however, variable and unpredictable contexts tend to entrain faster life history strategies, thus acting in the same direction of environmental harshness (Belsky, Schlomer, & Ellis, 2012; Brumbach, Figueredo, & Ellis, 2009; Ellis et al., 2009; Ross & Hill, 2012; Simpson, Griskevicius, Kuo, Sung, & Collins, 2012). Conversely, safe and predictable environments promote the development of slow life history strategies.

Environmental and genetic factors jointly contribute to determine an organism’s life history strategy. Theoretical models suggest that environmental and genetic effects on life history strategies should often coexist (e.g., Leimar, Hammerstein, & Van Dooren, 2006). While there is no room here for even a cursory treatment of this topic (see DeWitt & Scheiner, 2004; Ellis et al., 2009; Roff, 2002), it is important to note that all the life history traits studied in humans so far show at least moderate heritability (e.g., Figueredo, Vásquez, Brumbach, & Schneider, 2004; Kirk et al., 2001; MacDonald, 1997; Pettay, Kruuk, Jokela, & Lummaa, 2005). Furthermore, mechanisms of epigenetic inheritance may transmit environmental effects on life history strategies across multiple generations (Bateson et al., 2004; Champagne, 2010).

**Life History Strategies and Individual Differences in Humans**

The idea that life history theory may serve as an organizing framework for human individual differences was first advanced by Rushton (1985, 1987; Bogaert & Rushton, 1989), and subsequently framed in a developmental perspective by Belsky and colleagues (1991) and Chisholm (1993, 1999a). Belsky and colleagues hypothesized that harsh parenting, conflictual family relations, and insecure attachment would predict early sexual maturation, impulsivity, reduced cooperation, and exploitative interpersonal styles—the expected correlates of a fast life history strategy. Empirical studies have confirmed these associations and detailed how harsh parenting and insecure attachment predict early puberty (in females), precocious sexuality, unstable couple relationships, and promiscuous mating styles (reviewed in Belsky, 2012; Del Giudice, 2009; Gillath & Schachner, 2006; James, Ellis, Schlomer, & Garber, 2012). Chisholm (1999b; Chisholm, Quinlivan, Petersen, & Coall, 2005) found correlations between insecure attachment, present orientation (the inability to delay gratification and/or wait for larger rewards in the future), and shorter subjective life expectancy in adult women. In turn, present orientation and a shorter expected lifespan predicted earlier onset of sexual activity, a larger number of
sexual partners, and earlier age at first birth, consistent with a strategy of early reproduction and high mating effort (see also Laghi, D’Alessio, Pallini, & Baiocco, 2009).

The association between shorter life expectancy and early childbearing has been confirmed by epidemiological studies (Copping, Campbell, & Muncer, 2013; Nettle, 2011a). At the individual level, present orientation, impulsivity, and a short subjective life expectancy are all robustly associated with increased risk-taking, reduced cooperation, deviance, antisocial behavior, earlier intercourse, and larger numbers of sexual partners (e.g., Borowsky, Ireland, & Resnick, 2009; Brezina, Tekin, & Topalli, 2009; Chen & Vazsonyi, 2011; Curry, Price, & Price, 2008; Dunkel & Decker, 2010; Harris & Madden, 2002; Hill et al., 1997; Kahn, Kaplowitz, Goodman, & Emans, 2002; Kruger, Reischl, & Zimmerman, 2008; Lejuez et al., 2002; Wang, Kruger, & Wilke, 2009; White et al., 1994). These results strongly support the existence of a fast-slow dimension underlying a broad spectrum of individual differences. As predicted, the development of fast strategies is favored by the experience of harsh and unpredictable contexts (Belsky et al., 2012; Copping et al., 2013; James et al., 2012; Nettle, Coal, & Dickins, 2011; Simpson et al., 2012); in addition, attachment insecurity seems to be an important psychological mediator of these effects (see above).

Following a psychometric approach, Figueredo and colleagues (2004, 2005; Figueredo, Vásquez, Brumbach, & Schneider, 2007; Figueredo, Cabeza de Baca, & Woodley, 2012a) identified a heritable general factor accounting for a large proportion of variance in psychological traits reflecting a slow life history strategy. These traits include reciprocal, secure relationships with parents, partners, and friends; restricted sociosexuality (reduced desire for short-term, promiscuous sexual relationships); long-term planning, foresight, and persistence; responsibility and altruism; and religiosity and/or communitarian beliefs. Life history theory provides a functional explanation of why these traits covary with one another along a fast-slow dimension. Slower strategies predicts high investment and satisfaction in long-term romantic relationships, loyalty to the in-group, and low levels of interpersonal aggression and social deviance (Figueredo & Jacobs, 2010; Figueredo, Andrzejczak, Jones, Smith-Castro, & Monetro-Rojas, 2011; Figueredo, Gladden, & Beck, 2012b; Jones, Figueredo, Dickey, & Jacobs, 2007; Olderbak & Figueredo, 2010).

**Life history strategies and self-regulation.** Self-regulation occupies a central place in the network of life history-related traits. Deliberate control of behavior is required in order to engage in long-term relationships and cooperative enterprises, refrain from short-term sexual opportunities, avoid immediate risks, and so on. Low levels of self-control are primarily reflected in the construct of impulsivity and its two main facets, present orientation and lack of behavioral inhibition (Avila, Cuenca, Félix, Parcet, & Miranda, 2004; Reynolds, Ortengren, Richards, & de Wit, 2006). Behavioral inhibition is one of the main executive functions, a set of cognitive processes that underlie goal-directed behavior and depend strongly—though not exclusively—on prefrontal activity (Diamond, 2013; Miyake et al., 2000). The standard taxonomy of executive functions distinguishes between inhibition (deliberate overriding of dominant or prepotent responses), updating (constant monitoring and rapid addition/deletion of working memory contents), and shifting (switching flexibly between tasks or mental sets). Inhibition seems to work as a common factor in regulatory abilities, and accounts for most of the covariation between different executive functions (Miyake & Friedman, 2012).
Generally speaking, antisocial behavior is robustly associated with reduced executive performance (Morgan & Lilienfeld, 2000). In a number of studies, self-reported executive functions correlated strongly with measures of life history strategy, and appeared to mediate the impact of life history strategy on behavioral outcomes, including antisocial behavior and disordered eating (see Figueredo & Jacobs, 2010; Salmon et al., 2009; Wenner, Bianchi, Figueredo, Rushton, & Jacobs, 2013). However it must be noted that, while measures of behavioral inhibition and (to a lesser degree) memory updating show robust associations with impulsivity and self-control, measures of task shifting show no consistent relation with either (Hoffmann, Schmeichel, & Baddeley, 2012). Indeed, the ability to delay gratification—a key behavioral facet of slow strategies—has been associated with higher inhibition but lower shifting ability (see Miyake & Friedman, 2012). In all likelihood, behavioral inhibition is the key mediator of the association between executive functions and other life history-related traits. Supporting evidence comes from studies showing that inhibitory control predicts cooperativeness, empathy, and the ability to remain faithful to a romantic partner (Hansen, 2011; Pronk, Karremans, & Wigboldus, 2011). Moreover, motor inhibition tasks are especially strong predictors of antisocial behavior (Morgan & Lilienfeld, 2000), and the tendency to act without thinking is an especially strong predictor of risk-taking (Romer et al., 2011).

**Life history strategies and personality traits.** Personality traits reflect stable individual differences in motivation, behavioral dispositions, and self-regulation. As such, they show robust and predictable associations with the fast-slow continuum. In the framework of the Five Factor Model of personality (Costa & McCrae, 1995), the strongest associations are found between the personality factors of conscientiousness and agreeableness and slow strategy indicators such as restricted sociosexuality, relationship stability, risk aversion, and prosocial behavior (reviewed in Del Giudice, 2012; see also Holtzman & Strube, 2013). In addition, conscientiousness is a reliable predictor of longevity, in part because of its effects on health-related behavior (e.g., Bogg & Roberts, 2004; Chapman & Goldberg, 2011; Friedman, 1995; Martin, Friedman, & Schwartz, 2007; Weiss & Costa, 2005). Conscientiousness, agreeableness, and emotional stability (the reverse of neuroticism) load on a single higher-order factor (“metatrait”) called alpha or stability (Digman, 1997; DeYoung, 2006). As expected of a marker of slow life history strategy, alpha is a strong negative predictor of impulsivity (DeYoung, 2011; DeYoung, Peterson, & Higgins, 2002).

In contrast with agreeableness and conscientiousness, the personality factors of extraversion, openness to experience, and neuroticism (i.e., low emotional stability) correlate to various degrees with unrestricted sociosexuality, short-term mating, relationship instability, and risk-taking, as well as aggressive, disruptive, and antisocial behavior (Del Giudice, 2012). However it should be noted that, in a life history perspective, extraversion and openness are “hybrid” traits that include both fast-type and slow-type components. Some facets of extraversion tap warmth and affiliation, while others tap dominance and sensation seeking (Lucas, Deiner, Grob, Suh, & Shao, 2000; MacDonald, 1995); only the latter are functionally related to fast life history strategies. Consistent with this view, extraversion has been found to correlate with both short- and long-term mating orientation (Holtzman & Strube, 2013). Similarly, openness has two main facets, intellect and imagination (see Nettle, 2011b). Imagination correlates with positive schizotypy (see below), which in turn predicts unrestricted sociosexuality, reduced commitment in long-term relationships, and larger numbers of sexual partners (Nettle & Clegg, 2006; Del Giudice et al., 2010). Extraversion and openness load on metatrait beta or plasticity, which is a
positive predictor of sensation seeking and a negative predictor of self-control (DeYoung, 2011; DeYoung et al., 2002). Because of the hybrid content of extraversion and openness, however, metatrait beta cannot be univocally linked to the fast spectrum of life history strategies (Del Giudice, 2012).

At an even higher level of abstraction, it is possible to identify a general factor of personality (GFP; Musek, 2007), which has been proposed as a correlate of slow life history and high parenting effort (Figueredo et al., 2007; Rushton, Bons, & Hur, 2008). The GFP is positively correlated with both alpha and beta; however, there is still no consensus as to whether the GFP represents a methodological artifact or a real feature of human personality (e.g., Ashton, Lee, Goldberg, & de Vries, 2009; Just, 2011; Loehlin & Martin, 2011). Future research will hopefully clarify the ontological status of the GFP and determine its relevance to life history models of individual differences.

An illustrative example. The functional coherence of individual differences across domains is nicely illustrated by the longitudinal study of boys’ development by Moffitt and colleagues (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996). These authors identified a group of males (labeled “abstainers”) characterized by the virtual absence of antisocial behavior in childhood and adolescence. At 18 years of age abstainers were good students but also overcontrolled, fearful, timid, socially awkward, and likely to be virgins. At the age of 26, however, they had become successful adults in terms of education, occupational status, and economic security—in stark contrast with their highly antisocial peers (Moffitt & Caspi, 2005).

Abstainers were more likely to be married than any other group and enjoyed happy couple relationships; at the same time, they tended to delay having children, and—if fathers—had fewer children than their more antisocial counterparts. Their personality profiles showed high scores on all the facets of alpha (agreeableness, emotional stability, and conscientiousness). Abstainers also displayed low rates of psychopathology and problem behaviors, especially in comparison with highly antisocial males (Moffitt & Caspi, 2005). In short, abstainers bear the hallmarks of an extreme slow strategy, and exemplify the coordinated interplay of personality, self-regulation, sexuality, and attachment in the pursuit of long-term biological goals.

A Life History Framework for Psychopathology

In the previous section I discussed how life history strategies play a central role in the organization of physiology and behavior. They define the organism’s priorities and determine the allocation of effort and resources toward competing biological goals. Differences in life history strategy are the joint product of genetic and environmental influences on development, and are reflected in organized patterns of individual differences in motivation, affect, self-regulation, and personality. By organizing individual differences on such a broad scale, life history strategies set the stage for the development of psychopathology. More precisely, individual differences in life history strategy can be expected to determine individual differences in risk profiles for a broad range of mental disorders. As one moves along the fast-slow continuum of life history variation, some disorders and symptoms should become more frequent, while others should become less likely to occur. This is the functional basis for the distinction between fast spectrum and slow
spectrum disorders—that is, disorders that cluster at the fast or slow end of the life history continuum.

It is crucial to stress at the outset that, in this framework, the functional connection between life history strategy and psychopathology is an indirect one. As I discuss in detail below, causal pathways to psychopathology involve a multiplicity of traits and mechanisms—including temperament and personality, self-regulatory processes, and so forth. The general idea is that an individual’s configuration of life history-related traits may increases the likelihood of developing a certain disorder or cluster of disorders—often in interaction with other causal factors including developmental insults, deleterious genetic and/or epigenetic mutations, infections, nutritional deficits, and psychosocial stressors. The power of life history theory lies in the ability to integrate these diverse etiological processes within a common frame of reference. The result is a large-scale map of the psychopathological landscape organized along the fast-slow axis of life history variation. Such a map is an invaluable guide in understanding comorbidity patterns, since functionally related disorders—for example different disorders in the slow spectrum—can be expected to co-occur more frequently within the same individual. At the same time, the fast-slow distinction can be used to tease apart functionally distinct conditions that coexist within the same descriptive category. For example, later on I will argue that the diagnostic label of obsessive-compulsive disorder (OCD) comprises at least two functionally distinct clusters of conditions—a fast spectrum cluster characterized by endogenous obsessions and a slow spectrum cluster characterized by reactive obsessions (see Lee & Kwon, 2003). In total, a life history analysis helps “carving nature at its joints” by revealing commonalities between separate categories and suggesting important distinctions between phenotypically similar disorders (Keller & Nesse, 2006).

Mental disorders are complex biosocial phenomena, and as such they can be analyzed at many different levels. Needless to say, the broad perspective afforded by life history theory should be complemented by narrower functional accounts focusing on specific motivational/behavioral systems, cognitive mechanisms, genetic pathways, and so forth. With each narrower level of analysis, enhanced resolution may be gained at the cost of reduced generality. My present goal is to outline a framework as general and abstract as possible, while keeping in mind that a comprehensive evolutionary account of psychopathology will have to include a detailed model of human motivational and affective systems, specialized cognitive processes, and their neurobiological and molecular underpinnings.

Psychopathology and the Fast-Slow Continuum

So far, life history approaches to psychopathology have focused almost exclusively on the fast end of the fast-slow continuum. It is increasingly recognized that fast life history strategies can predispose individuals to a variety of disorders, either as maladaptive outcomes of life history-related traits or potentially adaptive but undesirable behavioral strategies (e.g., Barr & Quinsey, 2004; Belsky et al., 1991; Brüne et al., 2010; Figueredo & Jacobs, 2010; Frederick, 2012; Jonason, Li, Webster, & Schmitt, 2009; Mealey, 1995; Salmon et al., 2009). As I discuss below, there are indeed reasons to expect a disproportionate amount of pathology in association with fast life history strategies. However, most current models fail to address the potential role of slow strategies in setting the stage for the development of mental disorders (for an exception see
An Evolutionary Life History Framework for Psychopathology

Del Giudice et al., 2010). By their very nature, life history trade-offs involve costs in both directions. While the costs associated with fast strategies may appear more dramatic, those associated with slow strategies are no less real, nor less consequential for evolutionary dynamics. In applying life history theory to psychopathology, it is a mistake to idealize slow strategies by underplaying their potential costs in terms of both biological fitness and psychological well-being.

By their very nature, life history trade-offs involve costs in both directions. While the costs associated with fast strategies may appear more dramatic, those associated with slow strategies are no less real, nor less consequential for evolutionary dynamics. In applying life history theory to psychopathology, it is a mistake to idealize slow strategies by underplaying their potential costs in terms of both biological fitness and psychological well-being.

Consider for example inhibitory control and future orientation, two core psychological correlates of slow strategies. Individuals high on these dimensions are inevitably less able to take advantage of unexpected opportunities in the present, and may find it more difficult to adapt to changing or novel circumstances (Block & Block, 1980; Dickman, 1990). In other words, there are opportunity costs associated with high levels of self-regulation. If immediate impulses are suppressed in view of future rewards, there is an unavoidable risk that future rewards may never materialize. Also, self-control can lead to rigidity and conformity—the so-called “neuroses of health” (DeYoung et al., 2002). Thus, while it can be highly adaptive, self-regulation is clearly not an unmixed blessing (see also Block & Block, 1980; Eisenberg et al., 2001; Huey & Weisz, 1997; Robins, John, Caspi, Moffitt, & Stouthamer-Loeber, 1996).

Prosocial attitudes such as cooperativeness present a similar mixture of benefits and costs. While prosociality can be hugely rewarding, it also makes people vulnerable to cheating and exploitation, with potentially devastating consequences. Prosociality also has opportunity costs: while highly prosocial individuals are well-liked, they seldom reach the top of social hierarchies—unless they supplement prosociality with coercive and manipulative tactics (Hawley, 1999, 2011; Hawley, Little, & Card, 2008; Lease, Musgrove, & Axelrod, 2002). The moral emotions that motivate and regulate prosocial behavior include guilt, shame, and anger (Haidt, 2003; Rozin, Lowery, Imada, & Haidt, 1999; Tangney, Stuewig, & Mashek, 2007). These emotions are far from innocuous—in fact, they can become painful, consuming, and even disabling. Recent work on “pathological altruism” (see Oakley, Knafo, Madhavan, & Wilson, 2012) provides many vivid examples of the dark side of prosociality.

The cost-benefit balance of slow life history strategies is well illustrated by research on overcontrolled personality types (Block & Block, 1980). As the label suggests, overcontrolled individuals are characterized by low impulsivity and low behavioral flexibility. They are low in extraversion and openness and high in agreeableness and conscientiousness; they are prosocial, well-liked, sensitive to criticism, and display very low levels of aggression (Asendorpf & van Aken, 1999; Chapman & Goldberg, 2011; Robins et al., 1996). In other words, they fall squarely at the slow end of the fast-slow continuum. However, there is converging evidence that overcontrol is associated with higher risk for pathological conditions such as anxiety disorders and depression (e.g., Eisenberg et al., 2001; Huey & Weisz, 1997). Similarly, the highly self-controlled “abstainers” studied by Moffitt and colleagues (see above) grew up to become remarkably successful in many domains of life; however, they were not immune from depression and anxiety disorders, even if they experienced them at low rates relative to other groups (Moffitt & Caspi, 2005). A recent study by Sherman and colleagues (Sherman, Figueredo, & Funder, in press) showed that, when the confounding effects of behavioral normativeness are controlled for, slow life history traits tend to be associated with higher levels of social awkwardness, insecurity, and over-controlling personality traits.
From Life History Strategies to Psychopathology: Four Causal Pathways

The general statement that life history strategies set the stage for the development of psychopathology can be supplemented by a finer-grained analysis of the causal pathways that lead to the onset of mental disorders. Here I will consider four such pathways: (a) adaptive life history-related traits may be regarded as symptoms; (b) life history-related traits may be expressed at maladaptive levels; (c) adaptive strategies may yield individually maladaptive outcomes; and (d) adaptive life history-related traits may increase vulnerability to dysfunction. These pathways are logically distinct but not mutually exclusive, and may coexist in the etiology of any given disorder.

Adaptive life history-related traits may be regarded as symptoms. Sometimes, a phenotypic strategy may involve the expression of biologically adaptive traits that are nevertheless regarded as pathological (Nesse, 2004; Nesse & Jackson, 2006; Troisi, 2005). This is most likely to happen with fast life history strategies characterized by impulsive, exploitative, or aggressive tendencies. The resulting phenotype may be classified as a disorder, even if it does not reflect maladaptive or dysfunctional processes. Even if they are biologically adaptive, or used to be adaptive in ancestral environments, such strategies may often involve substantial costs in terms of health and emotional well-being. For example, it has been hypothesized that some forms of psychopathy should be regarded as adaptive strategies that allow psychopaths to increase their own reproductive success by exploiting others (e.g., Mealey, 1995). Even if “successful” psychopaths may enjoy high biological fitness, psychopathy is a source of trouble for society at large, and is legitimately regarded as a condition in need of treatment. In a life history framework, many apparent dysfunctions associated with psychopathy (e.g., reduced empathy, lack of guilt, impulsivity) may be better understood as design features of an extreme fast-spectrum strategy.

Another important category of adaptive traits that may be diagnosed as symptoms of a disorder is that of aversive defenses. Broadly speaking, defenses can be defined as mechanisms designed to protect individuals from physical and/or social harm. Most negative emotions—including fear, anxiety, disgust, and shame—can be conceptualized as defensive mechanisms, as they play crucial protective roles against physical danger, contamination by pathogens, social exclusion, and so forth (see Nesse, 2004; Nesse & Jackson, 2006). When defenses activate inappropriately and/or respond with excessive intensity, the outcome may be correctly recognized as maladaptive (see below). However, many protective mechanisms have strongly aversive effects (e.g., fever, vomiting, panic), and can be occasionally harmful to the individual. For this reason, they may give rise to undesirable conditions not only when they misfire but also when they respond appropriately in presence of actual threats. Sometimes, defensive processes can be altogether mistaken for disorders, especially if their logic is incompletely understood. Indeed, the “fallacy of mistaking defenses for diseases” is a pervasive feature of current diagnostic approaches (Nesse & Jackson, 2006).

The correlates of life history strategies often include up- or down-regulation of psychological and physiological defensive mechanisms. Up-regulated defenses have a lower
threshold for activation and/or respond with higher intensity when they activate. Defense up-regulation can be associated with both fast and slow strategies, although the specific type of mechanism involved is likely to differ between the two. In the context of fast life histories, sensitive defenses help protect the individual from immediate danger in risky, unpredictable environments. In the context of slow strategies, up-regulated defenses may help the individual prevent dangerous events and avoid potentially risky situations, even if the current environment is reasonably safe. Moreover, protecting oneself from even minor damages and losses contributes to the long-term maintenance of somatic investment—a key priority for slow life history individuals.

Whereas up-regulated defenses are an obvious source of pathological conditions, the effects of down-regulated defenses can be just as problematic. As pointed out by Nesse (1990; Nesse & Jackson, 2006), the fact that people rarely complain about “too little anxiety” or the “inability to feel fear” does not mean that such conditions do not exist or cannot be harmful to individuals, as well as their relatives and social partners. Down-regulation of defenses is most likely in the context of fast life history strategies, especially those involving a high degree of risk-taking. The underlying logic is that, in order to fulfill their purpose, such strategies require outright insensitivity to threats, dangers, social feedback, and so forth. For an extreme risk-taker, informational insulation from signals of threat can be an asset, not a weakness (see Del Giudice et al., 2011; Korte et al., 2005). The same logic can be applied to multiple domains. For example, the emotion of disgust is a behavioral defense against contamination and infection, and has been co-opted in the regulation of sexual behavior by increasing selectivity of mate choice (Rozin, Haidt, & McCauley, 2000; Schaller, 2011; Tybur, Lieberman, & Griskevicius, 2009). High sensitivity to disgust (especially sexual disgust) would interfere with a strategy of promiscuous, indiscriminate mating strategies; but while insensitivity to disgust may be adaptive in this context, it also increases the risk of contracting sexually transmitted diseases, with potentially severe fitness costs for the individual (Schaller, 2011).

**Life history-related traits may be expressed at maladaptive levels.** Even phenotypic traits that are biologically adaptive within a certain range may become maladaptive if they exceed the limits of that range. Sometimes, the expected fitness associated with a trait may slowly increase up to an optimal level, then decrease abruptly following a “cliff-edged” function. In such cases, selection for optimal trait levels may result in a high frequency of maladaptive phenotypes that overshoot the fitness optimum (Nesse, 2004b).

A trait can reach maladaptive expression levels owing to a combination of genetic, epigenetic, and environmental factors that contribute to push the phenotype in the same direction. In the simplest case, extreme levels of a trait may appear in the offspring of two individuals who are both high on that trait, yet still within the adaptive range. Thus, assortative mating—the tendency for mates to be more similar than average on a certain trait—can increase the risk for psychopathology due to extreme trait values. In general, when a disorder is caused by maladaptive expression of traits with substantial additive genetic and/or shared environmental variance, the relatives of the affected individual can be expected to show the same traits in a milder and probably adaptive form. Parent-offspring conflict and intragenomic conflict (see Schlomer, Del Giudice, & Ellis, 2011) are other likely causes of maladaptive trait expression. When evolutionary conflict is present, phenotypic development can be pictured as the result of
opposing forces, much like a game of tug-of-war. If for any reason this dynamic equilibrium is broken (for example because a mutation in the offspring makes it unable to counteract parental manipulation), the resulting unbalance may easily determine dysregulated or pathological outcomes.

In principle, the pathway leading from maladaptive trait expression levels to psychopathology may involve traits associated with both fast and slow life histories. However, there is some evidence that assortative mating on life history-related traits in humans tends to become stronger toward the slow end of the continuum (Figueroed & Wolf, 2009). If so, disorders that involve maladaptive expression levels of adaptive traits should occur more frequently in association with slow strategies, as similarity between parents increases the likelihood that offspring will inherit extreme genotypic combinations.

**Adaptive strategies may yield individually maladaptive outcomes.** In general terms, whether a trait is biologically adaptive or maladaptive depends on its overall contribution to an organism’s reproductive success. However, it is important to distinguish between the fitness contribution of a trait or strategy—averaged across all the individuals who express it—and the fitness of a particular individual. This distinction is crucial because a behavioral or developmental strategy may be fitness-enhancing on average, while imposing large fitness costs on some individuals (e.g., Cosmides & Tooby, 1999; Frankenhuiss & Del Giudice, 2012). In some cases, a strategy can be selected for even if most individuals who adopt it end up suffering severe damage—provided that potential losses are balanced by outstanding rewards for the lucky few. For instance, male elephant seals engage in ferocious fights that often cause harm and sometimes result in death. For most individuals who fight, the outcome in a net fitness loss. Still, fighting is an adaptive strategy: on average, males benefit from participating in fights, because not participating implies being shut out from reproduction and because top-ranking individuals enjoy extraordinary reproductive success.

Risky strategies are a prime candidate as a systematic source of individually maladaptive outcomes. Risk can be defined in a technical sense as unpredictable variation in outcomes (see Frankenhuiss & Del Giudice, 2012; Smallwood, 1996). Whereas some behavioral decisions offer a narrow range of possible outcomes (low-risk), others entail widely variable outcomes (high-risk), with the potential for large gains as well as large losses. By definition, risky strategies—such as aggressive competition for dominance—yield large gains in case of success but also impose heavy costs in case of failure. For example, people high in sensation seeking are overrepresented both in prison populations and among successful scientists, artists, and political leaders, suggesting that sensation seeking may instantiate a high-risk behavioral strategy (MacDonald, 1995). More generally, life history-related traits can steer individuals on high-risk pathways, thus increasing the likelihood of maladaptive and/or undesirable outcomes in case of strategy failure—even when the strategy is adaptive on average. This is more likely to happen in the context of fast life history strategies, which tend to promote risk-taking and favor the pursuit of large, immediate returns regardless of the potential costs. While some individuals engaging in high-risk strategies may end up developing mental disorders, other individuals expressing the same traits may enjoy desirable and/or biologically adaptive outcomes, depending on chance and unpredictable contextual factors.
Another important category of adaptive traits that systematically produce maladaptive outcomes is that of defensive mechanisms. By necessity, the calibration of defenses involves a trade-off between the rate of false negatives (failing to activate a defense mechanism when a threat is present) and that of false positives (mistakenly activating the mechanism when no threat is present). Defensive mechanisms are usually designed by natural selection to accept a high rate of false positives in order to avoid catastrophic false negatives; this is known as the smoke detector principle (Nesse, 2005). The smoke detector principle suggests that defensive mechanisms will often “misfire” or activate with excessive intensity, even when no actual threat is present. Occasionally, inappropriate activation of a defensive mechanism may cause serious harm to the individual. The logic of the smoke detector principle can be employed to shed light on the etiology of emotional symptoms such as panic attacks, anxiety, and phobic symptoms (Nesse, 2005; Nesse & Jackson, 2006). Individual differences in life history strategy are reflected in the calibration of behavioral and/or physiological defenses (see above), and indirectly affect the risk of inappropriate defense activation.

**Life history-related traits may increase vulnerability to dysfunction.** All biological and artificial mechanisms—no matter how well designed—are vulnerable to malfunctions, failures, and breakdowns. A psychological mechanism can malfunction because of accidents or environmental insults beyond its regulatory capacity (e.g., brain injury, exposure to toxins), deleterious genetic/epigenetic mutations, and attacks or manipulations by pathogens (see Cosmides & Tooby, 1999; Crespi, 2000, 2010). The continuous process of emergence and elimination of deleterious mutations is called mutation-selection balance; its dynamics determine the frequency and persistence of harmful variants in a population. Sometimes, a single mutation in a critical pathway is sufficient to cause a disorder; more often, disorders may result from the cumulative effect of many slightly deleterious mutations (mutation load), each with a small impact on phenotypic function. Since a large proportion of human genes are expressed in brain development, the likelihood that mutation load will have negative consequences on mental functioning is especially high. Mutation-selection balance has been proposed as a likely explanation for the persistence of common, heritable, and harmful mental disorders (Keller & Miller, 2006).

Exposure to pathogens (harmful viruses, bacteria, and other parasites) is another common cause of biological dysfunction. Infectious diseases—especially when they occur in early development—have been associated with increased risk for a broad range of mental disorders (see Patterson, 2011; Benros, Mortensen & Eaton, 2012). The role of pathogens in the etiology of mental disorders does not contradict that of genetic mutations. Infections, like mutations, can perturb developmental processes at critical stages; accordingly, mutation load and pathogen load may ultimately converge on the same neurobiological pathways and exert a cumulative effect on the risk for psychopathology.

While life history traits are designed to promote adaptation, they can nevertheless increase vulnerability to some types of dysfunction as a side effect. For example, some configurations of personality traits within the adaptive range (for example schizotypy or autistic-like personality) may become especially conducive to psychopathology when they are coupled with high mutation load or brain-damaging infections (see Del Giudice, 2010). Also, fast life history-related traits such as risk proneness and future discounting may indirectly increase an
individual’s exposure to environmental factors such as pathogens. Finally, up-regulated defensive systems are not only more prone to misfiring—they also become more vulnerable to genuine instances of malfunction and dysregulation (Nesse, 2001).

Sex differences

If life history strategies set the stage for psychopathology, sexual asymmetries in life history trade-offs should produce consistent patterns of sex differences in the epidemiology of mental disorders. The first key asymmetry concerns the mating versus parenting trade-off. On average, human males invest more in mating effort and less in parenting effort than females. The intensity of mating effort increases sexual selection for competitive traits such as risk-taking, dominance-seeking, and physical aggression (Archer, 2009; Kruger & Nesse, 2006; Wang et al., 2009; Wilson, Daly, & Pound, 2002). In total, higher mating effort in males should predispose them to fast spectrum disorders characterized by high levels of risk-taking, such as those in the externalizing spectrum (see Martel, 2013). In contrast, females have generally less to gain and more to lose from high-risk strategies than males, and can be expected to invest more effort in somatic maintenance and protection. As a consequence, they should be more prone to develop disorders that involve the up-regulation of protective defenses, and/or to exhibit more psychological and physiological symptoms reflecting defense up-regulation (see also McGuire & Troisi, 1998). This prediction applies to disorders across the fast-slow continuum, since up-regulated defenses can be functionally associated with both fast and slow life history strategies. The higher incidence of anxiety disorders in females (see Martel, 2013) is consistent with this prediction.

Another important asymmetry in life history strategy concerns the trade-off between current and future reproduction. As already discussed in the section on life history theory, this trade-off plays a more critical role in the organization of female life history strategies, since decisions concerning reproductive timing are more critical for females than for males. As a consequence, the timing of sexual maturation in females should be more sensitive to cues of danger and unpredictability. Indeed, the available data suggest that ecological stress in the first years of life anticipates gonadal puberty in girls, but not in boys (see Belsky, 2012; James et al., 2012). In addition, indices of sexual maturation in females can be expected to form a tighter cluster with other life history-related traits including motivation, personality, self-regulation, and so forth. It follows that maturation timing and rate should be stronger predictors of psychopathology in females than in males. This prediction is well supported by empirical research; the bulk of evidence indicates that individual differences in sexual maturation are more robustly associated with psychopathology in girls than in boys (Ge & Natsuaki, 2010; Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004; Mendle, Turkheimer, & Emery, 2007).

Correlates of Fast and Slow Spectrum Psychopathology

The conceptual distinction between fast and slow spectrum pathology provides a powerful heuristic criterion for the functional classification of mental disorders. Whatever the specific causal pathway (or combination of pathways) that determines the onset of a given disorder, fast spectrum conditions will be associated with traits such as low agreeableness and conscientiousness, impulsivity, disinhibition, and early sexual maturation (especially in females).
Conversely, slow spectrum conditions will exhibit a “signature” of slow life history-related traits in the areas of motivation, self-regulation, personality, and sexual maturation.

It is important to stress that correlations between life history-related traits and specific disorders may or may not imply a causal role of those traits in the etiology of the disorders. For example impulsivity, risk-taking, and social antagonism are likely to play a direct causal role in the etiology of externalizing symptoms (e.g., Lahey & Waldman, 2003). On the other hand, the robust correlation between externalizing symptoms and early sexual maturation (see below) does not necessarily mean that sexual maturation is directly involved in the onset of externalizing behavior. However, if the goal is to map disorders on the fast-slow continuum, this distinction is immaterial: regardless of their role in the etiology of a given disorder, life history correlates can be employed as convergent markers of the underlying life history strategy. In principle, this approach can be extended to include genetic, epigenetic, and neurobiological markers (e.g., Del Giudice et al., 2011; Figueredo et al., 2004, 2006; Worthman, 2009; Worthman & Brown, 2005). In this paper I mainly focus on the psychological level of analysis as a useful first approximation. A non-exhaustive list of the correlates of fast and slow spectrum psychopathology is presented in Table 1.

<table>
<thead>
<tr>
<th>Table 1. Correlates of fast and slow spectrum psychopathology.</th>
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<tr>
<td><strong>Fast spectrum psychopathology</strong></td>
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<tr>
<td>Motivation</td>
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<td></td>
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<tr>
<td>Self-regulation</td>
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<td></td>
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<tr>
<td>Personality traits</td>
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<td></td>
</tr>
<tr>
<td>Sexual Maturation</td>
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<tr>
<td>Environment</td>
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As can be seen in Table 1, the distinction between fast and slow spectrum disorders overlaps to some degree with the theory of undercontrolled and overcontrolled personality types and their role in psychopathology (Block, 2002; Block & Block, 1980; for a similar distinction see Tops & Boksem, 2010). This is hardly surprising, given the centrality of self-regulation in the organization of life history-related phenotypes. However, the present perspective has a much wider scope, as it integrates self-regulation in a broad conceptual network that includes mating, attachment, cooperation, and even physical and sexual maturation. Furthermore, in a life history framework the association between self-regulation and psychopathology need not be causal. While self-regulation profiles may directly contribute to the etiology of some mental disorders, in other cases the association may be largely or entirely spurious—that is, it may be due to covariation between self-regulation and other life history-related traits when only the latter are implicated in the genesis of a disorder.

A notable feature of Table 1 is the deliberate omission of emotions from the list of life history correlates of psychopathology. Of course, emotions are crucially involved in the etiology of many, perhaps most mental disorders (Keltner & Kring, 1998; Nesse, 1990). However, emotions can serve multiple motivational goals (Nesse, 2004; see also Keltner, Haidt, & Shiota, 2006), and the association between emotions and the underlying motivational processes is often remarkably non-specific, limiting the usefulness of emotions as markers of life history phenotypes. For example, anger can be triggered by aggressive competition, by threats to one’s dominance or status, by suffering or witnessing acts of injustice, by separation from an attachment figure, and so forth (Bowlby, 1973; Haidt, 2003). Anxiety, shame, and sadness are prominently associated with psychopathology, but their motivational specificity is also extremely low. In contrast, guilt is likely to be a reliable correlate of slow spectrum psychopathology because of its strong functional connection with cooperation, reciprocity, and caregiving (Haidt, 2003; Keltner et al., 2006). Even if careful analysis of emotional correlates may provide useful information about a given disorder, motivation is—all else being equal—a much better guide than emotion if the goal is to draw functional distinctions between disorders. An important implication is that diagnostic categories based on emotions and affect (e.g., anxiety disorders, depressive disorders) are especially likely to contain functionally heterogeneous conditions.

Finally, a life history perspective yields novel predictions about the environmental correlates of mental disorders (see Table 1). Ecological harshness and unpredictability tend to entrain development of fast life history strategies, while slow strategies are favored in safe and predictable contexts. As a result, many classic risk factors for psychopathology—such as stressful life events, low socioeconomic status, negative family relationships, trauma, and abuse—are predicted to increase the occurrence of fast spectrum disorders, but not that of slow spectrum disorders. On the contrary, slow spectrum disorders should be associated—at least on average—with safe, predictable environments, higher socioeconomic status, and reduced exposure to ecological and family stressors.
Applying the Framework

I now proceed to apply the framework developed in the previous section to a diverse set of common psychopathological conditions: externalizing disorders, schizophrenia spectrum disorders, autism spectrum disorders, obsessive-compulsive spectrum disorders, eating disorders, and depression. The goal is not to perform an exhaustive evolutionary analysis of these disorders (nor to systematically review the relevant empirical literature), but rather to demonstrate the heuristic and integrative potential of a life history approach to psychopathology and highlight the most promising directions for future research.

Table 2. Life history analysis of common mental disorders.

<table>
<thead>
<tr>
<th>Disorder category</th>
<th>Life history classification</th>
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<tr>
<td>Externalizing spectrum</td>
<td>Fast spectrum</td>
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<tr>
<td>Schizophrenia spectrum</td>
<td>Fast spectrum [possibly heterogeneous; age of onset]</td>
</tr>
<tr>
<td>Autism spectrum</td>
<td>Slow spectrum [possibly heterogeneous]</td>
</tr>
<tr>
<td>Obsessive-compulsive spectrum</td>
<td>Fast spectrum: endogenous obsessions</td>
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<td></td>
<td>Slow spectrum: reactive obsessions, OCPD</td>
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<tr>
<td>Eating disorders</td>
<td>Fast spectrum: dysregulated profile</td>
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<tr>
<td></td>
<td>Slow spectrum: perfectionistic and overcontrolled profiles</td>
</tr>
<tr>
<td>Depression</td>
<td>Heterogeneous [fast spectrum: depressed mood + somatic symptoms]</td>
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</table>

For each category, I examine the available empirical evidence to determine whether the relevant disorders can be provisionally characterized as fast or slow spectrum conditions (Table 2). I also discuss how current hypotheses in evolutionary psychopathology relate to the four causal pathways discussed in the preceding section. Whereas some psychopathological categories (e.g., externalizing disorders) show strong internal consistency in life history terms, other categories (e.g., obsessive-compulsive spectrum disorders) turn out to comprise an uneven mixture of fast and slow spectrum conditions. Moreover, a life history analysis indicates that standard diagnostic labels often fail to differentiate between functionally distinct conditions. For example, the classic distinction between anorexia nervosa and bulimia nervosa is empirically unreliable and only weakly related to individual differences in life history strategy; in contrast, eating disorder profiles based on personality (Westen & Harnden-Fischer, 2001) show a
remarkably good fit with the fast-slow distinction (see below). I conclude this section with an integrative summary in which I bring together individual disorders and outline a provisional life history taxonomy of common psychopathological conditions.

**The Externalizing Spectrum**

The externalizing spectrum comprises various disorders marked by aggressive, antisocial, and/or disruptive behavior (see Krueger et al., 2002, 2011). Externalizing disorders are also associated with high risk for substance abuse (Kendler, Prescott, Myers, & Neale, 2003a; McAdams, Rowe, Rijsdijk, Maughan, & Eley, 2011; Slade, 2007; Verona, Javdani, & Sprague, 2011). Disorders in the externalizing spectrum show high phenotypic and genetic correlations with one another, indicating the existence of a coherent, heritable dimension of externalizing behavior (Kendler et al., 2003a; Krueger et al., 2002; Lahey & Waldman, 2012). In the DSM-5, externalizing disorders—including oppositional defiant disorder (ODD), conduct disorder (CD), and antisocial personality disorder (APD)—are grouped in the category of “disruptive, impulse-control, and conduct disorders.”

In a life history perspective, externalizing spectrum disorders are prototypical instances of fast spectrum psychopathology. Externalizing symptoms are associated with impulsivity and undercontrol (e.g., Clark, 2005; DeYoung, 2011; Eisenberg et al., 2001; Huey & Weisz, 1997; Lynam, Leukefeld, & Clayton, 2003; Muris & Ollendick, 2005), early puberty timing and fast sexual maturation in both sexes (Mendle et al., 2007; Mendle & Ferrero, 2012), earlier onset of sexual activity (e.g., Armour & Haynie, 2007; Lévesque, Bigras, & Pauzé, 2010; van Goozen, Cohen-Kettenis, Matthyss, & Van Engeland, 2002), and larger numbers of partners in adolescence and young adulthood (e.g., Cui, Ueno, Fincham, Donnellan, & Wickrama, 2012). Low socioeconomic status, harsh or unpredictable parental discipline, parental conflict, family disruption, and child abuse—all cues of danger and unpredictability—are consistent predictors of externalizing behavior (Burt, Krueger, McGue, & Iacono, 2003; Farrington, 2005; Simpson et al., 2012). This further supports the notion that externalizing disorders are prototypical fast spectrum conditions.

An important component of the externalizing spectrum is the personality dimension of psychopathy (Jones & Miller, 2012). The psychopathic personality is characterized by shallow affect, callousness and lack of empathy, insincerity and manipulativeness, grandiosity, irresponsibility, and sensation seeking (Hare & Neumann, 2006). The distribution of externalizing behaviors and psychopathic traits is strongly male-biased, in both clinical and non-clinical populations (Cale & Lilienfeld, 2002; Crijnen, Achenbach, & Verhulst, 1997; Kessler et al., 2005; Leadbeater, Kuperminc, Blatt, & Hertzog, 1999; Martel, 2013; Slade, 2007).

As widely recognized in the evolutionary literature, psychopathic traits show all the markers of a fast life history strategy (e.g., Barr & Quinsey, 2004; Harris, Rice, Hilton, Lalumière, & Quinsey, 2007; Mealey, 1995). Psychopathic individuals are impulsive and exploitative; they tend to be sexually precocious, have many short-term partners, and frequently engage in sexual coercion (Harris et al., 2007; Jonason et al., 2009; Kastner & Sellbom, 2012; Lalumière & Quinsey, 1996; Lalumière, Mishra, & Harris, 2008; Mishra & Lalumière, 2008). Unsurprisingly, psychopathic traits correlate with measures of fast life history strategy, risk-
taking, and present orientation (Figueroed & Jacobs, 2010; Jonason, Koenig, & Tost, 2010; but see Gladden, Figueredo, & Jacobs, 2009). Finally, externalizing symptoms and psychopathic traits are negatively associated with the alpha personality metatrait and its components, agreeableness and conscientiousness (Decuyper, De Pauw, De Fruyt, De Bolle, & De Clerq, 2009; DeYoung, 2011; DeYoung, Peterson, Séguin, & Temblay, 2008; Essau, Sasagawa, & Frick, 2006; Jones, Miller, & Lynam, 2011; Krueger et al., 2011; Lynam & Derefinco, 2006).

Evolutionary models of externalizing spectrum disorders tend to stress the potential biological adaptiveness of aggressive, exploitative, and risky behavior—especially when coupled with promiscuous short-term sexuality (e.g., Barr & Quinsey, 2004; Belsky et al., 1991; Del Giudice et al., 2011; Ellis et al., 2012; Martel, 2013; Mealey, 1995; see Glenn, Kurzban, & Raine, 2011 for a review of alternative explanations). Accordingly, many evolutionary scholars see externalizing disorders as adaptive but undesirable constellations of traits. In some instances, externalizing disorders may represent maladaptive extremes of potentially adaptive traits (see MacDonald, 2012). It should be stressed that externalizing disorders can be adaptive even if their social outcomes are negative on average. This can happen if successful outcomes yield disproportionate fitness returns, even in a minority of cases (discussed in Frankenhuis & Del Giudice, 2012). For example, a study by Ullrich and colleagues (Ullrich, Farrington, & Coid, 2008) found negative correlations between psychopathic traits and biologically valuable outcomes such as status and wealth. However, overall trait-outcome correlations are not very informative unless patterns of outcome variability are also taken into account.

As already noted, high-risk behavioral strategies are likely to involve down-regulation of defensive mechanisms; indeed, externalizing disorders in adolescents and adults are often associated with reduced anxiety, fearlessness, and dampened responsivity of the stress response system (Alink et al., 2008; Fowles & Dindo, 2006; Lorber, 2004). However, defense down-regulation has only a marginal role in the DSM, and the definition of externalizing disorders revolves around antisocial behavior and its undesirable consequences.

The Schizophrenia Spectrum

Schizophrenia is a family of mental disorders characterized by delusions, hallucinations, and cognitive disorganization. Given the severe reduction in reproductive success associated with a schizophrenia diagnosis (e.g., Bassett, Bury, Hodgkinson, & Honer, 1996; Haukka, Suvisaari, & Lonqvist, 2003; MacCabe, Koupil, & Leon, 2009; Nanko & Moridaira, 1993), most evolutionary scholars regard this disorder as a maladaptive outcome of dysregulated socio-cognitive processes (e.g., Burns, 2004; Crow, 1995, 1997; Keller & Miller, 2006; McGuire & Troisi, 1998; see Stevens & Price, 1999 for an exception). Schizophrenia spectrum disorders (SSDs) are highly heritable (Tandon, Keshavan, & Nasrallah, 2008); at the same time, schizophrenia risk is increased by adverse environmental factors such as nutritional deficiencies, infections, and birth complications (e.g., Benros et al., 2012; Burns, 2004; McGrath & Murray, 2011). This suggests that accumulated deleterious mutations and environmental insults may converge on common neurobiological pathways, increasing the risk of cognitive breakdown.

Even if SSDs are biologically maladaptive conditions, there may be evolutionary advantages associated with schizotypal traits—a constellation of personality traits associated with
increased risk of psychosis (Claridge, 1997; van Os, Linscott, Myin-Germeyns, Delespaul, & Krabbendam, 2009). While some taxometric studies suggest that schizotypal traits may define a categorically distinct subgroup of individuals rather than a continuum with normal personality, there is still no consensus on this point and the evidence remains mixed (see Ahmed, Buckley, & Mabe, 2012; Coghill & Sonuga-Barke, 2012; Nelson, Seal, Pantelis, & Phillips, 2013). Various authors have proposed that schizotypal traits may be maintained by sexual selection processes based on mate choice. According to the sexual selection model of schizotypy (Nettle, 2001, 2006a; Shaner, Miller, & Mintz, 2004), schizotypy-increasing alleles affect brain processes so as to increase traits such as verbal and artistic creativity, thus conferring mating advantages on those individuals who do not develop a psychiatric condition. However, the outcomes of schizotypy may be either beneficial (mating success) or harmful (schizophrenia), depending in part on the individual’s genetic quality (i.e., lack of deleterious mutations) and developmental condition (e.g., good nutrition and low exposure to pathogens). In other words, according to this hypothesis verbal/artistic creativity functions as a fitness indicator (see Shaner et al., 2004), and schizotypy acts as an “amplifier” of individual differences in genetic quality and condition. The sexual selection model is thus consistent with a central role of mutation load in the etiology of SSDs, and is compatible with reduced fertility in schizophrenic patients and their close relatives (Del Giudice, 2010).

Consistent with the sexual selection model, positive schizotypal traits—unusual cognitive and perceptual experiences, tendency to magical ideation, reference and paranoid thoughts—are associated with verbal and artistic creativity, larger numbers of sexual partners, unrestricted sociosexuality, and reduced investment in long-term couple relationships (Beaussart, Kaufman, & Kaufman, 2012; Del Giudice et al., 2010; Haselton & Miller, 2006; Kinney et al., 2001; Nettle, 2006b; Nettle & Clegg, 2006; Miller & Tal, 2007; Rawlings & Locarnini, 2008). Moreover, large-scale studies of patients and their relatives show a robust familial association between schizophrenia and creativity (Kyaga et al., 2011). Schizotypal traits peak in adolescence/young adulthood and show a marked decline with age, mirroring typical changes in mating effort (Claridge et al., 1996; Fossati, Raine, Carretta, Leonardi, & Maffei, 2003; Venables & Bailes, 1994). In addition, positive schizotypy predicts higher levels of aggression in the non-clinical population (Fanning, Berman, & Guillot, 2012; Nederlof, Muris, & Hovens, 2012), and a hostile-dominant interpersonal style seems to be an enduring aspect of the personality of patients who manifest paranoid symptoms (Podubinsky, Daffern, & Lee, 2012). This suggests a degree of overlap between the schizophrenia spectrum and the externalizing spectrum. Finally, schizotypal traits are associated with low levels of agreeableness (Asai et al., 2011; Ross et al., 2002; but see Avia et al. 1995).

In light of these convergent findings, SSDs can be classified as belonging to the fast spectrum of psychopathology. According to sexual selection models, schizotypy can be understood as a high-risk strategy oriented toward short-term mating, whose negative outcomes become manifest as schizophrenia and other SSDs. Alternatively, the milder disorders of the schizophrenia spectrum (e.g., schizotypal personality disorder, brief psychotic disorder) may result from maladaptive levels of expression of potentially adaptive traits associated with fast life history strategies. This view is consistent with the hypothesis that schizotypal traits follow a cliff-edged fitness function, with an abrupt transition between optimal and maladaptive levels of expression (Nesse, 2004b). It should also be noted that most individuals who have psychotic
experiences at some point in their life recover completely, and never transition to a diagnosable SSD (van Os et al., 2009).

So far, there is only limited evidence concerning the relation between schizotypy and the timing of sexual maturation. The available data indicate that positive schizotypal traits tend to be higher in both early and late maturers, though the effect may be especially pronounced in early maturers (Gruzelier & Kaiser, 1996; Kaiser & Gruzelier, 1999). These findings are partially consistent with the idea of schizotypy as a fast life history-related trait; however, further research informed by a life history approach might reveal the existence of functionally distinct clusters within the schizophrenia spectrum. This would be consistent with data showing differences in genotype and symptom profiles between early- and late-onset schizophrenia (Lien et al., 2011).

The Autism Spectrum

The autism spectrum comprises disorders of variable severity characterized by impairments in social interaction, communication problems, and restricted and repetitive behaviors/interests. While autism spectrum disorders (ASDs) are substantially heritable, they are also highly heterogeneous in their genetic substrate (Betancur, 2011; Sanders et al., 2012). Furthermore, the three facets of the “autism triad” (social interaction, communication, and restricted/repetitive behavior) are largely dissociable, both phenotypically and genetically (Happé, Ronald, & Plomin, 2006; Happé & Ronald, 2008; Ronald, Larsson, Anckarsäter, & Lichtenstein, 2011). This heterogeneity must be kept in mind while discussing ASDs from a functional perspective.

Severe autism is almost certainly maladaptive, and some theorists have focused specifically on the negative aspects of ASDs. In particular, Shaner and colleagues (Shaner, Miller, & Mintz, 2008) hypothesized that autism—like schizophrenia—may represent the negative extreme of a fitness indicator. Unlike in the case of schizophrenia, however, the fitness indicator would be not sexually but parentally selected: under this hypothesis, children display their genetic quality to parents in order to effectively solicit their investment, and complex behaviors like social responsiveness and social engagement function as costly and sensitive fitness indicators. Autism would represent a catastrophic failure of these mechanisms, due to high mutation load and/or poor developmental conditions. The fitness indicator theory of autism is consistent with the large number of deleterious de novo mutations found in ASD patients (Awadalla et al., 2010; Sanders et al., 2012).

Shaner and coworkers’ (2008) emphasis on maladaptation should be balanced by accumulating evidence that autistic-like traits in the normative range—also known as the “broader autistic phenotype” (Wheelwright, Auyeung, Allison, & Baron-Cohen, 2010)—have a number of desirable and potentially adaptive correlates. Specifically, autistic-like traits predict higher systemizing abilities and attention to detail, better visuospatial skills and abstract spatial reasoning, and enhanced low-level sensory processing in the visual and auditory domains (Baron-Cohen, Ashwin, Ashwin, Tavassoli, & Chakrabarti, 2009; Grinster, van Beek, Maybery, & Badcock, 2009; Stevenson & Gernsbacher, 2013; see also Falter, Elliott, & Bailey, 2012; Mottron, Dawson, Soulières, Hubert, & Burack, 2006). The autistic facets of repetitive behaviors, restricted interests, and detail-oriented cognitive style are associated with the development of
outstanding talents in children (Happé & Vital, 2009; Ruthsatz & Urbach, 2012; Vital, Ronald, Wallace, & Happé, 2009). More generally, autistic-like traits are higher in people with technical-scientific interests and careers (Austin, 2005; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001; Ridley, Homewood, & Walters, 2011; Wheelwright et al., 2006). Accordingly, several theorists have argued that ASDs can be seen as extreme and usually maladaptive manifestations of otherwise adaptive traits (e.g., Baron-Cohen, 2003; Crespi & Badcock, 2008; Del Giudice et al., 2010).

In this perspective, Del Giudice and colleagues (2010) hypothesized that sexual selection may contribute to maintain autistic-like traits in the population despite the fitness costs of severe ASDs. Specifically, they argued that autistic-like traits in their non-pathological form contribute to a male-typical strategy geared toward high parental investment, low mating effort, and long-term allocation of resources—in other words, a male-typical manifestation of slow life history strategy. This hypothesis provides a parsimonious explanation of the male-biased distribution of both autistic-like traits and ASDs (Baron-Cohen et al., 2001, 2011). Several lines of evidence corroborate this hypothesis. Autistic-like traits predict reduced interest in short-term mating, increased investment of time and resources in one’s partner, and stronger commitment to long-term romantic relations—the opposite of positive schizotypy (Del Giudice et al., 2010). People high in autistic-like traits report shorter duration of friendships but longer duration of romantic relationships (Jobe & White, 2007); moreover, their partners are on average just as satisfied as those of people low in autistic-like traits (Pollmann, Finkenauer, & Begeer, 2009). Intriguingly, interest in sexual and romantic relationships is usually conserved in high-functioning ASDs, even if the development of courtship and sexual abilities follows a delayed trajectory (Hellemans, Colson, Verbraeken, Vermeiren, & Deboutte, 2007; Stokes & Kaur, 2005; Stokes, Newton, & Kaur, 2007). Indeed, people with ASD can be highly persistent in pursuing romantic interests, and often display obsessive preoccupation with their partner (Stokes et al., 2007).

In a life history perspective, ASDs are thus likely candidates for inclusion in the slow spectrum of psychopathology. Further evidence comes from the finding that sexual maturation is delayed in women high in autistic-like traits (Whitehouse, Maybery, Hickey, & Sloboda, 2011) as well as in women with ASD (Ingudomnukul, Baron-Cohen, Wheelwright, & Knickmeyer, 2007; Knickmeyer, Wheelwright, Hoekstra, & Baron-Cohen, 2006). Autistic-like traits may function adaptively as part of a slow life history strategy—especially in males—and only become maladaptive when they cross a certain threshold. The idea that ASDs are part of the slow spectrum of psychopathology is also consistent with the recent proposal that the main cognitive and behavioral correlates of the autistic spectrum (both adaptive and maladaptive) can be framed in a heterochronic perspective as delays or non-completions of typical developmental trajectories (Crespi, 2013). Of course, given the remarkable heterogeneity of ASDs, this functional explanation is likely to apply only to a subset of people diagnosed with autistic disorders. Different ASD subtypes may well require different explanations, potentially including the fitness indicator hypothesis by Shaner and colleagues (2008).

The existence of functionally distinct subtypes of ASDs may explain the inconsistent correlation of autism risk with socioeconomic status, which has been found to be positive in some studies and negative in others (e.g., Bhasin & Schendel, 2007; Leonard et al., 2011; Rai et al., 2012). A life history perspective may also contribute to explain the robust finding that autism risk
increases in the children of older parents, and especially mothers (Bhasin & Schendel, 2007; Gardener, Spiegelman, & Buka, 2009); if people high in autistic-like traits tend to delay reproduction, they will end up being over-represented among older parents in epidemiological studies. The increasing number of mutations in the sperm of older fathers is another plausible etiological factor in both ASDs and SSDs (Kong et al., 2012). However, if the present analysis is correct, deleterious mutations are only involved in a subset of ASDs, possibly limited to the more severe cases of autism.

The idea that ASDs can be characterized as slow spectrum disorders might seem inconsistent with the widely reported association between autistic symptoms and impaired executive functions (Russo et al., 2007). However, the contradiction is only apparent, since the executive deficits associated with ASDs and autistic-like traits in the normal range are limited to flexibility/shifting and—to a much smaller extent—memory updating (Ridley et al., 2011; Russo et al., 2007; Van Eylen et al., 2011). Reduced flexibility/shifting is the other side of the coin of restricted/repetitive behavior, and can be seen as a facet of behavioral persistence—a key feature of slow spectrum phenotypes (Table 1). Consistent with this view, reduced shifting abilities are associated with higher levels of self-restraint and increased delay of gratification (see Miyake & Friedman, 2012). As discussed in the section on life history strategies and individual differences, behavioral disinhibition is the only robust executive correlate of fast life history strategies and is not observed in autism, with the only exception of tasks involving saccade control (see O’Hearn, Asato, Ordaz, & Luna, 2008; Russo et al., 2007). In other words, the profile of self-regulation associated with autism and autistic-like traits is consistent with the hypothesis that ASDs are slow spectrum disorders.

Even if autistic-like traits show many signatures of a slow life history phenotype, they should be considered as part of an alternative behavioral strategy that deviates to some extent from the typical structure of life history correlates (Del Giudice et al., 2010). For example, there is accumulating evidence that people with mild forms of ASDs are not susceptible to audience effects on altruistic behavior, do not engage in distorted self-presentation to enhance their own reputation, and are less susceptible to the emotional effects of social ostracism (Chevallier, Molesworth, & Happé, 2012; Izuma, Matsumoto, Camerer, & Adolphs, 2011; Sebastian, Blakemore, & Charman, 2009). This combination of characteristics makes people high in autistic-like traits uncommonly transparent and trustworthy (Frith & Frith, 2011), which can be an asset in the context of cooperative relationships (including long-term romantic relationships). However, while agreeableness is usually associated with trustworthiness and honesty, autistic-like traits as a whole correlate negatively with agreeableness (Austin, 2005; Wakabayashi, Baron-Cohen, & Wheelwright, 2006). This suggests that individuals high in autistic traits may reach cooperative goals in ways that are atypical compared with the rest of the population.

Finally, the life history analysis presented in this section is consistent with Crespi and Badcock’s (2008) hypothesis that autism and psychosis are diametrical disorders of the social brain, involving opposite unbalanced patterns of “mechanistic” versus “mentalistic” abilities (see also Dinsdale, Hurd, Wakabayashi, Elliot, & Crespi, 2013). Besides showing different socio-cognitive profiles, ASDs and SSDs are characterized by opposite patterns of brain and body growth (Crespi & Badcock, 2008) and by diametrical patterns of genetic effects—for example up-versus down-regulation of molecular pathways and larger versus smaller numbers of gene
copies (Crespi, Stead, & Elliot, 2010; Gilman et al., 2012; see also Kalkman, 2012). A life history framework provides a broader context for the diametrical hypothesis by placing ASDs and SSDs at opposite ends of the fast-slow continuum, while acknowledging the possible existence of functionally distinct subtypes within both diagnostic categories.

**The Obsessive-Compulsive Spectrum**

Disorders in the obsessive-compulsive spectrum are primarily characterized by patterns of compulsive, repetitive thoughts and/or behaviors, usually associated with worry and anxiety. In addition to obsessive-compulsive disorder (OCD), the OC spectrum includes body dysmorphic disorder, hoarding disorder, and grooming disorders (skin picking and hair pulling). These disorders tend to co-occur, both within families and in the same individual (Phillips et al., 2010). There is considerable evidence that obsessive-compulsive personality disorder (OCPD)—a pervasive profile of orderliness, rigid perfectionism, and need to control one’s self and environment—is also part of the OC spectrum (Calvo et al., 2009; Phillips et al., 2010), even if the DSM-5 category of “obsessive-compulsive and related disorders” does not include OCPD. The phenomenology of OCD is highly heterogeneous; the content of OC symptoms may relate to a number of common themes including contamination/cleaning, obsessions/checking, symmetry/ordering, and hoarding (Mataix-Cols, Rosario-Campos, & Leckman, 2005; McKay et al., 2004).

A rich evolutionary literature on OCD has developed over the years (e.g., Abed & de Pauw, 1998; Boyer & Lienard, 2006; Brüne, 2006; Fiske & Haslam, 1997; Rapoport & Fiske, 1998; Szechtman & Woody, 2004; Woody & Szechtman, 2011). Obsessive-compulsive disorders are moderately heritable (Grisham, Anderson, & Sachdev, 2008) and can be severely impairing. Most theorists assume that OCD is either the maladaptive exaggeration of an adaptive trait or the result of a dysfunction in precautionary cognitive systems. However, the relation between OCD and mating/reproductive success has received very little attention (Fontenelle & Hasler, 2008), and the milder forms of the disorder are not necessarily maladaptive in the biological sense. Current models converge on the idea that the main functional substrate of OCD is an adaptive mechanism—the hazard-precaution system or security motivation system—specialized for dealing with potential low frequency threats such as food poisoning (Boyer & Lienard, 2006; Woody & Szechtman, 2011). Compared with manifest threats, potential threats pose a number of unique strategic problems. For example, they must be detected based on subtle, indirect cues, and there is no external feedback to determine when precautionary behaviors should be terminated. The logic of potential threats explains many features of compulsions (Woody & Szechtman, 2011); obsessions can be explained as the involuntary generation of potential risk scenarios, a mechanism designed to increase future harm avoidance (Abed & de Pauw, 1998; Brüne, 2006). Consistent with a threat prevention account and with the prediction that females should be more likely to develop symptoms reflecting up-regulated defenses, adult OCD patients are overwhelmingly women (Fontenelle & Hasler, 2008).

From the perspective of standard evolutionary models, OC disorders would seem to fit straightforwardly in the slow spectrum of psychopathology, as a combination of exaggerated trait expression, up-regulation of adaptive defenses, and dysfunctional protective responses. Indeed, hypersensitive precautionary defenses aimed at preventing future and/or potential threats can be
highly adaptive in the context of slow life history strategies. This hypothesis is consistent with the high levels of harm avoidance and guilt sensitivity observed in OCD patients (Pinto & Eisen, 2012; Shafran, Watkins, & Charman, 1996; see also O’Connor, Berry, & Weiss, 1999). Obsessive-compulsive personality disorder (OCPD) also fits this scheme, given its many overcontrol features and strong association with conscientiousness (Samuel & Gore, 2012).

This, however, is only part of the story, as a host of other findings indicate robust correlations between OC spectrum disorders—particularly OCD—and key markers of fast spectrum psychopathology. Obsessive-compulsive symptoms show moderate correlations with impulsivity (Ettelt et al., 2007; Smári, Bouranel, & Eiðsdóttir, 2008; Sulkowski et al., 2009), and reduced motor inhibition is often observed in OCD (e.g., Bannon, Gonsalvez, Croft, & Boyce, 2002; Cavedini, Zorzi, Piccinni, Cavallini, & Bellodi, 2010; Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; Chamberlain et al., 2007; Moritz et al., 2002; Penadés et al., 2007). Surprisingly, self-reported conscientiousness tends to be low in OCD patients, although this might depend on unrealistically high self-imposed standards (Kotov, Gamez, Schmidt, & Watson, 2010; Pinto & Eisen, 2012). Preliminary empirical data indicate that measures of life history strategy are uncorrelated with OC symptoms in non-clinical samples (Glass, 2012 [personal communication]). Even more important, OC spectrum disorders show high comorbidity with both autism spectrum (Anholt et al., 2012; Bejerot, 2007; Hollander, King, Delaney, Smith, & Silverman, 2003) and schizophrenia spectrum disorders (Lee & Telch, 2005; Poyurovsky & Koran, 2005; Poyurovsky et al., 2008; Sobin et al., 2000). In a life history framework these findings are paradoxical, and suggest that the OC spectrum may be functionally heterogeneous at a fundamental level.

The apparent paradox can be solved by turning to the crucial distinction between autogenous and reactive obsessions (Lee & Kwon, 2003). Autogenous obsessions have sexual, aggressive, and/or blasphemous content; they tend to be bizarre, ego-dystonic, and threatening in their own right. They often have no apparent trigger, or are triggered by remote/bizarre thought associations. In contrast, reactive obsessions concern realistic fears of contamination, mistakes, accidents, and/or disarray. They are triggered by cues of potential threats and are typically followed by preventive behaviors such as ordering or cleaning; anxiety is directed at the possible consequences of one’s actions rather than at the obsession itself. Patterns of autogenous vs. reactive obsessions in OCD are statistically robust and longitudinally stable, and are associated with distinct patterns of brain activity (Besiroglu et al., 2007, 2011; Moulding, Kyrios, Doron, & Nedeljkovic, 2007).

While evolutionary models of OCD based on threat prevention do a good job of explaining reactive obsessions, they have virtually nothing to say about autogenous obsessions. As it turns out, the autogenous-reactive distinction maps neatly on that between fast and slow spectrum disorders. Autogenous obsessions—but not reactive obsessions—are associated with positive schizotypy, indices of psychotic thought disorganization, reduced inhibitory control, higher levels of hostility, and substance abuse (Brakoulias et al., 2013; Lee & Telch, 2005, 2010; Lee, Kim, & Kwon, 2005; Lee, Yost, & Telch, 2009; see also Ettelt et al., 2007). On the contrary, reactive obsessions are associated with perfectionism, heightened responsibility and personal standards, and normal levels of motor and cognitive inhibition (Belloch, Cadebo, Carrió, & Larsson, 2010; Lee & Telch, 2010; Lee et al. 2005, 2009; Moulding et al., 2007). Latent class
analyses identify a fast-spectrum OCD subgroup showing autogenous (“taboo”) obsessions, low conscientiousness, and high comorbidity with anxiety and depression, and a slow-spectrum subgroup showing high conscientiousness, contamination/cleaning symptoms, and comorbidity with grooming disorders, panic disorder, and tics (Nestadt et al., 2009). Interestingly, tics are strong predictors of comorbid autistic traits in OCD (Ivarsson & Melin, 2008), supporting the existence of a cluster of slow spectrum disorders that includes ASDs.

In summary, the totality of evidence indicates that the OC spectrum comprises at least two functionally distinct clusters of disorders: (a) a slow spectrum cluster characterized by high conscientiousness, reactive obsessions, OCPD features (Coles, Pinto, Mancebo, Rasmussen, & Eisen, 2008), overlap with autistic traits (especially repetitive/restricted behaviors and interests; Hollander et al., 2003), and comorbidity with ASDs; and (b) a fast spectrum cluster characterized by low conscientiousness, impulsivity, autogenous obsessions, overlap with schizotypal features, and comorbidity with SSDs. The two clusters can be expected to show markedly different epidemiological profiles; for example, traumatic events and low SES should be more strongly associated with fast spectrum OCD, whereas slow spectrum OCD should often arise in safe and predictable environments. This would explain why research on the socioeconomic correlates of OCD has generated a multitude of contradictory findings (Fontenelle & Hasler, 2008). In contrast, OCPD is uniformly associated with high education levels, and OCDP patients have the highest SES of all personality disorders (Grant et al., 2004; Torgersen, Kringlen, & Cramer, 2001; Walsh et al., 2012). This is further evidence that OCPD can be categorized as a slow spectrum disorder.

Eating Disorders

Eating disorders (EDs) are defined by heightened concern with body shape/weight and associated behaviors such as dieting, binge eating, purging, and exercising. Eating disorders occur almost exclusively in females, and their age of onset peaks in adolescence (Hoek, 2006). The DSM-5 distinguishes anorexia nervosa (AN) from bulimia nervosa (BN) based on body weight, and two subtypes of AN—restricting AN and binge eating/purging AN—based on the occurrence of binging episodes. However, empirical data overwhelmingly indicate that these diagnostic categories are largely artificial: ED symptoms co-occur at high rates, and diagnostic crossover—i.e., change in diagnosis at different times—is extremely high, both between AN and BN and between AN subtypes (Eddy et al., 2008; Peat, Mitchell, Hoek, & Wonderlich, 2009; Westen & Harnden-Fischer, 2001).

Evolutionary models of eating disorders tend to focus specifically on dieting behavior. Two main alternative hypotheses have been proposed so far. First, dieting may work as a means to suppress fertility and delay or forego reproduction when the social environment is not optimal—for example when social support by relatives and partners is low, or when social competition is too harsh (Mealey, 2000; Surbey, 1987; Wasser & Barash, 1983; Voland & Voland, 1989). This hypothesis has received preliminary support in a study by Juda and colleagues (Juda, Campbell, & Crawford, 2004). Second, dieting may work primarily as a female strategy in mating and status competition (Abed, 1998; Ferguson, Winegard, & Winegard, 2011). Thinness is a reliable signal of youth, and dieting can increase one’s attractiveness because of men’s strong preference for younger partners (Buss 1989; Dunn, Brinton, & Clark, 2010; Kenrick...
& Keefe, 1992; Kenrick, Keefe, Gabrielandis, & Cornelius, 1996; Vaillancourt, 2013). In addition, dieting can enhance status in female groups (thus indirectly influencing mating success), especially when cultural emphasis on thinness is strong (Abed, 1998). This hypothesis is supported by the robust pattern of associations among perceived sexual competition, dieting behavior, and eating symptoms found in non-clinical samples (Faer, Hendriks, Abed, & Figueredo, 2005; Li, Smith, Griskevicius, Cason, & Bryan, 2010; Salmon, Crawford, Dane, & Zuberbier, 2008, Salmon et al., 2009); moreover, it is consistent with the finding that relational aggression in girls is preferentially directed against underweight peers (Wang, Iannotti, & Luk, 2010), and with the remarkable emotional salience of pride and shame in ED patients (Allan & Goss, 2012). Under both evolutionary hypotheses, the psychological processes that underlie dieting behavior are fundamentally adaptive, and lead to maladaptive outcomes (such as severe EDs) only when they become dysregulated or get trapped in vicious cycles (e.g., Abed, 1998; Faer et al., 2005; McGuire & Troisi, 1998; see Dwyer, Horton, & Aamodt, 2011 for an alternative view).

The mating competition hypothesis of eating disorders can be refined and extended by framing it in a life history perspective. While high levels of mating effort are associated with fast life history strategies, both fast and slow strategists can face intense competition for mates. The main difference is that fast strategists compete primarily to become desirable sexual partners, whereas slow strategists compete primarily to be chosen as long-term partners in committed relationships (thus shifting investment toward parenting effort); indeed, competition for desirable long-term partners can sometimes be fiercer than that for short-term mates. In female competition, bodily attractiveness plays a different role in short- versus long-term contexts. Men assign much more importance to bodily attractiveness when they are looking for short-term sexual partners, since a feminine body shape (including for example a low waist-hip ratio or large breasts) is a reliable signal of current fertility. When men judge a potential long-term partner, however, the relative importance of traits indicating overall reproductive value rather than current fertility (for example facial attractiveness cues such as symmetry) increases accordingly (Confer, Perilloux, & Buss, 2010; Currie & Little, 2009; Lu & Chang, 2012; Zelazniewicz & Pawlowski, 2011).

Since youth is a better index of reproductive value than of current fertility, females pursuing a slow life history strategy should be more willing to increase apparent youth—and hence thinness—at the cost of diminished body attractiveness. The prediction follows that, on average, slow life history females who face intense mating competition should desire (a) a thinner body than fast life history females, and (b) a thinner body than what men consider most sexually attractive. Furthermore, they should usually be more successful at achieving and maintaining their desired weight because of their higher conscientiousness and self-control. As a result, slow life history females should be statistically over-represented in AN compared with BN, and in the AN-restricting subtype compared with the AN-binge eating/purging subtype—even if single individuals are likely to move back and forth between diagnostic categories over time (Peat et al., 2009).

This prediction is fully supported by the available evidence. Patients with BN are higher in impulsivity, sensation seeking, and novelty seeking than AN patients (Cassin & von Ranson, 2005). They also tend to mature earlier and to have sex at a younger age (Mendle et al., 2007;
Wiederman, Pryor, & Morgan, 1996). Furthermore, AN shows considerably more overlap than BN with OCD, OCPD, and ASDs (Altman & Shankman, 2009; Godart et al., 2006; Halmi et al., 2003; Pooni, Ninteman, Bryant-Waugh, Nicholls, & Mandy, 2012). Compared with binging/purging anorexics, restricting anorexics are more agreeable and conscientious, less impulsive, lower in sensation seeking, and higher in motor inhibition (Bollen & Wojciechowski, 2004; Claes, Mitchell, & Vendereycken, 2012; DaCosta & Halmi, 1992; Keel et al., 2004; Rosval et al., 2006; Tasca et al., 2009; Waxman, 2009).

Even if standard diagnostic labels seem to reflect differences in motivation and self-regulation consistent with a fast-slow gradient, they are too volatile and unreliable to represent true alternative life history phenotypes (Eddy et al., 2008; Peat et al., 2009; Westen & Harnden-Fischer, 2001). In a functional perspective, personality profiles and comorbidity patterns are much better pointers to life history strategy than body weight and the presence/absence of binging behavior. Fortunately, empirical studies reveal a consistent structure of ED personality profiles that maps remarkably well on the fast-slow distinction. At the broadest level of analysis, it is possible to identify three personality subtypes in women with EDs: a high functioning/perfectionist subtype, an overcontrolled subtype, and a dysregulated subtype (Hopwood, Ansell, Fehon, & Grilo, 2010; Thompson-Brenner & Westen, 2005; Thompson-Brenner et al., 2008a; Thompson-Brenner, Eddy, Satir, Boiseau, & Westen, 2008b; Westen & Harnden-Fischer, 2001).

The high functioning/perfectionist subtype shows low comorbidity rates (mostly with OCD and OCPD) and the most favorable clinical outcomes. Despite suffering from potentially severe eating symptoms, individuals in this group tend to have high self-esteem and relatively intact family and couple relationships. Moreover, having experienced fewer than average stressful life events increases the likelihood of belonging to this subtype. In total, this profile is fully consistent with inclusion in the slow spectrum. On the contrary, dysregulated patients show high levels of impulsivity and antisocial/externalizing behavior, high comorbidity (especially with borderline personality disorder), and more stressful life events including high rates of sexual abuse—a pattern indicative of fast spectrum psychopathology. The overcontrolled subtype is characterized by high rates of depression, low self-esteem and passivity, restricted emotionality, and comorbidity with OCPD and avoidant personality disorder. While patients in the high functioning/perfectionist and overcontrolled groups can be diagnosed with either AN and BN, the dysregulated subtype is strongly associated with BN (Thompson-Brenner et al., 2008a, 2008b; Westen & Harnden-Fischer, 2001).

To sum up, eating disorders are associated with female competition at both ends of the fast-slow continuum, and range from potentially adaptive strategies to frankly maladaptive dysfunctions. While AN is especially prevalent at the slow end of the spectrum, BN can occur in association with both fast and slow strategies; this probably explains why previous research has failed to detect specific associations between life history strategy and AN versus BN symptoms (Salmon et al., 2009). In contrast with standard labels, the personality profiles of ED patients show a close fit with the fast-slow distinction. The high functioning/perfectionist profile—comprising both AN and BN—falls in the slow spectrum of psychopathology, and is likely to reflect heightened competition for status and/or long-term mating. The dysregulated profile—typically associated with BN—shows remarkable overlap with the externalizing spectrum, and is
likely to reflect competition in the short-term mating arena. The poor clinical outcomes associated with dysregulated EDs suggest that they may sometimes be understood as maladaptive outcomes of high-risk behavioral strategies. While it displays some markers of slow spectrum psychopathology, the overcontrolled profile is somewhat more difficult to classify. An intriguing speculation is that overcontrolled ED patients might be engaging in reproductive suppression following loss of status and/or social support (Mealey, 2000; Surbey, 1897), as suggested by their depressed mood, low self-esteem, and acute sense of social exclusion (Westen & Harnden-Fischer, 2001). While reproductive suppression is intrinsically future-oriented and thus consistent with a slow strategy (Del Giudice, 2009a, 2009b; Salmon et al., 2009), more research on this profile is needed before any firm conclusion can be drawn.

**Depression**

Depression is characterized by protracted episodes of distress and low, dejected mood. While the DSM-5 supports a unitary view of depression—epitomized by the inclusive diagnosis of “major depressive disorder”—the clinical presentation of depression is quite heterogeneous (Baumeister & Parker, 2011). Attempts to subtype depressive disorders based on empirical patterns of symptom co-occurrence consistently identify (a) a subtype characterized exclusively by depressed mood and feelings of worthlessness; (b) one or more subtypes characterized by somatic symptoms in absence of depressed mood; and (c) one or more subtypes in which depressed mood and somatic symptoms coexist (Carragher, Adamson, Bunting, & McCann, 2009; Chen, Eaton, Gallo, & Nestadt, 2000; Sullivan, Prescott, & Kendler, 2002).

Somatic symptoms of depression include sleep disturbances (insomnia or hypersomnia), appetite disturbances (increased or decreased appetite), psychomotor disturbances (agitation or retardation), fatigue, and pain. All these symptoms are functionally related to the stress response system (SRS), and in particular the hypothalamic-pituitary-adrenal axis (HPA). “Typical” symptoms—insomnia, decreased appetite, psychomotor disturbances—are associated with a hyperactivated HPA; “atypical” symptoms—hypersomnia, increased appetite, fatigue and pain—have been linked to HPA hypoactivation, which often occurs as an exhaustion phase following prolonged periods of hyperactivation (Baumeister & Parker, 2011; Miller, Chen, & Zhou, 2007; Taylor & Fink, 2008; Tops, Riese, Oldehinkel, Rijsdijk, & Ormel, 2008; but see O’Keane, Frodl, & Dinan, 2012). While the incidence of “pure” mood depression (i.e., depression without somatic symptoms) is similar in males and females, that of somatic depression is strongly female-biased, resulting in higher overall rates of depression in females (Angst, Gamma, Benazzi, Ajdacic, & Rössler, 2007; Carragher et al., 2009; Chen et al., 2000; Halbreich & Kahn, 2007; Baumeister & Parker, 2011, Silverstein, 2002; Sullivan et a. 2002). Women are especially likely to experience somatic depression in which typical and atypical symptoms alternate over time, suggesting cycles of HPA hyperactivation followed by exhaustion (Angst et al., 2007; Baumeister & Parker, 2011). Depression is only moderately heritable; the genetic factors predisposing to depression are virtually the same that predispose to generalized anxiety disorder (GAD), underscoring the strong overlap between stress, anxiety, and depression (Hettema, 2008; Lahey et al. 2008; Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011; see also Li, McGue, & Gottesman, 2012).

Most evolutionary theories of depression focus on low mood and its motivational and behavioral correlates (for exceptions see Korte et al., 2005; Raison & Miller, 2012). In the
prevailing view, depressed mood is an adaptive defensive mechanism, whereas clinical depression is usually maladaptive and reflects a dysfunction of the same mechanism (e.g., Allen & Badcock, 2003; Gilbert & Allan, 1998; Nesse, 2006; Nettle, 2004, 2009). A number of theorists have argued that depression may be an adaptation itself (e.g., Hagen, 1999; Price, Sloman, Gardner, Gilbert, & Rohde, 1994; Sloman & Price, 1987; Watson & Andrews, 2002); while this hypothesis appears reasonable in the specific case of postpartum depression (Hagen, 1999), there are reasons to doubt its applicability to depressive disorders as a whole (see Nesse, 2006; Nettle, 2004; Nettle & Bateson, 2012).

The function of low mood as a protective mechanism is twofold. First, low mood helps people disengage from the pursuit of central life goals that have become unproductive (Nesse, 2000). Second and more specifically, it promotes a risk-averse approach in unfavorable social circumstances—especially following losses in social support, close relationships, and social status or dominance (Allen & Badcock, 2003; Brown, Harris, & Hepworth, 1995; Gilbert, 1992; Kendler, Hettema, Butera, Gardner, & Prescott, 2003b; Nettle, 2009; Nettle & Bateson, 2012; Price et al., 1994). Such events tend to arouse shame and guilt, two emotions that are strongly associated with depression (Kim, Thibodeau, & Jorgensen, 2011; O’Connor, Berry, Weiss, & Gilbert, 2002). Predictably, males are more susceptible to status loss, while the depressogenic effects of reduced social support and social rejection are much stronger in females (Kendler, Myers, & Prescott, 2005; La Greca, Davila, & Siegel, 2009; McGuire & Troisi, 1998; Thompson, McKowen, & Asarnow, 2009). Low mood may also be useful in soliciting help from friends and relatives (Watson & Andrews, 2002), at least when it occurs with moderate intensity.

The main limitation of these models is that they concentrate on low mood but tend to ignore the stress-related components of depression. However, most subtypes of depression involve SRS-mediated somatic symptoms in addition to—or even in place of—depressed mood. In order to capture the full spectrum of depressive disorders, one has to consider two partly independent dimensions of individual differences, affective reactivity and stress reactivity. While affective reactivity determines one’s susceptibility to episodes of low mood (Nettle, 2004), stress reactivity is the crucial factor in the development of somatic symptoms. Thus, a complete evolutionary account of depression cannot be separated from evolutionary models of SRS functioning (e.g., Boyce & Ellis, 2005; Del Giudice et al., 2011; Korte et al., 2005).

Most relevant to the present discussion, the Adaptive Calibration Model (ACM; Del Giudice et al., 2011) explicitly employs life history theory to explain individual differences in SRS responsivity. In the ACM, high stress responsivity is associated with fast strategies in dangerous and unpredictable contexts, where it increases vigilance to danger, but also with slow strategies in safe and highly predictable environments, where it increases openness to opportunities and sensitivity to social feedback (Boyce & Ellis, 2005; Ellis, Jackson, & Boyce, 2006). Furthermore, males exposed to severely stressful contexts are expected to develop “unemotional” patterns of muted SRS responsivity more often than females (see Del Giudice et al., 2011 for details).

Taken together, evolutionary models of depressed mood and stress responsivity predict a complex relation between depression and life history strategy. Both fast and slow strategists can fail to obtain or maintain crucial social resources—status, dominance, and support—resulting in
episodes of depressed mood and risk for clinical depression. Several pieces of evidence support the idea that depression can occur in association with slow life history strategies. For example, depression is the diagnostic category that contains the highest proportion of individuals with secure attachment representations (Bakermans-Kranenburg & van IJzendoorn, 2009), and occurs even in individuals—such as the “abstainers” described by Moffitt and Caspi (2005)—that display negligible levels of externalizing behaviors. Furthermore, some subtypes of depression—in particular those characterized by pure depressed mood or pure somatic symptoms—are associated with very low rates of trauma, neglect, and abuse, comparable to those reported by non-depressed individuals (Sullivan et al., 2002). At the slow end of the continuum, males and females are both expected to develop relatively high levels of stress responsivity (Del Giudice et al., 2011), even if the actual intensity of stress responses is buffered by the availability of social support and lack of chronic stressors. As a result, symptom profiles at the slow end of the spectrum should not differ greatly between the sexes.

Moving toward the fast end of the continuum, both sexes face increasing threats to their ability to gain and maintain social resources. The availability of social support and stable, intimate relationships declines rapidly as environments become dangerous and unpredictable, exposing females to increased risk for depression mood. At the same time, sex differences in stress responsivity can be expected to become proportionally larger, as more males develop unemotional patterns marked by a hyporesponsive SRS (Del Giudice et al., 2011). Hyperactive SRS profiles can be adaptive in dangerous and unpredictable contexts, especially in females (Del Giudice et al., 2011); however, they also increase the risk of SRS dysregulation and dysfunction. In total, fast life history strategies should lead to increased risk for depression in both sexes, with females showing the highest rates of depressed mood and somatic symptoms. Consistent with these predictions, early and/or fast sexual maturation is a risk factor for depression in both sexes, with stronger effects in females (Graber, 2009; Mendle et al., 2007, Mendle & Ferrero, 2012). In addition, depression subtypes involving a combination of low mood and somatic symptoms are overwhelmingly more common in females, and are also associated with the highest rates of early trauma, neglect and abuse (Sullivan et al., 2002). Further support for the association between depression and fast spectrum pathology comes from studies showing that, in both adolescents and adults, depression often co-occurs with externalizing disorders (Herman, Ostrander, Walkup, Silva, & March, 2007; Vaidyanathan, Patrick, & Iacono, 2011). Aggressive, impulsive, and self-aggrandizing behaviors in childhood predict later onset of depression, especially in males (Block, Gjerde, & Block, 1991; Dussault, Brendgen, Vitaro, Wanner, & Tremblay, 2011; Gjerde, 1995; Lahey & Waldman, 2012). This is reflected in the overall association of depression with lower agreeableness, lower conscientiousness, and disinhibition (Kotov et al., 2010).

In conclusion, depressive disorders comprise a heterogeneous cluster of conditions, most of which are likely maladaptive. The many clinical subtypes of depression reflect different combinations of depressed mood symptoms and SRS-mediated somatic symptoms. From a life history perspective, the evidence indicates that depression may occur at both ends of the fast-slow continuum; this suggests the existence of functionally distinct clusters of depressive disorders, similar to those identified in the obsessive-compulsive spectrum or in the spectrum of eating disorders. Unfortunately, the current literature defines depression subtypes exclusively in terms of symptom co-occurrence; while they may show some overlap with life history strategy, those subtypes are unlikely to accurately reflect the functional distinction between fast and slow
spectrum psychopathology. The only plausible generalization from the available evidence is that combinations of depressed mood and high levels of somatic symptoms may be specifically associated with fast life history strategies, particularly in females. Further research in a life history framework should attempt to identify functional subtypes of depression based on motivation, personality, self-regulation, and comorbidity with other fast and slow spectrum disorders. For example, a promising criterion for slow spectrum depression is the presence of chronic guilt feelings and hyperactive altruistic concerns (see Kim et al., 2011; O’Connor et al., 2002; Quiles & Bybee, 1997). An in-depth life history analysis of depressive disorders may contribute to clarify the complex epidemiology of this group of disorders.

Summary and Integration

In this section I carried out an initial life history analysis of six categories of common mental disorders. Taken together, the results paint a coherent picture of how individual differences in life history strategy translate into specific patterns of risk for psychopathology. The constellation of fast spectrum conditions includes externalizing disorders, schizophrenia spectrum disorders, OCD with autogenous obsessions, the dysregulated subtype of eating disorders (typically expressed as BN), and depressive disorders characterized by a combination of mood and somatic symptoms. These disorders tend to co-occur, both within families and within individuals; many of them share elements of impulsivity, disinhibition, and/or bizarre ideation.

Slow spectrum psychopathology includes OCPD, OCD with reactive obsessions, autism spectrum disorders, the perfectionist and overcontrolled subtypes of eating disorders, and a cluster of depressive disorders of lesser severity. These comorbid disorders tend to share elements of inhibition, overcontrol, and cognitive rigidity. They are also characterized by lack of association with standard risk factors for psychopathology such as stressful life events, low SES, and early abuse; in some cases, they are actually associated with more favorable ecological and socio-economic conditions.

Among the disorders reviewed here, externalizing conditions and OCPD are the best candidates as adaptive or potentially adaptive phenotypes that are nevertheless labeled as disorders because of their socially and/or personally undesirable aspects. The same might apply to the milder conditions in the autistic and schizophrenic spectrum, although schizophrenia and severe autism are almost certainly maladaptive. Between adaptive phenotypic variants and destructive dysfunctions lies a grey zone of conditions that may be caused by maladaptive expression levels of potentially adaptive personality traits. Extreme, maladaptive variants of trait expression may be maintained in a population by natural and sexual selection for the adaptive version of the traits in question, including selection through assortative mating.

Other disorders in the fast and slow spectrum may be best understood as dysfunctions and/or individually maladaptive outcomes of up-regulated defensive mechanisms. Both reactive OCD and depression fit this profile. Obsessive symptoms depend on the activity of the hazard-precaution system, a defensive mechanism specialized for dealing with low-frequency potential threats. Depressive symptoms depend on the stress response system (including the HPA axis) and on the affective mechanisms that mediate low mood responses to social loss, failure, and defeat. Finally, eating disorders show strong functional connections with female competition for mating.
and status. While some milder or short-lived instances of disordered eating may represent adaptive or potentially adaptive strategies, severe EDs are more consistent with dysfunctional or maladaptive outcomes of sexual competition. Reproductive suppression is another defensive process that may trigger eating disorders, although this hypothesis remains much more speculative at this time.

This classification is of course still provisional, and many gaps and questions remain—for example about the possible functional heterogeneity of autism and schizophrenia, the role of reproductive suppression in disordered eating, or the identification of fast and slow spectrum subtypes of depression. However, even these initial results illustrate how a life history framework can bring an integrative perspective to evolutionary psychopathology, highlight connections between previously separate models, and suggest a host of novel empirical questions. The same approach can be easily extended to other disorders I have not reviewed in detail. As noted by Brüne and colleagues (2010), borderline personality disorder (BPD) bears all the hallmarks of fast life history strategies—impulsivity, unstable attachments, risk-taking, promiscuous sexuality, antisocial and paranoid personality features, and high comorbidity with externalizing disorders (see Brüne et al., 2010; Crowell, Kaufman, & Lenzenweger, 2013). Indeed, BPD may be best understood as a female-typical manifestation of the externalizing spectrum. Other likely examples of fast spectrum pathology are disorders in the bipolar spectrum; these conditions show substantial genotypic and phenotypic overlap with schizotypy and schizophrenia, including a familial association with enhanced creativity (see Crespi et al., 2010; International Schizophrenia Consortium, 2009; Kyaga et al., 2011; Nettle, 2001; Yu et al., 2010). A provisional classification of slow and fast spectrum disorders is shown in Figure 1. It is reasonable to expect that, in the coming years, the life history taxonomy outlined here will be extended to cover a large fraction of the most common psychopathological conditions.

**Figure 1.** Provisional life history taxonomy of common mental disorders. BPD = borderline personality disorder, OCD = obsessive-compulsive disorder, OCPD = obsessive-compulsive personality disorder.
Implications for Taxonomy

The life history framework advanced in this paper has far-reaching implications for the classification of mental disorders. Current taxonomic approaches include the DSM system of diagnostic categories—mostly based on symptom similarity—and a family of empirical approaches based on patterns of genetic and phenotypic correlations between disorders (e.g., Kendler et al., 2003a; Krueger, 1999; Krueger et al., 2002, 2011; Lahey et al., 2008, 2011; Verona et al., 2011; Watson, 2005; Watson, O’Hara, & Stuart, 2008).

Empirical taxonomic studies suggest the existence of broad, hierarchically organized clusters of disorders that overlap only in part with DSM categories. The fundamental distinction in empirical taxonomies is that between internalizing and externalizing disorders. In turn, internalizing disorders comprise a cluster of distress disorders (depression, GAD, post-traumatic stress disorder) and a cluster of fear disorders (panic disorder, agoraphobia, social phobia, and specific phobias; Clark & Watson, 2006). Bipolar and obsessive-compulsive spectrum disorders are usually regarded as additional clusters within the internalizing spectrum, although their exact placement is more problematic (Lahey et al., 2008; Slade, 2007; Watson, 2005). In a recent study, eating disorders were also included in the internalizing spectrum based on phenotypic correlation patterns (Forbush et al., 2010). A new factor-analytic study by Caspi and colleagues (2013) supplemented the internalizing and externalizing categories with a thought disorder factor comprising schizophrenia, mania (bipolar spectrum), and obsessive-compulsive disorder (OCD). Moreover, the authors identified a general, higher-order factor of psychopathological risk they labeled the p factor (see Caspi et al., 2013). In the present perspective, the p factor might capture the non-specific role played by harmful mutations and developmental insults, which increase the risk for a broad range of disorders across the life history spectrum (see above; see also Keller & Miller, 2006). To the extent that the p factor also reflects a general dimension of environmental stress, it may also show a degree of correlation with the fast-slow continuum.

While empirical taxonomies are valuable and informative, they are also limited by their lack of organizing theoretical principles. A life history framework can overcome those limitations and offer a more solid foundation for the taxonomy of mental disorders. In particular, I surmise that the fast-slow distinction (Figure 1) is both more inclusive and more accurate than the internalizing-externalizing distinction. It is more inclusive because it effortlessly integrates mood and anxiety disorders with personality disorders, schizophrenia spectrum disorders, and autism spectrum disorders—all within the same conceptual framework. In contrast, standard empirical taxonomies exclude SSDs, ASDs, and most personality disorders because those conditions are not primarily characterized by mood/emotional alterations and do not fit the conceptual distinction between “internalization” and “externalization” (the recent analysis by Caspi and colleagues [2013] is a partial exception). It is more accurate because it resolves many inconsistencies inherent in the basic internalizing-externalizing distinction and its further elaborations (see Watson et al., 2008).
Limitations of the Internalizing-Externalizing Distinction

A life history analysis shows that while externalizing disorders form a functionally homogeneous category, the internalizing spectrum consists of heterogeneous and functionally divergent conditions. To begin with, depression and GAD—often regarded as prototypical internalizing disorders—are in fact “bridge” diagnoses that overlap with both internalizing and externalizing disorders at the phenotypic, genetic, and developmental level (Block et al., 1991; Gjerde, 1995; Lahey et al., 2008, 2011; Vaidyanathan et al., 2011). Similar problems arise with OCD and eating disorders. While OCD is usually placed in the internalizing category, it shows atypically large correlations with externalizing disorders (Lahey et al., 2008), and a close relation with the schizophrenia/bipolar spectrum (Caspi et al., 2013). In a life history perspective, this occurs because OCD is a heterogeneous diagnosis with both fast and slow spectrum subtypes. The assignment of EDs to the internalizing spectrum on purely correlational grounds (Forbush et al., 2010) is also unsatisfactory. Here, the main problem is that standard diagnostic labels (AN and BN) do not reflect distinct functional types. Only the dysregulated subtype of BN shows substantial overlap with externalizing disorders; treating BN as a unitary construct can only yield misleading results. The low stability of internalizing symptoms across development (Haberstick, Schmitz, Young, & Hewitt, 2005; Krueger, Caspi, Moffitt, & Silva, 1998; Vollebergh et al., 2001) may be another cue to the functional inconsistency of this category.

The idea of a broad spectrum of internalizing disorders, with sub-categories characterized by similar affective profiles—fear disorders, distress disorders, and so forth—is both elegant and parsimonious. However, this hypothetical hierarchical structure breaks down if supposedly internalizing disorders—for example the dysregulated subtype of eating disorders—turn out to be functionally and phenotypically closer to the externalizing spectrum than to other internalizing disorders. Moreover, affective and emotional dimensions—such as negative affectivity, fear, and distress—are unreliable indicators of the underlying motivational traits, and are thus unlikely to capture functional differences between related clusters of disorders. In total, I wish to suggest that the internalizing-externalizing distinction may be problematic because it is in large part illusory. The obvious genotypic and phenotypic coherence of the externalizing spectrum may have led researchers to assume that internalizing disorders must form a symmetrical category with similar properties of coherence. If my analysis is correct, however, this assumption is mistaken, and the “internalizing spectrum” is a largely artificial collection of disorders with divergent functional properties. Of course, testing this hypothesis requires the ability to split DSM diagnostic categories into functionally meaningful subtypes, something that is not yet possible with current DSM-based datasets.

Conclusion

Researchers in evolutionary psychopathology face a pressing need to overcome the present state of theoretical fragmentation and move the field toward a truly integrative understanding of mental disorders. In this paper I outlined a general conceptual framework for the analysis of mental disorders based on the principles of life history theory. As I have shown, the framework can be fruitfully applied to a broad range of conditions, offering an integrative perspective on evolutionary psychopathology and suggesting a host of novel empirical questions. The life history taxonomy outlined in this paper is based on the novel distinction between fast
spectrum and slow spectrum psychopathology, and offers a promising alternative to both the atheoretical classification system of the DSM and the internalizing-externalizing distinction at the heart of current empirical taxonomies. Of course, such a broad-band approach is only the first step toward a comprehensive functional taxonomy of mental disorders; future models will have to progressively include specific motivational domains (e.g., mating, affiliation, harm prevention), specific behavioral and motivational mechanisms, and so forth. Crucially, a functional approach to taxonomy should not be expected to yield strictly hierarchical classifications; for example, a category of mating-related disorders would cut across the fast-slow distinction, and may well overlap with a category of disorders related to affiliation processes.

In future elaborations of the framework, its scope should be extended beyond motivation and behavior to include the cognitive, neurobiological, and genetic/epigenetic correlates of life history variation. While not formulated in an evolutionary perspective, the neurobiological theory of behavioral programs (Tops, Boksem, Luu, & Tucker, 2010; Tops & Boksem, 2010; Tucker, Luu, & Pribram, 1995; Tucker & Luu, 2007) is potentially consistent with a life history approach. Other promising models of individual differences in cognition and neurobiology (e.g., Del Giudice et al., 2011; Figueredo et al., 2006; Woodley, 2011) are explicitly based on life history concepts, facilitating theoretical integration. Moving to the genetic and epigenetic levels of analysis, promising candidates for integration include life history-informed approaches to the epigenetic effects of parental behavior (Meaney, 2007) and the diametrical model of autism and psychosis (Crespi & Badcock, 2008; Crespi et al., 2010; Del Giudice et al., 2010). Another important step will be to fully integrate the present framework with the recent sexual selection model of internalizing/externalizing disorders advanced by Martel (2013). A sexual selection perspective provides insight in the differences between male-biased disorders that typically emerge in childhood (e.g., conduct disorders, attention deficit-hyperactivity disorders) and female-biased disorders that develop in adolescence or early adulthood (e.g., depression, social phobia). In addition, it may help clarify the role and timing of environmental risk factors in the two sexes.

In conclusion, life history theory offers powerful tools for understanding not just individual differences in the normative range of personality and behavior, but also individual differences in the risk for a broad range of mental disorders. A life history approach calls for a revision of classical concepts (such as the internalizing-externalizing distinction) and the reorganization of existing diagnostic categories based on functional criteria. In return, it affords insight in crucial issues including the etiological role of environmental stress, the interplay between risk and protective factors, the meaning and distribution of sex differences, the structure of comorbidity patterns, and so forth. When framed in the right perspective, these apparently separate issues come together like the pieces of a puzzle, illuminating each other and revealing the contours of the broader picture. If future research will confirm its usefulness, the framework outlined in this paper could represent a significant step toward a truly integrative science of mental suffering.
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An Evolutionary Life History Framework for Psychopathology


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