Psychological Inquiry, 25: 261–300, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.884918

Routledge
Taylor & Francis Group

TARGET ARTICLE

An Evolutionary Life History Framework for Psychopathology

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In this article, 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.

Key words: *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, 2004b; 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 nonclinical range of individual differences in personality and behavior.

In this article, 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; G. T. Harris, Rice, Hilton, Lalumière, & Quinsey, 2007; Jonason, Li, Webster, & Schmitt, 2009; D. N. 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 (EDs; 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 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. Although 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 to refine them, connect them to one another, and ultimately integrate them within a common frame of reference.

Overview of the Article

I begin by introducing the basic concepts of life history theory in nonmathematical 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 (SSDs), autism spectrum disorders (ASDs), obsessive-compulsive (OC) spectrum disorders, EDs, 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. Because natural selection promotes reproductive success rather than happiness or health (Cosmides & Tooby, 1999; Gluckman, Low, Buklijas, Hanson, & Beedle, 2011; Nesse, 2001a, 2004b), biologically adaptive traits may or may not be socially desirable or conducive to health and well-being. In this article, I always employ the terms "adaptation" and "adaptive" in their biological sense.

Mental disorders are the main topic of this article, yet the concept of disorder has no straightforward biological definition (Nesse, 2001a). In an influential article, 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. Although 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 article 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; K. Hill, 1993; K. 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. Because 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 (B). 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 (K. 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; K. 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 quality. Although 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).

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,

[1]The term "strategy" denotes 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.

Ellison, & 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 (SRS), 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 SRS 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 the 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 beneficial only 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- versus future-oriented strategies should result in consistent individual differences in risk-related traits, such as boldness, exploration, and aggression (see also E. M. Hill, Ross, & Low, 1997; K. Hill, 1993; 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, 1999b; 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. Although 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; J. H. 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; J. H. 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; L. T. 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). Although 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, 1999b). 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 girls), precocious sexuality, unstable couple relationships, and promiscuous mating styles (reviewed in Belsky, 2012; Del Giudice, 2009b; Gillath & Schachner, 2006; James, Ellis, Schlomer, & Garber, 2012). Chisholm (1999a; 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, 2011). 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; P. Chen & Vazsonyi, 2011; Curry, Price, & Price, 2008; Dunkel & Decker, 2010; A. C. Harris & Madden, 2002; E. M. Hill et al., 1997; Kahn, Kaplowitz, Goodman, & Emans, 2002; Kruger, Reischl, & Zimmerman, 2008; Lejuez et al., 2002; X. T. 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, Campbell, & Muncer, 2013; James et al., 2012; Nettle, Coall, & Dickins, 2011; Simpson et al., 2012); in addition, attachment insecurity seems to be an important psychological mediator of these effects (see earlier).

Following a psychometric approach, Figueredo and colleagues (Figueredo, Cabeza de Baca, & Woodley, 2012; Figueredo, Vásquez, Brumbach, & Schneider, 2004, 2007; Figueredo et al., 2005) 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 ingroup, and low levels of interpersonal aggression and social deviance (Figueredo, Andrzejczak, Jones, Smith-Castro, & Monetro-Rojas, 2011; Figueredo, Gladden, & Beck, 2012; Figueredo & Jacobs, 2010; D. N. 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, although 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* (DeYoung, 2006; Digman, 1997). 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, whereas 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, 2011). Imagination correlates with positive schizotypy (see next), which in turn predicts unrestricted sociosexuality, reduced commitment in long-term relationships, and larger numbers of sexual partners (Del Giudice et al., 2010; Nettle & Clegg, 2006). 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). It is hoped that future research will 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 male participants (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, whereas 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 next, 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, as 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 argue that the diagnostic label of OC 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, as either 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 et al., 2009; Mealey, 1995; Salmon et al., 2009). As I discuss next, 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 Del Giudice et al., 2010). By their very nature, life history trade-offs involve costs in

both directions. Although the costs associated with fast strategies may appear more dramatic, those associated with slow strategies are neither 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, although 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. Although prosociality can be hugely rewarding, it also makes people vulnerable to cheating and exploitation, with potentially devastating consequences. Prosociality also has opportunity costs: Although 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, and sensitive to criticism, and they 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 earlier) 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, 2013) 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 overcontrolling 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 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, 2004b; 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, 2004b; Nesse & Jackson, 2006). When defenses activate inappropriately and/or respond with excessive intensity, the outcome may be correctly recognized as maladaptive (see next). 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, 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 coopted 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 although 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 (e.g., because a mutation in the offspring makes it is 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 (Figueredo & 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; Frankenhuis & 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 Frankenhuis & 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. Although 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, 2001b, 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 earlier), 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. Because 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 Benros, Mortensen, & Eaton, 2012; Patterson, 2011). 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.

Although 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 (e.g., 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, 2001a).

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; X. T. 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, as 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, because 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 1st 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 next) 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; Figueredo et al., 2006; Worthman, 2009; Worthman & Brown, 2005). In this article I mainly focus on the psychological level of analysis as a useful first approximation. A nonexhaustive list of the correlates of fast and slow spectrum psychopathology is presented in Table 1.

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*. Although 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, 2004b; see also Keltner, Haidt, & Shiota, 2006), and the association between emotions and the underlying motivational processes is often remarkably nonspecific, 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, whereas 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, SSDs, ASDs, OC spectrum disorders, EDs, 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.

For each category, I examine the available empirical evidence to determine whether the relevant

Table 1. *Correlates of Fast and Slow Spectrum Psychopathology.*

	Fast Spectrum Psychopathology	Slow Spectrum Psychopathology
Motivation	Social antagonism	Social compliance, conformity
	Unstable attachments	Stable attachments
	Precocious sexuality	Delayed sexuality
	Sexual promiscuity, high sex drive	Sexual restraint, low sex drive
	Sensation/novelty seeking	Preference for routines
	Risk taking	Risk aversion, harm prevention
Self-regulation	Disinhibition, impulsivity	Inhibition, restraint
	Discounting of future rewards	Discounting of immediate rewards
Personality traits	Low conscientiousness	High conscientiousness
	Low agreeableness	High agreeableness
Sexual maturation	Early, fast maturation	Late, slow maturation
Environment	Harsh, unpredictable	Safe, predictable
	High exposure to stressors	Low exposure to stressors

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., OC 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 (AN) and bulimia nervosa (BN) is empirically unreliable and only weakly related to individual differences in life history strategy; in contrast, ED profiles based on personality (Westen & Harnden-Fischer, 2001) show a remarkably good fit with the fast–slow distinction (see next). 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., 2011; Krueger et al., 2002). Externalizing disorders are also associated with high risk for substance abuse (Kendler, Prescott, Myers, & Neale, 2003; 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

Table 2. *Life History Analysis of Common Mental Disorders.*

Disorder Category	Life History Classification
Externalizing spectrum	Fast spectrum
Schizophrenia spectrum	Fast spectrum
	[possibly heterogeneous; age of onset]
Autism spectrum	Slow spectrum
	[possibly heterogeneous]
Obsessive-compulsive spectrum	Fast spectrum: endogenous obsessions
	Slow spectrum: reactive obsessions, obsessive-compulsive personality disorder
Eating disorders	Fast spectrum: dysregulated profile
	Slow spectrum: perfectionistic and overcontrolled profiles
Depression	Heterogeneous
	[fast spectrum: depressed mood + somatic symptoms]

existence of a coherent, heritable dimension of externalizing behavior (Kendler, Prescott, et al., 2003; Krueger et al., 2002; Lahey & Waldman, 2012). In the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed. [*DSM-5*]; American Psychiatric Association, 2013), externalizing disorders—including oppositional defiant disorder, conduct disorder, and antisocial personality disorder—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 & Ferrero, 2012; Mendle et al., 2007), earlier onset of sexual activity (e.g., Armour & Haynie, 2007; Lévesque, Bigras, & Pauzé, 2010; van Goozen, Cohen-Kettenis, Matthys, & 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 (S. 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 nonclinical 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; G. T. Harris et al., 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 (G. T. Harris et al., 2007; Jonason et al., 2009; Kastner & Sellbom, 2012; Lalumière et al., 2008; Lalumière & Quinsey, 1996; Mishra & Lalumière, 2008). Unsurprisingly, psychopathic traits correlate with measures of fast life history strategy, risk-taking, and present orientation (Figueredo & 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; S. Jones, Miller, & Lynam, 2011; Krueger et al., 2011; Lynam & Derefinko, 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 SRS (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, & Lonnqvist, 2003; MacCabe, Koupil, & Leon, 2009; Nanko & Moridaira,

1993), most evolutionary scholars regard this disorder as a maladaptive outcome of dysregulated sociocognitive processes (e.g., Burns, 2004; Crow, 1995, 1997; Keller & Miller, 2006; McGuire & Troisi, 1998; see Stevens & Price, 1999, for an exception). 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-Germeys, Delespaul, & Krabbendam, 2009). Although 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; G. F. Miller & Tal, 2007; Nettle, 2006b; Nettle & Clegg, 2006; 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 nonclinical 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, Sugimori, Bando, & Tanno, 2011; S. R. Ross, Lutz, & Bailey, 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, 2004a). 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. Although 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, 2008; Happé, Ronald, & Plomin, 2006; 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; Grinter, 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 nonpathological 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., 2011; Baron-Cohen et al.,2001). 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). It is intriguing that 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 men—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 noncompletions 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 overrepresented 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 involved only 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, as 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, although 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 sociocognitive 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 OC spectrum are primarily characterized by patterns of compulsive, repetitive thoughts and/or behaviors, usually associated with worry and anxiety. In addition to 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 OC 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). OCDs 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 female individuals 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). 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. OC 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 nonclinical samples (Glass, 2012; Glass, personal communication, August 13, 2012). Even more important, OC spectrum disorders show high comorbidity with *both* autism spectrum (Anholt et al., 2010; Bejerot, 2007; Hollander, King, Delaney, Smith, & Silverman, 2003) and schizophrenia spectrum disorders (Lee & Telch, 2005; Poyurovsky et al., 2008; Poyurovsky & Koran, 2005; Sobin et al., 2000; Suhr, Spitznagel, & Gunstad, 2006). 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 versus reactive obsessions in OCD are

statistically robust and longitudinally stable and are associated with distinct patterns of brain activity (Besiroglu et al., 2011; Besiroglu et al., 2007; Moulding, Kyrios, Doron, & Nedeljkovic, 2007).

Although 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, Kim, & Kwon, 2005; Lee & Telch, 2005, 2010; 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 et al., 2005; Lee & Telch, 2010; Lee et al., 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). Of interest, 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 socioeconomic status (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

EDs are defined by heightened concern with body shape/weight and associated behaviors such as dieting, binge eating, purging, and exercising. EDs occur almost exclusively in females, and their age of onset peaks in adolescence (Hoek, 2006). The *DSM-5* distinguishes AN from BN based on body weight, and two subtypes of AN—restricting AN and binge eating/purging AN—based on the occurrence of bingeing episodes. However, empirical data overwhelmingly indicate that these diagnostic categories are largely artificial: ED symptoms co-occur at high rates, and diagnostic crossover—that is, 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 EDs 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;; Voland & Voland, 1989; Wasser & Barash, 1983). 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, Gabrielidis, & 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 nonclinical samples (Faer, Hendriks, Abed, & Figueredo, 2005; N. P. 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 (J. 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 EDs can be refined and extended by framing it in a life history perspective. Whereas 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, because a feminine body shape (including, e.g., 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 (e.g., facial attractiveness cues such as symmetry) increases accordingly (Confer, Perilloux, & Buss, 2010; Currie & Little, 2009; Lu & Chang, 2012; Zelazniewicz & Pawlowski, 2011).

Because youth is a better index of reproductive value than of current fertility, women 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 women who face intense mating competition should desire (a) a thinner body than fast life history women 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 women should be statistically overrepresented 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 bingeing 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, Eddy, Franko, et al., 2008; Thompson-Brenner, Eddy, Satir, Boiseeau, & Westen, 2008; 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. Whereas 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, Eddy, Franko, et al., 2008; Thompson-Brenner, Eddy, Satir, et al., 2008; Westen & Harnden-Fischer, 2001).

To sum up, EDs are associated with female competition at both ends of the fast–slow continuum and range from potentially adaptive strategies to frankly maladaptive dysfunctions. Whereas 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. Although 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). Although 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. Although 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; L. S. 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 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; G. E. Miller, Chen, & Zhou, 2007; Taylor & Fink, 2008; Tops, Riese, Oldehinkel, Rijsdijk, & Ormel, 2008; but see O'Keane, Frodl, & Dinan, 2012). Although the incidence of "pure" mood depression (i.e., depression without somatic symptoms) is similar in male and female individuals, 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; L. S. Chen et al., 2000; Halbreich & Kahn, 2007; Baumeister & Parker, 2011, Silverstein, 2002; Sullivan et al., 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 X. 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, 2013). 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; P. J. Watson & Andrews, 2002); whereas 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; G. W. Brown, Harris, & Hepworth, 1995; Gilbert, 1992; Kendler, Hettema, Butera, Gardner, & Prescott, 2003; 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, men are more susceptible to status loss, whereas the depressogenic effects of reduced social support and social rejection are much stronger in women (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 (P. J. 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. To capture the full spectrum of depressive disorders, one has to consider two partly independent dimensions of individual differences, *affective reactivity* and *stress reactivity*. Although 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 (Del Giudice et al., 2011) explicitly employs life history theory to explain individual differences in SRS responsivity. In the adaptive calibration model, 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, male individuals exposed to severely stressful contexts are expected to develop "unemotional" patterns of muted SRS responsivity more often than female individuals (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, men and women 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 depresses mood. At the same time, sex differences in stress responsivity can be expected to become proportionally larger, as more male individuals 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 female individuals (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 female individuals 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 female individuals (Graber, 2009; Mendle & Ferrero, 2012; Mendle et al., 2007). In addition, depression subtypes involving a combination of low mood and somatic symptoms are overwhelmingly more common in female individuals, and are 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 male individuals (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 OC spectrum or in the spectrum of EDs. Unfortunately, the current literature defines depression subtypes exclusively in terms of symptom co-occurrence; whereas 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 female individuals. 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, SSDs, OCD with autogenous obsessions, the dysregulated subtype of EDs (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, ASDs, the perfectionist and overcontrolled subtypes of EDs, 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 socioeconomic 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 gray 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, EDs show strong functional connections with female competition for mating and status. Although 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 EDs, 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 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, borderline personality disorder 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, Cheung, Leung, Chua, & McAlonan, 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.

Implications for Taxonomy

The life history framework advanced in this article 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, Prescott, et al., 2003; Krueger, 1999; Krueger et al., 2011; Krueger et al., 2002, Lahey et al., 2008; Lahey et al., 2011; Verona et al., 2011; D. Watson, 2005; D. 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, posttraumatic stress disorder) and a cluster of *fear disorders* (panic disorder, agoraphobia, social phobia, and specific phobias; Clark & Watson, 2006). Bipolar and OC 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; D. Watson, 2005). In a recent study, EDs 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 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 nonspecific role played by harmful mutations and developmental insults, which increase the risk for a broad range of disorders across the life history spectrum (see earlier; 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.

Although 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

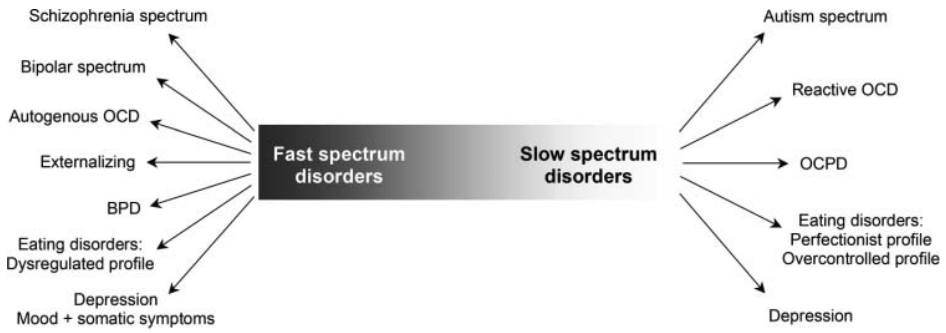

Figure 1. Provisional life history taxonomy of common mental disorders. BPD = borderline personality disorder; OCD = obsessive-compulsive disorder; OCPD = obsessive-compulsive personality disorder.

more *accurate* than the internalizing–externalizing distinction. It is more inclusive because it effortlessly integrates mood and anxiety disorders with personality disorders, SSDs, and ASDs—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 D. Watson et al., 2008).

Limitations of the Internalizing–Externalizing Distinction

A life history analysis shows that although 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; Lahey et al., 2011; Vaidyanathan et al., 2011). Similar problems arise with OCD and EDs. Although 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 subcategories 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 EDs—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 data sets.

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 article 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 article 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. Although not formulated in an evolutionary perspective, the neurobiological theory of behavioral programs (Tops & Boksem, 2010; Tops, Boksem, Luu, & Tucker, 2010; Tucker & Luu, 2007; Tucker, Luu, & Pribram, 1995) 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 article could represent a significant step toward a truly integrative science of mental suffering.

Note

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Acknowledgments

I am grateful to Mattie Tops, Jay Belsky, Bruce Ellis, Amanda Klimczuk, Willem Frankenhuis, and Romina Angeleri for the inspiration, feedback, and encouragement they provided throughout the writing of this article.

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Psychological Inquiry, 25: 301–306, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.904136

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COMMENTARIES

A Framework for Psychopathology Based on Life History Theory: A Landmark Formulation

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The target article provides a set of proposals for the application life history theory (LHT; as part of an evolutionary approach) to the classification of psychopathology and mental disorder. The model extends the application of LHT from a few well-studied mental disorders (primarily the externalizing disorders) to the whole of psychopathology and provides evolutionary psychiatry with a set of much needed and clear organizing principles.

Proposals for the application of evolutionary theory to the classification of mental disorder are, of course, not new. Nesse and Jackson (2006) argued that evolutionary theory was the missing biological foundation for psychiatric nosology and proposed that analysing the *motivational structure* of an individual's life (which is another term for considering their life history [LH] strategy) was an important aspect of applying evolutionary principles to the understanding of psychopathology. Also, Feierman (2006) proposed that human ethology can become the evolutionary biological basis for the classification of mental disorders. However, such considerations have not yet made their way into mainstream psychiatric thinking.

A major weakness of modern psychiatry has been the lack of a unifying theoretical framework. As a result, the discipline has been characterized by conceptual pluralism and a multiplicity of approaches that have tended to drift further apart over time (Abed, 2000; Brüne, 2008; McGuire & Troisi, 1998).

A number of major evolutionary psychiatric texts have been published over the past few decades (Brüne, 2008; McGuire & Troisi, 1998; Stevens & Price, 1996, 2000). Nevertheless, evolutionary science remains largely ignored by both mainstream psychiatry and by the wider medical community, and this has prompted calls to those in charge of setting the undergraduate medical curricula to recognize and correct this (Stearns, Nesse, Govindarajuc, & Ellison, 2010). It also remains true that evolutionary biology still does not feature on the curriculum of any postgraduate psychiatric training program anywhere in the world (Abed, 2000). Unlike evolutionary psychology, which is a flourishing and expanding academic discipline, evolutionary psychiatry remains a minority interest, and it is notable that there still isn't a single peer-reviewed journal dedicated to the subject currently in existence.

The reasons for the apparent lack of engagement with evolutionary ideas are, no doubt, complex but may be, in part, related to uncertainty or a lack of clarity regarding the clinical utility of such a theoretical approach. It appears that rather than considering evolutionary science as a foundational and basic science of psychiatry, psychiatrists have tended to view it as an interesting alternative approach (Nesse, 2012). It is, of course, unrealistic to expect a high-level metatheoretical framework such as evolutionary theory to yield quick or immediate results that would change practice at the clinical level but such changes are likely to come with time given the insights that become possible once various psychopathologies are subjected to evolutionary analysis.

The strengths of Del Giudice's (DG's) framework compared to rival proposals are its reliance on a single universal biological process (LH strategy) and its potential for detecting heterogeneity. The application of the framework provides the predictive power for detection of latent disorders or subtypes of disorders based primarily on the novel distinction between slow and fast spectrum disorders. DG makes a powerful and persuasive case for LH strategies organizing individual differences across multiple domains and hence providing a meaningful biological basis for understanding psychopathology. Thus, the theoretical coherence and potential usefulness of this distinction strengthens the author's case for supplanting the existing distinction of externalizing–internalizing disorders with the slow-fast spectrum distinction as a universal organising scheme for human psychopathology.

A further strength of the LHT framework is that it does not necessarily exclude or supplant other causative factors and explanatory theories. In fact, it is

clear that the LH framework positively requires other explanatory theories and explanations as it can neither help with the thorny question of the demarcation of psychopathology from adaptive responses nor explain certain salient features of mental disorders without reference to other explanatory systems. For example, whereas the framework predicts that males will be overrepresented in fast spectrum disorders (e.g., externalizing disorders), it does not offer an explanation for the stark female preponderance in eating disorders except through reference to sexual selection. These issues are fully recognized by the author and do not detract from the usefulness of the model; nevertheless, it is important to be aware of the limitations of the framework (although one might argue that this is simply a characteristic of the framework and not a limitation).

The framework must therefore integrate other known evolutionary causes for vulnerability to disease and disorder (such as mismatch), which are recognized major causes of both medical and psychiatric disorder (Brüne, 2008; Gluckman & Hanson, 2006; Nesse, 2005; Stearns et al., 2010). I discuss the potential relevance of mismatch to eating disorders and schizophrenia later on in my commentary.

The externalizing disorders (as would be expected) are presented as the prototypical conditions where DG's framework works particularly well. The model manages to provide a comprehensive and satisfying explanation for their aetiology, explain some of the most salient features of these disorders, and provide a sound biological basis for their classification. The framework also provides a useful explanatory framework for autistic spectrum disorder (ASD), obsessive-compulsive disorder (OCD), eating disorders, and schizophrenia to varying degrees. The benefits the framework offers to the understanding of depression are less clear at this stage.

With regard to ASD, the framework generates impressive insights into the reproductive advantage of mild or subclinical autistic traits and provides a coherent explanatory framework for the reproductive niche that such individuals occupy, namely, a male-typical slow spectrum strategy with low investment in mating and high parental investment. Clinical autism is the extreme version of this and is maladaptive. However, the true picture may prove more complicated with recent findings that a substantial minority of attention deficit/hyperactivity disorder individuals (fast spectrum) manifest with autistic traits to a significantly higher degree than controls (Kotte et al., 2013). This raises the possibility of heterogeneity (a possibility recognized by the author) of ASD.

The framework also works well in the analysis of OCD by uncovering the latent heterogeneity of this disorder with the reactive subtype being a slow spectrum, whereas the newly identified endogenous variant being a fast spectrum disorder. Similarly with eating disorders (ED), the analysis supports a rethinking of the current classification of ED patients into anorexia nervosa (AN), bulimia nervosa (BN), and ED not otherwise specified, which show considerable overlap, into more meaningful categories.

The case of schizophrenia also shows promise with regard to the adaptive advantage of schizotypy as a fast spectrum phenotype and, similar to the autistic scenario, the schizophrenic disorder is the maladaptive variant of this. Finally, in the case of depression, in contrast to the other categories discussed in the target article, the framework seems to generate fewer insights, and very few solid predictions seem to emerge for this disorder(s). Also, the author seems to dismiss rather too readily the suggestion that depression *can be* adaptive. For example, there is good reason to believe that the current diagnostic systems that rely primarily on symptom checklists devoid of context can lead to the overdiagnosis of depression and to an overestimation of its prevalence (e.g., Nesse, 2009; Wakefield, 2007). If this is accepted, then at least a proportion of diagnosed depression is an adaptive response to loss (similar to bereavement reaction) and to unpropitious situations (Nesse, 2000).

DG does acknowledge that depression is a complex and heterogeneous disorder with a complex relationship to the slow–fast spectrum of LH. At this point it is not clear what added value there is in identifying slow as opposed to fast life history spectrum subtypes of depression with regard to psychopharmacology, although one can imagine this distinction being relevant and valuable to designing and implementing psychological therapy interventions for depressed patients.

I now make some specific comments on ED and schizophrenia.

Eating Disorders

DG provides a detailed and up-to-date review of the evolutionary literature of these disorders. In summary, the evolutionary formulations are the reproductive suppression hypothesis and the female sexual competition hypothesis. However, DG does not distinguish between two variants of the hypothesis of reproductive suppression. One proposes that AN is a form of reproductive self-suppression (Voland & Voland, 1989), whereas the other contends that AN is the result of reproductive suppression of subordinate females by dominant females through female intrasexual sexual competition (Mealey, 2000). Both variants of the reproductive suppression hypothesis are relevant, more or less, exclusively to AN as the other variants of ED have a body mass index within the normal range or above. Furthermore, there is no

evidence that AN patients are submissive or subordinate, and therefore, Mealey's (2000) hypothesis that AN is a "loser's strategy" has received little empirical support (Faer, Hendriks, Abed, & Figueredo, 2005).

The prevalence of BN is more than 3 times that of AN in epidemiological surveys (Hoek & van Hoeken, 2003), and if we add patients with ED not otherwise specified and those with subclinical disordered eating patterns, the proportion of females with amenorrhoea will be a very small fraction of the total female population with eating problems. Hence, if eating disorders in general represent a strategy for reproductive suppression, then it appears to be a strategy that is rarely successful given that the vast majority of females remain fertile while continuing to show evidence of clinical or subclinical symptoms.

Also, although reproductive suppression can be compatible with slow spectrum psychopathology (delayed reproduction and low mating effort), it would struggle to explain the fast spectrum traits within the ED population.

The sexual competition hypothesis (SCH; Abed, 1998), on the other hand, proposes that ED in all their forms arise from intense female intrasexual competition for mates. In this model, the pursuit of thinness is a female strategy for mate attraction and retention (through the display of signs of youth and hence maximizing mate value) and is adaptive in its mild to moderate forms and that it is the extreme form of this adaptation that we identify as ED. Hence, SCH can accommodate the whole range of ED phenomena (both clinical and subclinical) and would be compatible potentially with both fast and slow spectrum variants. It is therefore, currently the most parsimonious explanation for the whole range of these disorders.

In a questionnaire study of a nonclinical female population in the United States, we found a significant correlation between disordered eating and the intensity of female competition for mates, which is clearly supportive of SCH (Faer et al., 2005). Also, in an extended replication of the aforementioned study of a nonclinical female population in the United Kingdom, we found a similarly significant correlation between competition for mates and disordered eating (which is also supportive of SCH) and a significant correlation between fast LH strategy (Low K strategy) and disordered eating. Our explanation for this latter finding was that in a nonclinical population there are likely to be a predominance of BN-type eating patterns (hypothesized to show a fast LH strategy) with very few truly AN individuals (Abed et al., 2012). A similar explanation (low or absent AN cases) may be considered for the finding in another study of a nonclinical population by a different group of researchers, of a significant correlation between high executive function (associated with slow LH) and low levels of disordered eating (Salmon, Figueredo, & Woodburn, 2009).

Further support for the specific predictions made by the SCH (Abed, 1998) came from the study of another nonclinical population by another group of researchers, whereby male homosexuals were found to show a higher vulnerability to disordered eating, whereas lesbian subjects showed the opposite trend (Li, Smith, Griskevicius, Cason, & Bryan, 2010). Both these findings were explicitly predicted in the original hypothesis (Abed, 1998). In contrast to SCH, the reproductive suppression model would have no explanation for these latter findings.

We hypothesized, therefore, that in a clinical sample, although both AN and BN will score highly on female competition for mates, AN would show evidence of slow LH and BN patients would be on the fast end of the LH strategy (the actual terminology used was high and low K strategies corresponding to slow and fast LH strategies, respectively; Abed et al., 2012). We intended, therefore, to test this prediction through studying a clinical population of ED patients using a psychometric approach. Unfortunately, we were unable to continue with this study due to the retirement of a key collaborator.

However, subjecting AN and BN to a life history analysis, DG was able within the target article to demonstrate through the review of existing research findings that patients with BN are higher on impulsivity, sensation seeking, and novelty seeking than AN patients; that they tend to mature earlier and to have sex at a younger age; and that AN shows considerably more overlap than BN with OCD, obsessive-compulsive personality disorder, and ASD. In addition, that compared with bingeing/purging anorexics, restricting anorexics are more agreeable and conscientious, less impulsive, lower in sensation seeking, and higher in motor inhibition. Hence, providing evidence in support of the contention that ED psychopathology occurs at both ends of the slow–fast LH spectrum and demonstrating the taxonomic power of the LH framework.

It is also noted that DG has proposed an interesting and useful modification to the SCH formulation, namely, that the nature of female competition differs in the two variants of ED according to where they lie on the slow–fast spectrum continuum. Competition in the slow spectrum ED is through the display of signs of youth (an indicator of reproductive value), whereas the fast spectrum ED is through the display of signs of fertility. The original SCH formulation (Abed, 1998) suggested that all ED stems from competition for the display of signs of youth. DG's modification gives SCH greater explanatory power and is a welcome improvement of the hypothesis.

It is clear that the LH analysis of EDs can help illuminate some of the underlying differences between the different variants of these disorders and hence

provide an improved and more scientific taxonomy of these disorders. Nevertheless, it is clear that the LH framework must also rely on other explanatory frameworks to explain the existence of these disorders in the first place (in this case, sexual selection) as well as to explain their most prominent features (their female preponderance, their particular geographical distribution, their recent emergence, etc.). Finally, the question of mismatch is worth a mention. Mismatch is a potentially major cause of psychopathology as well as medical disorders (Brüne, 2008; Stearns et al., 2010).

Although anorexia is said to have existed prior to modern times, it was first described in the late 19th century and has increased in frequency over the 20th century when it came to prominence in Western societies (Gordon, 1990; Russell, 2000). The other variants of ED (primarily BN) appear to be novel disorders that are specific to Western or Westernized societies (Russell, 2000). Whether EDs have arisen de novo in relatively recent times in Western societies or showed a sharp increase in prevalence in such societies, the issue of mismatch becomes relevant as an explanatory model. The mismatch arises from the interaction of the evolved female sexual strategies for mate value maximization suited for the small-scale social environment of the environment of evolutionary adaptiveness, with the modern urbanized Western societies where large numbers of autonomous females of reproductive age live in close proximity to each other and are exposed to ubiquitous media images of high-quality female competitors. These and other factors are hypothesized to lead to a level of intensity of female intrasexual competition not encountered in the environment of evolutionary adaptiveness. Both the main evolutionary formulations for ED discussed earlier assume a degree of mismatch to explain the recent rise of these disorders, but in the case of SCH this assumption is explicit and discussed in some detail (Abed, 1998).

Although, DG does not deal with the question of mismatch within the LHT framework, it may be possible to accommodate it within the fourth proposed pathway where traits produce the vulnerability to develop a disorder (alongside other causes of vulnerability). However, the primary vulnerability to ED is likely to have arisen, through sexual selection thus explaining the lopsided sex ratio and not directly related to LHT. Nevertheless, LHT does provide an explanation for the vulnerability to developing the variants of eating disorders through the slow–fast spectrum distinction.

Schizophrenia

DG has classified schizotypal disorder and schizotypal traits (particularly the positive schizotypal traits) as a type of fast LH spectrum disorder. The basis for doing so is the observation that individuals with schizotypy and the relatives of schizophrenic patients possess verbal and artistic creativity suggesting that these traits have been sexually selected for high investment in mating as opposed to parental effort. Hence, according to this model, schizotypal traits can be beneficial and lead to reproductive success through investment in mating effort, but their harmful version can lead to schizophrenia. The clinical syndrome of schizophrenia, which is maladaptive, is caused, according to the target article, by mutational load and developmental factors (nutritional factors and exposure to pathogens).

However, social factors known to cause a significant increased risk of schizophrenia are not considered by the author. Specifically, recent epidemiological findings of schizophrenia show significantly increased risk in first- and second-generation migrants (Bourque, van der Ven, & Mella, 2011; Cantor-Graae & Selten, 2005). However, no increased risk is observed when migration entails a move from social exclusion to social inclusion (van Os, 2012). There is also increased risk associated with urbanization (Krabbendam & van Os, 2005), which shows a dose–effect relationship to the risk of developing the disorder.

In addition, there is increased risk to ethnic minorities associated with living in neighborhoods or localities where there is low density of same-group ethnic population (Das-Munshi et al., 2012). This risk is increased further in "visible minorities," for example, through salient racial differences. Furthermore, the prognosis of schizophrenia appears to show a gradient whereby the more traditional a society the better the prognosis despite the lack of services and modern interventions in those societies (Jablensky et al., 1992; Sartorius et al., 1986; World Health Organization, 1973). Added to these findings, the lack of documented cases of schizophrenia in the anthropological literature in pristine hunter–gatherer populations (E. F. Torrey, personal communication, 2010) or, in fact, in any literature prior to the 18th century (Evans, McGrath, & Milns, 2003; Torrey, 1980, 1987) points toward the probability that schizophrenia has arisen as a result of a mismatch between the design of the social brain and the novel modern social environment.

We hypothesized, therefore, that the pathogenic environmental elements relate to the modern social structures where large numbers of strangers and nonkin (outgroup individuals) live in permanent close proximity to each other (Abed & Abbas, 2011, 2014). We proposed that the nature of the pathogenic environmental stressor is either a paucity of kin or ingroup members or a high density of outgroup members in the individual's social environment during critical stages of development, or some combination of both. The hypothesized nature of the vulnerability we proposed, is either

an oversensitivity to the existence of strangers (outgroup individuals) in the social environment or an impairment in the individual's ability to recategorize or redesignate outgroup individuals as ingroup members, or some combination of both. However, the relationship between schizotypy and the putative oversensitivity to outgroup individuals is yet to be explored and understood.

Given that the maladaptive nature of schizophrenia is not in dispute, the outgroup intolerance hypothesis may be compatible with the LHT framework and may fit within the fourth pathway of the framework where the schizotypal traits (and/or other traits yet to be identified) confer vulnerability to developing schizophrenia in particular types of social environments. However, the full LH analysis of this putative vulnerability must await further corroborative evidence and a better understanding of its precise nature.

Conclusion

This is a well-researched and well-argued proposal that I find very little to disagree with. The model is so persuasive that I wondered at times how no one had not thought of the slow–fast spectrum disorder distinction before. However, it is not a stand-alone framework (nor is it meant to be), and for it to work well, it requires to take into account layers of explanatory systems both at the ultimate and proximate levels. My remarks regarding the probable importance of mismatch to eating disorders and to schizophrenia do not detract from the importance or the value of this framework. It only underscores the need to take into account not only proximate causation of the various types of psychopathologies discussed (which DG fully acknowledges) but also other layers of ultimate causation.

It is likely, in my view, that DG's LH framework will prove to be a landmark publication and a significant milestone in the development of evolutionary psychiatry and one that may lead to greater acceptability of the application of Darwinian principles to psychiatry and psychopathology by mainstream psychiatrists. The approach demonstrates the value and power of evolutionary explanations in providing insights into the nature of mental disorder not attainable through the traditional mechanistic, proximate approaches alone. The framework meets the challenges of being theoretically sound (taking account of ultimate as well as the proximate causation), being empirically based and having the potential for practical real-life taxonomic utility. It represents, in my view, a clear advance in evolutionary psychiatric thinking.

Note

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Psychological Inquiry, 25: 307–310, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.910487

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Psychopathology in Life History Perspective

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The fundamental goal of all living things, evolutionary theory teaches us, is the dispersion of genes in future generations. As a result, many if not most features of living organisms, including their behavior, have been shaped by natural selection to promote, directly or indirectly, reproductive fitness. Some four decades ago now, renowned evolutionary biologist Theodosius Dobzhansky (1973) observed that "nothing in biology makes sense except in the light of evolution" (p. 125). If we substitute "the life sciences" for "biology" and acknowledge that psychology and human development are life—not just social—sciences, then it becomes clear that an evolutionary perspective has much to offer these fields, including the study of psychopathology. Yet the fact remains that with the exception of a few specialized areas of inquiry—like the social-psychological study of mating—the behavioral and social sciences have been slow to embrace an evolutionary perspective.

Whereas students of psychology and human development, including those concerned with seriously disturbed functioning, are devoted to illuminating the *how* of human functioning, inquiring in ever more sophisticated ways into genetic, epigenetic, endocrinological, and neurological processes, rarely does such work consider the ultimate, evolutionary question of *why*. Thus, the study of mechanism reigns supreme in the psychological sciences, but the issue of function, especially ultimate function—in terms of fitness benefits (and costs)—is all too rarely considered.

Almost a quarter of a century ago now this realization dawned on me after reading Draper and Harpending's (1982) evolutionary reinterpretation of the effects of father absence on human development. This intriguing work introduced me to the concept of reproductive strategies and thus life-history theory, the central organizing framework that Del Giudice (this issue) employs to consider the nature and function of diverse psychopathologies. Indeed, it led me to embrace the fast–slow strategy distinction around which Del Giudice (this issue) frames his analysis of psychopathology when it came to thinking about my principle field of study—socialization and how parents, families, and early experiences in life shape human development (Belsky, 2000, 2007, 2012; Belsky, Steinberg, & Draper, 1991). Especially important was that it enabled me to see past traditional mental health or disease models, which regard some ways of functioning as "good" or "optimal" or "healthy" and others as not and thus appreciate that nature does not share the same values as we highly educated Westerners—and many others for that matter. Instead, it cares first and foremost about, as already noted, the dispersion of genes in future generations; and so, even if what we, as civilized individuals who regard caring, cooperation, and intimacy highly, to name just three manifestations of supposedly "optimal" functioning, place a premium on such ways of behaving, they are not *inherently* better than other ways of functioning. And that is because over the course of evolutionary history, behaving in contrasting ways has aided and abetted reproductive fitness, directly or indirectly; in consequence, genes contributing to such diverse ways of functioning have been selected and remain with us today.

The implication of this view, of course, is that much of what we in the psychological sciences regard as "healthy" or "optimal" may not always be. After all, there are clear opportunity costs associated, for example, with delayed discounting of the future. After all, one could die before securing advantages associated with delaying gratification, or the benefits to be claimed could expire or be claimed by someone else. Consider in this regard the value we place on "saving for the future," something often regarded as a veritable moral value and thus reflection of "character." Yet in a highly inflationary economy, saving is a fool's errand; and the wise thing to do financially is not simply to spend what one has—before the purchasing power of funds deteriorate—but to even take on debt, given that the value of the money repaid at some future date will be substantially less than of the money borrowed.

A similar analysis applies to ways of functioning that we typically regard as anything but optimal. All too often defenses involving negative emotions—fear, anxiety, shame—are conceptualized in disease terms (Nesse & Jackson, 2005), as Del Giudice (this issue) reminds us. Sure they may be unpleasant, but that is not to say that they are fundamentally dysfunctional. The fact that they are not always perfectly calibrated to what is being defended against simply reflects the fact that natural selection does not produce perfection, just phenotypes that are more functional than many alternatives. Indeed, they do not even have to pay off all the time and can even misfire much of the time—Nesse's (2005) smoke-alarm

principle that Del Giudice (this issue) also highlights. What nature seems to wisely appreciate with respect to such defences is that it is better to err on the side of caution than to be caught off guard and unprotected. Indeed, what all too often goes unappreciated in analyses of psychological disturbances is that certain ways of functioning can fail to pay off much, even most of the time, but may nevertheless have been selected because when they did, they do so in spades—and the possibility remains that this is still the case. As Del Giudice (this issue) reminds us, schizophrenia certainly has costs, but these may represent the trade-off involved in the pursuit of a hyper creative approach to life. For some, even most individuals, this psychopathology is principally costly, but for others it yields disproportionate benefits, or at least once did.

The ultimate point to be made is that there is no single way to get the ultimate job of life done. For some individuals or in some circumstances, one approach to life may pay off reproductively—or at least did so in the past and so its genetic and evolutionary legacies remain with us today—whereas in other cases different ways of being better serve that goal (or once did). What this implies most fundamentally is that scholars of human functioning should be asking not only *how* behavioral or psychological phenomena—including psychopathological ones—operate, but *why* they exist in the first place; in what way might they have enhanced reproductive fitness? In other words, why were they selected?

Questions like these, experience teaches me, lead many to embrace, no doubt unknowingly, the naturalistic fallacy, presuming, for example, that because humans may have been shaped by natural selection to engage in murderous violence or to become depressed—because in some manner, shape, or form these ways of functioning probabilistically furthered reproductive goals and interests—then there is nothing we can do about it, nor anything we should do about it. Nothing could be further from the truth. What evolutionary-minded scholars assume, in fact, is just the opposite: that an understanding of psychopathology enriched by evolutionary insight could, even should, better enable us to prevent and/or remediate such disturbance in the first place.

It is in this spirit that Del Giuidice (this issue) has recast thinking about psychopathology, knowing him as I do. Like him, and as already noted, I have come to appreciate the integrative utility of thinking about psychology and human development in life-history terms, distinguishing faster versus slower rates of development (Belsky & Pluess, 2013; Belsky et al., 1991). What makes this framework so powerful is its capacity to tie together so many facts and ideas. Many besides me have called attention to the intellectual silos we inhabit. Some, for example, study the stress response system; others focus on conduct disorder; still others investigate brain mechanisms related to executive functioning. All appreciate that at some level we are dealing with a whole that is greater than the sum of its parts, even as we specialize in the investigation of one or a few parts. But what is often lacking is a means of putting Humpty-Dumpty together. That is exactly what life history theory offers, as Del Giudice (this issue) so clearly appreciates. As he succinctly states, "Life history strategies are best thought of as functionally complex phenotypes, resulting from the integration of a suite of morphological, physiological, and behavioral traits" (p. 263).

It is this very feature of life history thinking that originally fascinated me—and still does. What became clear to me years ago is that life history theory offers a means to integrate phenomena central to socialization research (e.g., parenting, attachment security, cooperation, antisocial behavior) with the study of somatic development—in the form of pubertal timing. Not only did the resulting insight yield what I regarded as an "uncanny prediction"—that rearing experience would regulate somatic development in the form of pubertal timing—but it provided the basis for an evolutionary theory of socialization (Belsky et al., 1991), one that stimulated much research, with much of it supporting the core prediction that, at least in the case of female individuals, less supportive rearing environments would promote earlier sexual maturation (for review, see Belsky, 2012; Ellis, 2004). Such a result was anticipated because under conditions that posed threats to individuals, including to their safety and thus longevity, natural selection should, theoretically, accelerate development to increase the likelihood of the individual reproducing before dying.

In the time since we endeavored to recast traditional socialization theory in evolutionary perspective, numerous efforts have been made to expand, extend, revise, and enhance our theoretical model (for review, see Belsky, 2007). Thus, "fellow evolutionary-developmental travelers" have highlighted the need to distinguish (a) paternal and maternal influence (Ellis & Garber, 2000), (b) environmental harshness and unpredictability (Ellis, Figueredo, Brumbach, & Schlomer, 2009), and (c) more and less consistent contextual cues (Frankenhuis & Panchanathan, 2011) while underscoring the importance of (d) extrinsic mortality and morbidity (Chisholm, 1993), as well as (e) future orientation (Chisholm, 1993); (f) the differential susceptibility of individuals to environmental influence (Belsky, 1997, 2000, 2005); (g) the differential development of boys and girls (James, Ellis, Schlomer, & Garber, 2012), perhaps especially in middle childhood (Del Giudice, 2009); and (h) the role of the stress-response system in the contextual

regulation of reproductive strategy (Del Giudice, Ellis, & Shirtcliff, 2011). Especially noteworthy is that very recent applications of life history theory have made it clear that what was originally conceptualized as a theory of socialization, interpersonal development, and reproductive strategy can develop into an evo-devo theory of reproduction, health, and longevity (Belsky, 2014). This claim is based on Del Giudice and associates' (Del Giudice et al., 2011; Ellis & Del Giudice, in press; Ellis, Del Giudice, & Shirtcliff, 2013) adaptive calibration model of the stress response system, which recasts thinking about allostasis and allostatic load in evolutionary perspective, along with Rickard, Frankenhuis, and Nettle's (2014) proposal that humans may not just have evolved to monitor the external environment in order to regulate development in a fitness-enhancing manner as Belsky et al. (1991) theorized, but internal bodily cues as well, including inflammation, oxidative stress, and telomere length.

The point to made, then, is that life history theory and the conceptual and organizing distinction between fast and slow life history strategies central to it has much to offer psychology and human development scholars studying diverse subject matters—and in ways that tie together what are often separate fields of inquiry. It is no wonder, at least to me, then, that Del Giudice (this issue) uses this theoretical framework to shed new light on psychopathology. Although there are no doubt many reasons to question some of his claims and interpretations, though he is clear in many places that what he offers are speculations rather than factual assertions, it would seem indisputable that he is offering something new and original. In light of very recent work suggesting that many diverse psychopathologies load on a single p factor, reflecting more versus less disturbed functioning (Caspi et al., 2014), there are certainly empirical grounds for questioning Del Giudice's (this issue) general hypothesis that many disturbances can be arrayed on a bipolar dimension anchored at one end by putatively fast-spectrum disorders and at the other by slow-spectrum ones. But note that central to the latter's but not the former's analysis are critical—and theoretically driven—distinctions between types of eating disorders (fast/dysregulated vs. slow/perfectionist, overcontrolling), types of depression (fast/mood+somatic symptoms vs. slow/depression alone), and types of obsessive-compulsive disorder (OCD; fast/autogenous OCD vs slow/reactive OCD, OCPD). One is forced to wonder, then, if distinct measurements of the disorder and disorder dimensions that Del Giudice interprets in fast versus slow life history terms (see the target article Figure 1) were subject to factor analysis, whether a singular p factor with high loadings on all disorders would emerge. By revealing a measurement challenge to p-factor enthusiasts—to make the distinctions that Del Giudice highlights—a means of formally testing competing frameworks for understanding similarities and differences across supposedly distinctive psychopathologies emerges.

Two points need to be made with regard to this claim, both of which Del Giudice (this issue) fully appreciates. First, most exploratory factor analyses are designed to eliminate bipolar factors, replacing them with (more?) easily interpretable unipolar ones (i.e., simple structure). This means that the way in which factor analyses are conducted will need to be seriously considered when it comes to testing competing theoretical and empirical frameworks. The second point is that Del Giudice's (this issue) bipolar model does not preclude a p-factor structure. And this is because if deleterious mutations and other insults compromise development in both fast and slow backgrounds, a general dimension of dysfunction would be expected to emerge, but be largely, even if not entirely independent, of the fast-slow psychopathology dimension he predicts should emerge. Indeed, as he notes, one might actually expect some modest and positive degree of association between a p-like factor and the fast polarity of an anticipated bipolar factor because environments conductive to fast life histories also increase the likelihood of the very developmental insults that should engender true dysfunction in the first place.

Del Giudice's (this issue) effort to reframe thinking about psychopathology in life history terms calls attention to many more questions and issues that merit empirical consideration. As a result, it is not just "old wine in a new bottle." Once again, then, it should be clear that an evolutionary analysis does not simply yield, as too many uninformed critics contend, only a series of "just-so" stories. In fact, what often goes unappreciated by those who seem more ideologically opposed to evolutionary analysis than anything else is that, as David Buss once pointed out to me, *all* hypotheses qualify as just-so stories! As an empiricist, as well as an insightful theorist, Del Giudice surely appreciates that some of his analysis is likely to prove more off than on the mark. This, of course, is the fundamental (risk-taking) nature of theoretical analysis. But it is also the stuff that fuels scientific excitement—the ongoing dance between theory and evidence. We should thus be thankful to Del Giudice (this issue) for composing such a fine piece of music.

Note

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Psychological Inquiry, 25: 311–321, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.914120

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Life History Theory as Organizing Principle of Psychiatric Disorders: Implications and Prospects Exemplified by Borderline Personality Disorder

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Introduction

Life history theory (LHT) concerns an organism's differential allocation of resources to physical growth and reproduction. Less technically expressed, there is a trade-off between an organisms capacity to invest energy in somatic growth, as opposed to investment of energy in reproductive activity, resulting in life history (LH) strategies shaped by natural selection. Accordingly, growth rate, age and body size at sexual maturation, number and size of offspring, mortality rate, length of lifespan and so on, are biological traits modeled by environmental contingencies (Stearns, 1992).

The concept of LHT was originally applied to differences between species, referred to as "r/K selection" (MacArthur & Wilson, 1967). That is, "r" (for growth rate) selected species tend to grow small bodies, mature and reproduce early (and often only once in their lifetimes), have large numbers of offspring, provide little care for offspring, and have relatively short lifespans (among vertebrates, most fishes follow this LH pattern; among mammals, r-selection is typical for small rodents). In contrast, K-selected species (K stands for capacity) grow larger bodies, reproduce multiple times over an extended period, have smaller numbers of offspring (litters), invest substantially in offspring survival, and have longer individual lifespans compared to r-selected species (Stearns, 1977). This LH pattern is typical for large mammals such as whales, elephants, and primates (including humans).

Aside from species-typical patterns of reproduction along the r-K continuum, there is abundant evidence that within-species differences exist regarding LH strategies (Stearns, 1992). Put another way, ecological (environmental) conditions (interacting with genetic factors) determine whether an individual adopts a faster or slower LH strategy, whereby the relevant environmental factors include current and future availability of resources, as indicated by observable cues or predicted based on experience acquired in early developmental stages (Ellis, Figueredo, Brumbach, & Schlomer, 2009). Critical aspects involved in decisions over faster or slower LH strategies concern the timing of biological maturation, current versus future reproduction, quality versus quantity of offspring, and quality versus quantity of parental care in offspring and mating (Belsky, 2012; Del Giudice, this issue; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011).

A wealth of research has shown that the principles of LHT apply to humans in the same way as in any other organism (e.g., Ellis et al., 2011). It is necessary to point out, however, that terms such as "strategy" or "decision making" by no means imply conscious reflection. Although the speed of biological maturation, onset of sexual activity, and intensity of care for offspring is regulated by sex hormones, the stress response system, and neuropeptides (Bribescas, Ellison, & Gray, 2012; Del Giudice, Ellis, & Shirtcliff, 2011; Feldman, Gordon, & Zagoory-Sharon, 2011), Del Giudice points out that, in addition, LH strategies have profound impact on psychological traits such as cooperation, reciprocity, risk taking, interpersonal aggression, and pair-bonding, as well as executive functioning, inhibitory control, and personality traits (Del Giudice, this issue).

In fact, abundant evidence suggests that individual differences in early environmental conditions coin individual LH strategies in quite predictable ways (Belsky, Steinberg, & Draper, 1991; Hochberg & Belsky, 2013). Central to this is the observation that the quality of parenting profoundly influences the way children develop "inner working models" (IWM; Bowlby, 1969), which in turn serve as a guideline for predicting future resource availability. In other wording, children who grow up in a stable, emotionally safe, and predictable familial environment learn to see the world as a "safe haven" in which stable relationships with trustworthy others (family, peers, partners) indicate the availability of resources in the future. Accordingly, from the perspective of attachment theory, securely attached individuals tend to pursue slower LH strategies, that is, they tend to mature later, delay reproduction; are generally risk averse; and form stable, long-term intimate relationships with partners. Such individuals are also cooperative, are empathetic, display low levels of interpersonal aggression, and have good inhibitory control over impulses. In terms of personality traits, they score high on Conscientiousness and Agreeableness. In contrast, children who are exposed to environmental cues such as harsh

parenting, violence, or other sources of danger are more likely to develop an IWM suggesting that future resource availability is unpredictable, thereby shifting LH strategies toward faster development, including earlier biological maturation, sexual activity, and earlier reproduction (Belsky et al., 1991; Ellis, 2004; Ellis et al., 2011). A faster LH strategy is associated with insecure attachment patterns, increased delay discounting (Chisholm, Quinlivan, Petersen, & Coall, 2005), impulsivity, larger numbers of sexual partners, lack of reciprocality, reduced inhibitory control, and increased risk-taking behavior. Moreover, such behavior is associated with higher scores on Extraversion, Openness to Experience, and Neuroticism (summarized in Del Giudice, this issue).

Del Giudice (this issue) now expands this view to suggest that LHT could serve as an organizing principle for psychiatric conditions. In essence, he proposes that psychiatric disorders can be categorized along the fast or the slow end of LH strategies. In support of this novel approach, Del Giudice (this issue) rightly points out that (evolutionary) psychopathology lacks an organizing principle that is capable of explaining overlapping symptomatology of different disorders, comorbidity, and the relationship of "disorder" with "normal" psychological functioning. If LHT could fill this conceptual chasm, it would be of invaluable great heuristic value for the understanding of human cognition, emotions, and behavior in terms of both normal functioning and disorder.

To succeed as a metatheoretical framework for psychiatric conditions in the long run, however, LHT needs to demonstrate its compatibility with behavioral, neuropsychological, and biological findings (including genetics, neuroimaging) pertaining to a disorder, syndrome, or "endophenotype." Another, even more difficult problem that the LHT approach would need to overcome relates to the standard medical model of psychiatric conditions, which suggests that a disorder represents a deficit or deviation from a statistical norm. In other words, clinicians won't easily buy the idea that psychiatric conditions can be seen as "strategies" (Mealey, 1995; Troisi, 2005) emerging from complex gene–environment interactions, which, from the viewpoint of subjective well-being, appear dysfunctional or "abnormal" yet from a biological perspective characterize a way of maximizing reproductive success.

In this commentary, I argue that both of the aforementioned concerns can successfully be addressed, even though most psychiatric conditions seem to present a mixture of fast and slow LH strategies, rather than being unidirectionally fast or slow. For the sake of clarity and conciseness, I choose borderline personality disorder (BPD) as an example of a largely fast LH strategy.

BPD Seen Through the Lens of LHT

In brief, BPD is characterized by unstable interpersonal relationships, frantic fear of abandonment, and difficulties in emotion regulation, as well as impulsivity and risk-taking behaviors (American Psychiatric Association, 2013; Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). Approximately 1% to 6% of the general population fulfils the diagnostic criteria of BPD, with significant variation between populations (Sansone & Sansone, 2011). Bio-psycho-social models of BPD suggest that patients frequently develop mistrustful inner working models that lead them to predict and experience others as untrustworthy and rejecting (Fonagy, Target, & Gergely, 2000), based on adverse experiences with primary caregivers leading to insecure attachment (Agrawal, Gunderson, Holmes, & Lyons-Ruth, 2004). A substantial number of patients with BPD have suffered childhood trauma, including physical, emotional, or sexual abuse (Bierer et al., 2003; Zweig-Frank & Paris, 1991). In line with LHT models of socialization (Belsky et al., 1991; Chisholm, 1999), and consistent with Del Giudice et al.'s (2011) adaptive calibration model (ACM), the experience of early adversity coins an individual's expectations with regard to future resource availability in terms of interpersonal relationships (i.e., trustworthiness, reciprocity and empathetic concern). This suggests that individuals exposed to trauma, neglect or abuse tend to maximize short-term benefits from interpersonal relationships, that is, pursue a fast LH strategy (Brüne, Ghiassi & Ribbert, 2010). Accordingly, LHT predicts that the following features, indicative of a fast LH strategy, would be associated with BPD: high stress-responsivity, emotional dysregulation, lack of concern for others' mentalities, impulsivity (poor impulse control), increased risk-taking behavior (including sexual risks), lack of trusting relationships, instability of romantic relationships, and high number of short-term sexual relationships, as well as increased vigilance towards partners' faithfulness, early biological maturation, and poor investment in own offspring. Beyond behavioral and psychological traits associated with BPD, LHT must also be compatible with neurobiological findings from neurophysiology (e.g., responsivity to stress), neuroimaging, and genetics. Predictions from LHT should also be consistent with patterns of comorbidity among different psychiatric disorders, all the more, as diagnostic boundaries between conditions are fraught with anachronism and unreliability (Brüne, 2008). As for BPD, empirical evidence suggests that the condition frequently co-occurs with depression, posttraumatic stress disorder (PTSD), eating disorders, and attention deficit/ hyperactivity disorder (ADHD). Although ADHD can arguable be conceptualized as a fast LH strategy, the situation is

more complex for depression, PTSD, and eating disorders, which seem to be consistent with both slow and fast LH strategies. These issues are dealt with in more detail in the following sections.

Traits Associated with BPD Following a Fast LH Strategy

Neuropsychology and Personality

One key feature of BPD concerns patients' difficulties in regulating their emotions in appropriate ways, which may account for several symptoms including idealization and derogation of others, impulsivity, and risk-taking behavior. These signs and symptoms can be conceptualized as behavioral expression of high stress responsivity. According to the ACM (Del Giudice et al., 2011), high stress responsivity promotes a fast LH strategy in dangerous and unpredictable contexts, whereby it increases vigilance to threat and down-regulates one's sensitivity to social feedback (Boyce & Ellis, 2005). Consistent with this interpretation, several studies have shown alterations of the hypothalamic-pituitary-adrenal stress axis in BPD, which correlate with symptom severity and a history of childhood trauma (Carvalho Fernando et al., 2012). In fact, early adversity in general has been found to be associated with persistent changes of stress responsivity, possibly via epigenetic mechanisms (Murgatroyd et al., 2009). In line with these findings, research into emotion perception suggests that patients with BPD display heightened vigilance or avoidance reactions to negative emotions such as fear and anger (Brüne et al., 2013; Jovev et al., 2012). At the same time, patients with BPD are often strikingly "alexithymic," that is, they have difficulties in reflecting upon own and others' emotions, whereby alexithymia in BPD has been found to be related to stress intolerance and impulsivity (Gaher, Hofman, Simons, & Hunsaker, 2013). This apparent "empathy paradox" (Dinsdale & Crespi, 2013), however, is plausible considering LH strategies emerging from early adversity. For example, psychoanalytic accounts of superior emotion perception abilities in BPD suggest that inconsistent or neglectful parenting may trigger an enhanced sensitivity to subtle emotional cues of parents, such that the child can maintain a coherent mental image of its caregiver (Krohn, 1974). Along similar lines, Linehan (1993) argued that patients with BPD may be hypersensitive to emotional cues that potentially signal rejection or abandonment. Such biased emotion perception impacts on social interaction, if it interacts with difficulties in emotion regulation arising from overactivation of the attachment system (Fonagy et al., 2000). Overactivation of the attachment system leads to a functional down-regulation of mentalizing abilities, partly, as a means of self-protection against continuing traumatization by an abusive caregiver (Fonagy et al., 2000). Accordingly, hypersensitivity to negative emotions may further contribute to distorted views of others (Gunderson & Lyons-Ruth, 2008), such that others are generally perceived as untrustworthy (Nicol, Pope, Sprengelmeyer, Young, & Hall, 2013). In turn, seeing others as untrustworthy and uncooperative may enhance one's own (unconscious) opportunistic attitude toward short-term exploitation of resources (Ebert et al., 2013).

This view is also compatible with research showing enhanced impulsivity and delay discounting in patients with BPD. In fact, if one's IWM suggests poor resource availability in the future (compatible with a fast LH strategy), immediate resource acquisition is a logic consequence. As Del Giudice (this issue) argues, "down-regulation of defenses is most likely in the context of fast life history strategies," requiring "outright *insensitivity* to threats, dangers, social feedback, and so forth" (p. 270). In line with Del Giudice's predictions, empirical evidence suggests that patients with BPD are poor in impulse control and in tolerating delay of gratification, that is, they prefer immediate (lower) gains over (higher) future monetary gratification (Völker et al., 2009).

In relation to this, research involving theories of personality development suggests that fast LH strategies would be associated with high scores on Extraversion, Openness to Experience, and Neuroticism (Del Giudice, 2012), as well as with low scores on Agreeableness and Conscientiousness, whereby high scores on the latter two dimensions were more characteristic of slow LH strategies (Del Giudice, this issue). Indeed, Extraversion (boldness), Openness to Experience, and Neuroticism (i.e., emotional instability) have been found to correlate with unrestricted sexuality, short-term mating, relationship instability, risk taking, and disruptive behavior not only in humans (Del Giudice, 2012) but also in animals in comparable ways (M. Wolf, van Doorn, Leimar, & Weissing, 2007).

With regard to BPD, patients have been found to score higher on novelty seeking and lower on cooperativeness compared to nonclinical and clinical controls (Fossati et al., 2001) in a study using the Temperament Character Inventory (Cloninger, Svrakic, & Przybeck, 1994). Specifically, both novelty seeking and cooperativeness in BPD were largely independent of attachment style and quality of parental care. These findings are consistent with the hypothesis of a fast LH strategy in BPD. Our own research has utilized neuroeconomic games and responsivity of patients to the intranasal administration of a single dose of oxytocin to study LH-relevant

behavior in BPD. For example, in a study using a Dictator Game version, in which participants had the option to punish observed unfairness occurring during an interaction of two characters, we found differences in personality traits between BPD patients and controls, which had diametrically opposite impact on participant's motivation to engage in third-party punishment. In line with Del Giudice's (this issue) predictions regarding the association of personality traits with a fast LH strategy, patients with BPD scored higher than controls on Neuroticism (with no difference in Openness to Experience), higher on Machiavellianism, and lower on Agreeableness and Conscientiousness. Contrary to predictions, however, patients scored lower on Extraversion than controls. Most interestingly, in BPD third-party punishment correlated with Neuroticism and Machiavellianism, and inversely with Agreeableness (as a measure of empathetic concern for others), which was the reverse in nonclinical controls. This finding is consistent with the interpretation that patients with BPD seemed to pathologically identify with the disadvantaged person in the Dictator Game, whereby antisocial traits motivated patients to punish unfair behavior, rather than empathic concern for others (Wischniewski & Brüne, 2012).

Along similar lines, research into interpersonal trust and cooperation has revealed that individuals with BPD have difficulties in maintaining and reestablishing reciprocal trusting relationships. For example, King-Casas et al. (2008) used a so-called trust game (TG), where one player (the investor) is endowed with a sum of money units (MU), of which he or she can "invest" a proportion of his choice in another player (the trustee). The trustee then decides how much he or she is willing to return to the investor (as a measure of reciprocality and cooperation). Mistrustful investors are less likely to spend a substantial share, because they would expect an insignificant return by the trustee. Conversely, mistrustful trustees unlikely reciprocate, if the TG is played iteratively with the same investor, because they probably expect the investor to defect over time. BPD patients, as trustees, initially returned as many MUs as controls. However, contrary to controls, patients' willingness to reciprocate diminished over successive rounds. Moreover, when the investor's behavior was experimentally manipulated such that the trustee was frustrated by the lack of the other players' cooperation, psychologically healthy subjects could be coaxed back into cooperation by overly generous investments, whereas BPD patients did not respond to cajoling (King-Casas et al. 2008). In further support of a fast LH strategy in BPD, Unoka, Seres, Aspán, Bódi, and Kéri (2009) found that BPD subjects, in the role of an investor in a TG, transferred fewer MUs than patients with depression and healthy controls (Unoka et al., 2009), depending on symptom severity such as stress-related paranoia and difficulties in interpersonal relations, as well as with a lack of confidence in the trustee (i.e., reduced trust). Likewise, another study reported that patients with BPD, as investors, adjusted their investment in that they transferred fewer MU to unfair trustees while ignoring—unlike nonclinical controls—the trustee's neutral or negative facial expression (Franzen et al., 2011). All this is compatible with the view that BPD patients act in quite opportunistic ways and disregard emotional signals of others that might guide one's decision of whether to cooperate with others.

Another area of research that indirectly supports the hypothesis of a fast LH strategy in BPD concerns the experimental administration of oxytocin (OT). Rather than being a "prosocial" hormone in general, it has turned out that OT seems to enhance an individual's prepotent interpersonal behaviors "for better or worse," depending on one's prediction concerning another's trustworthiness and cooperation, which in turn are closely linked with attachment and the presence of absence of childhood adversity (Ebert et al., 2013). Consistent with this view, BPD patients showed a reduction in cooperation and trust in an economic game (called the "Assurance Game," which is similar in design to a classic Prisoner's dilemma) after the intranasal administration of a supraphysiological dose of OT (Bartz et al., 2011). In this study, the lack of trust was influenced by the participants' attachment styles, in that individuals with highly anxious-avoidant attachment showed the largest decrease in trust upon administration of OT, relative to the placebo group, whereas trust increased in those with high anxiety and low avoidance scores under OT.

In our own study, we adapted a classic TG by introducing faces of attractive and less attractive counterparts (as trustees), based on the hypothesis that individuals who favor short-term over long-term gains (i.e., pursue a fast LH strategy) would be more likely to reciprocate an offer made by an attractive investor than subjects who invest more in long-term relationships. With regard to BPD, we predicted that in the placebo condition, patients with BPD would generally invest more in attractive trustees but less in unattractive counterparts compared to controls, irrespective of their mistrustful inner working model or experience of childhood trauma. If, however, OT enhanced social salience, the experience of childhood trauma could lead to increasing distrust, thereby overriding one's interpersonal opportunism. In line with these predictions, we found that both BPD subjects and healthy controls tended to invest more money in attractive than in unattractive trustees. Notably, BPD patients transferred fewer MUs as investors in the OT condition as compared to placebo, whereby emotional neglect significantly predicted patients' investment in the OT condition (Ebert et al., 2013). To conclude,

neuroeconomic studies corroborate the assumption that patients with BPD have acquired an opportunistic interpersonal orientation in terms of resource acquisition, that is, pursue a fast LH strategy. In addition, the finding that BPD patients invest their own resources differentially depending on others' physical attractiveness is compatible with their actual sexual behavior and how intimate relationships are built.

Sexuality, Mating, and Parenting

According to Del Giudice et al.'s ACM, a fast LH strategy would predictably be associated with increased risk taking, earlier intercourse, and larger numbers of sexual partners. In addition, biological maturation is expected to be accelerated (Belsky et al., 1991; Ellis et al., 2011). Indeed, a large population-based study revealed that early age at first sexual intercourse predicted lifetime number of sexual partners and future risk-taking behavior in general (Olesen et al., 2012). In our own (small-scale) explorative study, we found that patients with BPD, compared to controls, had experienced more childhood adversity and more short-term than long-term romantic relationships but did not differ with regard to age at menarche (Brüne et al., 2010). Recently, our group has collected data from a larger sample of BPD patients and psychologically healthy controls. Notably, individuals with BPD had, on average, significantly more sexual partners in the past 12 months than healthy controls. They also expected to have more sexual partners in the near future than controls. Moreover, BPD patients reported a greater willingness to engage in risky endeavors, specifically risks affecting health and social behavior (as opposed to financial risks). Again, there was no difference in biological maturation between the groups. These findings, as well as research into mating behavior of patients with BPD, are largely consistent with a fast LH strategy. A number of studies have shown that individuals with BPD are more likely to experience breakups of relationships (Labonte & Paris, 1993) and that woman diagnosed with BPD report lower levels of marital satisfaction and greater likelihood of experiencing partner violence (Bouchard, Sabourin, Lussier, & Villeneuve, 2009). Consistent with this pattern, Tragesser and Benfield (2012) showed that individuals with borderline features engage more in costly mate retention tactics, whereby monopolization of time, emotional manipulation, commitment manipulation, violence against rivals, submission and debasement, and verbal possession signals were more frequently observed in men, and jealousy induction, derogation of competitors, and derogation of the mate was more prevalent in women with BPD features. Although partly defensive in nature, and therefore perhaps intuitively more characteristic of slow LH strategies, these mate retention tactics are more likely to work effectively in the short term, but less so in the long run, because they are costly to the pursuer and aversive to one's mate, which may, in fact, increase the likelihood of a breakup (Shackelford, Goetz, Buss, Euler, & Hoier, 2005).

With regard to parenting practices among patients with BPD, there is a surprising paucity of data. Invalidating parenting may be one mechanism involved in the transgenerational transmission of BPD personality traits (Linehan, 1993). In line with the hypothesis of a fast LH strategy in BPD, one would indeed predict relatively little parental investment in offspring in terms of emotional availability and nurturing. Indeed, mothers with BPD seem to display critical and intrusive behaviors as well as role confusion (i.e., fear of being abandoned by own offspring) and frightened or frightening behaviors. Specifically, the oscillation between overinvolvement and withdrawal as well as between hostility and coldness seems to be characteristic of mothers with BPD (Stepp, Whalen, Pilkonis, Hipwell, & Levine, 2012). Accordingly, there is at least partial support of poor parenting of individuals with BPD. Our own observation in in-patients with BPD seems to corroborate this conclusion. We observed that a relatively large number of patients with BPD come from a family background in which the biological father is absent, or multiple consecutive stepfathers have been present during childhood and adolescence of the affected individual. Moreover, several patients have half-siblings from relationships of their mothers with multiple partners. Likewise, we observed among in-patients with BPD that a substantial number of women have been forced to give their children into foster care or under the auspices of youth welfare services, which, from an evolutionary perspective makes sense in light of the assumption of a fast LH strategy.

Neuroimaging Data and Genetics of BPD

One of the most critical potential objections against LHT applied to psychopathology refers to research into neuroimaging and genetics suggesting structural or functional brain deficits and/or genetic vulnerability. Both aspects concern the classical medical model of psychiatric disorders, according to which differences in brain structure between affected and unaffected individuals and/or differences in genetic makeup reflect abnormalities from a statistical norm. In fact, there is abundant evidence showing that childhood maltreatment is associated with reductions in volume of limbic areas (e.g., Dannlowski et al., 2012; Teicher, Anderson, & Polcari, 2012) and the corpus callosum (Teicher et al. 2003), and that impulsivity in BPD is associated with alterations in blood flow in frontal cortical regions (R. C. Wolf

et al., 2012). This commentary cannot do justice to the wealth of neuroimaging findings in BPD. The relevant, often overlooked, issue here is that alterations in brain metabolism or even brain structure do not necessarily reflect defective functioning. Following Teicher et al. (2003), early environmental stress, for example, in the form of childhood neglect or abuse, is possibly not simply toxic to the brain, thus interfering with (normal) brain development. Instead, as Teicher et al. (2003) put it, "exposure to significant stressors during a sensitive developmental period causes the brain to develop along a stress-responsive pathway" (p. 39). That is, early adversity may elicit "a cascade of stress responses that organizes the brain to develop along a specific pathway selected to facilitate reproductive success and survival in a world of deprivation and strife" (p. 39) This fundamentally different view of structural and functional brain imaging findings is in full accordance with the view that early experiences not only shape the psychological development of IWM and how individuals adapt their LH strategy according to their predictions of future resource availability but also leave a mark on how the hardware (i.e., the brain) supports the operation of one's individual software (i.e., IWM). In the case of BPD, this suggests that changes in limbic structure may actually support a fast LH strategy.

Along similar lines, research into psychiatric genetics has largely focused on the diathesis-stress model (Monroe & Simons, 1991), according to which subjects are vulnerable to develop a disorder if carrying a genetic variant that meets some sort of adversity or negative life event. Conversely, some genetic variation may protect against the development of a disorder even in the presence of severe adversity (Polanczyk et al., 2009). The diathesis stress model can, however, not explain why so many "vulnerability genes" have undergone recent positive selection in human evolution. This is contradictory in itself, because it is implausible to assume that natural selection has favored allelic variants, which increase vulnerability to adversity. Instead, this strongly suggests that these genes exert hitherto undetected or overlooked beneficial effects with regard to reproductive fitness (which is not necessarily the same as "good for health"; Del Giudice, this issue; Ellis et al., 2011). Accordingly it has been argued that a particular genetic variation that predisposes to pathology if associated with early adversity can have beneficial effects when environmental contingencies are developmentally more supportive (Belsky, 2012; Boyce et al., 1995). This suggests that it is more accurate to speak of differential susceptibility or plasticity conferred by genetic variation—that is, responsivity to both positive and negative conditions—rather than focusing one-sidedly on vulnerability (Belsky et al., 2009). Consistent with this assertion, it has been demonstrated that the low-activity MAO-A variant, which has been found to be linked to antisocial behavior in individuals who experienced childhood adversity (Caspi et al., 2002) is associated with *lower* than average prevalence of antisocial personality when children grow up in supportive environments (Widom & Brzustowicz, 2006). Likewise, the s-allele of the 5-HTTLPR, which has been deemed to be a "risk allele" for depression, confers *lower* risk for depression under favorable environmental conditions (Taylor et al., 2006), and children carrying the 7-repeat variant of the DRD4 gene, which increases the risk for ADHD in adverse conditions, develop ADHD and externalizing problems *less than average* if their mothers are responsive to their children's emotional needs (Bakermans-Kranenburg & van Ijzendoorn, 2006). Moreover, adult carriers of the COMTval and DRD4 7-repeat alleles show the highest responsivity to their children's needs when stress levels are low, whereas their responsivity is lower than average when stress levels are high (van Ijzendoorn, Bakermans-Kranenburg, & Mesman, 2008). Taken together, these findings indicate that allelic variation involved in dopamine and serotonin turnover play an important role in differential susceptibility to environmental conditions, possibly mediated by one's responsivity to reward and punishment, as well as stress regulation (Bakermans-Kranenburg & van Ijzendoorn, 2007; Caspi, Hariri, Holmes, Uher, & Moffitt, 2010; Ellis et al., 2011). Overall, it seems that plasticity genes can have additive effects, that is, the susceptibility to the environment may increase with the number of plasticity alleles (Belsky & Beaver, 2011). Surprisingly, Del Giudice hardly discusses possible implications for LH strategies from the perspective of differential susceptibility. With regard to the psychobiological development of girls, however, research has shown that girls carrying the GG variant of the estrogen receptor-alpha (ESR1) gene who grow up with less sensitive mothers are younger at menarche than AA carriers, whereas GG carriers raised by sensitive mothers experience menarche later than carriers of the AA genotype (Manuck, Craig, Flory, Halder, & Ferrell, 2011), suggesting that the same genetic variants can promote a faster or slower LH strategy. More specifically to BPD, a recent review conceded that despite evidence for heritability of around 40% of BPD, the search for candidate genes involved in BPD has been disappointing, which could relate to the "tendency to look for genetic effects on disease rather than genetic effects on vulnerability to environmental causes of disease" (Amad, Ramoz, Jardri, & Gorwood, 2014). Future genetic research into BPD should therefore be more hypothesis driven, and LHT might be a good empirical framework for this kind of approach.

Traits Associated With BPD Putatively Following a Slow LH Strategy

Even though the overall pattern of behavior in BPD, as well as the underlying cognitive and emotional processes, implies a fast LH strategy, some traits associated with the syndrome are rather suggestive of a slow LH strategy. These could, in part, reflect compensatory mechanisms for behaviors at the fast end of the continuum. As Del Giudice (this issue) explains, although risky strategies may yield large gains in case of success, they also impose considerable costs in case of failure. Among such defensive strategies, the frantic avoidance of abandonment, even if only imagined, is one of the most relevant diagnostic features of BPD (American Psychiatric Association, 2013). In line with this assumption, BPD patients usually score higher on the dimension "harm avoidance" of the Temperament Character Inventory (Cloninger et al., 1994) compared to controls (Fossati et al., 2001). Of interest, Fossati et al. (2001) reported that differences in harm avoidance were mediated by attachment patterns and measures of parental care, suggesting that early experiences with primary caregivers impact on the degree of defensive behavior. Consistent with this finding, our own and other studies revealed that the administration of OT seemed to reduce interpersonal opportunism in BPD, whereby the trust-lowering effect of OT (i.e., more defensive action) was related with insecure attachment in one study (Bartz et al., 2011) and severity of childhood trauma in another (Ebert et al., 2013).

Another feature, typically found in individuals with BPD, is the tendency of patients to invalidate themselves, which may underlie feelings of emptiness and self-disgust. In fact, disgust seems to be a relevant factor impacting on patients' self-concepts, whereby the degree of disgust is often linked to the severity of traumatizing experiences (Rüsch et al., 2011). According to Del Giudice (this issue), high sensitivity to disgust interferes with a fast LH strategy, particularly in relation to sexual behavior. Conversely, insensitivity to disgust may bare the risk of contracting sexually transmitted diseases. Following this line of reasoning, the presence of disgust would be a clear indicator of a slow LH strategy. In support of this hypothesis, Benecke and Dammann (2004), analyzing patients' facial expressions, found that BPD patients displayed high amounts of anger, contempt and disgust during therapeutic interaction, whereby contempt and disgust particularly occurred patients were talking about themselves (Benecke & Dammann, 2004). Similar findings emerged in another study in BPD during an attachment-related task revealing traumatization by attachment figures (Buchheim, George, Liebl, Moser, & Benecke, 2007). These findings are compatible with our own research showing that patients with BPD rate themselves as significantly more unattractive compared to controls, in spite of their sexually promiscuous behavior.

The spectrum of comorbid disorders associated with BPD is also suggestive of a mixed, largely fast, LH strategy. Although ADHD is arguably quite typical for a fast LH strategy, the case for PTSD and depression is more complex. Studies suggest that comorbidity rates of these disorders with BPD are considerable (Luca, Luca, & Calandra, 2012; Pagura et al., 2010). PTSD, for instance, seems to feature the extremes of variation of defense mechanisms akin to arrested flight, submission, freezing, and dissociation (Cantor, 2009; Silove, 1998). This pattern of behavior therefore indicates a slow strategy, although it might be difficult, in general, to attribute defense mechanisms exclusively to a slow LH strategy (Del Giudice, this issue). However, as Del Giudice points out, depression can be situated at both ends of the fast–slow LH spectrum. As for the fast end, hyperactive stress regulating mechanisms can have adaptive properties in dangerous environments (i.e., promoting fast LH strategies), yet they may also bare the risk of dysfunction. Accordingly, depression could be a costly consequence of a failure of stress regulation. Consistent with this interpretation, depression is more likely to occur in fast maturers, somatic symptoms associated with depression are linked with early adversity, and depression in adolescence often co-occurs with externalizing behaviors, and generally with lower agreeableness, conscientiousness, and poor inhibitory control (summarized in Del Giudice, this issue). It is equally plausible, however, to assign depressed mood a role in slow LH strategies, because it may shield an individual from pursuing unattainable goals and help avoid risks (Gilbert, Gilbert, & Irons, 2004). With regard to BPD, either explanation may apply, that is, depression could be the cost for failure of a high-risk (fast) strategy, or a self-protective mechanism in the sense of down-regulating strategic action to cope with stress caused by a fast LH pattern.

Along similar lines, Del Giudice (this issue) argues that eating disorders may reside at both ends of the continuum of LH strategies, based on the relevance of sexual competition for mates. Accordingly, a slow LH strategy would promote women to desire a thinner body than what men perceive sexually most attractive, which in turn would increase the woman's value as a long-term mate. Consequently, slow LH strategies should be more characteristic of anorexia nervosa (AN) than bulimia nervosa (BN). Consistent with this hypothesis, Del Giudice suggests that BN is associated with earlier sexual maturation and activity; patients with BN also show more externalizing

behaviors than patients with AN. In accordance, BPD seems to be more often associated with BN than AN (Rosenvinge, Martinussen, & Ostensen, 2000). However, more evenly distributed comorbidity rates have been reported in other studies (e.g., Chen, Brown, Harned, & Linehan, 2009).

Concluding Comments

Del Giudice (this issue) has proposed LHT as a framework for nosological categorization of psychiatric disorders. LHT is indeed a highly valuable concept that Del Giudice convincingly applies to a broad range of psychopathological conditions.

Although such a conceptual article is necessarily constrained to painting a larger picture, it is nevertheless imperative to assess the compatibility of the framework with the available sources of insight, which include, at least, behavioral, neuropsychological, imaging and genetic research. This is crucial, if a metatheoretical concept such as LHT is to survive skepticism of clinicians against the utility and applicability of evolutionary concepts to psychiatry (Troisi, 2012). Moreover, it is necessary to demonstrate that such a new perspective can add to the understanding of the nature of psychiatric disorders, and help to advance treatment. Arguably, these tasks are easier to accomplish in syndromes with a well-defined phenotype. Conversely, it might be more difficult to reconcile predictions from LHT with the scientific evidence in very broadly defined phenotypes such as "schizophrenia."

In this commentary, I have tried to demonstrate, using BPD as an example, that LHT is a valuable concept to explain psychological and neurobiological traits, though the picture is complex, and individual differences prevalent even within a fairly precisely conceptualized phenotype.

Abundant evidence support the view that BPD reflects a fast LH strategy (Brüne et al., 2010), even though some features seem to be more typical for a slow LH strategy. The latter may, for example, occur secondarily as (dysfunctional) strategies to cope with distress caused by traits associated with the fast end of the continuum. For instance, I have argued elsewhere that patients with BPD may engage in self-injurious behaviors or temper tantrums, not only to reduce tension and distress but also to prevent imagined or real abandonment. In an evolutionary perspective, self-injury or tantrums can be seen as threats to parents' inclusive fitness, given the overall amount of investment that human parents have already provided in preceding years. Hence, self-imposed threat to the physical existence by offspring is perhaps the strongest signal on the side of the offspring to increase parental care and nurturance (Brüne, 2008).

Aside from behavioral traits, it is essential to reconsider findings from psychiatric neuroimaging and genetic studies. The traditional "medical" perspective suggests that deviations from a statistical norm represent "deficits" (i.e., brain damage or vulnerability genes). With respect to neuroimaging in BPD, however, many such "deficits" may, in fact, reflect complex adaptations to early adversity, which may even be functional in dangerous and unpredictable environments, but dysfunctional in safer environments (Teicher et al., 2013). Of note, studies have shown that anatomical "abnormalities" found in patients with a history of childhood adversity are reversible upon psychotherapy (Davidson & McEwen, 2012), suggesting that functional or structural brain variation is not necessarily impervious to modification.

Along similar lines, LHT suggests that the one-sided view on psychiatric genetics (vulnerability concept) should, in part, be replaced by one that views genetic variations as expression of plasticity "for better or worse," depending on the interaction of genes with the environment (Belsky & Beaver, 2011; Brüne et al., 2012). This view may have profound implications for the understanding of BPD, because BPD patients may actually be among the genetically most plastic individuals who, due to early adversity, have developed dysfunctional interpersonal strategies (Stanley & Siever, 2010). A more general extension to this assertion suggests that plasticity genes may, in fact, act at both ends of the LH spectrum. That is, the very same allelic variation can promote a slow or a fast LH strategy, depending on early environmental contingencies. Although central to the application of LHT to psychiatric conditions, Del Giudice has made surprisingly little use of this source of evidence in support of his model.

Seeing psychiatric disorders as dysfunctional outcomes of developmental plasticity has also eminent therapeutic implications. First, this view certainly influences (or ought to influence) therapists' attitude. Rather than perceiving an individual patient as a "victim" of his or her poor genetic endowment or his or her behavior as a consequence of brain damage, the LH perspective suggests that complex gene–environment interaction has produced a particular phenotype that is malleable to the better if adequately dealt with, that is, the provision of access to psychotherapy.

An example for this diametrically opposite view refers to the interpretation of King-Casas et al.'s (2008) trust game findings (see earlier). Kishida, King-Casas, and Montague (2010), commenting on the study, suggested that "borderline personality disorder confers a *diminished capacity* to represent expectations for social partners, and as a consequence individuals with BPD *cannot take corrective action* [emphasis added] (social control signal) that might

serve to reestablish cooperative interaction" (p. 553–554). An alternative interpretation that is much more in line with LHT would propose that, rather than reflecting a cognitive deficit, it is the motivational structure of patients with BPD that lead them not to take corrective action by reinstalling cooperation. That is, individuals whose IWM suggest that others are untrustworthy may not be *motivated* to respond to attempts to coax them back into cooperation (Ebert et al., 2013). Along similar lines, Ellis et al. (2011) warned us to conclude from research into developmental plasticity that it might be useful to bolster highly susceptible individuals against adversity. Instead, it is therapeutically much more useful to provide them with responsive social environments from early on (Ellis et al., 2011). Although this might turn out to be a mammoth task for health care providers, LHT eventually teaches us that prevention and early detection of psychological problems is the key for the improvement of psychological health. Del Giudice's work can take us a leap forward in this direction.

Note

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Psychological Inquiry, 25: 322–324, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.912560

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An Evolutionary Framework for Psychological Maladaptations

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Mental disorders represent abstract constructs, in contrast to physiologically based disorders, such as type 1 or type 2 diabetes, that can be unambiguously diagnosed. This distinction is commonly ignored, either through reification of psychopathological conditions or through neglect of what should be the main goal of psychiatry: transforming it into an applied discipline that fits the standard medical model of understanding diseases as dysfunctions of specific adaptive systems (Nesse & Stein, 2012). Although adaptive functions are much more complex for cognition and emotion than for metabolism of glucose, the core principle should be exactly the same: To understand and categorize disease-associated maladaptations, we must connect them with the adaptations that have become dysfunctional. But how?

Historically, two main routes have been followed to link normal adaptive functions with disease, for mental disorders: top-down and bottom up, in each case from psychiatry and psychology, and across a great divide to neurology, physiology, development, and genetics. In my view, these paths have led either into the conceptual wasteland of *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; American Psychiatric Association, 2013) or into a genomic wilderness with more and more phenotype variation "explained" statistically, but not functionally, by increasingly large sets of increasingly small genetic effects.

Enter evolutionary biology, and efforts to construct unified frameworks for psychiatry based on its privileged position as the only scientific discipline that focuses specifically on the causes and sources of adaptation. Phenotypes, from finch beaks to amygdala activation patterns, thus vary within and between species, and such variation is associated with both costs and benefits in contexts related to adaptive functioning, survival, and reproduction. One of the largest scale phenotypes subject to natural selection is life history, the schedules, durations, and patterning of events salient to survival and reproduction. As described by del Guidice, comparative analyses across sets of species, such as mammals, have identified a "fast–slow" continuum, with "faster" species characterized by a combination of traits including earlier maturity, lower survival rates, and higher fertility but less investment in each offspring (e.g., Promislow & Harvey, 1990). Directly comparable patterns have been described within and across human populations that differ in extrinsic mortality risk: Higher mortality, as from disease, famine, or low socioeconomic status, has been linked with "fast" traits such as early reproduction, short interbirth intervals, and lower investment in each child (e.g., Nettle, 2011b; Störmer & Lummaa, 2014). How, though, can such a continuum extend to mental adaptation, and psychiatry?

Life History of the Psyche

Del Guidice seeks to extend the "fast–slow" life history to the brain, to psychological adaptation, and to psychopathologies as arrayed along a fast–slow axis. His exposition centers on more or less adaptive bundles of social, sexual, and parenting behaviors spread across a vast, yet ultimately linear, "psychological landscape." This is an extremely ambitious undertaking, and it is difficult to be unsympathetic to any such striving for conceptual, and empirical, unification across formerly disparate fields. Several key issues can usefully be addressed in considering the application of life history theory to psychiatry in this manner.

First, from large-scale studies in evolutionary biology, the degree to which a single fast–slow axis exists remains an open question. One of the more recent high-profile analyses (Bielby et al., 2007) indeed found that a model of two fast–slow axes—representing, first, offspring size versus number, and second, timing of reproduction—fit the data considerably better than one. Can psychiatric disorders be mapped onto two such separate continua? Do psychological traits really vary as clearly coadapted packages, structured predominantly by one or two ecological-evolutionary spectra?

Second, and more generally, the evolution of fast and slow life history traits is driven predominantly by schedules of age-specific mortality. Connections of survivorship patterns with facultative, or genetically based, variation in human psychology and behavior would thus appear central to a life-history based framework for psychiatry, but such links are presently tenuous at best, and restricted to specific situations such as timing of menarche. More-direct dovetailing of life history traits with psychology and behavior comes from studies of animal personality

variation, especially the shy–bold continuum, which has been shown to strongly impact on patterns of survival and reproduction in natural populations (e.g., Dingemanse, Both, Drent, & Tinbergen, 2004). Might a higher dimensional analogue of this continuum map onto human psychological variation better than a dimension of fast versus slow? Should *Diagnostic and Statistical Manual of Mental Disorders* constructs be squashed, stretched, sliced, and squeezed onto life-historical contours, or might a more-coherent structure be derived de novo, from some first principles?

Third, and most generally, life history theory centers on trade-offs between, for example, investment in growth verses reproduction, allocation to current versus future reproduction, and focus on quality versus quantity of offspring. In theory, life-history trade-offs should thus be reflected in cognitive trade-offs, such that specialization or allocation in one psychological domain is inversely associated with that in another. Cognitive trade-offs between, for example, default network and task-positive network brain activation patterns (Jack et al., 2012), certainly exist, but how might they relate to psychiatric disorders? Might extremes of trade-offs, such as between social and nonsocial cognition (Crespi & Leach, in press), characterize many psychopathologies independently of life-history, with fast and slow traits as consequences, rather than causes, of fundamental cognitive axes that evolved in the contexts of a much broader swath of psychological and behavioral domains? Do, for example, relatively autistic individuals invest more in parental effort than mating effort, and if they do, would this pattern reflect adaptive life-history covariation; trade-offs; or, perhaps more parsimoniously, less developed skills in social cognition that constrain success from mating effort?

From Adaptation to Disorder, and Back

Enter evolutionary biology again, as guide in our quest to develop and apply the standard medical model to psychiatry. By basic evolutionary theory, variation in nature is maintained by balances of costs and benefits: Higher blood glucose may provide benefits in one context, such as faster energy deployment or protection of the brain's supply, but it incurs costs in other contexts, such as tissue damage. Variation in human personality has been well characterized by the five-factor model, and positions along each of its axis can be linked, with increasing empirical rigor, to costs and benefits in various fitness-related contexts (Nettle, 2007, 2011a). We can thus connect evolutionary biology with human personality, in a scaffold of robust, empirically documented costs, benefits, and trade-offs (e.g., Alvergne, Jokela, & Lummaa, 2010; Bernard, 2010; Gurven, von Rueden, Stieglitz, Kaplan, & Rodriguez, 2014; Nettle & Penke, 2010). The second key connection, of normal human personality variation with abnormality in mental disorders, has been the nexus of intense interest, research, and progress for many years (Widiger & Costa, 2012). As a result, personality disorders can now be conceptualized and characterized as "extreme and/or maladaptive variants of the domains and facets" of the five-factor model (Widiger & Presnall, 2013, p. 516), and personality disorders can be associated, with increasing success, to Axis 1 disorders (Widiger, Simonsen, Krueger, Livesley, & Verheul, 2005). Taken together, these works frame a nascent medical model for psychopathology, grounded in three interlocking tiers: evolutionary theory; foundational work on personality; and current, pragmatic parsing of psychiatric disorders.

The N-Dimensional Brain

Bridges connecting personality-related adaptation and trade-offs with personality disorders, and more severe psychiatric conditions, represent only a first step toward a medical psychiatry, given that personality represents only one of many adaptive neurologically, developmentally, and genetically based systems. Under this evolutionary framework, for example, autism can be understood in the context of underdeveloped social cognition and developmental noncompletion (Crespi, 2013); bipolar disorder and depression can be understood as dysregulation of the emotional, motivational adaptive systems that underly social-behavioral, fitness-associated goal-seeking (Johnson, Fulford, & Carver, 2012; Nesse, 2004), and schizophrenia can be conceptualized and studied in terms of alterations to causal salience perception (van Os, 2009), language (Crespi, 2008), and social cognition (Crespi & Badcock, 2008), all of which involve, to some degree, maladaptive hyperdevelopment of human-elaborated brain components and functions (Crespi, 2013; Crespi & Leach, in press). In these cases, and all others, the nominal "disorders" remain reified, but the adaptive systems subject to maladaptive alterations do not; they, like glucose regulation mechanisms, really *are* real and have evolved by natural selection and other core evolutionary processes. Characterizing such systems should proceed most effectively through integration of top-down conceptual with bottom-up mechanistic approaches, especially studies that fill the broad gaps between psychology and neuroscience. In this context, our overarching goal is to determine, for each individual beset with mental difficulties, what adaptive systems are subject to underdevelopment, overdevelopment, loss of homeostatic regulation, or pathological effects from mutational or deleterious-environmental input.

Diagnostic categories will fade in importance as knowledge of brain development and function come into better focus, and our abilities to personalize therapies (as, perhaps ironically, already done in psychoanalytic traditions) expand and mature. Our ultimate objective is thus not to reform psychiatric nosology but to destroy it.

By the paradigm described here, there are at least as many "axes" of adaptation for the brain as for the body, all shaped by selection to fit our evolutionary history of diverse social, ecological, and environmental challenges—and as many axes of associated disease. Life history, and a fast–slow continuum, is one for which usefulness to understanding psychopathologies is now beginning to emerge.

Note

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Psychological Inquiry, 25: 325–329, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.920596

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Life History Selection and Phenotypic Diversification

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Del Giudice (this issue) has written an excellent review of how a reexamination of our understanding of the phenomenology of many conditions commonly considered pathological can be facilitated by an analysis based on the conceptual framework of life history theory. We completely agree with this premise and propose that to adequately consider the relations among the psychiatric conditions discussed by Del Giudice, it is desirable to apply the distinction made by Crawford and Anderson (1989) between *true pathologies* (which presumably depress individual fitness) and *pseudopathologies* (which may not depress individual fitness but may actually enhance it at the possible cost of group fitness, in addition to being perceived as undesirable traits by modern societies).

Risk Preferences

Let us first consider what might be interpreted as pseudopathologies that may be functionally consistent with different life history strategies. The understanding of risk aversion, various implications of which are discussed by Del Giudice, has advanced considerably in the last two decades. Although *risk* itself is defined by economic theory as simply variability in outcomes, it is important to take into consideration certain more fine-grained dimensions of this construct, and these particular scenarios include whether individuals are faced with statistical distributions of outcomes for which the defining probabilities vary in a predictable fashion and whether these probabilities can be known to some degree, as we might expect to observe a complex interplay between these antecedent ecological circumstances and life history speed in shaping the expression of psychopathology. Owing to the fact that fast life history strategists are adapted to unstable, unpredictable environments (Ellis, Figueredo, Brumbach, & Schlomer, 2009), it is theoretically expected that the types of risks taken by them are frequently those defined in the literature as "ambiguous," in that the outcome probabilities are likely to vary unsystematically and therefore to be unknowable in advance. Risk taking under uncertainty of outcome probabilities requires iterative risk taking for there to be sufficient payoff (Liu & Colman, 2009). Moreover, considering that the probabilities of consequences are variable and probably unknown in this scenario, systematic effortful information processing and complex rational analysis to decide on whether to take the risk are less beneficial than for contexts in which the outcome probabilities are constant and may thus be known (which is more likely in stable, predictable environments and slow life history strategists). Fast and frugal heuristics (Gigerenzer, Todd, & the ABC Research Group, 2000) are expected to be the main decision process in this scenario, whereas systematic effortful information processing, which is phenomenologically similar to *worry* (Dash, Meeten, & Davey, 2013), may instead be deployed by slow life history strategists (Kennair, Fernandes, & Glass, 2014). Considering this, and considering that worry is a time- and energy-consuming phenomenon that may not be affordable for fast life history strategists, anxiety (which is an emotion deployed when considering prospective risks) should be accompanied by worry in slower life history strategists more so than by faster life history strategists.

Another dimension in which risk preferences, life history speed, and pseudopathological conditions may covary refers to whether individuals would incur greater fitness costs by taking risks over possible gains or by taking risks to avoid possible losses. Seminal works by Kahneman and Tversky (e.g., Kahneman & Tversky, 1979; Tversky & Kahneman, 1992) have demonstrated that people tend to present stronger loss aversion and are thus more prone to taking risks to prevent losses than to accrue gains, but individual differences exist in this respect. Individuals with longer-term investments and who have more emotional or material resources appear to present stronger loss aversion (e.g., Isen & Geva, 1987; Rusbult, 1983), thus it could be inferred that slower

life history strategists (who have much to lose; Wolf, van Doorn, Leimar, & Weissing, 2007) present stronger loss aversion than faster ones, although evidence is indirect and research on this variation is still scarce. If confirmed, this would have implications for the triggers of increased states of anxiety and other negative emotions across the life history continuum: They should be more pronounced over uncertainty over possible gains for faster life history strategists than for slower ones, whereas for slower life history strategists they should be more pronounced over uncertainty with respect to possible losses.

Differentiation and Integration Effort Allocation

We now turn to the question of true pathologies, by which we mean psychiatric conditions that do depress individual fitness and should therefore be considered unequivocally maladaptive. In the context of true pathologies, we note that Del Giudice describes the theoretical work of Woodley (2011) on the cognitive differentiation and integration of mental abilities via life history strategy as one of a handful of "promising models of individual differences." Consistent with this there are subsequent empirical and theoretical refinements of this model that have substantial implications for the theory under consideration.

To summarize, the Cognitive Differentiation-Integration Effort (CD-IE) model posits that life history speed regulates the degree to which cognitive abilities are correlated with one another via the positive manifold (*g* factor), independent of a correlation at the individual differences level between *g* and speed of life history (*K* factor). Meta-analyses indicate a disattenuated and synthetic correlation between the two ranging between .02 and .06 (Figueredo et al., in press; Woodley, 2011). Individuals living in stable and predictable environments can afford to specialize their cognitive ability profile via the allocation of "differentiation effort" in order to facilitate adaptation to specialized sociocultural niches in ontogenetic time. Effort allocation of this sort is especially favored in crowded environments where differentiated mental abilities can translate into increases in aggregate efficiency of resource utilization (and consequently a rising carrying capacity) for a cooperative social group. Conversely, environmental instability and unpredictability favors a capacity for contingency management, and hence the allocation of "integration effort" into the strengthening of the positive manifold among abilities. The advantage of exhibiting a balanced ability profile is that unstable and transient sociocultural niches, requiring a myriad of different abilities, can be occupied and vacated when necessary. Tests of this hypothesis involving both convenience samples of undergraduate college students and large, representative samples of the young adult population of the United States (Woodley, Figueredo, Brown & Ross, 2013) corroborate the original predictions made in Woodley (2011).

CD-IE theory has been extended to encompass the broader manifold that exists amongst the investment domains comprising human life history, *Super-K*, which is the higher-order common factor underlying the widest array of convergent indicators of life history speed (Figueredo, Vásquez, Brumbach, & Schneider, 2004). This generalization of the principle has been termed the *Strategic Differentiation-Integration Effort* (SD-IE) model. It holds that not only are individuals within slow life history populations more *cognitively* specialized but they are also more *strategically* specialized in terms of how they allocate resources among the specific fitness domains that constitute their overall life history strategy, further facilitating functional *polyethism* (ethological polymorphism). Conversely, those with fast life histories are *strategic generalists*, who allocate equal amounts of effort into producing a generalized fast life history strategy, further facilitating the capacity for contingent switching. The presence of significant SD-IE effects has been confirmed at the individual differences level in both student and various nationally-representative samples of the U.S. population (Figueredo, Woodley, Brown & Ross, 2013) and at the group differences level, using various methods (Armstrong, Fernandes, & Woodley, 2014; Dunkel, Cabeza de Baca, Woodley, & Fernandes, 2014; Fernandes & Woodley, 2013; Woodley & Fernandes, 2014; Woodley, Fernandes, & Madison, 2014).

As was mentioned, both the SD-IE and CD-IE models have substantial implications for Del Giudice's thesis, some of which have already been explored. For example, both CD-IE and SD-IE have been posited as potential contributors to the complex phenomenon of autistic-like personality disorders and related phenotypes (such as *Savantism*). Savants, for example, exhibit lower levels of *g*; however, they are highly specialized in terms of their ability profiles (often exhibiting prodigious talent within narrow memory domains). Savantism is frequently associated with autistic-like personality (Treffert, 2009), which, based on Del Giudice's schema, is associated with slow life history. Woodley (2011) posited that high differentiation effort stemming from slow life history could account for this phenotype, despite Savant's exhibiting lower *g*. Similarly, Figueredo et al. (2013) posited that those with autistic-like personalities constitute a distinct morph of slow life history strategy, as illustrated by contrasting the characteristics of the typical high-K population (who are characterized by *high* social effectiveness, as evidenced by high GFP; Figueredo et al., 2004) with those of individuals exhibiting autistic-like personality (who are typically

characterized by *low* social effectiveness). Similarly, those with autistic-like personalities exhibit a strong tilt toward visuospatial ability and away from verbal ability (Del Giudice, Angeleri, Brizio & Elena, 2010). It was posited that this variegation among these two equally high-K groups might reflect frequency-dependent selection for cognitive and sociocultural niches that are socially specialized and "niche construction" (environmental engineering) specialized respectively.

The broader implication of CD-IE and SD-IE for Del Giudice's model is that, if the covariance among psychiatric conditions relates to an underlying life history factor, we would expect the covariance to be weaker among those conditions that fall toward the high-K pole of the life history speed continuum and stronger among those that fall toward the low-K pole. The implication of this is that the putative mechanisms that may be domain specific to each disorder category, and which Del Giudice suggests should be studied more closely, are more likely to play a role in the etiology of conditions manifest among those with slow as opposed to fast life history strategies. In summary, therefore, there should be a greater diversity of psychiatric conditions among those with slow relative to those with fast life history strategies, with more interindividual variability in terms of the specific symptoms with which individuals present at diagnosis.

This is important for Del Giudice's thesis, as tests involving populations that are range-restricted in terms of levels of K, might yield differential results based on the level of K, leading those unacquainted with CD-IE and SD-IE to erroneously discard the theory on the basis of the seemingly greater modularity of symptoms presented by those with slow relative to those with fast life histories. These theories predict this variation in the levels of underlying covariance, and thus need to be kept in mind when testing Del Giudice's model.

Effects of Assortative Mating

There is yet another potentially selective mechanism to consider that might govern the dynamics of comorbidity among different forms of psychopathology, producing higher degrees of polymorphism in psychopathology at the slower end of the life history speed continuum and lesser degrees of polymorphism in psychopathology at the faster end of the life history speed continuum: Vásquez and Figueredo (2002) proposed a theory in which the normal mechanisms of assortative mating among humans should automatically generate such a systematic pattern of covariation.

A common observation among clinicians that work with persons who suffer from psychiatric conditions is that it is relatively rare to find an individual that falls within a single commonly accepted diagnostic category (as per, e.g., the current version of the the American Psychiatric Association's, 2013, *Diagnostic and Statistical Manual of Mental Disorders*). Instead the typical client is a person with multiple concurrent diagnoses (Benjamin, 1996), which is a condition referred to in the biomedical sciences as multiple comorbidities. This is an empirical observation, but the question of causality remains.

Del Giudice suggests that one possible mechanism underlying this phenomenon might be pleiotropic deleterious mutations. This model would necessarily predict genetic correlations among the comorbid conditions. However, the existence of the requisite genetic correlations among psychiatric conditions that are presupposed by this model has yet to be empirically substantiated.

The so-called Genetic Dregs Hypothesis, on the other hand, does not presuppose common genetic causes among the conditions commonly observed to be comorbid. Instead, it derives the prediction from Fitness Indicator Theory (Miller, 2000) that high mate value individuals should assortatively mate with each other by discriminating against potential sexual partners that lack the requisite indicators of genetic quality (meaning a lower load of deleterious mutations, whether pleiotripic or not). This dynamic has the necessary consequence that low mate quality individuals are left with no one but each other as prospective sexual partners. On the operative metric of mate value, it is probably the case that many psychiatric conditions are of comparably low marginal mate value and are therefore functionally interchangeable as assets or liabilities (with emphasis on the latter) in the hypothesized mating market. In the likely absence of sufficient competencies to make specific diagnoses of these psychiatric conditions, low mate value individuals with different conditions would probably consider themselves to be roughly matched in mate value and wind up as mutually acceptable sexual prospects. In contrast, by merely discriminating against prospective mates with any such psychiatric conditions, high mate value individuals should wind up selecting sexual partners with none of these mate value liabilities. An unfortunate combination of polygenes contributing to psychiatric conditions that are roughly equivalent in their marginal effects upon depressing mate value should accumulate in the region of the gene pool (the metaphorically "shallow end") containing the residue of low mate value individuals that were rejected as partners by their higher mate value counterparts. This aggregation of traits indicating lower fitness represents the genetic dregs, analogous to the precipitates inevitably produced when making wine. Offspring produced by assortatively mating individuals of low mate value that might have been formerly

diverse in their profiles of psychiatric conditions would instead acquire profiles of comorbidity spanning multiple psychiatric conditions, in spite of the initial lack of any logically necessary causal dependencies amongst these distinct psychological traits.

As it has been shown that slow life history strategists have evolved systematically higher standards of mate value for partner selection (which serve the function of at least partially avoiding the potential hazards of the higher levels of homozygosity entailed by positive assortative mating; Figueredo & Wolf, 2009), it is therefore likely that these genetic dregs are more prone to accumulate in the portion of the mating pool composed of individuals with faster life history strategies (given their generally lower standards of mate value for sexual partners and increased tolerance for higher degrees of disassortative mating). These dynamics should therefore automatically generate higher levels of comorbidity among different forms of psychopathology at the faster than the slower end of the life history speed continuum, as well as systematically higher levels of psychopathology in general at the faster end of that continuum.

In conclusion, we have proposed several additional formulations of the problem as potentially complementary to those of Del Giudice and outline conditions for their possible empirical disconfirmation. We have done this not to criticize or contest Del Giudice's proposals but instead to help elaborate on this highly promising theoretical framework.

Note

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Psychological Inquiry, 25: 330–333, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.916190

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On Challenges Facing an Ambitious Life History Framework for Understanding Psychopathology

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Marco Del Giudice's (this issue) efforts to build a novel, innovative, theory-inspired framework for understanding psychopathology around a continuum of life history trajectories are admirable. The purely descriptive, empirically driven distinction between externalizing and internalizing conditions is unsatisfying for several reasons: It *is* purely descriptive; it represents empirical data awaiting theoretical understanding, At the same time, it is empirically inadequate to account for a variety of disorders (though, it might be noted, it need not aspire to do so); too readily, it encourages a rather simplistic, and untested, simplistic notion about disorders: Commonly, they're what happens when people experience strong negative emotions—the emotions irritate either the self or, through acting out, other people. Del Giudice's framework, by contrast, is fundamentally theory driven. It is inspired by integrative theory within evolutionary biology. Rather than seeing broad swaths of psychopathology as simply the outcomes poorly regulated emotion, Del Giudice's framework is built around the idea that behavior (and physiology) is adaptively coordinated in various ways. Out of these adaptively coordinated suites, however, may arise, through multiple pathways, disorder. Again, I applaud these efforts.

As I view it, Del Giudice's proposal is a very ambitious promissory note. He doesn't claim its full ambitions to be realized at this time. He does put enough argument and evidence on the table to say that the framework has utility, and its potential for having far-reaching utility is well worth a serious conversation. That conversation—yielding a full vetting of how far the framework can take our understanding of psychopathology—could go on for some time. And, to my mind, it's very deserving of a long conversation. My commentary should be viewed as a small contribution to the early stages of that conversation. It challenges some of the assumptions (explicit or implicit) of Del Giudice's framework in its most ambitious form. (In other ways, perhaps I ask Del Giudice to be explicit about assumptions—or at least clarify them to me.) I offer these challenges from the perspective of a (hopefully) useful Devil's Advocate, by no means hardheaded unbeliever.

The Ambitious Form of the Framework

In the first half of his target article, Del Giudice does a very nice job of accomplishing several aims. He clarifies what he means by disorder. In particular, though "true" disorders may involve harmful dysfunction, as he uses the term a disorder could also be functionally adaptive, even if socially undesirable. He offers a brief description of the fundamentals of life history theory, a broad theoretical perspective within evolutionary biology. He then presents the fundamental bases of his life history framework for understanding psychopathology.

First, life history strategies are broad, coordinated suites of coadaptive features shaped by selection to optimize inclusive fitness, under constraints. Del Giudice focuses on individual differences that result from a major continuum capturing much within-species variation in life history parameters, the fast–slow continuum. In short, fast strategists transition from a pre-reproductive period to a reproductive period at a relatively young age; they are more sexually permissive; they invest less in long-term cooperative relationships and hence tend to be more aggressive and less agreeable, and so on; they discount the future at a steeper rate and, hence, are less cautious and more risk taking. Slow strategists, by contrast, mature relatively late, invest more in stable long-term romantic relationships, are less aggressive and more cooperative, and are relatively cautious.

Second, following from a life history framework, strategies along the fast–slow continuum may be adaptive (or, in ancestral human conditions, have been adaptive) in certain circumstances: for example, faster strategies when mortality risks are greater and less predictable, slower strategies when mortality risks are lesser and more predictable.

Third, life history strategies indirectly contribute to psychopathology through a number of different pathways. (a) Adaptive variants may be socially undesirable and hence labeled disorders. Extreme versions of fast and slow strategies, though arising through developmental processes shaped by selection because of their net benefits, may nonetheless not be tolerated in modern society. (b) Maladaptive variants may appear because fitness functions are cliff-edged, such that slight overshooting of a target phenotype

can be maladaptive. (c) Strategies that are adaptive on the whole need not result in good outcomes in every instance. By its very nature, risk taking increases variance in favorability of outcomes, and some individuals experience poor outcomes even when the average net gain of all individuals taking the risk is positive. (d) Life history strategies can lead to vulnerabilities to mutations and other developmental perturbations.

Fourth, in its most ambitious form, this framework accounts for the vast majority of psychopathological conditions. That is, the very ambitious version of the life history framework is that most psychopathology results from one of the four pathways just enumerated. Del Giudice dedicates a substantial portion of the last half of his article to making the case that major clusters of psychological disorders— ranging from psychopathy to schizophrenia, autism to depression, obsessive-compulsive disorders to eating disorders—can be fit within the framework and, hence, are outcomes of one or more of the pathways just presented.

I emphasize that, to have substantial utility, the most ambitious form of Del Giudice's framework need not be correct. Even if a few major clusters of psychopathology are most appropriately explained within the contours of the framework, it would be very useful. Again, however, my commentary pertains to the most ambitious form.

An Evolutionary Framework for Understanding Disease

Disease itself is not a sensible target of evolutionary explanation. Why bodies and brains are vulnerable to disease, however, is subject to evolutionary explanation (Nesse, 2011). Reasons are abundant; I list just a few here.

(a) *Evolutionary events that impose costs on organisms*. Mutations are not selected (though mutation rate can be subject to selection). Mutations are more often deleterious than beneficial. Selection operates to remove deleterious mutations. But strength of selection on individual mutations may be very weak, meaning that individuals carry many mildly deleterious mutations (see Keller & Miller, 2006).

(b) *Coevolutionary processes that prevent optimal adaptation*. Hosts and pathogens coevolve in response to one another, selection favoring features of pathogens that better take advantage of hosts while favoring features of hosts that better defend against pathogens. Neither hosts nor pathogens are perfectly adapted to one another. Similarly, the interests of mothers and fetuses conflict, such that fetuses may benefit from a faster rate of nutrient transfer to them than is optimal from the mother's perspective. At any given point in time in the coevolutionary process fueling selection for adaptations and counteradaptations in each party, some mothers and some fetuses will not fare well in this conflict. This conflict, then, may explain many of the maladies of pregnancy, harming mothers and/or fetuses (Haig, 1993). (See also Crespi, 2008.)

(c) *Trade-offs under constraints*. A fundamental tenet of life history theory is that organisms allocate energetic and other resources to fitness-enhancing traits, and to do so in such a way that reproductive success, integrated across the life span, is optimized. Optimization occurs under the constraint of limited resources, however, meaning that no feature to which resources are allocated will be perfect. (Instead, the instantaneous marginal net benefit from all allocations, integrated across the life span, is expected to be equal.) This outcome purportedly explains why organisms senesce (e.g., Kirkwood, 1977). Individuals who maintained perfect soma through repair processes could be better allocating some of the energy dedicated to repair to some other features (e.g., reproduction). But more generally, it helps explain why individuals do not perfectly resist pathogens, do not heal perfectly from wounds, do not sense dangers with perfect fidelity, and must grow before being reproductively capable.

(d) *Mismatches between ancestral and modern environments*. Adaptations that offered net benefits in ancestral conditions need not deliver them now. A favorite example pertains to the rise of "diseases of civilization" in Western countries and the United States in particular: metabolic and cardiovascular disease. Adaptations for energy storage and utilization that worked well under conditions of limited nutrient supply do not work well under conditions of abundance (e.g., Nesse, 2011).

Do Vulnerabilities to Mental Disorders Operate Exclusively (or Near Exclusively) Through Life History Strategies?

Life history strategies, again, consist of coordinated suites of features, calibrated and organized through developmental and physiological (e.g., endocrinological) processes. Surely, some of the vulnerabilities to disease can, in principle, operate through effects on life history strategies to yield maladaptive outcomes. Indeed, Del Giudice mentions examples: for example, some mutations or harmful intrauterine event may lead to an overly extreme expression of a fast or slow strategy; the same may lead to failures in risk taking entailed by a strategy (e.g., in a purported case of schizophrenia, in which mutations are claimed to derail sexually selected expression of creativity; e.g., Shaner et al., 2003). But an ambitious form of the life history framework argues that most

all psychological disorders either (a) are not true disorders, or (b) are disorders produced by one of the processes already noted earlier: overexpression of a life history strategy; a case of individual failure in the context of a life history strategy; vulnerability to maladaptation due to a life history strategy—that is, that the vulnerabilities leading to psychopathology due to mutations, coevolutionary processes, trade-offs, mismatches, and so on *do not, in any major way, occur through pathways other than those involving life history strategies*. In my view, one major challenge facing Del Giudice's framework is to build a strong argument in this regard. Next I discuss a few specific challenges that come to mind.

An a priori Basis, Why Should It Be So?

It is not obvious to me that, as a rule, psychopathology *ought* to result from a pathway involving life histories. Mutations or toxins that affect physical disease can target very specific traits, not necessarily coordinated suites of traits (though effects on specific traits can instigate a cascade of other effects, albeit not necessarily ones organized strategically). Would the same not be true of disorders with behavioral syndromes? I can imagine, specifically, disorders neurodevelopmental or neurodegenerative in nature as likely candidates. Hence, for instance, Huntington's disease is caused by a dominant autosomal mutation, and results in loss of coordination, cognitive deficits, and a broad range of psychiatric symptoms (including depression, anxiety, compulsion, hypersexuality). The mutation affects Htt, involved in the transcription of a particular protein of unknown function. Parkinson's disease has diffuse specific etiology (likely mutation in some cases, exposure to toxins in others) but defined neuropathology. It too affects motor function but affects a wider breadth of psychological features in time. I do not see that these neurodegenerative disorders manifest through one of the four pathways through which life history strategies can affect outcomes. (For discussion of possible common pathways of neurodegenerative disorders, see Ross & Poirier, 2004.) More to the point of disorders that Del Giudice discusses, on an a priori basis, I remain unconvinced that neurodevelopmental disorders too affect arrays of features that share early developmental pathways, yet not coordinated in ways characteristic of life history strategies. The life history interpretations of schizophrenia and autism spectrum disorders that Del Giudice discusses are interesting but remain highly conjectural. (The fact that males rather than females are prone to these disorders may simply reflect the fact that males are the sex more vulnerable to disruptions early in development in general, whether psychological or not, perhaps owing to greater male developmental striving; e.g., Rutter, Caspi, & Moffitt, 2003.) More to the point, however, I do not see an argument that, a priori, a life history interpretation is likely to be appropriate.

Heterogeneous Causes of Behavioral Syndromes?

Certain personality features are associated with early puberty and, perhaps, relatively fast senescence. Others may be associated with late puberty and relatively slow senescence. In this sense, fast and slow life histories may manifest in behavioral traits. But to the extent that these associations are weak or modest, one must be careful, it seems to me, about *defining* life history strategies in terms of behavioral syndromes. Behavioral outcomes may be overdetermined, such that, at times, for instance, aggressivity is *not* the outcome of fast life history. Indeed, it seems that, if a person matures late and senesces slowly yet acts aggressively, his or her life history is, in terms of its defining features, slow. How prevalent such persons are depends on how tight the correlations are between defining life history parameters and personality features. How tight are they?

How Well Do We Understand Fast-Slow Variations in Life Histories?

Del Giudice's framework presumes that life histories are driven by cues of extrinsic hazards and their predictabilities. Of life histories that are fast (or slow), however, there may be multiple causes. Hence, for instance, in species in which sexual selection is strong, more fit males dedicate much effort to mating and have lower viability, that is, they are fast strategists (Kokko, Brooks, McNamara, & Houston, 2002). Less fit males are relatively slow strategists. Strategies in these instances are not contingent on extrinsic hazards but, rather, condition-dependent variation in benefits to allocating effort to mating versus somatic maintenance. Might some variation in life histories within human populations have similar causes, rather than be contingent on cues of extrinsic mortality rates? What might be the implications for variation in behavioral profiles among individuals who are, by definition, fast (or slow) strategists?

Are Trade-Offs and Strategies Other Than Those Along a Fast–Slow Dimension Involved?

This observation leads to my next concern. Del Giudice focuses on the fast–slow dimension along which life history strategies lie, with psychopathologies reflecting extremes or disruptions of fast and slow strategies. Yet, even of those forms of psychopathology that stem from overexpression or disrupted expression of contingent trade-offs, it's not clear why

all should be understood in terms of fast–slow trade-offs. Hence, for instance, some researchers have argued that some forms of depression result from a chronic inflammatory state, predisposed by a variety of conditions, but including diet and composition of gut flora (e.g., Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008). Chronic inflammation may well be involved in mediating trade-offs between immune function and other efforts. But it's not clear why inflammation-induced depressive symptomatology must (or should) be understood as an outcome of a pathway through which fast–slow life histories affect psychopathology.

Are Outcomes Mediated Through Life History Variations, Moderated by Life History Variations, or Both?

Del Giudice notes a number of disorders that appear to have different variants, with different variants within cluster categorized as fast or slow spectrum disorders (e.g., depression, eating disorders, obsessive-compulsive disorders). But here, are the distinct variants to be understood as having been mediated by life history strategies via the four pathways specified? Or are the precise manifestations of particular disorders moderated by life history variation (or, more generally, personality variations)? That is, do certain disorders, etiologically homogeneous, nonetheless give rise to distinct symptom patterns due to interactions with personality features? Is the mediator–moderator distinction important to Del Giudice's life history framework?

Summary

I end where I began. I like bold, far-reaching proposals. And Del Giudice's life history framework for understanding psychopathology certainly qualities as both bold and far-reaching. Again, I suspect that his target article will instigate a very long conversation about the merits of this framework. In my view, the very ambitious version of the framework, which accounts for most of psychopathology in terms of one of four pathways by which life history strategies have their effects, faces challenges, both theoretical and empirical in nature. That said, I look forward to efforts on behalf of the framework to meet these challenges.

Note

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Psychological Inquiry, 25: 334–336, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.916195

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Taylor & Francis Group

Life History Theory as a Powerful Framework for Clinical Psychology

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In the target article, Del Giudice (this issue) offers a bold and provocative new framework for the field of evolutionary psychopathology that, if substantiated, may have far-reaching implications for the disciplines of psychiatry and clinical psychology in general. (With apologies to my psychiatrist colleagues, I shall hereafter use the term "clinical psychology" for simplicity, as it is my own field, but in the context of this commentary, the usages are essentially interchangeable.) To evolutionary social scientists, the application of such a powerful and fundamental concept as *life history theory* (LHT; for a review of the success and scope of this theory, see Stearns, 2000) to mental disorder may seem immediately appropriate, sensible, and promising. However, the field of psychology has not fully moored itself to the consilient foundation of science formed by physics, chemistry, and biology (Tooby & Cosmides, 1992; Wilson, 1998), and so many clinical psychologists may perceive Del Giudice's model as coming out of left field.

Despite Charles Darwin's interest in the topic of mental illness (Walmsley, 1993), clinical psychology has been one of the last branches of psychology to incorporate evolutionary perspectives in an extensive fashion (Carmen et al., 2013). The reasons for the field's overall resistance to evolutionary perspectives are beyond the scope of this commentary; suffice it to say that LHT is a theory that has paid its dues in the biological sciences (Stearns, 2000) and has illuminated the fields of developmental (Ellis, 2004; Figueredo et al., 2006), social (Kaplan & Gangestad, 2005), and personality psychology (Gladden, Figueredo, & Jacobs, 2009), among others. The question at hand, therefore, is not whether LHT is an appropriate framework to apply to the topic of psychopathology but whether Del Giudice's (this issue) particular formulation turns out to be correct. It is my position that, at the very least, Del Giudice's evolutionary life history framework for psychopathology is an ambitious, well-thought-out, and exciting step toward the integration of clinical psychology into the evolutionary life sciences, and it may well be a good deal more.

One of the main strengths of LHT as it relates to psychology is its ability to parsimoniously package into a single whole what would otherwise be (and indeed, usually is, in the current literature) a cumbersome collection of psychological constructs with vaguely intuitive relationships to one another, a tangled nomological net, the edges of which are impossible to find. By contrast, LHT connects, in one clean sweep, constructs as diverse as sociosexuality, social cohesion, risk-avoidance, parenting attitudes, antisocial behaviors, impulsivity, life expectancy, childbearing age, and attachment style, just to name a few (Del Giudice, this issue; Figueredo et al., 2006.). Few other midlevel theories in psychology come to mind as having such explanatory power. Whether LHT has the additional capacity to shoulder the burden of explaining psychopathology remains to be seen, but Del Giudice (this issue) does a commendable job of setting forth an eminently falsifiable theory, with straightforward, well-articulated predictions that are open for immediate targeting by critics who wish to test it (as is the case with any good theory). Figueredo and colleagues (2005) identified a factor they refer to as *K*, which is essentially a measure of how fast or slow an individual's life history strategy is; simple correlations between individuals' *K* scores and their levels of various psychopathology are sufficient to test Del Giudice's predictions, and thus his model.

Implications for Taxonomy

At the risk of putting the cart before the horse, I would like to consider a few of the implications for clinical practice if the LHT model of psychopathology, or another model like it, were to gain wide empirical support. As Del Giudice implies, such a model could hypothetically form the basis for an alternate classification system of mental disorders—especially if the long-stumbling *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) fails to regain its footing. Its most recent iteration, the *DSM-5* (American Psychiatric Association, 2013), has spurred a startlingly rapid hemorrhaging of support from a number of different schools of psychological and psychiatric thought. The psychodynamic community has designed a psychodynamic alternative to the *DSM* (McWilliams, 2011), and the biomedical community, led by the National Institute of Mental Health, has created its own system, the Research Domain Criteria (RDoC; Insel et al., 2010). The small but growing contingent of evolutionarily informed clinical psychologists and psychiatrists is sure to follow suit sooner or later, as the atheoretical

nature of the *DSM* becomes more and more frustrating to a community who believes that the study of the human mind should indeed have a central theory: the theory of evolution.

Imagining how an evolutionarily informed alternative to the *DSM* would look is a fascinating and challenging exercise, as the knowledge base that would be needed to create such a classification is nowhere near complete. (On the other hand, one might rebut, this problem never stopped the architects of the *DSM*; incompleteness of knowledge is not a valid argument against the progressive pursuit of said knowledge—this is what science is all about.) One principle likely to be included in such an evolutionarily informed taxonomy would be Wakefield's (1992, 2005) concept of *harmful dysfunction*. This definition of disorder essentially states that true disorders satisfy the dual criteria of being (a) harmful to the individual or society, and (b) dysfunctions of naturally selected mechanisms, that is, failure to properly serve their evolved function. The harmful dysfunction analysis is not without its critics (Lilienfeld & Marino, 1995; McNally, 2011), but it is one of the only mainstream attempts to inject evolutionary thought into the existing *DSM* system, and it has the ability to provide conceptual clarity when new disorders are proliferating among charges of overpathologization (e.g., Kutchins & Kirk, 1997; Washburn, 2013). Separating true mental disorders from inconvenient or unpleasant but functionally normal human behavior has important implications (Cosmides & Tooby, 1999); these considerations can change the way we treat psychological distress as well as the way we view ourselves and our mental health. Mismatch between our current and our ancestral environments may render certain design features of the human animal suboptimal or distressing in contemporary society (Glantz & Pearce, 1989; Nesse & Williams, 1994), but it would be inaccurate to label this as mental illness. This concept of normal functioning versus pathology corresponds to the first of Del Giudice's four causal pathways from life history strategies to psychopathology ("Adaptive life history-related traits may be regarded as symptoms," p. 269), whereas his other three causal pathways (see pp. 261–300 of the target article) represent crucial perspectives on the ultimate causes of disorder that are nevertheless absent from the atheoretical *DSM*.

Another perspective from the evolutionary behavioral sciences likely to show up to some degree in an evolutionary taxonomy of mental illness would be the notion that the mind consists of discrete yet interconnected evolved systems that serve particular functions (Kennair, 2003). This proposition enjoys the unusual position of being, at the same time, both exceedingly mundane and extremely controversial, depending on the nature and extent of the psychological systems one is positing (e.g., the existence of neural pathways representing the spatial location of perceived objects is relatively uncontroversial [Robertson, Treisman, Friedman-Hill, & Grabowecky, 1997], unlike the claims that social processes also have dedicated mental modules, which have generated substantial debate [Frankenhuis & Ploeger, 2007]). Although ongoing scientific debate and evidentiary aggregation systems like PsychTable.org (Balachandran & Glass, 2012) work to settle the empirical details, some evolutionarily informed researchers of psychopathology may already favor a taxonomy that conceptualizes mental disorders as disruptions in particular evolved systems (Kennair, 2003). It is worth noting that this conceptualization actually bears some resemblance to the research domains of National Institute of Mental Health's RDoC (viz., negative and positive valence systems, cognitive systems, systems for social process, and arousal/modulatory systems; Insel et al., 2010), although the approach, methods, and assumptions of the physiologically oriented RDoC are obviously very different in nature from those of an evolutionary taxonomy. Del Giudice's discussion of the LHT model, being evolutionarily informed, references this perspective in the discussion of OCD-like behaviors, low mood, and a number of other behavior and emotional patterns as functional. Although I personally am not yet convinced of the adaptive origins of eating disorder behavior (e.g., Mealey, 2000), one need not agree with every purported adaptationist hypothesis to accept the premise that the brain contains a number of evolved systems that can become dysregulated for any number of reasons.

One invaluable contribution of Del Giudice's model to a hypothetical *DSM* replacement is the provision of a biological theory (viz., LHT) to explain why and how genetic and environmental factors result in particular phenotypic outcomes such as mental disorder. The valuable field of behavioral genetics (Moffitt, 2005) purports to answer this question, but to an evolutionist, the behavioral genetics literature contains a hole the exact size and shape of LHT; learning about how the interplay of genes and environment result in developmental outcomes is one thing, but understanding the ultimate forces that have shaped these dynamics is another matter entirely. In the LHT framework of psychopathology, gene–environment relationships play the crucial role of the feedback system that guides an individual's life history strategy as a function of his or her particular context and experiences. The LHT model is also fully consonant with *ecological systems theory* (Bronfenbrenner, 1979), in that the various levels of an individual's environment affect his or her development; in turn, the individual exerts an influence on his or her immediate environment, creating a dynamic system. The agreement of the LHT model with existing

developmental models of psychopathology work in its favor and make its acceptance within the field much more likely.

Conclusion

Del Giudice's (this issue) LHT model of psychopathology represents a triumphant theoretical integration of developmental and evolutionary perspectives into the realm of clinical psychology. By providing a parsimonious model of mental disorders based upon fast or slow life history strategies, Del Giudice has created the type of model that could form the basis for an upcoming evolutionary taxonomy of mental disorders. The model's assumptions and predictions are clear, and the model is explicated in such a way that it can be easily tested. This empirical support, of course, is the next stage in the LHT model's life, and only time will tell if Del Giudice's predictions will pan out. What he offers here is no less than a fully testable comprehensive model of mental disorder; I am cautiously but eagerly optimistic about the potential of this new framework to inform and even transform the way we understand, classify, and treat mental disorders.

Note

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Psychological Inquiry, 25: 337–340, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.915708

Routledge
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Fast and Slow Sexual Strategies Are Not Opposites: Implications for Personality and Psychopathology

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In the target article, Del Giudice (this issue) uses life history theory (LHT) to help explain personality and psychopathology. Although we admit the explanatory power of LHT is enticing, especially to minimalists, and although we have employed LHT previously in theorizing about the evolution of individual differences (Holtzman, Augustine, & Senne, 2011; Holtzman & Strube, 2011), here we offer a skeptical message about LHT in the spirit of scientific progress.

A central assumption of LHT is that short-term mating (the mating strategy preferred by fast life history strategists) is the polar opposite of long-term mating (the mating strategy preferred by slow life history strategists). This means that short-term mating activities, such as having one-night stands, and long-term mating activities, such as getting married and raising children, are assumed to be directly pitted against one another. On the surface, the notion that there is an opposition between these two sexual strategies makes some sense, because time and energy spent engaging in one strategy is time and energy that cannot be spent engaging in the other strategy; however, recent empirical evidence suggests that this opposition may not be as strict as is implied by LHT.

For instance, in a groundbreaking study, Jackson and Kirkpatrick (2007) explored the potential of a multidimensional approach to investigating sexual strategies by developing a measure that contained items aimed at assessing both orientation toward short-term mating and orientation toward long-term mating. They reasoned that if short-term and long-term mating are in fact relatively separate dimensions, then they should be only moderately inversely correlated. In contrast, if short-term and long-term mating are polar opposites on a single dimension, then they should be very strongly inversely correlated. In accordance with their prediction, Jackson and Kirkpatrick (2007) found that the short-term mating orientation and long-term mating orientation scales were only modestly negatively correlated. Thus, the authors concluded that short-term and long-term mating orientation are not opposites on the same dimension but instead should be measured on relatively separate dimensions.

Figure 1 depicts this two-dimensional (2D) model of sexual strategies. In this model, the short-term mating axis extends from the bottom right (low short-term mating orientation) to the top left (high short-term mating orientation), and the long-term mating axis extends from the bottom left (low long-term mating orientation) to the top right (high long-term mating orientation). Whereas traditional orthogonal axes would indicate that short-term and long-term mating orientation are entirely independent of one another ($r = .00$), and a single axis would indicate that short-term and long-term mating orientation are direct opposites of one another ($r = -1.00$), here the axes are intentionally situated between these two extremes, thus reflecting the moderate negative correlation between short-term and long-term mating orientation (Holtzman & Strube, 2013; Jackson & Kirkpatrick, 2007).

This 2D model has an important explanatory advantage over the 1D LHT model. Because LHT pits short-term strategies directly against long-term strategies, LHT can capture only those individuals who use either short-term or long-term sexual strategies exclusively. In contrast, the 2D model of sexual strategies can describe four different varieties of people: those who use short-term strategies only, those who use long-term strategies only, those who use a combination of short-term and long-term strategies, and those who use neither short-term nor long-term sexual strategies. These four varieties constitute the quadrants of Figure 1.

Clearly, there are instances in which a person might use more than one type of sexual strategy or might not use any sexual strategy. Take, for example, an extraverted and open-minded man who, though he has had several children with his wife and engages in parenting activities with her, has a few one-night stands with extramarital partners on the side. Admittedly, there is somewhat of a trade-off between these two activities; it is obvious that a person cannot be in two different geographical locations (a long-term mating context and a short-term mating context) at once. However, this does not imply that a few short-term sexual encounters preclude the man in the example

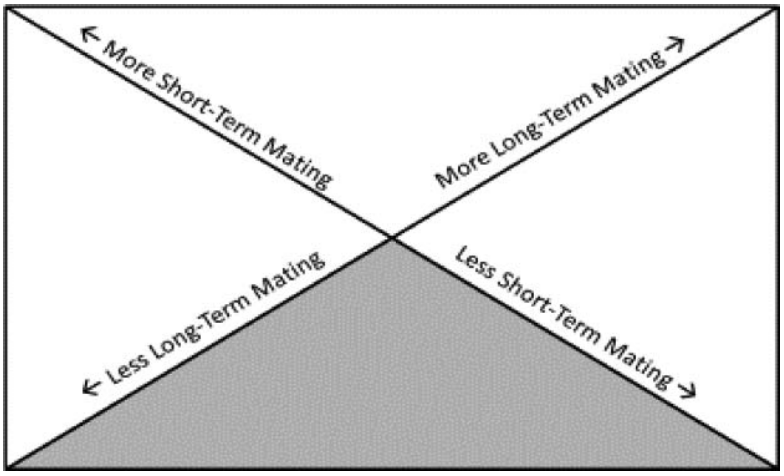

Figure 1. Two-dimensional model of sexual strategies.

from engaging in long-term mating and, by extension, parenting activities (or vice versa). In this example, LHT's presumed opposition between short-term and long-term sexual behavior is largely an illusion; it is clearly possible for a person to engage in both.

On the other hand, consider the case of a young schizoid individual who spends most of her days engaging in solitary activities, such as gardening or simply sitting in a chair. As time goes on, she experiences increasing bouts of catatonia, often remaining silent and motionless for extended periods. This person has neither sexual desires nor sexual prospects, and thus is very low on both short-term and long-term mating orientation. Other examples of individuals who may be low on both short-term and long-term mating include people with various sexual dysfunctions described in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; American Psychiatric Association, 2013). Among these dysfunctions are male hypoactive sexual desire disorder, which is characterized by low or no sexual desire or fantasies in males, and female sexual interest/arousal disorder, which is characterized by low or no sexual desire or fantasies in females (American Psychiatric Association, 2013). Like those with schizoid personality disorder, individuals with these sexual dysfunctions are disinclined to engage in either short-term or long-term mating.

Because people who use a combination of short-term and long-term sexual strategies and people who use neither strategy clearly exist, it is important to examine the personalities of these individuals in addition to the personalities of individuals who use one strategy or the other. However, because the 1D LHT model does not differentiate between those high on both short-term and long-term strategies and those low on both short-term and long-term strategies, it cannot be used to discern the personalities of these two varieties of people. This is a serious flaw of the LHT model. We argue that the 2D model of sexual strategies provides a promising way of conceptualizing the personalities of individuals such as the man in the preceding example who has extramarital affairs, and the woman in the preceding example who is uninterested in sexual activity altogether. Indeed, some recent evidence suggests that this approach merits further exploration. Holtzman and Strube (2013), for instance, examined the relationship between sexual strategies and personality using separate scales to assess short-term and long-term mating orientation. The authors suggested that if long-term mating captures variance in personality above and beyond that captured by short-term mating, then a 2D sexual strategies model is better suited for investigating individual differences in personality and psychopathology than is a traditional 1D model.

Indeed, whereas Del Giudice struggles to explain the evolutionary basis for Extraversion and Openness using the single LHT dimension, Holtzman and Strube (2013) found that these traits can be mapped onto a 2D model. Specifically, in a U.S. sample, Extraversion correlated positively with both short-term mating orientation and long-term mating orientation. Similarly, Openness correlated positively with short-term mating orientation and, contrary to what would be expected if short-term and long-term mating were direct opposites, did not correlate negatively with long-term mating but rather was unrelated to it. Thus, in line with our example of the extraverted and open-minded man who engages in short-term sexual affairs while still maintaining a long-term relationship, Extraversion and Openness may be two traits that are better accounted for by a 2D sexual strategies model than a 1D model.

Furthermore, whereas Del Giudice does not address the possibility of people who do not engage in sexual activity at all, the 2D model of sexual strategies may provide a useful way of modeling the

personalities of these individuals. Specifically, Holtzman and Strube (2013) found that schizoid personality disorder was strongly negatively correlated with long-term mating but was not related to short-term mating (again in contrast to the positive correlation with short-term mating that would be expected if short-term and long-term mating were direct opposites). Schizoid personality disorder, as in our example of the woman who spends her days in isolation and has no interest in pursuing sexual activity, may therefore be one disorder that maps better onto a 2D model than a 1D model. These points seriously call into question the LHT framework as a general model for individual differences. The minimalistic psychometric approach of LHT forces unidimensionality on a multivariate problem—the problem of personality. In turn, it completely sacrifices our ability to detect both the personalities of people who engage in multiple sexual strategies and the personalities of people who engage in no sexual strategies.

Although this rejection of a strict LHT framework may at first elicit aversion from evolutionary psychologists, we argue that the postulate can be retained that individual differences—including those in psychopathology—emerged through the process of evolution. The key to making the transition in conceptualization, we argue, is the more thorough integration of the concept of mutation–selection balance, which is captured by Keller and Miller's (2006) evolutionary watershed model of psychopathology. The watershed model posits that some forms of psychopathology result from the slow buildup of very slightly maladaptive mutations across many generations. As the evolutionary game is played, nature strongly selects against seriously harmful (e.g., deadly) mutations, but it does not strongly select against mild mutations. As a consequence, mild mutations can accumulate in certain branches of the genetic tree. Psychopathology, as Keller and Miller (2006) pointed out, is one outcome of this process.

Del Giudice does (albeit briefly) discuss the notion of mutation–selection balance as one possible cause of psychopathology, and we applaud him in this attempt to integrate mutation–selection balance into evolutionary personality psychology. However, we feel that this attempt at integration entails some problems. By acknowledging that some types of psychopathology may result from the buildup of relatively harmless mutations, it seems Del Giudice may be implicitly advocating for another type of multidimensional model (distinct from our 2D sexual strategies model), wherein mutation load constitutes a second dimension, orthogonal to the fast–slow LHT dimension. According to such a model, different types of psychopathology still fall at particular points along a fast–slow continuum but are expressed only at particular levels of mutation load. Although this type of model would indeed be preferable to the 1D model for which Del Giudice explicitly advocates, it still involves an LHT dimension, which again contains a false opposition between fast (short-term) and slow (long-term) sexual strategies. Our model in Figure 1 does not have this problem.

In addition to avoiding the problem of a false opposition between short-term and long-term sexual strategies, our model explicitly incorporates mutation–selection balance theory. We argue that there is a dumping ground (depicted by the grayed-out bottom quadrant of Figure 1) that captures high mutation load. That is, people who fall within the top three quadrants of Figure 1 (i.e., those high in short-term mating only, those high in long-term mating only, and those high in both short-term and long-term mating) carry fewer mutations than people who fall within the lower (gray) quadrant (i.e., those low in both short-term and long-term mating). As in Keller and Miller's (2006) evolutionary watershed model of psychopathology, the accumulated mutations of individuals who fall within this lower quadrant may lead to certain types of psychopathology, specifically those types of psychopathology that are associated with low orientation toward both short-term and long-term mating and are therefore associated with lower reproductive success (e.g., schizoid personality disorder).

Thus, expanding upon the ideas put forth by Del Giudice, we argue that there is a cliff-edged function that separates the upper three quadrants in Figure 1 from the lower (gray) quadrant. A cliff-edged function in this instance refers to a phenomenon in which the values assigned to one of the variables in a multivariate space drop dramatically. In this case, the variable for which values drop is reproductive success (which, although not pictured, constitutes a third axis extending three-dimensionally through the center of Figure 1), and the point at which reproductive success drops is when one crosses the threshold from the white quadrants to the gray quadrant in Figure 1. It may be helpful to think of this gray zone as a valley or canyon—a low point representing minimal reproductive success. At this juncture in the history of evolutionary personality psychology, it remains unclear which one of the top three quadrants of Figure 1 is optimal in the sense of yielding the highest reproductive success; in fact, there may even be multiple optima, which could be visualized as mountains rising out of the page in Figure 1. Locating the optimum or optima remains a topic for future research.

In sum, we favor a 2D model of sexual strategies over the 1D LHT model. Not only does it have the potential to double the variance accounted for in individual differences in personality and psychopathology, but it also explicitly integrates the concept of mutation–selection balance.

Note

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Psychological Inquiry, 25: 341–345, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.897200

Routledge
Taylor & Francis Group

The Virtues of Evolutionary Psychology for Studying Human Vices

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The target article author (MDG) rightly points to an area of psychology in desperate need of a reformulation along the lines of an evolutionary/functional analysis. For too long, the study of psychopathology, in all its forms, has suffered from a lack of parsimony and coherence. Indeed, to us—as outspoken proponents of the utility of evolutionary psychology—the major strengths or virtues of the adaptationist paradigm are that it provides a set of a priori assumptions concerning the most important questions to ask about mental health and illness (i.e., questions about adaptive mechanisms, ultimate functions, and ancestral-modern environmental mismatches; Buss, 2000; Nesse & Williams, 1994; Wakefield, 1992), provides the most appropriate methods for evaluating functional hypotheses (see Schmitt & Pilcher, 2004), and allows for a proverbial trimming of the various esoteric topics that undermine the scientific study of mental health and individual differences (Confer et al., 2009). MDG does a tremendous job at pulling together work from various subdisciplines of psychology to make his case that researchers can use life history theory to better understand the nature of psychopathology. In this commentary, we expound upon topics with which we agree with MDG, points where we think he did not go far enough, and discuss how the Dark Triad (i.e., psychopathy, narcissism, and Machiavellianism) have been fruitfully studied from a life history perspective.

The task of organizing the psychological sciences within evolutionary biology has some powerful implications for the study of psychopathology. It is important to note that by doing so we can distinguish the pathological "forest" from the "trees." This is because evolutionary psychologists ask themselves "why" questions as opposed to "how" questions. "How" questions (a.k.a., proximal) involve the mechanisms that trigger a given response (viz., behaviorism, social psychology). "Why" questions (a.k.a., ultimate) are concerned with the reasons why people are responsive to certain stimuli in the first place. However, as MDG rightly points out, the most common way of diagnosing psychopathologies is the "community standard." This presents scientists and clinicians with a moving target and slippery slope way of determining if someone has a mental disorder.

From an evolutionary approach, dysfunction may take on a more stringent definition than the community standard or subjective wellbeing approaches. Once researchers have identified the function of a trait in its relevant context (which itself needs to be identified), they can go about ascertaining whether someone is suffering from a disordered version of that trait. Indeed, the term "dysfunction" alone assumes we already know what a given trait is supposed to do, an assumption we feel has not been sufficiently met outside of general personality traits like the Big Five. A dysfunction of an evolved physiological mechanism would be indicated, for example, if one's blood failed to clot after one's skin was cut, if one failed to sweat in response to external heat, or if one's larynx failed to rise to close off the passage to the lungs when food is swallowed (see Buss, 2000; Wakefield, 1992). There are at least three criteria by which we could judge whether a psychological trait is not functioning as it should in its relevant context (Buss, 2000). First, the mechanism fails to activate in lieu of its triggers (e.g., a narcissist receives praise but fails to feel better about himself). Second, a trait is activated in an inappropriate context (e.g., being extraverted at a funeral). Third, the mechanism fails to coordinate behavior with other mechanisms (e.g., wanting social interaction but relying solely on Facebook instead of making actual friends).

MDG rightly points out that most work on psychopathologies has focused on those on the fast end of the spectrum. Personality traits like psychopathy (along with narcissism and Machiavellianism) have received considerable attention lately as a potential adaptive strategy (e.g., Jonason, Koenig, & Tost, 2010; Jonason, Valentine, Li, & Harbeson, 2011). This is a rather obvious tendency among researchers for at least two reasons. First, work on traits like the Dark Triad have been glamorized by the tendency of both the media to portray them as hero or at least antiheroes (Jonason, Webster, Schmitt, Li, & Crysel, 2012; Leistedt & Linkowski, 2014) and for researchers to use various

"sexy" characters to make their point about the adaptive value of the Dark Triad (e.g., James Bond; see Jonason, Li, & Teicher, 2010).

Second, fast spectrum disorders are incongruent with the rather slow way of life that characterizes modern Western societies with its heavy investment in offspring and cooperative, mutualistic relationships. Those with fast spectrum disorders have an agenda that is directly contradictory to the people they live with and, therefore, are more easily seen as disordered from a community standard. This makes them stand out more than slow spectrum disorders. If we assume researchers make a name for themselves by saying something nonobvious, the focus on fast spectrum disorders seems like a foregone conclusion. Instead, MDG takes a much more balanced approach, noting there are slow and fast spectrum disorders, each with their respective costs and benefits (see also Nettle, 2006). Indeed, to this point, MDG convincingly argues that each disorder is about how one deals with opportunities. Those characterized by a slow spectrum rarely take opportunities, but those characterized by a fast spectrum disorder cannot take opportunities fast enough.

We fear one criticism that might be leveled against this theoretical contribution is that is does not provide a means by which clinicians can actually state in a definitive fashion who has a disorder or doesn't (Holcomb, 2001). However, this would be a specious criticism. The MDG approach might actually clarify some of the boundary conditions of various disorders aiding in clearer diagnoses. Clinicians have particularly strong needs to classify individuals as having a disorder or not for legal and medical purposes. By better defining various conditions, there may actually be fewer disorders to work with. Even if we assume that all the disorders that are presently in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; American Psychiatric Association, 2013) are true disorders (as opposed to some variant of preexisting ones), the MDG approach may at the very least move us from the community standard model to a more quantitative model. Instead of relying on the community standard, disorders could be indexed on some quantitative and theoretically relevant life outcomes like number of sex partners, number of friends, or income. This would also allow for the assessment of relative severity of disorders to be more than subjective experience.

Such an approach would take some serious work, but we feel this would fundamentally change the way we think about disorders, moving them from the either–or (false) dichotomy. It would be more consistent with personality research outside of the clinical settings. It would move from thinking about cutoffs to thinking about distributions of scores in a population. MDG assumes (as we do) that personality traits are best understood on continua as opposed to dichotomies (Penke, Denissen, & Miller, 2007). Again, although we recognize the desire/need to classify individuals as we just noted, we feel this is patently flawed. Quantitative researchers have repeatedly pointed out the statistical and theoretical problems with breaking continua into groups—problems like diminished power, potential spuriosity, and obscuring nonlinear relationship (MacCallum, Zhang, Preacher, & Rucker, 2002; Maxwell & Delaney, 1993). The tendency to do so serves people's predilection for simple answers. Just because something is hard or more complicated does not mean it is wrong or should be avoided. It just means more work is needed; more nuanced analyses are required.

One area we do not feel MDG went far enough is to explore the possibility that the various "psychopathologies" or "disorders" are not either per se but, instead, are adaptive solutions to environmental contingencies (Denissen & Penke, 2008). Although he discusses the types of contexts under which we would expect to find certain traits he appears to miss the more important point that various contexts may actually elicit certain responses and that when these are in their disordered form it because of a mismatch between how one's brain is tune and their current environment. For instance, some of the symptoms of posttraumatic stress disorder could be described as contingent responses to stressful and harsh environments (Christopher, 2004). During recurring crises and calamities, a person's brain may be set to run at a faster pace, favoring immediate outcomes and reproduction. In the context where the adaptive response was calibrated, the traits are functionally useful and not working against the person's interests. It is only when the war veteran returns home and is confronted with the abundantly slower life history defining conditions of modern Western living that trouble arises.

A related area where we feel MDG did not go far enough was in reference to cross-cultural psychology. The history of cross-cultural psychology reveals it to be a rather descriptive science whereby a given pair or some small set of countries/cultures/states are compared on a psychological dimension or the strength of associations between psychological constructs is contrasted across cultures (Church, 2009; van de Vijver & Leung, 2001). However, without evolutionary psychology, there are very few a priori reasons to expect particular outcomes or associations across cultural forms. In contrast, evolutionary psychology has yielded a plethora of insights into why cultures vary along psychological dimensions, including predictable associations among psychological constructs and factors such as pathogen load (Gangestad, Haselton, & Buss, 2006; Schaller & Murray, 2010) and morbidity (Ellis, Figueredo, Brumbach, &

Schlomer, 2009), providing strong reasons to predict particular cross-cultural effects. We would argue one could easily extend this to study cross-cultural psychopathology. Most efforts to do that to date have noted the difficulties in applying the same mental health and illness standards across different countries (Van de Velde, Bracke, Levecque, & Meuleman, 2010), and to some degree assert the need for psychopathology to be contextually understood (Friedman et al., 2010). However, this also seems to us to be moving the goalposts. A more functional analysis of personality traits and disorders and understanding how personality traits interact with particular environmental contingencies will allow for a better understanding of the geographic and ecologically embedded distribution of various psychological disorders (Ein-Dor, Mikulincer, Doron, & Shaver, 2010; Schmitt, 2008).

Taking the aforementioned points about environmental calibration, a missed point is that a life history paradigm may also (surprisingly) provide guidance for how to fashion better interventions. Our understanding of the manner by which various therapy treatments are devised is based on the manner by which individuals assume the brain works and interacts with the body. For instance, a Freudian approach assumes the brain works like a hydraulic system where pressure builds up and needs to be released, and it works on a system of conflicts between the id, ego, and superego. Alternatively, a neuroscience model envisions that brain and behavior problems stem from structural or mechanical problems with physical aspects of the brain. In contrast, we would suggest the life history model would propose an information-processing model of the brain, behavior, and their related dysfunctions. However, unlike other therapies that involve information processing (e.g., rational emotive therapy, cognitive behavioral therapy), the life history model provides a priori content that should and should not prove important in shaping people's psychology and, therefore, to improving potential dysfunctions. For instance, providing someone who has problematic narcissism with information that acts as proxies for stability in one's environment may be able to slow the pattern of information processing toward a less selfish way of life than the way that tends to characterize narcissism. This would not only trim away much of the proliferous noise in the diagnosis of disorders but also may provide a guiding framework for what are the useful and useless treatment procedures.

In short, what we are arguing for here is an adaptive information-processing model of the generation of personality traits in their ordered and disordered fashions. Information about one's environment is processed by one's brain to make sense of the world and make predictions about the future. For those falling into fast spectrum disorders, information will have tuned their brain to move faster because it perceives they are living on a shorter time line. This has important implications in that it actually dismisses the idea of personality psychopathologies in the first place. It suggests that all people have the ability to find themselves in fast or slow spectrum disorders. Such flexibility is surely part of our evolutionary history. The sensitivity to environmental inputs whether over the course of one's childhood or in flash events in their lives is an apparently important system to allow individuals to better adjust their behavior to immediate circumstances. An individual who employed only one set of tactics or strategies would produce fewer offspring in the long run compared to someone with a more protean approach to life (Jonason & Webster, 2012).

Last, we wish to challenge an implicit assumption MDG makes. He treats life history strategies in a bidirectional way, with life history strategies being described on a single continuum. We wonder whether this is overly simplistic. Take, for instance, the multidimensional nature of sexual strategies (Penke, 2011). According to strategic pluralism (Gangestad & Simpson, 2000; Jonason, Li, & Cason, 2009; Schmitt, 2005), individuals' sexual strategies exist on two relatively orthogonal dimensions that can run simultaneously. That is, a woman can simultaneously engage in friends-with-benefits or booty-call relationships while she looks for a more serious partner. This allows her to satisfy her needs for socioemotional support and sexual satisfaction while she attempts to find the "best deal" of a partner with whom she can invest in making a baby (Jonason, 2013). In as much as sexual strategies are subsumed under the larger heading of life history strategies (Dunkel & Decker, 2010), it seems rather obvious that individuals may simultaneously engage in fast and slow life history strategies. Take, for instance, the first author of this commentary. His approach to publishing could be described as producing a large number of low-investment publications (offspring) while working on high-investment publications (offspring). By having access to both approaches, individuals may have even more flexibility in their decision making and can reap greater rewards. That is, they can, on the fly, recalibrate (to a degree) their approach to life. Indeed, some do this in that when they go on vacation may act differently than when they are at home. For instance, the behavior of many American college students on spring break (a 1-week holiday in March or April) is markedly different to how they behave at home or at their university. It may be that this bidimensional nature that underlies the apparent overlaps in some of the disorders noted by MDG and the idea of comorbidity. Such an approach might be tested through the use of cluster analysis to allow

researchers to better define each trait on its own and in relation to others.

One final oversight to which we would like to draw attention is the reliance on the Big Five personality traits and psychopathy (on its own) to describe various disorders. We wish to highlight the work on what is called the Dark Triad, which focuses on the overlap between psychopathy, narcissism, and Machiavellianism (Paulhus & Williams, 2002). This work falls well within the realm of this review, having been repeatedly studied from a life history paradigm. For instance, using the information provided in Table 1 in the target article makes it clear that the Dark Triad traits—mostly psychopathy given its "darker" nature (Rauthmann, 2012)—can be treated as fast spectrum traits. Those high in the Dark Triad traits evidence sexual promiscuity (Jonason, Li, Webster, & Schmitt, 2009), unstable attachments (Jonason, Li, & Czarna, 2013), risk taking (Crysel, Crosier, & Webster, 2013), impulsivity (Jones & Paulhus, 2011), future discounting (Jonason, Koenig, et al., 2010), low conscientiousness and agreeableness (Paulhus & Williams, 2002), and exposure to (familial) stressors (Jonason, Lyons, & Bethell, 2014). Despite all of this, we would suggest the Dark Triad traits are *not* necessarily disorders. For instance, the limited empathy associated with the Dark Triad (Jonason & Krause, 2013; Jonason, Lyons, Bethell, & Ross, 2013) might be adaptive in as much as not empathizing with one's victims will facilitate the exploitive, cheater strategy embodied by the traits. We contend they are not necessarily fast spectrum disorders, but we agree they are "fast" traits.

We have used our work on the Dark Triad to highlight how traits that most would consider dysfunctions might be adaptive if properly understood in an evolutionary perspective. We feel MDG has added important nuance and balance that complements our work. We look forward to upcoming work that will expand on his article in basic and applied contexts.

Note

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Psychological Inquiry, 25: 346–351, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.915707

Routledge
Taylor & Francis Group

Evolutionary Psychopathology and Life History: A Clinician's Perspective

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Introduction

Life history theory is one of the major approaches within evolutionary theory, combining the adaptationist approach with developmental constraints and including how organisms must invest resources strategically (Kaplan & Gangestad, 2005). Del Giudice has offered a truly biological approach to evolutionary psychology through his previous work on life history theory (e.g., Del Giudice, 2009) and is one of the rising stars of the field. It is therefore most welcome when Del Giudice in his current article considers evolutionary psychopathology from this perspective, something I suggested might be fruitful and necessary a decade ago (Kennair, 2003). Del Giudice offers a veritable tour de force, spanning an enormous amount of literature and developing an original approach by considering all mental disorders from a life history perspective, or more precisely from a fast versus slow life history perspective. There is an impressive amount of work behind this article, and although I cannot do justice to all points he raises, I do attempt to provide a clinician's perspective to some of the conditions that I am most familiar with.

It is true; the field of psychopathology lacks theoretical integration (Kennair, 2011, 2012; Nesse & Stein, 2012). There is little consensus on what constitutes mental disorder, and despite work from an evolutionary approach (e.g., Wakefield, 1999), we will probably need research from an evolutionary modular and functional approach for such approaches to fulfill their potential. A comprehensive life history perspective might have the explanatory power to aid such integration, at least as part of the process of expanding the developmental and individual differences level of analysis to an evolutionary psychology approach to psychopathology (Kennair, 2003, 2011, 2012).

Although I am very fond of Del Giudice's work, and welcome his interest in evolutionary psychopathology, I have two points I would like to raise: (a) the possible limitations of reducing life history theory to a fast versus slow approach, and (b) the possible consequences of some of the disorders' treatability for a life history approach. I take the clinician's perspective in the current commentary. As both an evolutionary psychologist and a clinician, I believe it is the latter that may contribute the most interesting perspective for further development of the proposed taxonomy.

Life History Theory and Psychopathology

It is somewhat surprising that in his target article Del Giudice reduces life history theory to two history types: *fast* versus *slow*. It would seem that this reduction loses the full promise of a comprehensive life history approach (see also Bielby et al., 2007). There have historically been many suggested categories of pathology, and most fail. Most clinicians are not looking for yet another set of categories, when what we need at this point may be something more applicable and clinically substantial. Yet again, there is nothing more practical than a good theory (Lewin, 1945): Maybe a deeper understanding of the phenomena we treat will help us help our patients better (Kennair, 2012)?

Is life history theory primarily a theory of individual differences? I would argue that it is not. It may explain differences in expressed behavior and traits, but in general it is a theory of how a specific species will allocate resources through life, based on ecological and resource challenges, and how these compromises (given limited resources) will influence the organism's later "choices."

In concluding, Del Giudice points out the following: "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" (p. 286).

This is important: First, it assuages my worry that the fast–slow distinction is expected to explain all aspects (see, e.g., Bielby et al., 2007), although this must be considered fundamental to the target article's thesis. Second, it shows that there are many different aspects of a functional approach (see, e.g., James & Ellis, 2013). I look forward to the future development of the model, fleshing out how different disorders are formed by maybe several functional perspectives and processes.

The Four Proposed Causal Pathways

The target article describes four causal pathways that lead to the onset of mental disorders.

"Adaptive life-history-related traits may be regarded as symptoms." As I address elsewhere, most cases of mental disorder are maybe best regarded as states, rather than as traits. Sometimes states or behavior are classed in diagnostic manuals as expressions of psychopathology, despite being the evolved output of functioning adaptations (Kennair, 2003, 2011; for a debate, consider Horowitz & Wakefield, 2012). Further, it might be relevant to keep undesirable behavior in others separate from aversive emotions experienced by the individual (Kennair, 2003, 2011). Some cases of the first type may be "treatable conditions" (Cosmides & Tooby, 1999), and some of the second may be fully functioning defences (Nesse & Williams, 1996) and probably ought not to be treated without due consideration. Having said this, I fully recommend considering that, for example, rumination in depression might not be an adaptive defence (Wells, 2009; see Watson & Andrews, 2002). I recommend that any evolutionary taxonomy of mental disorder adopts Wakefield's (1999) criteria of dysfunction. Without it we end up categorizing both dysfunction and function as disordered, and surely that must be, from an evolutionary perspective, a more fundamental division in the taxonomy of pathology than fast–slow life history. As such I disagree with the inclusion of this "pathway."

"Life-history-related traits may be expressed at maladaptive levels." If traits associated with life history may result in maladaptive behavior, and this is coupled to specific life histories, then this might be important to specify more clearly—including the consequences for selection. If one could provide a substantial case for specific life histories, for example, slow life histories (but preferably more precise or comprehensive) causing maladaptive levels of, for example, behavioral defenses, then this would provide support for some evolutionary theories of depression given that one indeed can build a convincing case that depression really is adaptive, at least at lower levels. And that, for example, reduced activity (Nesse, 2000, 2006), risk taking (Nettle, 2009), depression-induced social negatiation (Hagen, 1999), or rumination (Andrews & Thomson, 2009; Watson & Andrews, 2002) are defenses or mechanisms to solve adaptive problems. This is not currently the case.

"Adaptive strategies may yield individually maladaptive outcomes." Yet again, if the mechanisms involved are functioning, it is hard to consider that this is psychopathology. Whether life history is associated with panic attacks is not elaborated in the target article. Although I take an evolutionary perspective to phobias (Kennair, 2007; Sandseter & Kennair, 2011), it is not clear that panic arises from adaptive processes (Kennair, 2007). From a cognitive behavioral perspective, panic is treated efficiently by reattributing misinterpretation of symptoms of anxiety as signs of somatic or psychological catastrophe (which causes rapidly increased anxiety through positive feedback) and by removing nonrelevant safety behaviors (Clark, 1986; Wells, 1997). Why our ancestors would benefit from monitoring and misinterpreting anxiety symptoms as signs of heart attacks, fainting (which is possible only due to blood injury phobia—Kennair, 2007; Marks, 1988—yet again a disorder that has a more convincing adaptive explanation than panic), choking, or going mad is far from clear to me. The same is the case with general anxiety and worry; I would dispute that even mild worry is helpful, given the nature of worry (Wells, 2009). Yes, some claim it is verbal problem-solving behavior (Borkovec, Robinson, Pruzinsky, & DePree, 1983), but typically there is no solution-behavior; primarily worry is problem focused. Despite many people engaging in worry (Wells & Morrison, 1994), *it would seem that worry is not the way they actually do solve problems.*

"Life-history-related traits may increase vulnerability to dysfunction." Anything can potentially malfunction, including mental mechanisms, due to a legion of reasons (Buss & Greiling, 1999; Keller & Miller, 2006; Nesse, 2005). This is true pathology (Kennair, 2003, 2011; Wakefield, 1999). This pathway is probably the most important potential contribution of the article. If future research shows that specific life histories are associated with increased incidence of dysfunction, and preferably specific and modularly discrete dysfunction (à la the Stone, Cosmides, Tooby, Kroll, & Knight, 2002, paradigm), then this would be a major breakthrough for the article's thesis. I would therefore have liked to see more substantial evidence of this pathway, which indeed would be the most explicit life history to pathology *pathway* of the four.

What Does the Model Offer?

The life history approach to psychopathology is a broad and general model. In several places Del Giudice stresses the importance of considering a legion of other relevant factors, levels of analysis, and approaches. As such it is often difficult to pinpoint the hard predictions of the model. Some disorders will cluster together with slow life history and others with fast life history disorders. This backdrop may explain several other features of individual differences, and may explain why an individual may have many similar disorders. This may be true. We expect links between neuroticism and a slow life history, and our measures of these might both include risk aversiveness. But neuroticism is already measured by asking about depressiveness and anxiety. One needs to avoid circularity. Also, I am little more reserved

about how clear the evidence is for the features of fast or slow (target article, Table 1) being typical of patients suffering from the listed disorders (target article, Figure 1). This being said: I look forward to future research investigating these possible and original predictions when considering actual patient populations. This future research needs to consider whether there actually exists a systematic pattern between the correlates of fast or slow life history (target article, Figure 1) and features of patients from the specified fast or slow diagnoses. Some these would surprise me, however, including autogenous obsessions (I do not believe it would be found to be fast life histories; rather they would look as slow as other obsessive-compulsive disorders (OCDs), if indeed that is a relevant appropriate categorization) and depression (I am far from certain that these patients would prove to have slow life histories), but it would be most interesting.

Efficient Treatment and Consequences for a Life History Approach

As a clinician involved in treatment trials for disorders such as OCD, phobias, depression, and generalized anxiety disorder (GAD), I am interested in not only how an evolutionary approach may improve our understanding of disorders but primarily how this approach may improve our efficiency in treating mental disorder.

OCD

The treatment of choice for OCD (especially compulsions) is exposure with response prevention (ERP; Kozak & Foa, 1997; NICE, 2005; Walsh & McDougle, 2011), although this intervention is limited by availability. In a study considering dissemination of effective treatments, by teaching inexperienced student therapists ERP for OCD, our students were able to match results from international clinical trials (Solem, Hansen, Vogel, & Kennair, 2009).

The suggested subcategory of OCD called Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS; Swedo & Grant, 2005) might have been of relevance to the current target arrticle. I discuss this case as an illustration. PANDAS is a controversial theory (Macerollo & Martino, 2013; Walsh & McDougle, 2011) but suggests that extremely rapid onset of OCD symptoms in childhood may be due to strep infections (Swedo & Grant, 2005). Note that it is the body's reaction to the infection, rather than the infection itself, that is presumed to be associated with OCD. There are several problems with the theory (Macerollo & Martino, 2013; Swedo & Grant, 2005; Walsh & McDougle, 2011), but maybe the greatest problem in the current context is that the sequela or other types of organic change caused by or associated with the infection are reversed as efficiently with exposure therapy as other forms of OCD (Storch et al., 2006; Swedo & Grant, 2005; Walsh & McDougle, 2011). The case of PANDAS also lends itself to adaptationist hypotheses about how a developmental infectious stressor might increase OCD adaptively (as a defense against further infection). It might even be possible to fit it into the current life history theory. I am not convinced that this would be either relevant or necessary. And in any case, PANDAS (if indeed this subcategory exists, maybe it just is OCD) is reversible, and in the current environment it would best constitute what Cosmides and Tooby (1999) insightfully categorize as a treatable condition.

Despite the controversial status of PANDAS, the treatment community has, alas, hopped on the bandwagon. There has been a large increase of antibiotic treatment of presumed PANDAS, without indication (Gabbay et al., 2008), and subsequent research has not confirmed the existence of PANDAS (Macerollo & Martino, 2013).

For obsessions, metacognitive therapy (Fisher & Wells, 2008; Kennair, 2004) may be more relevant than ERP. Yet again, treatment is promising and relatively swift (Fisher & Wells, 2008). In the current context it is worth noting that this approach does not consider the content of the obsessions, rendering the division of OCD into reactive and autogenous superfluous. My reading of this extensive literature and my clinical experience leads me to conclude that there is overlap between these categories (e.g., as mentioned in Belloch, Cabedo, Carrió, & Larsson, 2010, as cited by Del Giudice, this issue) and that far from all findings are in line with the fast versus slow features (target article, Table 1). Also, many of the findings are from nonclinical populations. The idea that some types of OCD obsessions are more impulsive is also important to challenge (NICE, 2005): I have treated patients who fear harming their children. The first intervention, if they have started to avoid their child, is to reestablish contact between parent and child. They need to learn that they are not going to do any harm. If they truly have OCD, these thoughts are unwanted, ego dystonic. That is what the "unwanted" in the YBOCS (a standard measure of obsessions and compulsions) items indicates; but note: The patient fears that having impulses will cause behavior. This is a false belief, a metacognition, called Thought-Action Fusion (Fisher & Wells, 2008). If the diagnosis is correct, the last thing they will do is harm their child; the "impulse" motivates safety behaviors, not the "aggressive" or sexual acts. Also "aggressive" is a misnomer for these "impulses," as they are thoughts, not emotions, and the patient is anything but

aggressive. The arguments for the fast life history type OCD are therefore those I find to be weakest. It is also important to note that self-description questionnaires used with overly self-critical participants may result in true descriptions of how they view themselves, but nonvalid descriptions of how they are compared to other participants or populations that the questionnaires were developed for. Smári, Bouranel and Eiðsdóttir (2008) discussed this possibility in their discussion of impulsivity. Del Giudice, also, is aware of this problem, and notes this possibility for other aspects such as low conscientiousness in OCD. Many references used to build the case for a fast life history type OCD are with nonpatient samples. Smári et al. (2008) accurately noted that their results need replicating and are mainly interpreted as how obsessive-compulsive symptoms may arise in normal populations.

A new taxonomy of OCD needs to provide improved knowledge for treatment. If it does not, and if there in any case is overlap of the types, there is limited value. I worry that treatment is too efficient for the condition to be a phenotype brought about by life history compromises. Further, OCD is more an expression of a state rather than trait. There are a large number of studies attempting to subdivide OCD based on content. I believe that is a dead end. On the other hand, it would be very exciting if some of Del Giudice's ideas informed an evolutionary based empirical investigation of OCD.

Depression and GAD

I do understand Del Giudice's conclusion, after reviewing the literature, that depression probably consists of several distinct clusters—maybe there are disorders that are more gastro-intestinally driven and some that are better explained by social circumstances (Kennair, 2003). From a clinical perspective we still have scant evidence that such divisions matter. Or that any of the proposed adaptive functions are relevant. If one rather considers proximate maintaining psychological factors the disorder seems more coherent, and treatable. In our current clinical trials we treat patients when they fulfill criteria for major depressive disorder. Further subdivisions do not seem relevant, apart from indicating degree of severity.

Depression and GAD are two disorders that share the genetic basis to a large degree (Kendler, Neale, Kessler, Heath, & Eaves, 1992). They also seem to be maintained by similar proximate factors: Rumination and worry are perseverative mental processes (Wells, 2009). Whereas rumination looks back at failures and mishaps and past events, worry looks ahead with apprehension toward potentially threatening events. GAD is therefore a lot more appropriate as a model of danger monitoring, than OCD (Boyer & Lienard, 2006), which is a disorder that focuses more on preventing danger than discovering danger. In these disorders changing the beliefs that worry or rumination are adaptive in any way, showing that it is under the patient's control and thereafter discontinuing worry or rumination is considered the effective mechanism (Wells, 2009). This approach reduces rumination without engaging in any problem solving, and thus will provide one of the first empirical tests of Watson and Andrews's (2002; see also Andrews & Thomson, 2009) evolutionary model of depression.

But if a tendency toward perseverative mental processes is a result of life histories, then I am surprised how efficiently we change these phenotypes. I am aware that because life history itself promotes plasticity, this might not be a correct understanding of the proposed model, but at the same time I wonder whether the extreme plasticity I have described above may be compatible with the current trait-based model and whether such plasticity will cause a life history perspective to lose predictive value.

Considering scientific clinical trials, there is reason for optimism about how malleable these states are. The life history theory approach therefore seems to run into a problem: If changing behavior is as simple as state-of-the-art effective therapy suggests that it is, then maybe a trait-based approach is less relevant. After years of considering evolutionary approaches, I have to admit that none have thus far contributed to my efficiency as a therapist, whereas proximately developed methods have (e.g., Wells, 2009).

The Future of Evolutionary Clinical Psychology and Evolutionary Psychopathology

I am skeptical of trait-based approaches to malleable, treatable conditions. I believe that many of these disorders are treated with such ease and efficiency that I would consider a different approach. In clinical practice, one would first treat the pathological states before setting any personality disorder with any certainty. Also I believe that an evolutionary taxonomy must start with Wakefield's (1999) harmful dysfunction definition and not attempt to shoehorn all current disorders, nondisorders, and treatable conditions that may be found in the existing diagnostic manuals into a single taxonomy. The disorders are too disparate, ranging from what may be evolved adaptive behavior to true malfunction of mental mechanisms. And in between there will be results of by-products of evolution, like our ability to consider our own thoughts and emotions, form metacognitions, and make up unhelpful ways of attempting to control our aversive thoughts or emotions (see, e.g., Kennair, 2007; Wells, 2009).

I therefore end up with the following questions that I hope may be addressed in further work on this

important topic, and that will make Del Giudice's approach more available to clinicians.

Is the model as trait based and change resistant as I perceive it to be? If disorders may be cured within a few weeks of treatment changing psychological processing, would this be a challenge to the model? Could the model predict helpful interventions or treatment approaches for different conditions? When treatment works for one disorder, does the model expect a trait consistent expression of life history through other disorders, akin to Freudian symptom substitution? I would like to point out that currently we are not certain how effective new methods are at relapse prevention for, for example, depression, which has been one of the major problems within depression treatment for years (Baldwin, 2000). Relapse may therefore be due to features that are consistent with Del Giudice's approach.

Is it possible to develop an approach that goes beyond the fast–slow approach to life history, and would this consider primarily mechanism breakdown associated with life history (as in the pathways mentioned above), or would it primarily consider currently nondesirable results of functioning adaptations?

Is it possible to further elaborate on whether the current approach primarily involves the fastest or slowest life histories? Would it be possible, within this framework, to explain why an individual develops one disorder and not another? And how does the model handle overlap, for example, that a substantial percentage of patients may be diagnosed with both autogenous and reactive obsessions?

I am certain that life history theory will be one of the major theoretical approaches in general evolutionary approaches to the science of human behavior. The current contribution may therefore be an important first step. The field will have to become more aware of development, genetic influences, and individual differences if mainstream evolutionary psychology shall be able to address problems within psychopathology and mental health (Kennair, 2011). Figueredo et al. (2005) pointed out the problematic large theory to data ratio within evolutionary personality psychology; since then, this might have improved a little. Alas, that criticism is even truer for the field of evolutionary psychopathology. Evolutionary psychopathology is to a much larger degree a basic science of an applied field: mental health care. Contributions thus sorely need to come at a clinically applicable level, although even pioneers of the field have tempered optimism of this (e.g., Nesse, 2005). More work on conceptualization or on hypothesized function is therefore of less relevance. Unless, as Del Giudice attempts, it actually achieves two objectives: (a) generate greater interest in evolutionary psychopathology among evolutionary psychologists, and (b) generate empirical testing and research of evolutionary approaches to mental disorder. There is such promise in the target article.

Meanwhile, methods within mainstream clinical psychology continue to be developed and evaluated in clinical trials. As both an evolutionary psychologist writing on psychopathology, and a clinician who has taken part in several clinical trials the last 10 years, I worry that innovation with no need of an evolutionary approach shows greater improved effect of treatment of, for example, depression and generalized anxiety than any evolutionary perspectives currently promise. Mental health care has many future challenges, and understanding the nature of mental disorder is obviously one of these (Kennair, 2012). As such, one must especially welcome contributions that not only dare to aim to achieve this but also express the intention to integrate the field.

Note

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Psychological Inquiry, 25: 352–354, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.897199

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Application of an Integrated Evolutionary Psychological Framework to Psychopathology

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Evolutionary psychological theory has the potential to serve as a useful framework for the classification and organization of developmental psychopathology (e.g., see Bjorklund & Ellis, 2005; Ellis, 2004; Geary, 2010). The original theory of natural selection suggests that heritable individual differences in traits that confer a survival advantage are more likely to be passed on to subsequent generations (Darwin, 1859), including extreme variants of such traits that may increase risk for psychopathology (e.g., extreme anxiety). Further, subsequent evolutionary psychological theories such as sexual selection and life history theory have the potential to elucidate several key defining features of psychopathology including sex differences in prevalence and developmental course. For example, sexual selection suggests that between- and within-sex variability in traits that facilitate reproductive success are more likely to be passed on to subsequent generations (Darwin, 1871) with extremes in such traits potentially differentially increasing risk for psychopathology in males and females (e.g., extreme sensation-seeking increasing risk for externalizing disorders in males; Geary, 2010; Martel, 2013). Further, evolutionary developmental theory such as life history theory suggests that the timing of trait development and expression is important for survival and reproduction with extreme variability in such timing increasing risk for psychopathology (Del Giudice, this issue; Ellis, 2004; Ellis et al., 2012). For example, early pubertal maturation seems to increase risk for psychopathology such as substance abuse (Kaltiala-Heino, Koivisto, Marttunen, & Frojd, 2011; Lynne-Landsman, Graber, & Andrews, 2010; Negriff & Trickett, 2012). Of course, such evolutionary-based influences are complex and interact with and are shaped by proximal environmental and cultural factors (Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999; Ellis, Shirtcliff, Boyce, Deardorff, & Essex, 2011).

Del Giudice's (this issue) current use of evolutionary developmental theory, specifically life history theory, to provide a framework by which to organize and classify psychopathology is thus commendable in that fast versus slow life history strategies seems to have utility for explaining the developmental timing and onset of at least some types of psychopathology (e.g., substance abuse; Kaltiala-Heino et al., 2011; Lynne-Landsman et al., 2010; Negriff & Trickett, 2012). Further, a fast versus slow life history strategy may influence the development of particular types of traits that, in extreme form and perhaps particularly in certain contexts, may increase risk for specific clinical disorders. For example, as Del Giudice (this issue) discusses, a fast spectrum life history strategy, commonly associated with exposure to harsh and unpredictable environmental stressors, is often linked with low conscientiousness and agreeableness, as well as high impulsivity. It is intuitive that such a life history strategy would increase risk for externalizing disorders. Therefore, Del Giudice's (this issue) proposed organizing evolutionary-based life history framework is promising in that it may be able to suggest alternative ways to classify psychopathology.

Yet, use of a fast versus slow life history distinction specified by Del Giudice (this issue) may not reflect the complexity involved in the classification of such a broad array of clinical disorders. For example, it is difficult to see the connection between a fast life history strategy and schizophrenia (or its spectrum disorders) with its core symptoms of delusions, hallucinations, disorganized speech, disorganized or catatonic behavior, or negative symptoms (American Psychiatric Association [APA], 2013). Linkages between impulsivity, low conscientiousness, low agreeableness, and psychotic symptoms remain underdeveloped. Further, it is difficult to see how psychotic symptoms, or even subthreshold symptoms, would facilitate a fast life history strategy to reproduction.

In addition, the connection between a slow life history strategy and psychopathology also seems somewhat implausible, as a slow life history strategy is associated with safe, predictable environments and low exposure to stressors, as well as high conscientiousness and agreeableness (Del Giudice, this issue). Particularly difficult to view through this lens are autism spectrum disorders (ASD), classified by Del Giudice (this issue) as a slow spectrum disorder. Yet ASD share a great deal of overlap with common externalizing disorders, argued by Del Giudice (this issue) to be best captured as fast spectrum disorders. For example, ASD seems to be highly comorbid with attention deficit/hyperactivity disorder (Frick & Nigg, 2012), and ASD are likewise often characterized by low levels of effortful control and executive

dysfunction (Geurts, Verte, Oosterlaan, Roeyers, & Sergeant, 2004; Pennington & Ozonoff, 1996; Samyn, Roeyers,, & Bijttebier, 2011). Therefore, ASD would seem to be misclassified as a slow life history strategy.

Although it seems reasonable that some internalizing disorders, such as some instances of depression and obsessive-compulsive disorder (OCD), may be best represented as slow spectrum disorders, as Del Giudice (this issue) postulates, it is unclear that subtyping depression, OCD, and even eating disorders using this type of approach is more parsimonious than the current *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) approach (APA, 2013). For example, Del Giudice (this issue) argues that there may be subtypes of depression with one subtype characterized by both mood and somatic symptoms, best classified as a fast spectrum disorder, with other subtypes better classified as slow spectrum disorders. Yet the validity of these subtypes is far from established (APA, 2013). For OCD, Del Giudice (this issue) relies on a distinction between the content of obsessions for classification within a life history framework. That is, he argues that cases of OCD with autogenous (i.e., sexual, aggressive, and/or blasphemous) content are best classified as fast spectrum, whereas cases of OCD with reactive (i.e., contamination, mistakes, accidents, disarray) content are best classified as slow spectrum. Yet it is unclear where a person with both types of obsessions would be classified. Finally, eating disorders are subdivided into a dysregulated profile, which is classified as fast spectrum, and a perfectionistic, overcontrolled profile, which is classified as slow spectrum. Yet it is unclear whether all eating disorders, including binge eating disorder, can be so neatly classified. In addition, it seems unlikely that such a subtyping approach will enhance parsimony. It is also unclear how such a subtyping approach could be applied, as many of these distinctions would require extensive history of symptom profiles and/or novel personality assessment.

Yet a novel classification approach based on a combination of natural selection, sexual selection, *and* life history theories may provide clarification and do more justice to the complexity of psychopathology. As shown in Table 1, some disorders may be due to extreme individual variability in traits that confer a survival advantage, or facilitate natural selection. For example, separation anxiety disorder, posttraumatic stress disorder, and dissociative disorders are all examples of disorders that may be best described as extreme variants of traits that confer some survival advantage by providing protection from predators and/or severe stressors and hence increasing the odds of survival. In contrast, other disorders may be best represented as extremes of between-sex differences in traits that are sexually selected, or confer reproductive advantage: for males, by facilitating competition for mates and/or mating opportunities and, for females, by facilitating mate choice, female competition for mates, and/or effective childrearing. In this way, conduct disorder and antisocial personality disorder may be sexually selected in males, and generalized anxiety disorder, depression, and borderline personality disorder may be sexually selected in females (see Martel, 2013). Finally, life history strategies may interact with sexually selected trait predispositions to influence the trajectories of certain forms of developmental psychopathology. For example, chronic exposure to harsh and unpredictable environments may predispose boys to fast spectrum psychopathology during childhood including autism spectrum disorders, attention-deficit/hyperactivity disorder, oppositional-defiant disorder, and substance use disorders, whereas low exposure to stressors in safe, predictable environments may increase girls' vulnerability to slow

Table 1. *Integrated Evolutionary Psychological Framework for Psychopathology.*

Integrated Evolutionary Theory		
Natural Selection	Separation anxiety disorder	
	Posttraumatic stress disorder	
	Dissociative disorders	
Sexual Selection	*Competition for mates (>males)*	*Mate choice / Parenting (>females)*
	Conduct disorder	Generalized Anxiety Disorder
	Antisocial personality disorder	Depression
		Borderline personality disorder
Life History	*Fast*	*Slow*
	Autism spectrum disorders	Social phobia
	Attention-deficit/hyperactivity disorder	Eating disorders
	Oppositional-defiant disorder	
	Substance-related disorders	

spectrum psychopathology during adolescence including social phobia and eating disorders.

In summary, application of a fast–slow life history strategy framework, as advocated by Del Giudice (this issue), has great implications for advancing our understanding of classification of clinical disorders, perhaps particularly externalizing disorders. Yet his life history analysis is not as simply applied to some disorders (e.g., the internalizing disorders, schizophrenia and ASD). His theory is promising in that it suggests alternative ways in which to segment or carve up currently-defined *DSM* categories. Yet these subdivisions do not seem to be any more parsimonious than the current cumbersome *DSM* categories. In sum, whereas Del Giudice's (this issue) evolutionary life history framework may provide a useful starting point for informing classification of psychopathology, integrated consideration of life history theory alongside other evolutionary theories such as natural selection and sexual selection will likely provide more refined advances in classification of psychopathology.

Note

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Psychological Inquiry, 25: 355–359, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.916595

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Sources of Behavioral Variability and the Etiology of Psychopathology

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All organisms, including humans, are products of natural selection. As a consequence, any comprehensive explanation of behavior requires at least some engagement with evolutionary theory. No other theoretical approach can so effectively and parsimoniously account for behavior across a wide range of biological taxa and in multiple domains. Life history theory is a similarly powerful metatheoretical framework firmly derived from evolutionary principles. It provides an understanding of how organisms allocate limited time and energetic resources to such essential biological functions as survival, growth, reproduction, and parental investment. Although life history theory is a relatively newer development in the biological sciences—and an even newer development in the human behavioral sciences—it, like evolutionary theory, enjoys substantial cross-taxa empirical support.

Both evolutionary theory and life history theory are particularly valuable for understanding the etiology of psychopathology in that they provide a blueprint of the Platonic ideal of how to "carve nature at its joints." For this reason, Del Giudice's (this issue) work provides a substantial and important contribution to the literature on the etiology of psychopathology. In this commentary, we expand on a key point that Del Giudice makes in his analysis: Psychopathology (and behavior more generally) is a product of the interaction of both stable individual differences (i.e., life history strategies) and situational and environmental factors (both persistent and acute; pp. 263, 265). We consider the case of risk-taking behavior—an area of our own expertise—as an example of the importance of examining acute environmental and situational inputs for behavior. We conclude with a brief discussion of some implications of an interactive approach for the prevention and treatment of psychopathology.

Behavioral Plasticity and the Influence of Acute Situational and Environmental Factors

Del Giudice (this issue) suggests that life history strategies must be functionally self-consistent to be maximally adaptive (p. 262). This characterization of life history strategies is most consistent with the idea that these strategies are analogues of stable individual differences in personality, where personality describes patterns of consistent behavior across situations and contexts. Someone with a "fast" life history strategy, for example, would exhibit behavior consistent with this strategy across multiple contexts. Others have made similar arguments suggesting that stable individual differences in multiple different domains, including personality traits, may be products of specific life history strategies (e.g., Buss, 2009; Mishra, in press; Simpson, Griskevicius, & Kim, 2011; Wolf, van Doorn, Leimar, & Weissing, 2007). In nonhuman animals, the term "behavioral syndrome" has been used to describe patterns of consistent behavior across contexts (effectively animal "personalities"; reviewed in Sih, Bell, & Johnson, 2004; Sih, Bell, Johnson, & Ziemba, 2004). Growing evidence suggests that behavioral syndromes are also in part products of life history trade-offs (e.g., Biro & Stamps, 2008).

Research evidence clearly suggests that individual differences in personality (in humans) and behavioral syndromes (in nonhuman animals) account for important variance in behavior. However, a substantial portion of behavioral variability appears to be a more plastic product of acute environmental and situational inputs. For example, in a meta-analysis, Bell, Hankison, and Laskowski (2009) showed that approximately 35% of the variance in nonhuman animals' behavior could be accounted for by stable individual differences. Of course, we do not mean to imply that behavior can be neatly separated into products of stable individual differences and acute environmental or situational factors (this approach recalls the flawed "person–situation" dichotomy in social psychology; Fleeson & Noftle, 2008; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). Behavior is necessarily the product of a complex interaction between nonindependent individual differences and environmental factors. However, we do seek to emphasize that any comprehensive understanding of a behavioral phenomenon (especially one as complex as psychopathology) requires explicit acknowledgment of the influence of more acute social and environmental inputs.

A perfectly adaptive organism would have infinite behavioral plasticity so as to respond optimally to any and all stochasticity in their environments. Of

course, infinite plasticity is not possible due to necessarily bounded physiological and cognitive limitations. In particular, life history trade-offs impose constraints on phenotypic flexibility due to the necessity of clusters of traits and behaviors co-occurring together (e.g., late growth is incompatible with early reproduction). However, behavioral plasticity is not lost as a product of stable life history strategies. Rather, life history strategies can be considered stable individual differences that predispose—but do not necessitate—certain patterns of behavior. We illustrate the importance of considering more acute environmental and situational influences on behavior by considering the etiology of risk-taking behavior, an area of our own expertise. Risk taking is a particularly useful behavior to examine because there is relatively extensive work demonstrating that it is both a product of stable individual differences (i.e., life history strategies) and acute environmental factors (reviewed in Mishra, in press).

The Case of Risk-Taking Behavior

In the context of life history theory, risk-taking behavior is typically considered to be part of a "fast" life history strategy associated with future discounting, impulsivity, and short subjective life expectancy (reviewed in Del Giudice, this issue; Mishra & Lalumière, 2008). However, risk taking has a complex etiology, and many forms of risk taking appear to be less a manifestation of a life history strategy and more a product of acute situational or environmental inputs. In fact, risk-taking behavior appears to be highly plastic, even within individuals (reviewed in Mishra, in press). Here, we describe some different pathways that have been proposed to lead to varied patterns of risk-taking behavior over the lifespan (Quinsey, Skilling, Lalumière, & Craig, 2004).

Adolescent-limited risk taking is a normative pattern of risky behavior in multiple domains (e.g., promiscuous sexuality, reckless driving, substance use and experimentation, interpersonal conflict, etc.) mostly confined to adolescence and early adulthood (Moffitt, 1993). Adolescent-limited risk taking is in large part a product of acute situational and environmental factors. The adolescent years are marked by particularly intense social competition (just ask anyone about their high school years!), and young people are at steep competitive disadvantage relative to older people who have had greater time to accumulate embodied capital, social status, and resources (Wilson & Daly, 1985; reviewed in Mishra, in press). As a consequence of these competitive pressures, younger individuals engage in greater risk taking in an attempt to obtain outcomes that may not be attainable through safer, low-risk means.

Most people desist from risk-taking behavior as they leave their teenage years behind (Moffitt, 1993). This reduction in risk-acceptance corresponds with acute changes in the costs and benefits of risk taking. Marriage, a stable job, and having children are reliably associated with reductions in risky behavior later in life (reviewed in Mishra & Lalumière, 2008). Notably, those who lose this stability later in life (e.g., by being widowed, or divorced) engage in subsequently greater risk taking (Daly & Wilson, 2001), suggesting relatively high plasticity in behavior. Of course, not everybody desists from risk taking after the teenage years; there are those who continue to engage in persistent risk-prone behavior, largely due to more stable individual differences linked with particular life history strategies (e.g., low embodied capital resulting in persistent competitive disadvantage; Mishra, in press; Mishra, Barclay, & Lalumière, 2014; Mishra & Lalumière, 2008).

Other evidence for the plasticity of risk-taking behavior comes from experimental studies demonstrating support for risk-sensitivity theory. Risk-sensitivity theory posits that decision makers engage in risk taking when low-risk options are unlikely to meet one's desired goals or outcomes (Mishra, in press; Mishra & Lalumière, 2010). In circumstances of need—great disparity between one's present and desired or goal states—risk taking allows for obtaining outcomes that might otherwise be unavailable or unattainable through safer, low-risk means. Several experimental studies have shown that acute manipulations of the perception of need lead to immediate changes in risk propensity, consistent with risk-sensitivity theory (reviewed in Mishra, 2014). These immediate changes in risk taking have also been shown to occur independent of stable individual differences in risk propensity (e.g., Mishra & Lalumière, 2010; Mishra, Daly, Lalumière, & Williams, 2012).

For example, Mishra et al. (2012) showed that people from disadvantaged socioeconomic backgrounds who had demonstrably engaged in persistent risky behavior (e.g., problem and pathological gamblers, ex-convicts, drug addicts) are actually risk-sensitive decision makers who modulate risk acceptance as a product of environmental cues. Risk-persistent participants in this study clearly exhibited a constellation of behaviors consistent with a stable "fast" life history strategy. In their everyday environments, they engaged in domain-general risk taking, including substance abuse, problem gambling, and property and violent crime. However, in laboratory tasks that manipulated the perception of need in domains they were unfamiliar with (i.e., in computer game–like tasks involving foraging for digital apples to "survive" and earn money), participants exhibited adaptive risk-sensitive behavior. That is, they were

risk accepting when far from a goal (i.e., in a situation of high need), but acutely shifted to risk avoidance when risk taking was unlikely to provide positive outcomes (i.e., when the costs of risk taking exceeded the benefits).

These results suggest that although those in impoverished environments appear to engage in stable, risk-persistent behavior in their everyday environments, they are also sensitive to acute environmental and situational cues regarding the costs and benefits of risk taking. Thus, what may appear to be a "fixed" fast, risk-prone life history strategy may instead be a plastic product of environmental inputs that consistently facilitate risk taking. Risk-persistent individuals may experience what has been termed *enduring situational evocation*, whereby certain consistent environmental features elicit persistent patterns of behavior (Buss & Greiling, 1999). Such persistent behavior may thus be a result of behavioral plasticity, not stable life history strategies or individual differences. Consistent with this hypothesis, some evidence suggests that people often make risk-sensitive decisions independent of stable individual differences in risk-propensity (Mishra et al., 2012; Mishra & Lalumière, 2010; reviewed in Mishra, in press).

Of course, at the other end of the spectrum, there are individuals who demonstrate very low levels of behavioral plasticity in the domain of risk taking. Those who fit the criteria for psychopathy (an extreme manifestation of antisocial personality disorder) show a pattern of persistent risk-taking behavior across the lifespan that appears to be largely a product of genetic influences (Lalumière, Mishra, & Harris, 2008; Mealy, 1995). Similarly, those who have been severely physiologically disadvantaged early in life by experiencing such neurodevelopmental insults as head trauma/brain injury, maternal substance abuse, or obstetrical complications also engage in persistent and relatively inflexible risk taking across the lifespan (reviewed in Mishra, in press; Mishra & Lalumière, 2008). This is especially true when these neurodevelopmental insults interact with impoverished social environments involving such negative experiences as parental divorce, sexual and physical abuse, and poor nutrition, among others (Moffitt & Caspi, 2001; Rutter, 1997). These more stable patterns of risk-taking behavior behavior—psychopathy and (what has been termed) *life-course-persistent offending*—likely correspond to more stable life history strategies that are a product of more persistent inputs (i.e., genes and early developmental environments).

A consideration of the etiology of risk taking is useful for more generally understanding the complexity of behavior of any kind, including psychopathology. Explaining behavior requires elucidation of multiple causes: genetic influences, early developmental environments, stable environmental cues, and more acute environmental or situational factors. Any comprehensive framework for understanding behavior must therefore explicitly acknowledge multiple sources of variation for behavior, and caution must be exercised when invoking any particular explanations (e.g., individual differences vs. situational factors; Fleeson & Noftle, 2008; Roberts et al., 2007).

Implications for Prevention and Treatment of Psychopathology

Del Giudice's life history analysis of psychopathology provides a naturalistic understanding of what factors predispose people to suffering from certain disorders. This approach focuses on explicating the sources of stable individual differences in life history strategies, which in turn are associated with certain clusters of psychopathology. Although the life history approach is certainly very useful in elucidating the etiology of psychopathology—"carving nature at its joints"—it has fewer direct implications for treatment. One of the virtues of a focus on identifying acute environmental and situational cues that facilitate psychopathology is that this approach offers a direct pathway for treatments and interventions. The life history strategy approach to psychopathology, by contrast, offers more by way of prevention strategies (similar to the ecologically relevant prescriptions provided by Ellis et al., 2012, for reductions in adolescent risk taking). Collectively, the two approaches provide a complementary, powerful, and comprehensive framework for treatment, intervention, and prevention of psychopathology.

The case of depression provides an excellent illustration of the importance of considering both acute situational/environmental cues and life history predispositions for the treatment and prevention of psychopathology. Growing evidence suggests that depression serves the adaptive function of focusing one's analytical attention on an instigative problem through substantially increased rumination (reviewed in Andrews & Thomson, 2009). Research suggesting that depressed people have enhanced ability to solve analytical problems is consistent with this hypothesis (e.g., Braverman, 2005; Storbeck & Clore, 2005; reviewed in Andrews & Thomson, 2009). This understanding of depression emphasizes the importance of targeting acute environmental and situational factors that facilitate depressive symptoms for treatment. The adaptive model of depression has other implications for treatment, including reduced use of antidepressant drugs (which interfere with people's ability to ruminate), and a focus on analytical thinking in cognitive behavioral therapy sessions (Andrew & Thomson, 2009; Andrews, Thomson, Amstadter, & Neale, 2012).

By contrast, a life history approach to the problem of depression would be most effective when guiding prevention strategies as opposed to treatment strategies. A fast–slow framework of understanding individual differences helps to identify those individuals who are most susceptible to psychopathology like depression. Prevention strategies could thus be designed and targeted to reduce the baseline risk of the development of psychopathology by highlighting conditions that facilitate susceptibility to mental disorder. It is clear that both life history–facilitated predispositions and acute environmental and situational factors must be considered to most effectively address both prevention of psychopathology (through addressing root causes of dysfunctional or maladaptive life history strategies) and treatment of psychopathology (after symptoms have already manifested) wherever possible.

Finally, one particularly important implication of Del Giudice's naturalistic, evolutionary-based framework of psychopathology (as well as other evolutionarily guided frameworks) is that there are normative, "natural" biological mechanisms that give rise to such dysfunctional or unwanted outcomes as mental disorder. In our view, communicating this reality to those suffering from mental disorder for psychopathology allows for a far more productive starting point for interventions and treatments. One can imagine that it would be much more affirming to those seeking treatment to start with an understanding of their experience as a product of natural processes (rather than as "disease" that can be cured in a biomedical sense). Of course, we must be careful to not to commit the naturalistic fallacy—the mistake of proclaiming that what is natural must necessarily be desirable, good, or excusable (especially in the case of more antisocial forms of psychopathology, e.g., psychopathy, that cause harm to others). Rather, we advocate facilitating understanding among sufferers of psychopathology that normally functioning mechanisms often give rise to dysfunctional, disordered, and/or unwanted behaviors. This approach is particularly valuable in our present sociocultural climate where those who suffer from psychopathology are stigmatized and marginalized because of their suffering from a "disease." Such a starting point would allow for more productive engagement with effective treatment strategies that reduce distressing outcomes for sufferers of mental disorder.

Del Giudice's evolutionary life history approach for understanding psychopathology is an important and substantial addition to the scientific literature. Del Giudice provides a compelling framework for understanding the sources of individual differences in susceptibility to psychopathology. In this commentary, we suggest that this laudable approach is effectively complemented with a consideration of more acute situational and environmental influences on the development and manifestation of psychopathology. Understanding psychopathology as a product of both stable individual differences in predispositions
(i.e., life history strategies) and more acute situational and environmental factors allows for a more comprehensive approach for prevention and treatment of mental disorder. Nature does not give up its secrets easily, and frameworks such as Del Giudice's are a needed step in the right direction.

Note

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Psychological Inquiry, 25: 360–362, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.911639

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Life History Theory's Best Chance: Illuminating Cluster B Personality Disorders

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It was a pleasure to read Marco Del Giudice's (this issue) target article introducing an evolutionary life history framework for psychopathology. We agree that life history strategies are a likely factor in certain types of abnormal behaviors. There are certainly instances in nature that suggest that organisms vary in the way they allocate their time and resources in the service of optimizing reproductive strategies. In hominids, such disparate reproductive strategies could conceivably lead to changes in behaviors and unique enduring (personality) features. It is an especially compelling idea because variation in reproductive strategies has a long phylogenetic history, and therefore may have fundamentally affected the design of the mammalian neurobehavioral system. Because life history theory has been an understudied concept in the field of psychopathology, Del Giudice's target article is particularly welcome.

Our main critique of the theory is that it is perhaps overreaching and may not apply to every type of psychopathology. We would caution one basic premise in the target article, that is, the supposition that the field of evolutionary psychopathology (i.e., evolutionary psychiatry) is fragmented and requires a unifying principle. Similar to machines, organisms can break down in a variety of unrelated ways (Nesse, 2005). Whether an automobile is disabled by a flat tire or dirty spark plug requires no special theory to connect each type of failure. Similarly, there are many unrelated forms of renal disease (and their only tangible link is that they simply occur in the same organ). Renal dysfunction can be manifested by such disparate conditions as, for example, renal cell carcinoma, polycystic disease, and postinfectious glomerulonephritis. There is no compelling reason to search for a unifying theory to explain these varied kidney ailments. Similarly, the search for a unifying principle of psychopathology may be equally fruitless.

Another potential hitch is the possible misapplication of life history principles to modern psychiatric conditions—ailments that have no substantive evolutionary history. Eating disorders are perhaps one of the clearest examples. To our knowledge, the epidemic of classic eating disorders is mostly a modern Western phenomenon; such conditions do not appear to be associated with hunting-and-gathering societies. Therefore, the application of an evolutionary psychopathological theory to an eating disorder—a modern behavioral artifact—may be misplaced. Evolutionary forces will have certainly shaped the underlying neurocircuitry involved in the process of eating behaviors. However, such forces would not have shaped the actual behavioral features of an eating disorder. Instead, eating disorders are probably an accidental by-product of contemporary social problems interacting with the normal neurocircuits that support natural eating behaviors.

This is a general criticism that we have toward many evolutionary theories of psychopathology—there is frequently a failure to investigate the deviant behavior inside hunting-and-gathering societies. In our own research, we have always explored the evidence for each psychiatric ailment inside hunting-and-gathering societies, and we acknowledge that such evidence is often scant and inconclusive. However, our view is that some perfunctory attempt should always be made to establish the possible phylogenetic history of every psychiatric condition.

We also believe that life history theory is dubiously applied to schizophrenia, obsessive-compulsive disorder (OCD) and several subtypes of depression. For example, to tie life history theory to schizophrenia, Del Giudice (this issue) claims that "environmental insults" and "accumulated deleterious mutations" form the basis of schizophrenia (p. 276). However, we believe these are only secondary factors in the development of the condition. Instead, the preponderance of evidence shows that schizophrenia is a highly heritable condition, approaching 85% heritability in some calculations (Cardno et al., 1999). Moreover, the major candidate genes of schizophrenia are neither uncommon nor especially sinister (DTNBP1 [dysbindin], NRG1 [neuregulin 1], COMT, DISC1, RELN; Harrison & Weinberger, 2005). Research articles dealing with hypothesized schizophrenia genes often banter pathological terms (i.e., deleterious mutations), but this is simply because pathological genes are felt to exist and not due to any conclusive evidence.

For readers familiar with our own research, it will come as no surprise that we believe that the better model to explain the core phenotype of schizophrenia is to trace it back to shamanism (Polimeni, 2012, Polimeni & Reiss, 2002) and ancient religious prophets (Stevens & Price 2000). It is generally agreed that the most prominent phenotypic behaviors related to schizophrenia are delusions, hallucinations, and disordered thinking. However, it turns out that more than 90% of delusions and hallucinations are magico-religious (e.g., telepathy, curses, paranoid, spiritual)—an observation that has been conspicuously ignored by mainstream psychiatric researchers. We have therefore concluded that schizophrenia is a vestigial behavioral complex rooted in shamanism—the historical generators of magico-religious belief systems. Of note, we do not disagree with much of the research cited in the target article about the possible evolution of schizotypal traits (Nettle, 2006; Nettle & Clegg, 2006); however, we believe such research better supports the shamanistic theory of schizophrenia.

At first look, the application of life history strategies to obsessive-compulsive spectrum disorders seems to hold promise, but here too we have certain reservations. It is agreed that OCD probably represents some sort of "hazard-precaution system"—and in fact, we proposed a similar notion almost 10 years ago, contained in our group-selection evolutionary theory of OCD (Polimeni, Reiss, & Sareen, 2005). It is also true that OCD patients generally have a tendency to be methodical and cautious, and such hesitant behaviors could certainly be framed as postponing gratification to enhance investment in somatic development (i.e., a delayed reproductive strategy). Therefore, we agree that—at first sight—life history strategies could have some connection the OCD phenotype. However, under closer scrutiny, there are a number of nagging problems that we feel are not suitably highlighted in the target article.

Like schizophrenia, OCD spectrum disorders tend to have high heritability (Mathews et al., 2007; van Grootheest, Cath, Beekman, & Boomsma, 2005). Moreover, in our own clinical experience, OCD patients seem to have varied childhood life histories.

It is generally agreed that the common OCD symptoms are (a) checking, (b) washing, (c) counting, (d) needing to confess, (e) hoarding, and (f) requiring symmetry and precision (Rasmussen & Eisen, 1992). We have always found it compelling that the most common compulsions and obsessions have the potential of helping individuals, as well the tribe (Polimeni et al., 2005). Evolutionary forces are undoubtedly complex—and we believe that individual, kin, and group selection could conceivably all play a role in the presence of obsessive-compulsive behaviors. It is acknowledged that the worst extremes of OCD are probably maladaptive, as well as trichotillomania (and skin-picking), which appear to be extreme variants of normal grooming behaviors.

In our clinical experience, we have not noticed a fast spectrum category, containing low conscientious, impulsive OCD patients. In our view, the three referenced articles that claim prominent impulsivity in OCD are weak and not in accordance with common clinical experience (Ettelt et al., 2007; Smári, Bouranel, & Eiðsdóttir, 2008; Sulkowski et al., 2009). All three articles compare sets of self-reported scales of dubious validity. Actual impetuous behaviors such as sexual promiscuity, frequent fights or speeding would have been better measures of impulsivity. In our clinical experience, OCD patients tend to show little impulsivity, unless the rater counts the object of the compulsion as an impulsive behavior (which is misguided in our view). Ironically, a few days after writing the outline for this report, one commentator (JP) interviewed a patient with obsessive-compulsive personality disorder that surprisingly reported having received about 20 speeding tickets in his lifetime. However, it turned out that all of the tickets had been accrued during a period when he was delivering pizzas—and due to the patient's conscientiousness, he felt especially compelled to deliver the pizzas on time!

When it comes to depression, we agree that "depression is quite heterogenous" and that it "is only moderately heritable." Because *DSM -5* propagates an atheoretical, unitary view of depression, the analysis of potential etiologies has been woefully neglected. In our view, the great majority of depressive syndromes can be traced to five general etiologies: (a) hierarchal status conflict (e.g., financial, job loss), (b) attachment issues (e.g., marital separation), (c) endogenous depression (e.g., bipolar disorder), (d) organic brain syndromes (e.g., Parkinsonian depression, dementia, head injury), and (e) childhood trauma or neglect (e.g., borderline personality disorder, antisocial personality disorder).

The mood disorders field is inherently confusing because some presentations of depression were probably adaptive to our ancestors, whereas others were undoubtedly maladaptive. For example, many bouts of depression that nowadays present to the family physician's office seem to originate in hierarchal tensions (e.g., job loss, work conflicts, financial problems) or attachment issues (e.g., marital issues). In several articles dating back to 1967, John Price has methodically outlined depression's long phylogenetic history and its adaptive qualities within primate and hominid species (Price, 1967; Price, Sloman, Gardner, Gilbert, & Rohde, 1994). The "normal" expression of depressive behaviors may not be so adaptive in modern life (without threats of tribal ostracism); however, the great majority of depressive presentations appear to spring from a normal neurobehavioral apparatus.

Where life history theory seems exceptionally appropriate is in its potential application to borderline personality disorder and antisocial personality disorder (and perhaps other "Cluster B" conditions such as hysterical personality disorder and narcissistic personality disorder). Studies have shown that most borderline personality disorder patients have childhood histories consisting of psychosocial trauma and neglect (Bandelow et al., 2005; Winsper, Zanarini, & Wolke, 2012; Zanarini et al., 1997). It has always been perplexing why such patients should be so emotionally sensitive and impulsive, rather than inured to adverse social environments. The idea that an adverse environment could shift an organism to emphasize reproductive effort over somatic effort makes evolutionary sense. The intense emotional reactions (to perceived separation), aggressivity, and promiscuity often seen in borderline patients are all in accordance with a fast life history strategy. It is however possible that the self-mutilating behaviors characteristic of the most severe forms of borderline personality disorder could represent a transformation toward a maladaptive response (akin to pathologic feather picking in socially isolated psittacine birds; Jenkins, 2001) or those impulsive behaviors observed in Harlowe's monkeys (Harlow, Dodsworth, & Harlow, 1965).

In summary, while Marco Del Giurdice's life history theory fits well with Cluster B/externalizing spectrum disorders (and possibly Cluster C disorders) we suggest that the model does not as easily capture and codify schizophrenia, autism, obsessive-compulsive disorder, eating disorders, depression, and many other diagnostic categories not explicitly mentioned in his article. We look forward to greater synthesis and research in this area, some of which is described by Dr. Del Giurdice in his conclusions.

Note

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Psychological Inquiry, 25: 363–368, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.916191

Routledge
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Life History Theory in Psychopathology: More Than an Elegant Heuristic?

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The application of life history theory (LHT) to the social, behavioral, and psychological sciences has become increasingly popular and provides unique perspectives on human development and behavior (e.g. Ellis et al., 2012). Like others before him (e.g., Nesse & Stein, 2012; Williams & Nesse, 1991), Del Giudice (this issue) uses LHT as a lens through which to understand a broad range of human psychopathologies. Evaluation of his contribution should include not only the quality of what Del Giudice adds to current LHT conceptualizations of psychopathology but also what he leaves out. We believe that what has been left out in the target article constrains the potential impact of Del Giudice's contribution. In this introductory article on *evolutionary psychopathology*, Del Giudice does an elegant job of adding new perspective to psychopathologies that occupy the LHT spectrum. These articulations bring us to right up to prediction's door (see p. 261) but do not cross the threshold. This is important, given that the ability of a theory to make predictions is a core aspect of determining its value. The problem with the current work, elegant articulations aside, is that does not cast LHT as a predictive model, able to make novel predictions of evolutionary psychopathology. It is our position that what is needed in order to unleash LHT's potentially enormous implications for understanding psychopathology is a fundamental change in how LHT is applied, not only in the current context but in the broader literature as well. The needed change involves reconfiguring life history applications within an empirical framework that actively allows the investigation of both genetic and environmental causes of individual differences. Because life history research is inherently driven by theory, it is equally important that theoretical developments, such as Del Giudice (this issue), are oriented to encourage research questions that consider how genetic influences contribute, both alone and in concert with environments, to individual differences in LHT strategies.

Like the vast majority of human development and family theories, most operationalizations of life history theory assume environmental causality, generally attributed to conditional adaptation, when applied to human phenotypic development. It has long been a curious irony that in many empirical studies that use LHT, possible genetic contributions are generally ignored or are discussed only in passing. Although LHT "practitioners" may show greater appreciation for genetic associations (rGE) and Gene × Environment interactions (G×E), few empirical studies of LHT explicitly incorporate genetic components (although see Ellis, Schlomer, Tilley, & Butler, 2012). Indeed, many of the disorders Del Giudice describes as lying on the slow or fast end of the life history continuum show significant heritability. Although we agree that life history characteristics are not genetically fixed, life history strategies are genetically canalized to some degree. For example, under evolutionary selection humans as a species are on the slow end of the life history continuum. Life history applications to human development are essentially about determining *how slow* is one's strategy, bound by biological constraints (e.g., humans can't have litters). More important, however, is the notion that conditional adaptations that typify life history plasticity can be thought of as a G×E. Although specific mechanisms are not entirely clear (although see Essex et al., 2013; Meaney, 2010), humans (and other organisms) have evolved the genetic capacity to adjust developmental trajectories based on their social and physical environment. The notion that life history strategies manifest as a result of G×E has been discussed at length within the theoretical literature and we do not claim G×E has been neglected in the theoretical realm. Rather, the empirical literature in LHT could be more closely aligned with theoretical tenets by more directly integrating genetic components. Undoubtedly, LHT can be a useful heuristic for organizing disorders and disparate perspectives in psychopathology. The life history logic for psychopathology risk laid out by Del Giudice is indeed elegant. However, if LHT is going to go beyond providing evolutionary insights, no matter how elegant, of what we

already know about human psychopathology, LHT and the research done within this framework, must be restructured to call for examinations of genetic and G×E contributions to individual differences in LHT behaviors. Thus our major critique of this article is that Del Giudice does not go far enough with regard to life history implications for psychopathology. We suggest applying LHT to psychopathology can be more than a heuristic by extending beyond the limits of most developmental and family theories by more explicitly incorporating rGE and G×E. In doing so, LHT can be utilized as a genetically informed predictive model for psychopathic etiology. Our commentary centers on elaborating on these two points.

Life History Research Needs to Engage Heritability Directly

It has been more than 20 years since Belsky, Steinberg, and Draper (BSD; 1991) published their seminal paper that brought LHT to human developmental researchers. The BSD model provided the potential to causally explain links between early environments and child outcomes as a function of our evolutionary past. In addition, this publication introduced the notion that life history characteristics such as pubertal timing are the result of conditional adaptations to early environmental experiences. Much of the current research on life history strategies in human developmental research continues to be based on this general premise, that adverse early life events trigger responses from humans to adopt a "fast" strategy, organized around impulsivity, risk taking, early maturation, and immediate gratification rather than slower maturation, delayed gratification, and so on. Unfortunately there is another premise has also been carried forth, embodied by the following from the BSD publication: "In order to test this prediction, it will be necessary. . .to discount behavior genetic explanations" (p. 664). Clearly times have changed regarding this way of thinking (including B, S, and D; see also Del Giudice, Ellis, & Shirtcliff, 2011); however, lack of widespread engagement in heritability suggests its spirit persists. What Del Giudice has done should be applauded, like others before him, for adding functional understanding to risk for psychopathology. In particular, this work adds an important evolutionary view to our understanding of slow spectrum psychopathologies, and in doing so shines light on the often neglected slow end of the spectrum. However, as we argue is common in the life history literature, this work largely ignores the fact that where individuals fall on the slow–fast continuum is also strongly influenced by genetic variability.

Early Critiques of Conditional Adaptation

The issue of heritability (h^2) and LHT is not new. Soon after the publication of the BSD model, researchers began probing the validity of the assumption that fast strategy behaviors are adopted due to early life adversities. Early critiques of BSD's early life conditionality assumption was led by David Rowe and his colleague A. J. Figuerdo (see Rowe, 2002; Rowe, Vazsonyi, & Figueredo, 1997; see also Cleveland, Wiebe, van den Orrd, & Rowe, 2000). These critiques focused on the importance of considering whether life history traits, such as "fast" mating effort or risk behaviors were better explained by one of two competing mechanisms: conditional versus alternative mechanisms (Crawford & Anderson, 1989). As stated by Rowe et al. (1997), the conditional mechanism posits that individuals have *identical genetic propensities* for adopting fast strategies and where an individual falls on the fast–slow life history spectrum is causally linked to their environmental experiences. On the other hand, the alternative mechanism posits differences in life strategies are related to genetic variation across individuals. The weight of Rowe's research in this area, as well as the work that Figueredo has amassed since Rowe's earlier work (see Figueredo, Vásquez, Brumbach, & Schneider, 2004; Figueredo et al., 2006), provides clear support for the alternative strategy perspective.

One of the earliest studies by Rowe's research group that targeted the conditionality assumption underlying the BSD model examined whether differences in adolescent behavior problems across different family structures were due to genetic or environmental sources of variance (Cleveland et al., 2000). Rather than focusing primarily on LHT, the article hypothesis targeted the general assumption of environmental causality with regard to the association between family structures and behavioral problems, as it commonly existed in sociological and psychological studies. The article did note, however, that LHT shares the core assumption of causal environmental transmission. Mean level differences in latent factors commonly associated with behavioral genetics were estimated using additive genetics (A), shared environments (C), and nonshared environments (E) across four different family types. The four types of family structures examined were two-parent full-sibling families, two-parent half-sibling households, mother-only full-sibling families, and mother-only half-sibling families. The two family structures with the greatest mean level differences in adolescent behavior problems were two-parent full-sibling and mother-only half-sibling families, with the least and most behavior problems, respectively. The key finding of the study was that nearly all (81–94%) of the mean-level differences in adolescent behavior

problems between the two extreme family types were due to genetic influences. This finding supported the conclusion that the link between family structure and amount of adolescent behavioral problems was due to genetically based self-selection into family structures rather than environmental experiences conditionally triggering differences in behavior problems across these family structures. These findings, although primarily couched in terms of examining assumptions of family socialization theories more generally, were equally problematic for the early life conditionality assumption underlying the BSD model as well as current thinking in LHT research.

In 2002, Rowe more directly examined the BSD model using Add Health Data. If the underlying conditionality assumption of the BSD model was correct, twins within the same household would show similar correlations between menarcheal age and sexual onset age regardless of genetic similarity (i.e., whether the twin pairs were MZ or DZ twin pairs). However, the cross-trait cross-twin correlations between menarcheal age and sexual onset age (e.g., correlations between Twin1 menarcheal age and Twin2 sexual onset age) were significant for MZ pairs and similar to within-trait cross-twin associations (e.g., the correlation between Twin1 and Twin2 for sexual onset age) but not for DZ pairs. In fact, the genetic correlation—the similarity of the genetic influences—impacting the covariation between menarcheal age and sexual onset age was .72. A prior examination of the conditionality assumption, this time for mating effort, also provided more evidence for alternative rather than conditional strategies (see Rowe et al., 1997).

As Del Giudice (this issue) notes, many of the outcomes reviewed in his article show high heritabilities. This evidence, in light of previous work, should be reason for pause among life history researchers. It suggests that more empirical research needs to be focused toward a more deliberate understanding of how genetic influences contribute to life history strategies, and by extension evolutionary psychopathology. As previously noted, evidence regarding genetic influences on individuals' position on the fast–slow spectrum is substantial. This research alone provides basis to toss out the assumption that individuals have *identical* genetic life history biases characteristic of conditional thinking (see Rowe et al., 1997). Rather, there are likely individual differences in canalized life history strategy that are correlated with life-history-relevant environmental exposures. This genetic bias may also mitigate the influence of environments independent of rGE. For example, intervention efforts to reduce risk behavior characteristic of fast strategies may need to be stronger or more enduring for individuals with greater genetic propensity toward the fast end of the spectrum (see also Lykken, 1995). In addition, more recent research has indicated genetically based variability in environmental sensitivity. At the level of treatment for psychopathy, the degree to which symptoms are the result of life history traits gone awry, the relative influence of each of these factors could be critical for designing effective treatments.

Over a decade has passed since the initial critiques of conditionality were published. We view it as problematic that LHT and family socialization models continue to share similar underlying assumptions. LHT, by virtue of its evolutionary logic, is ideally suited to move past conventional family socialization models by more explicitly incorporating genetics within empirical research. These arguments should not be misconstrued to mean we see environmental influences on life history strategies as spurious or the product of rGE. Rather, our goal is to emphasize this continued similarity and encourage the reconfiguration of LHT so as to reduce this overlap. However, our view is that genetically based life history biases might be a better starting point than putative environmental effects when formulating hypotheses about psychopathic etiology, and life history strategies in general. Research on understanding the links between environments and LH traits needs to continue, but in a more genetically informed fashion. Although genetic biases in life history strategies have been mentioned elsewhere (e.g., Del Giudice et al., 2011), our opinion is life history empirical research would be well served if this issue were brought to the forefront.

G×E in Life History Research

The path toward a better understanding of gene–environment interplay within empirical research on life history can be drawn through novel G×E research. Indeed, much of the recent G×E research has been cast in a life history framework. Differential susceptibility theory (DST; Belksy & Pluess, 2009; Boyce & Ellis, 2005; Ellis, Essex, & Boyce, 2005; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011) provides an integrated approach to gene–environment causality, or at least the beginning of such. However, many G×E studies that invoke DST seem to do so in light of findings that show individual differences in environmental effects by genotype rather than the specific DST interaction form (see Reiss, Leve, & Neiderhiser, 2013). Nonetheless, despite being a relatively new endeavor, DST has shown substantial evidence that genes modify reactions to environments. Because many DST studies leverage the experimental and quasi-experimental designs, these findings are more convincing than many in behavioral sciences. Many of these studies, however, do not draw hypotheses from life history

theory per se. This is unfortunate, because better integration of life history theory in G×E research has the potential to mitigate nonreplication criticisms through better directing G×E hypotheses (e.g., Duncan & Keller, 2011). For example, considering the diversified bet-hedging approach to DST, within family variation in environmental sensitivity is the result of evolutionary processes that favored diversity in susceptibility (see Ellis et al., 2011, for a review). However, if sensitivity is hypothesized to be the adaptive result of environmental pressures over evolutionary time, it is expected that sensitivity would be specific to the pressure associated with its evolution, that is, life history relevant cues. The open issue regarding domain-specificity in sensitivity could be better addressed by using theoretically informed hypotheses from LHT.

Research within DST has demonstrated that there is much to be gained by empirically examining how genes and environments work together. In the vein of considering gene–environment transactions the two classic strategies or mechanisms of life history adaptation, conditional versus alternative strategies, are still relevant. However, ability to predict life history strategy, and by extension risk for psychopathology, may be determined by three important factors based in alternative and conditional strategies and environmental calibration or influence on these strategies: (a) genetic bias toward a life history strategy, (b) genetic susceptibility to the environment, and (c) environmental exposures. The ability for environmental exposures to entrain life history strategies may be conditional on the former, genetically linked individual characteristics. Of course, the picture becomes less clear when phenotypic forms of environmental susceptibility are also considered (see Ellis et al., 2011). However, we reiterate that evaluating genetic bias, as well as environmental susceptibility, may be the best place to start when determining life history strategy. Given the high heritabilities of many psychopathogies, predicting risk from LHT should start in genetics. Research on G×E provides such a starting point.

LHT: A Predictive Model for Psychopathology

Prevention/intervention studies provide a unique opportunity to study G×E hypotheses derived from life history theory. Such studies are well suited for LHT hypotheses for several reasons. First, prevention/intervention programs provide an environmental exposure that is unrelated to genetic propensities. By virtue of random assignment to intervention and control groups, exposure to the intervention is not confounded by rGE. As a result, developmental changes that are associated with intervention participation cannot be interpreted as the result of self-selection into a particular environment. Second, and extending from the first, randomized prevention/intervention designs permit causal interpretation of effects associated with intervention participation. When coupled with genetic information, causal hypotheses regarding both genetic and environmental influences can be tested. Third, randomized prevention/intervention trials provide more power to detect genetic and environmental main effects as well as G×E (see Brody et al., 2013; McClelland & Judd, 1993). Last, longitudinal prevention/intervention trials can further increase power through repeated measurements (Jaffee & Price, 2007).

At present, there are no studies published that describe a prevention/intervention trial designed to influence life history strategies specifically (and by extension risk for psychopathology). Such a study, however, would contain several components, all designed to influence life history etiology. First, the intervention should start early, during the child's first 5 to 7 years of life, and multiple levels of influence should be targeted. For example, the intervention would be designed to reduce, or at least minimize exposure to, environmental cues of elevated extrinsic morbidity/mortality such as witnessing or being victim to violence or exposure to neighborhood disorder. In addition, such an intervention might include components designed to increase parental investment, such as parent training or encouraging positive shared activities. Last, child-centered characteristics would also be considered, such as genetic liability for high-risk behavioral phenotypes or personality dimensions. A life-history-based intervention would be maximally effective when all three levels (i.e., environment, parent, child) are influenced, and both the parent and child phenotype are well matched to their environment. Last, an ideal prevention/intervention study would include a genetic component, designed to elucidate genetic underpinnings of life history traits so that genetically linked life history biases can be more explicitly incorporated. An epigenetic component would be useful as well, to examine longitudinal change and stability in methylation pattern and potential environmental influences on these patterns as they relate to life history characteristics.

This ideal design aside, there are some prevention/intervention studies that contain at least a few of these elements. For example, PROSPER is a community-based longitudinal project designed to study the impact of a partnership mode of delivering preventive interventions through a university-school-cooperative extension collaboration. PROSPER consists of 28 participating school districts in Iowa and Pennsylvania randomized into control and intervention conditions. During their seventh-grade year, adolescents in the intervention were delivered programs that target social

norms, personal goal setting, decision making, and peer group affiliation. Data collection began with the adolescents were approximately 11 to 12 years old and followed annually through the end of high school. More recently, genetic data were collected on a subset of PROSPER participants (i.e. gPROSPER). Initial genetic studies in PROSPER indicate the intervention was effective at reducing long-term change trajectories in externalizing behavior problems, particularly among genetically sensitive adolescents exposed to adverse home environments (Schlomer et al., 2013). If extreme life history strategies can be conceptualized as risk for psychopathology, then it would be expected high externalizing during adolescence would predict adult psychopathology. Of importance, if the intervention was effective at altering trajectories of life history strategies, predictions can be made regarding what ecological influences should be causally related to developmental psychopathology mediated by life history strategy. At an individual level, interventions could potentially be tailored to influence life history strategy, the multiple determinants of which could provide information about what environmental exposures would be needed, how strong they need to be, and perhaps how long they need to be implemented to affect life history strategies and subsequent risk for adult psychopathology.

Another set of designs still generally untapped by LHT researchers are adoption designs. Adoption research provides the opportunity to examine G×E processes in designs that can substantially reduce rGE confounds. And although there is a broad range of reasons why children are placed into the adoption systems, many of those reasons are related to the circumstances of the birthmother that may be correlated with fast LHT tendencies. Similarly, adoptive environments likely provide a good sampling of environments that support slow-LH strategies. In short, there are several designs researchers can use that provide information about genetic propensity for life history strategies. We encourage researchers to utilize such data that permit a more complete picture of life history environmental determinants.

Summary and Conclusion

We urge LHT researchers to consider heritability, or some version of genetic inheritance, within their research programs. Although genetic contributions are well recognized within the theoretical literature on LHT, empirical research has not followed suit. Early work in this area, as well more recent findings, provide strong evidence that continuing to either ignore or pay lip service to the reality that life history traits are strongly influenced by genetic underpinnings limits the potential of life history research to make an impact on behavioral sciences. Considering heritability does not mean being limited to performing behavioral genetic ACE (additive genetic, common environmental, unique environmental) decompositions. Going forward there is no reason that LH research cannot do better than other developmental and family theories that do not consider genetics as a complementary component. In addition to behavioral genetic research, studies that combine measured genetic influences, either single gene or multigene measures, with life-history-relevant measures of the environments and parenting would provide real insight into life history strategies. In this similar vein, etiology of evolutionary psychopathology would benefit greatly by providing more than cursory treatment of genetic contributions to life-history-mediated risk.

Note

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Psychological Inquiry, 25: 369–375, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.915709

Routledge
Taylor & Francis Group

Addressing Our Inner Salmon in an Evolutionary Framework for Psychopathology

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Life history theory is an elegant instrument for describing major differences in patterns of life history traits across plant and animal taxa (Charnov, 1993; Roff, 1992, 2002; Stearns, 1992). Typical life history traits discussed in the classic evolutionary biology literature include size at birth, growth pattern, age of sexual maturation, size at maturity, age of first reproduction, number and sex ratio of offspring produced, age- and size-specific reproductive investments, age- and size-specific mortality schedules, and length of lifespan (see Stearns, 1992). A basic assumption of the classic optimality approach to life history theory is that, given adequate genetic variation, the evolution of species has involved natural selection of optimal combinations of these traits. However, genetic and other constraints, and trade-offs have reduced the set of possible combinations. Life history theory predicts trade-offs between energetic investment in growth, maintenance, and reproduction across species, of which a trade-off between the main constituents of reproductive investment, mating and parental effort, may be the most common (McGlothlin, Jawor, & Ketterson, 2007). It is easy to see how if organisms possess finite resources that trade-offs affecting life history traits would necessarily evolve over evolutionary time. If the "pie of finite resources" is divided up between life history traits, taking a large slice of one type of trait leaves less of the pie to be divided into other forms of investment. Among vertebrate species, for example, salmon have very different life histories than primate species. Their life history consists of relatively rapid growth, early maturation and first reproduction, small size, little parental care, and the production of a high number of offspring, followed immediately by death in semelparous species, eclipsing a postreproductive period. In contrast, the life history of human beings consists of relatively slow development, late puberty and first reproduction, iteroparity, large body size, low number of offspring, followed by high parental investment (extended to grandparental investment) and a long life span, including a female postreproductive period.

Life history theory can be traced back to the beginning of the 20th century with the emergence of the idea that a life history is a set of adaptive traits associated in ways amenable to mathematical analysis (Stearns, 1976). Classic research in life history theory began with the construction and examination of stochastic mathematical models predicting expected species differences and trade-offs when the relevant life history parameters were varied or held constant. Such models have been tested empirically by studies determining the phenotypic or genetic relationships among life history traits across species or populations, by experimental manipulation of a trait to examine the effect on another trait or trade-off, and artificial selection studies (e.g., see Roff, 1992, 2002). The salmonid, by the way, turn out to be an excellent group on which to examine the assumptions of life history theory due to the large number of related species, considerable life history variation (e.g., semelparity vs. iteroparity), the existence of widespread populations inhabiting different geographical locations, and a repository of data available due to the species' commercial value (Hendry & Stearns, 2004). In contrast, the life history of our species, *Homo sapiens*, the only living hominin, can only be closely compared with the less than complete archaeological evidence of extinct members of the genus *Homo* or our closest extant primate relatives, such as the chimpanzee, gorilla, and orangutan (e.g., Robson & Wood, 2008). Human populations are also widespread, but with many migrations more recent, and modern conditions and culture impacting species-typical life history events and current selection pressures in unprecedented ways (see Stearns, Byars, Govindaraju, & Ewbank, 2010).

When the typical human life history emerged is not known, but fossilized dental evidence indicates that the life history of ancient members of the genus *Homo* was closer to that of African apes than to that of modern humans (C. Dean et al., 2001; M. C. Dean, 2006), suggesting a fairly recent appearance. An early *Homo sapien* fossilized juvenile found in Morocco dated to 160,000 years ago exhibited dental development equivalent to that of same-aged modern European children, but the earlier existence of a prolonged life history has not yet been documented (Smith et al., 2007). Directional selection on an ancestral hominin presumably produced the later maturity, lower offspring number, and high level of parental investment typical of modern humans. In modern subsistence or nonindustrial societies, taller women appear to have the advantage as greater height is

associated with lower infant mortality (Monden & Smits, 2009; Polette & Nettle, 2008; Sear, 2006; Sear, Allal, Mace, & Mcgregor, 2004). Because later menarche allows the achievement of greater height, larger size in early hominin women likely conferred a fitness advantage in the pleistocene environment of hunter-gatherers, leading to slower development and later reproduction. The exceptionally slow life history of humans, with sexual maturity not occurring until late in the second decade of life, appears to be part of an adaptive suite of traits, including a long period of juvenile learning, intense parental care, slow brain development, heightened encephalization, enhanced cognitive abilities, and long lifespan, distinguishing our species from other primates (Kaplan et al., 2007; Kaplan, Hill, Lancaster, & Hurtado, 2000; Potts, 2004; Russon & Begun, 2004). Presumably over the course of human evolutionary history the genetic architecture and developmental processes underlying these traits have become both fixed and linked, and we all embody variants of them today, compared to those versions of homologous genes contained within our distant cousins, the salmon.

Early life history theory included attempts to find a few overriding optimal combinations or solutions to the problem of partitioning energetic investments explaining the relationships among most life history traits across most species. At one time, species falling at two general endpoints of an expected continuum of life histories, for example, ranging from those with more salmonid features to those more typical of primates and longer lived animals, were referred to as *r*- and *K*-strategists, respectively (MacArthur & Wilson, 1967; Pianka, 1970). In this context "strategies" refers to the coevolution of all life history traits coming about by coordinated selection processes. The use of the terms *r*- and *K*- strategies and selection seemed useful descriptors but eventually fell out of favor in the biological sciences when this dichotomy was shown to have little useful explanatory value in understanding the complex selection forces operating on different species in varied ecologies and with different phylogenetic histories (Parry, 1981; Reznick, Bryant, & Bashey, 2002; Roff, 1992, 2002; Stearns, 1992). To the extent that the dichotomy of "slow" versus "fast" life history strategies employed in Del Giudice's (this issue) target article is synonymous with the *r*/*K* distinction (Del Giudice & Belsky, 2010) it is subject to the same limitations and long divorced from modern life history theory. Stearns, the primary founder of life history theory, prefaced a section in his seminal work of 1992 recommending the rejection of the *r*/*K* dichotomy as follows:

> In the 1960s and 1970s interest in life history evolution was stimulated by the identification of a dichotomy between species that matured early, had many small offspring, made a large reproductive effort, and died young and species that matured later, had a few large offspring, made a small reproductive effort, and lived a long time (MacArthur and Wilson 1967; Pianka 1970). The explanation was microevolutionary and based on differences in mode of population regulation. It argued that short-lived species with high reproductive rates had evolved under density independent conditions and called them '*r*-selected', implying that such circumstances selected for a high intrinsic rate of increase (*r*). Long-lived species with low reproductive rates were thought to have evolved under density-dependent conditions and were called '*K*-selected', implying that such circumstances selected for ability to withstand high densities of conspecifics (*K* represents saturation density).
>
> This explanation was suggestive and influential but incorrect. First, it was couched at the level of population regulation rather than demographic mechanism and confused the statistical description of population processes with the selection pressures that act on individual organisms. (p. 206)

Likewise Roff (1992), a cofounder of life history theory, summarized the problem with this dichotomy:

> To summarize, the concept of *r*- and *K*-selection has been useful in helping to formalize the definition of fitness in density-regulated populations, but attempts to transfer the concept to actual populations without regard to the realities of the complexities in life history have probably been detrimental rather than helpful. The terms *r*- and *K*-selection should be interpreted strictly in terms of models of density dependence (Boyce 1984; Elgar and Catterall 1989), and given the confusion that now surrounds the issue, it may be preferable to avoid use of the terms altogether. (p. 46)

Stearns outlined five further reasons for not employing the *r*/*K* dichotomy, including that its use in classifying life histories fails for about 50% of species for which reliable data exist (Stearns, 1977; Wilbur, Tinkle, & Collins, 1974). Although *r*/*K* selection theory presumably describes broad trends in differences in the life histories of different taxa, there are many exceptions to these trends. For example, one of the longest lived species on the earth, the giant Redwood tree (*Sequoia*), develops quickly, produces millions of seeds over its lifetime, and can live for millennia (Stephenson, 2000). Among animal species, the tiny cave-dwelling salamander or "human fish," *Proteus anguinus*, is estimated to live a century and achieves reproductive maturity at an age similar to humans (Voituron, De Fraipont, Issartel, Guillaume, & Clobert, 2011). Some turtle and tortoise species (e.g., the Galapagos tortoise) have been known to have exceptionally long lives lasting close to two

centuries, yet they exhibit high reproductive effort (Kirkwood, 1985). Even within one subset of species of one class, the mammalia, the covariation of life history traits is not unidimensional (Bielby et al., 2007).

Furthermore, just what selection processes were producing *r*- versus *K*-selected species was a bit of a mystery, and this topic was barely addressed by early proponents, including MacArthur and Wilson (1967; see Reznick et al., 2002). One of the issues, however, that the debate over *r/K* selection highlighted is that, aside from the major effects of extrinsic age-specific risk of mortality and the predictability of temporal and spatial environmental variation, classic life history theory per se did not, nor did it intend to, predict the particular selective forces or ecological factors shaping the life histories of individual species (see Stearns, 1992).

Moreover, predictions about the effect of extrinsic mortality (defined as environmental causes of death such as disease, conflict, accidents, and predation) and temporal or spatial environmental heterogeneity (e.g., seasonal changes, food patchily distributed) turned out to be somewhat variable. For example, high levels of extrinsic adult mortality and environmental heterogeneity were originally suggested to predict an early reproduction, rapid senescence, and short life span, whereas high rates of juvenile extrinsic mortality predicted the reverse (Stearns, 1992). With regard to rate of maturation, this is because in a population with high adult mortality, those genotypes producing individuals that reproduce later will be selected against in favor of genotypes reproducing earlier, before they have the chance to succumb to environmental fatalities. However, subsequent models have shown these relationships are far more complex than originally outlined. For example, if extrinsic mortality is not random but condition-dependent, affecting some age groups or individuals differentially, then this can reverse the classic prediction (Abrams, 2004; Chen & Maklakov, 2012; Reznick, Bryant, Roff, Ghalambor, & Ghalambor, 2004; Shokhirev & Johnson, 2014; Williams, Day, Fletcher, & Rowe, 2006). Traits that allow individuals to avoid disease, predation, conflict, or accidents would be selected, and in turn increase longevity and reduce the benefits of early reproduction. Human behavior, cognitive processes, and sociality are potentially such traits. Long-lived large bodied species have the ability to conditionally alter their allocation of energy and behavior over ontogeny to withstand and survive changes in their environment. In addition, maturation rate and lifespan may become uncoupled if, for example, high extrinsic adult mortality increases the resources available to surviving individuals, producing both faster growth and reduced senescence as a result (Reznick et al. 2002). Any comprehensive model of human development and psychology derived from life history theory would need to incorporate or consider these additional nuances of the theory.

Although classic life history theory focused on the stochastic mathematical relationships between life history traits in consideration of only a few key environmental factors, this does not mean the particular ecological circumstances of individual species are not important. Different species have varying phylogenetic histories and inhabit different and complex ecologies where competing selection pressures mold life histories. In the end, the tests of life history theory are only as good as they capture the relevant parameters for each individual species. Unidimensional models cannot be expected to capture all the crucial differences in multidimensional selection histories. Early on Stearns (1976) recognized that a multitude of environmental factors would need to be taken into account or controlled in testing models of the evolution of the life history of a specific species including food availability, temperature, competitors, and predators. Over time life history analyses have become increasingly supplemented with functional and ecological approaches to help identify how a particular species came to exhibit the unique pattern of life history traits and trade-offs currently observed.

If *r/K*-like dichotomies are no longer used to explain broad differences across species their value in explaining within species differences in life history traits and human psychological traits, such as racial differences in intelligence, is likewise problematic (e.g., Graves, 2002a, 2002b). In addition, whether mainstream life history theory can be extended directly to account for individual differences among members of the same species has always been a somewhat debatable point (see Stearns, 1992). Past selection pressures that shaped the life history of a species may or may not play a concurrent or the same role in presently observed interindividual variation in life history traits or trade-offs. Furthermore, correlations observed between environmental conditions and life history traits do not demonstrate that these factors have actually played a role in the evolution of the traits. The reconciliation of individual selection models with species-level selection processes in shaping life histories is long overdue and would provide a useful framework for successfully integrating life history theory into developmental psychology and psychopathology. Initially life history theory devoted less attention to the relevance or causes of interindividual variation on life history traits, other than generally attributing it to phenotypic plasticity (Roff, 1992; Stearns, 1992). But the considerable phenotypic, and presumably genotypic, variation in life history traits observed within all species surely begs

further explanation. If some salmon exhibit more primate-like life histories (e.g., iteroparity, longer life span) and some humans exhibit life histories in the salmonid direction (e.g., faster maturation, high reproductive effort), what are we to make of or attribute these differences to? How do we explain this variation, and can we consider individual departures from a species' typical life history pattern just noise or is it meaningful?

Life history traits and trade-offs are heritable, although generally their heritabilities are lower than those for other traits (see Roff, 2002; Stearns, 1992). Trade-offs are classically indicated by negative genetic correlations in response to selection, and typically negative phenotypic correlations between traits (Roff, 1992, 2002), but extrinsic or intrinsic factors can readily obscure the latter. For example, a trade-off between growth and reproduction in humans would typically produce a negative genetic correlation between body size and number of offspring, but exposure to early disease or prohibitions against early marriage could obliterate this correlation at the level of the phenotype. This means that in any given human population at any given time it may not be possible to observe the evolved trade-off typical of the species. Thus when considering variation in life history traits and their trade-offs within a species we must distinguish between genetic and phenotypic variance and consider the potential sources of each, including novel environmental influences.

There are a least five potential sources of genetic variance in life history traits within a population: (a) *mutation selection balance*, (b) *heterosis*, (c) *antagonistic pleiotropy*, (d) *frequency dependence*, and (e) *environmental heterogeneity* (see Roff, 1992, 2002, and Stearns, 1992, for good descriptions of these sources). If genetic variation in human life history traits can be attributed to one or a combination of these factors, then phenotypic variance may be a result of (a) *noise or random variance* due to environmental or genetic variance with neutral effects on selection; (b) *adaptive plasticity*, whereby a given genotype maintains fitness by producing different phenotypes in different environments (referred to as a reaction norm) or conditions (referred to as conditional strategies); (c) *maladaptive plasticity*, where a phenotype is pushed outside of the species-typical reaction norm by extrinsic or intrinsic factors with negative fitness consequences; or (d) *alternative life history strategies*, whereby two or more distinct phenotypes or *morphs* are produced by different genotypes maintained by frequency dependent selection (see Roff, 1992; Stearns, 1989, 1992; West-Eberhard, 2003).

Alternative life history strategies are exhibited across and a property of different species, but more rarely observed or observable within species (Stearns, 1992). Alternative life history strategies are usually associated with distinct genotypes and morphs, rather than with traits exhibiting continuous variation across individuals. Typically, alternative life history strategies within a species or population are evidenced by bi- (or tri-) modality in a trait or phenotype brought about by disruptive selection. A classic example involves the distinct morphs found in male bluegill sunfish, whereby genotypically small males engage in sneak fertilizations and large males engage in territorial defense and parental investment as alternative means of maximizing fitness (Gross & Charnov, 1980). Most human traits vary in a continuous fashion, which tends to argue against the notion of evolved alternative strategies per se in favor of multiple genetic and environmental influences in the production of the phenotype. However, there may be some possible exceptions. Migliano,Vinicus, and Lahr (2007) suggested that pygmies may have diverged in height and weight from other human populations because of the fitness advantage of earlier growth cessation and earlier reproduction in ancestral environmental conditions of high mortality. Becker, Verdu, Hewlett, and Pavard (2010), however, noted problems with the sampling technique, threshold employed to define the populations, and mathematics of the model, underscoring the problems of constructing accurate life history models.

Adaptive phenotypic plasticity in life history traits likely underlies much of the individual differences in life history traits seen within species (Roff, 2002; Stearns, 1989; West-Eberhard, 1989, 2003). The ability of a genotype to adjust development adaptively to different environmental conditions has evolved to produce a reaction norm that calibrates the expressed phenotype with environmental conditions. In a sense, phenotypic plasticity uncouples genotypes from selection processes, reducing genetic evolution although environments may change or vary (Stearns, 1982, 1989). However, plasticity can also serve to further enhance natural selection through processes whereby selection on beneficial phenotypes drives the evolution of genotypes in a concurrently advantageous direction (see Stearns, 1989; West-Eberhard, 2003, for discussion of these processes). At the same time any correlated behaviors or psychological processes may mitigate or intensify selection pressures on life history traits. For example, migration may have evolved as a means to overcome environmental variation, in turn lessening its selective effects on life history traits (Roff, 2002).

Over a species' evolution, life history traits may become genetically correlated with morphological, physiological, and behavioral (including psychological) traits (Roff, 1992). For example, genes underlying rate of development may be the same or linked to those producing morphological traits, such as adult

size or dimensions, through allometric processes resulting in later maturers generally achieving greater adult height (e.g., Tanner, 1962). Life history traits involving reproductive investments have likely become linked to endocrinological systems, including the androgenergic system involved in the trade-off between mating and parenting effort in males (McGlothlin et al., 2007). Likewise genes underlying a trade-off favoring future over current reproduction may become linked to risk-averse behaviors or personality traits (Wolf, van Doorn, Leimar, & Weissing, 2007). Although the author of the target article indicates that there is not necessarily a causal relationship between life history traits and mental disorders, they are apparently correlated. The implication is that through the course of human evolution the genetic complexes underlying life history traits or trade-offs have had pleiotropic effects or become linked to those involved in the neurobiological pathways leading to an increased risk of psychopathology. The problematic employment of a discarded unidimensional concept aside, this suggestion is compatible with a life history framework. However, what specific selection pressures may have resulted in genetic correlations between life history traits and risk factors for a good number of psychological disorders differentiated by sex are not readily discernable in the proposed model. They could well exist, but we need more evidence, potential mechanistic pathways, or at least plausible candidates to chase up.

Del Giudice states that his goal in presenting the slow–fast life history framework is not to replace other functional explanations of mental disorders but to provide a higher organizing principle. However, at this point in time, it may be more profitable to focus on the functional level and build up to a more general framework (if one exists) that would be compatible both with midlevel functional theories and the modern subtleties of life history theory. My intention here is constructive, as it is exciting to see the inroads evolutionary and functional analyses of human personality traits and psychopathology have made into traditionally psychological topics over the last few decades, as the target article masterfully reveals. Having drawn upon functional perspectives in considering conditions, such as anorexia nervosa and depression (Surbey, 1987, 2011), I am predisposed to them and have a bit of a stake in the future of evolutionary approaches. Also, my early reports of relationships among early menarche, childhood stress, and father absence (Surbey, 1990, 1998) situated the phenomena within a general life historical perspective while maintaining a pluralistic approach in considering both past selection pressures and current environmental circumstances producing such phenotypic relationships and variation. Therefore, the recognized marriage of applicable evolutionary perspectives, including modern life history theory, with the traditional field of human psychopathology is indeed an event I hope to attend.

The search for higher level theoretical approaches and organizing principles has and always will be the goal and mainstay of evolutionary biologists and psychologists alike. However, when phenomena may be explained at lower levels (e.g., by phenotypic plasticity) applying higher level explanations may not be parsimonious, even if they appear elegantly simpler and intuitively satisfying. An analogous situation would be the application of group selection in explaining the evolution of a trait, such as altruism, when an explanation at the individual level of selection does the trick. And if that higher level of explanation (e.g., Wynne-Edwards' [1962] early model of group selection) may be flawed, it is even better to hold off until a better overarching model can be formulated. Although species and individuals clearly do differ in tempo of development, a fast–slow continuum probably does not represent a wholesale *explanatory* dimension of individual differences any more than a "small–large" or even "feminine–masculine" dimension might, both of which could likely be substituted with little change in the proposed model. Any such continuum represents just one dimension in a multidimensional selection landscape shaping human biology and psychology. Moreover, that the majority of disorders discussed by Del Giudice are classified as heterogeneous in terms of the slow/fast spectrum suggests that this dichotomy is not the far-reaching explanatory heuristic sought.

There is some value in maintaining a diverse, albeit seemingly fragmented approach (Kennair, 2003, 2011), in considering the evolutionary processes and selective agents involved in the production and maintenance of psychopathology in human populations, as these are complex disorders in a species with a complex evolutionary history. Evolutionary theories and concepts, such as intragenomic conflict, sexual selection, frequency dependence, antagonistic pleiotropy, modularity, parent-offspring conflict, and intergenerational effects, promise some utility or have already begun to make advances in viewing psychopathology through an evolutionary lens (e.g., see Baron-Cohen, 1995; Baron-Cohen, Knickmeyer, & Belmonte, 2005; Crespi & Badcock, 2008; Del Giudice, Angeleri, Brizio, & Elena, 2010; Haig & Wharton, 2003; Keller & Miller, 2006; Martel, 2013; Nettle & Clegg, 2006; Shaner, Miller, & Mintz, 2004, and other examples in the target article). Although grand theories and a high-level deductive approach are the long-term goal of the evolutionary-minded, in the meantime, addressing our inner salmon alongside the complex etiology of human psychopathologies may require a seemingly more plodding midlevel evolutionary approach.

Note

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Psychological Inquiry, 25: 376–384, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.916194

Routledge
Taylor & Francis Group

Slow Life History Strategies and Slow Updating of Internal Models: The Examples of Conscientiousness and Obsessive-Compulsive Disorder

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Skillfully interweaving work from various disciplines, Del Giudice (this issue) suggests dimensions of personality and psychopathology that reflect, as I interpret it, underlying behavioral/physiological programs. These programs may take the form of systems of integrated behavioral and physiological control that each evolved to optimize behavior and physiology in specific environmental and situational conditions (e.g., Del Giudice, Ellis, & Shirtcliff, 2011; Ellis, Figueredo, Brumbach, & Schlomer, 2009; Mehrabian, 1995; Tops, Boksem, Luu, & Tucker, 2010; cf. Block, 2002). Such conditions may vary on parameters such as predictability, stability, harshness and levels of resources. The functions of those systems explain properties of the disorders and how the disorders relate to personality and each other.

More specifically, Del Giudice reviews evidence that mental disorders can be categorized at either end of a continuum ranging from slow life history strategies (e.g., late maturation and reproduction, high parenting effort, low risk taking) to fast life history strategies (e.g., early maturation and reproduction, low parenting effort, high risk taking). The slow and fast life history strategies are optimal in terms of reproductive fitness in predictable and stable, versus unpredictable or unstable environments, respectively. Del Giudice discusses evidence that the two types of life history strategies and the associated mental disorders can be discriminated on the basis of personality differences. For example, high Conscientiousness relates to slow life history strategies. I agree with the broad outline of the author's thinking, and in this commentary I link this life history framework for psychopathology to a neurobiological mechanism.

In his introduction, Del Giudice states that "although 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" (p. 262). The aim of the fast–slow framework is to capture the broadest and most general level of this explanatory hierarchy, helping to connect other explanations to one another, and ultimately integrate them within a common frame of reference. To facilitate the connecting of different level of explanation, in this commentary I present a neurobiological mechanism behind a fundamental dimension of individual differences that may relate to the fast–slow continuum. I do this in terms of the theory of Predictive And Reactive Control Systems (PARCS; Tops et al., 2010; Tops, Boksem, Quirin, IJzerman, & Koole, 2014). Specifically, I argue that psychopathology can arise at both ends of a continuum reflecting the degree to which novel and salient information is processed and forwarded through corticostriatal loops by a right hemisphere control system and the degree to which internal models are updated in light of novel evidence by left hemisphere control. Focusing on what PARCS can add to the framework in the target article, I intend to show that the evolutionary life history framework described by Del Giudice and the PARCS framework supplement each other and synergistically increase explanation and connection at different levels.

PARCS

PARCS suggests that reactive control systems evolved early in evolutionary history for the purpose of behavioral control in unpredictable environments. This system is composed of lateral limbic system structures such as the ventral striatum, anterior hippocampal formation, and amygdala, as well as ventrolateral cortical structures such as the inferior frontal gyrus (IFG), and anterior insula (AI). This system is thought to specialize in the processing of novelty and biological salience in order to control behavior in unpredictable as well as in urgent and emergency situations. It functions in a feedback-guided manner to the immediate situation and focuses attention narrowly on the local situation.

Predictive control systems, alternatively, are comprised of dorsomedial structures such as the posterior cingulate cortex, precuneous, angular gyrus, parahippocampal cortex, posterior hippocampal formation, medial prefrontal cortex, and dorsolateral prefrontal cortex. It is believed that this network of systems is largely an outgrowth of evolutionary pressures that emerged in highly predictable and stable environments (Tops et al., 2010; Tops, Boksem, et al., 2014). PARCS suggests that the dorsal predictive

system function is to run simulations to predict future events. Craik (1943) suggested that imagining the future using internal models allows for testing alternative possibilities, and making better predictions regarding situational outcomes. In the same sense, the dorsal predictive system engages in creating internal models that predict future outcomes through simulation, and updates those models slowly, in line with the idea that it responds to environmental predictability.

The right IFG is involved in appraisal and conditioning and detects novel, unpredicted, and salient stimuli that require elaboration or scrutiny from reactive control. The left IFG takes control when elaboration or scrutiny is needed to ensure consistency of new information with internal models, which may lead to ruminative processing. In this manner it can take new information and communicate with predictive systems to update internal predictive models promoting greater predictive control in the future (Tops, Boksem, et al., 2014). This function involves the verbalization and semantization functions in the left IFG and relates to the "left brain interpreter" that was proposed to explain findings in split-brain patients. A left brain interpreter refers to the construction of explanations (in terms of internal models) by the left brain in order to make sense of the world by reconciling novel information with what was known before (Gazzaniga, 2000). Similarly, a right hemisphere mechanism for anomaly or novelty detection has been proposed, versus a left brain mechanism for maintaining our current beliefs (internal models) about the world (Ramachandran, 1995). Finally, according to a model by Perlovsky (Perlovsky & Ilin, 2013) the language semantic area in the left IFG guides the development of internal models using information and restrictions from culture and collective wisdom that have accumulated in language.

Important dimensions of individual differences in personality and susceptibility to specific mental disorders appear to relate to the degree to which novel and salient information is processed and the degree to which internal models are updated in light of the novel evidence. The associations in the target article between unpredictable or harsh environments, personality and psychopathology involving fast strategies are relatively intuitive, whereas the association between predictable and safe environments and personality (Conscientiousness) and psychopathology involving slow strategies is surprising (but well explained). In this commentary, I focus on the latter associations to illustrate how reactive and predictive controls are important for understanding personality traits and their association with mental disorders on the basis of individual differences in the degree of processing of novel information and updating of internal models.

Conscientiousness and Adaptation to Environmental Predictability

To better understand the Conscientiousness trait, I first discuss basic animal traits that I think Conscientiousness in humans evolved from. Reactive and predictive controls can be related to basic personality differences that are found in various animal species. Reflecting underlying predictive and reactive control systems, there is evidence that the basic traits evolved to be adaptive in predictable and stable, and in unpredictable or changing environments or circumstances, respectively.

A fundamental personality difference seems to be the degree in which behavior is guided reactively by environmental stimuli (Benus, Den Daas, Koolhaas, & Van Oortmerssen, 1990). Aggressive animals easily develop routines (i.e., a rather intrinsically driven rigid type of behavior) and show reduced impulse control (behavioral inhibition) in operant conditioning paradigms. Nonaggressive animals in contrast are more flexible and react to environmental stimuli all the time, that is, they show larger cue dependency and conditioned immobility. For that reason, Koolhaas et al. (1999) suggested the terms *proactive coping* and *reactive coping*. Studies of animals in feral populations indicate that the proactive and the reactive coping style represent fundamental biological trait characteristics that can be observed in many species. These coping styles play a role in the population ecology of the species. The optimal proportion of each temperament in a population changes with the predictability and stability of the environment. The reactive and proactive traits developed during evolution because they are adaptive in unpredictable or changing environments and predictable and stable environments, respectively. Their differential degree of flexibility may explain why proactive animals are more successful under stable colony conditions, whereas reactive animals do better in a variable or unpredictable environment, for example, during migration (see Koolhaas et al., 1999).

The aggressive proactive trait does not necessarily constitute the most adaptive trait in predictable and stable human societies. I argue that human evolution may have favored the development of a Conscientiousness personality strategy from the proactive personality. The rigid and aggressive predictive control of the proactive personality may have evolved into, or may have been supplemented by, a variant that exploits the advantages of collaboration and of moral and authority rule structures to protect obtained (in-group) resources and against aggressive competition. Predictability enables long-term investments if those investments can be protected from aggression and other threats. This idea seems compatible with the suggestion by Del Giudice that Conscientiousness-related disorders are sensitive

to potential threats in favorable environments. Conscientiousness combines proactive personality aspects such as competitiveness and rigidity with social constraint. In the trait of social constraint, the feedback-guided elaborative control of the left reactive system appears to keep tight control over impulses from the simultaneously active predictive system. This control is associated with low transfer of novel information from the right to the left hemisphere and consequently slow revision of internal models by new information (cf. Tucker, Luu, & Pribram, 1995). This way, internal models of social and moral rules are kept stable.

In PARCS, Conscientiousness reflects a mixture of reactive and predictive control in the form of reactive feedback-guided control over feedforward aggressive impulses. Hence, Conscientiousness combines reward seeking with constraint from social values. Conscientiousness has relationships with impulse control, self-discipline, reappraisal coping, problem solving, dutifulness, conservatism, traditionalism/conventionality, religious fundamentalism, moralistic, rigidity, intolerance of unpredictability and ambiguity, and effortful control of behavior in the service of long-range goals (Carver & Connor-Smith, 2010; Connor-Smith & Flachsbart, 2007; De Fruyt, McCrae, Szirmák, & Nagy, 2004; Hirsh, DeYoung, Xu, & Peterson, 2010; Koenig & Bouchard, 2006; MacDonald, 2008; Roberts, Chernyshenko, Stark, & Goldberg, 2005; Segerstrom, 2005; Tellegen, 1985). For some of those correlates, such as conservatism, traditionalism, conventionality and rigidity, it is easy to see that they involve slow updating of internal models. Reappraisal coping may also function to align novel experience with internal models, decreasing pressure to update models. In this commentary, I discuss the relationships of slow updating with impulse control, morality, disgust sensitivity and intolerance of unpredictability.

In the target article, Del Giudice makes the point that Conscientiousness is related to a slow-spectrum OCD group that shows contamination/cleaning symptoms, obsessive-compulsive personality disorder features, predominance of females, guilt, shame and disgust sensitivity and moral concern, worry, intolerance of uncertainty, and need for predictability (Hummelen, Wilberg, Pedersen, & Karterud, 2008) and comorbidity with grooming disorders, panic disorder, and tics, as well as with an overlapping restrictive anorexia group. OCD and associated perfectionistic traits are further characterized by attempts to monitor closely and take control over processes that would otherwise operate in proactive, feedforward ways (Tops & Wijers, 2012).

Relationships have been reported between Conscientiousness and left cerebral hemisphere or IFG/AI activation (see Tops & Boksem, 2010). Similarly, in two studies, trait social norm compliance/traditionalism correlated during affective tasks with activation in the left IFG/AI (Brown, Acevedo, & Fisher, 2013). In anorectic individuals, high-calorie food stimuli activated left IFG (BA47/11) proportional to scores of restraint and cognitive control (Rothemund et al., 2011). In the next sections I show that features of Conscientiousness-related OCD can be interpreted as concerns about in-group stability or inclusion, constraint of proactive and aggressive impulses that may threaten in-group stability and inclusion, and strategies to limit the intake of novelty and the inclusion of the foreign. Those features and strategies tend to be associated with left IFG/AI activation.

Moral and Social Inclusion or Stability Concerns

The left IFG takes control when elaboration or scrutiny is needed to ensure consistency of new information with internal models, which may lead to ruminative processing of moral and social rule concerns and feelings of guilt and shame (see Tops, Boksem, et al., 2014). Induction of embarrassment, indignation/anger, shame, and guilt activated left IFG (BA 47 or 45; Michl et al., 2014; Shin et al., 2000; Spence, Kaylor-Hughes, Farrow, & Wilkinson, 2008; Takahashi et al., 2004; Wagner, N'Diaye, Ethofer, & Vuilleumier, 2011; Zahn et al., 2009). Similarly, processing of transgressions of social norms or social deception activated left IFG (BA 47; Berthoz, Armony, Blair, & Dolan, 2002; Lissek et al., 2008). Shame and embarrassment and left IFG (BA 47) activation were larger for social transgression with audience compared to unwitnessed (Finger, Marsh, Kamel, Mitchell, & Blair, 2006). Across three studies employing different paradigms, the processing of negative morally laden stimuli was found to be highly left-lateralized (Cope et al., 2010). Regions of engagement common to the three studies showed coactivation of the left IFG (especially BA 47), temporoparietal junction, and dorsal system areas. Left IFG activity was especially prominent in the study comparing controversial morally wrong to noncontroversial morally wrong, a condition that may require elaborate processing of moral and social dilemmas. Finally, left IFG (BA 47) was active together with medial prefrontal cortex, left temporal pole, and left thalamus during a judgmental task for appropriateness of facial affect compared to a gender matching task (Kim et al., 2005).

In a classical study, subjects were injected with sodium amobarbital into the right and left carotid arteries for neurosurgical purposes and were asked to recount verbally an emotional life event before and after the injection (Ross, Homan, & Buck, 1994). Following left-sided inactivation, subjects mentioned

"basic emotions" (i.e., appraisal-related emotions such as feeling afraid). Following right-sided inactivation, subjects' emotional stories were factually the same as before the injection, but they contained significantly more social content. According to the authors, when only left hemisphere functionality was intact, subjects retrieved "social emotions" associated with the memory to the exclusion of basic emotions. Those social emotions appeared to reflect concerns about moral or social appropriateness, desirability, and inclusion (e.g., feeling "stupid," "silly," "sorry," "embarrassed," "unaccepted"). The results by Ross et al. show right hemisphere involvement in basic emotional appraisal versus left hemisphere involvement in reappraisal of emotions in terms of social and moral internal models.

Studies in which information was presented to one hemisphere at the time produced related results. A series of studies demonstrated person-based learning in the right hemisphere and group-based learning and in-group favoritism effects only in the left hemisphere (Sanders, McClure, & Zárate, 2004; Zárate, Sanders, & Garza, 2000; Zárate, Stoever, MacLin, & Arms-Chavez, 2008). Another study found that endorsing likable personality items profited from presentation to the left hemisphere, whereas rejecting unlikable items profited from presentation to the right hemisphere (Marsolek, DeYoung, Domansky, & Deason, 2013). Similarly, in an fMRI study, desirable information activated left IFG but undesirable information activated right IFG (Sharot, Korn, & Dolan, 2011).

Converging evidence pointing to a role of left hemisphere frontal activation in social desirability has been found in studies of frontal activation asymmetry in EEG experiments. The trait social desirability is a positive correlate of Conscientiousness that reflects motivation to present oneself in a favorable manner and to actively avoid social disapproval through conformity (Crowne, 1979). More recently evidence has been summarized that scales measuring social desirability should be redefined as measures of interpersonally oriented self-control that identify individuals who demonstrate high levels of self-control, especially in social contexts (Uziel, 2010). Trait social desirability is associated with relative left frontal activation asymmetry (e.g., Kline, Blackhart, & Joiner, 2002; Pauls, Wacker, & Crost, 2005; Tomarken & Davidson, 1994). Moreover, this association is stronger or specifically found when social evaluative concerns are triggered, such as when subjects are tested by opposite-sex experimenters compared to same-sex experimenters (Kline et al., 2002) and when presented with personality feedback in the presence of an opposite-sex confederate compared to privately (Crost, Pauls, & Wacker, 2008). The evidence appears consistent with the proposal that Conscientiousness and left IFG/AI are implicated in processing socio-moral information to constrain proactive impulses.

Slow Updating by Keeping the Novel Out: Disgust

Disgust is tied to aversion and fear of novel stimuli ("neophobia"), related to a function to protect against infection and food poisoning by avoiding unfamiliar stimuli that are not known to be safe (Nordin, Broman, Garvill, & Nyroos, 2004). As disease is often spread between individuals, social types of disgust evolved that help protect the in-group from out-group threats. Moreover, Conscientiousness appears associated with types of disgust that help maintain the protective structure of the in-group by controlling aggressive impulses.

Disgust and disgust-based emotions are also importantly involved in OCD, eating disorders, depression, social phobia, and the sexual disorders. Power and Dalgleish (1997) suggested that there is a subgroup of obsessional patients whose problems are not related to anxiety but that may be disgust based. In healthy volunteers, disgust sensitivity was found to be related to symptoms of agoraphobia and OCD (Muris et al., 2000). Similarly, Ware, Jain, Burgess, and Davey (1994) found significant correlations between disgust sensitivity, "fear" ratings toward revulsion animals, and the Washing subscale of the Maudsley Obsessive-Compulsive Inventory. In another study, washing and checking behaviors were better predicted by disgust than by anxiety or depression (Mancini, Gragnani, & D'Olimpio, 2001).

Based on semantic analyses of emotion terms, Power and Dalgleish (1997) and Johnson-Laird and Oatley (1989) concluded that the complex emotions of shame, guilt, contempt, and loathing, together with some forms of embarrassment, are derived from the basic emotion of disgust. Power and Dalgleish (1997) derived emotions such as hatred, contempt, and loathing from a combination of disgust and anger and derived embarrassment, especially as shame, from a combination of primarily disgust and fear.

Disgust sensitivity appears to have an important role in the control of anger and moral value in individuals who are high on Conscientiousness. Disgust sensitivity predicts lower levels of trait, behavioral, and daily aggression (Pond et al., 2012). Moreover, disgust sensitivity is positively associated with in-group attraction and the desire to exclude out-group members (Faulkner, Schaller, Park, & Duncan, 2004; Navarrete & Fessler, 2006). Disgust is recognized as having two components, physical and moral (Jones & Fitness, 2008). In their seminal review of the topic, Rozin and Fallon (1987) noted that disgust is one of the most powerful ways of transmitting cultural and moral values. Disgust sensitivity, especially

concerning moral and sexual disgust, correlates positively with Conscientiousness (Druschel & Sherman, 1999; Tybur, Lieberman, & Griskevicius, 2009). Women on average score higher on Conscientiousness and disgust sensitivity scales than do men (Davey, 1994; Haidt, McCauley, & Rozin, 1994). Conservatives are more easily disgusted than liberals (Inbar, Pizarro, & Bloom, 2009), and authoritarianism is associated with stronger disgust (Hodson & Costello, 2007). As recent research indicates, moral disgust (and anger) might also underlie motivations to punish norm-violating third parties (e.g., Kurzban, DeScioli, & O'Brien, 2007). Notably, increased disgust sensitivity may also indirectly increase the need for self-control and constraint, to inhibit aversions.

I propose that disgust sensitivity may function to transmit and protect cultural and moral values in order to control proactive aggressive impulses that are simultaneously present in Conscientious individuals. However, although physical disgust is associated with IFG/AI activition, it remains unclear whether this is also the case for moral disgust (Chapman & Anderson, 2012). Nevertheless, activation of the IFG/AI in response to disgusting stimuli is predicted by disgust sensitivity (Borg, de Jong, Renken, & Georgiadis, 2013; Calder et al., 2007; Caseras et al., 2007; Mataix-Cols et al., 2008). In addition, greater activity in the left IFG/AI in response to disgusting stimuli explained higher disgust sensitivity in women compared to men (Caseras et al., 2007). Moreover, as discussed in the previous section, the left IFG/AI is activated in studies of scrutiny of moralistic content and social appropriateness, social inclusion concerns, and associated emotions such as shame and guilt.

Slow Updating by Keeping the Novel Out: Novelty-Induced Grooming

In rodents, one of the major behavioral changes that occur in response to novelty or stress is self-grooming, known as "novelty-induced grooming." It has been suggested that this behavioral response plays a deactivating role in restoring behavioral homeostasis (Delius, Craig, & Chaudoir, 1976; Gispen & Isaacson, 1981). Washing symptoms of OCD may reflect grooming to avert infection. Other forms of grooming that are comorbid with OCD, such as hair pulling, skin picking, and nail biting, may decrease the processing of external unfamiliar stimuli by diverting attention away and shift it toward the internal and familiar self.

Notably, grooming is also performed between individuals, including maternal grooming from mother to offspring. This suggests that self-grooming may also activate representations of caring close and familiar others. Maternal grooming involves the neuropeptide oxytocin that, relevant to the present discussion, has been associated with in-group favouritism in humans (De Dreu, 2012). Animal research showed that maternal grooming and the oxytocin receptor system that regulates this behavior exhibits a high degree of plasticity in response to changes in environment in the postnatal period, with implications for the transmission of behavioral response to novelty and maternal care across generations (Champagne & Meaney, 2007). Oxytocin potently enhances novelty-induced grooming behavior (Drago, Pedersen, Caldwell, & Prange, 1986) and neonatal administration of oxytocin increases novelty-induced grooming in the adult rat (Noonan, Continella, & Pedersen, 1989).

The present framework suggests the interesting hypothesis that grooming in OCD patients may reflect another behavioral strategy to limit the processing of novel stimuli and may involve oxytocin (Leckman et al., 1994). However, this hypothesis remains speculative, as the evidence so far is sparse. For instance, a functional role of oxytocin in habituation of arousal following novelty-induced grooming has not yet been established. However, this would fit recent results and theory that oxytocin facilitates the habituation of novelty-induced coping responses (Tops et al., 2013; Tops, Koole, IJzerman, & Buisman-Pijlman, 2014).

Slow Updating by Keeping the Novel Out: The Corpus Callosum

In addition to behavioral strategies, the intake of novel information into internal models may also be limited by neuroanatomical characteristics that limit interhemispheric transfer of information. The size of the corpus callosum, a bundle of neural fibers beneath the cortex that comprises the primary mode of interhemispheric communication, may be a relevant parameter in the amount of interhemispheric transfer. More consistent hand preference (i.e., how consistently, or strongly, an individual prefers to use one versus the other hand over a wide variety of tasks) appears associated with smaller corpus callosum size (e.g., Luders et al., 2010) and with decreased right hemisphere activation (e.g., Propper et al., 2012). Accordingly, consistent versus inconsistent handedness is associated with decreased versus increased interhemispheric interaction and access to processes localized to the right cerebral hemisphere, respectively (Prichard, Propper, & Christman, 2013). Recently, evidence was reviewed that consistent handedness is associated with failure to update preexisting beliefs in light of new evidence (Prichard et al., 2013). These findings suggest that consistent-handers relative to inconsistent-handers show decreased transfer of novel information from right to left IFG and subsequent integration in internal models.

The correlates of consistency of handedness seem generally reflective of differences in the updating of internal models. Consistent-handers show characteristics of Conscientiousness. They score higher than inconsistent-handers on a measure of submission to authority, are more likely to identify with a conservative political party, and express less positive attitudes toward out-groups (Lyle & Grillo, 2014). The association between consistent handedness and conservatism or Conscientiousness may be mediated by reduced updating of internal models with novel information. This interpretation is supported by the finding that conservative individuals, reminiscent of the proactive trait, are less able to alter habitual response patterns to deal with a novel task (Amodio, Jost, Master, & Yee, 2007). Similarly, conservatism is associated with resistance against social system change (see for a meta-analysis Jost, Glaser, Kruglanski, & Sulloway, 2003), which may relate to the proactive trait being adaptive in a stable environment. Moreover, consistent-handers display a preference for familiar brands, higher disgust sensitivity, and eating disorders (see Prichard et al., 2013). They also show a stronger asymmetry toward the left of the arcuate fasciculus (bundle of axons) connecting the IFG with the posterior superior temporal gyrus (Propper et al., 2010).

Flexible Predictive Control

The association of predictive control with rigidity in the case of Conscientiousness needs to be put in context. Although the rigid, proactive control from which the Conscientiousness trait derived is adaptive in predictable and stable environments, evolutionary expansion of capacities for formation of internal models, through increased encephalization and learning, enabled flexible predictive control that can be applied in a wider variety of environments and circumstances, including relatively less stable ones (see Chiappe & MacDonald, 2005; Jones, 2011; Potts, 1998). However, traits associated with lower or rigid predictive control are still prevalent, indicating that they may have retained adaptivity in certain circumstances or in populations made up of individuals with diverse traits and strategies.

Conclusion

At the beginning of this commentary, I stated my intention to show that the evolutionary life history framework described by Del Giudice and the PARCS framework supplement each other and synergistically increase explanation and connection at different levels. I proposed that a continuum derived from PARCS may be related to the slow–fast continuum, facilitating further integration of evidence and connection between PARCS and the life history framework for psychopathology. Specifically, I argued that psychopathology can arise at both ends of a continuum reflecting the degree to which internal models are updated in light of novel evidence by a left hemisphere control system. Some slow strategies require, and facilitate the development of, internal models that are kept stable by slow updating. By contrast, fast strategies and fast updating are associated with reactive control in unpredictable or unstable environments. The life history framework connects these mechanisms to processes such as reproductive strategies, partner selection, and associated behavioral characteristics. PARCS connects those mechanisms and processes to additional behavioral and brain characteristics, such as those related to seeking or avoiding novelty.

The frameworks may predict aspects of disorders that did not receive much attention in research yet. For instance, PARCS suggests that disorders featuring high Conscientiousness involve regulation of strong aggressive impulses. Indeed, although research on this topic is scarce, anger attacks are present in about half of patients with OCD, who are prone to become upset or angry in situations in which they are not able to maintain control of their physical or interpersonal environment (Painuly, Grover, Mattoo, & Gupta, 2011).

The life history framework for psychopathology, especially when combined with PARCS, offers many testable research ideas that are likely to advance the field and increase our understanding of mental disorders. The benefits of such research endeavors are likely to be enormous. By understanding functional relationships and differences between disorders, and processes and mechanisms causing disorders (and for whom), we will be better equipped to develop scientifically based interventions for treatment and prevention. I look forward to the continued advancement of research in this area, and to future applications of it.

Note

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Psychological Inquiry, 25: 385–388, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.916192

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Functional Classification of Psychiatric Disorders: A Luminous Future?

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After three decades of consensus-based diagnostic categories in mental health, there is great frustration with our relative failure to develop a classification system of psychiatric disorders that meets the criteria of scientific validity and clinical utility. The recent release of the new edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013) has been accompanied by a collective flurry of debate and controversy about the impact and relevance of its symptom-based classification (Nemeroff et al., 2013). In an effort to address the long-standing critiques and clear limitations of descriptive approaches to psychiatric diagnosis, many researchers have stressed the necessity to explore new and more powerful methods of classifying psychopathological conditions (Hyman, 2011a). Along these lines, Del Giudice (this issue) provides a nice review of how a life history framework can offer an alternative formulation for the nosology of psychiatric disorders.

The author claims that a reorganization of existing diagnostic categories based on functional criteria helps "carving nature at its joints" and could represent a significant step toward a truly integrative science of mental suffering. In other words, his proposal argues for a better validity of the functional classification compared with the atheoretical classification system of the *DSM* or the internalizing–externalizing distinction. Broadly speaking, the validity of a scientific classification is the extent to which it reflects the aspects of the real world with which the science in question is concerned. However, this "absolute validity" is beyond the reach of many behavioral sciences, including psychiatry. Hence, "lesser validities" are generally adopted in the psychiatric literature dealing with the problem of diagnostic systems (e.g., face validity, construct validity, content validity; Fulford, Thornton, & Graham, 2006). One of these "lesser validities" is especially important for clinicians: predictive validity. Predictive validity refers to the extent to which a classification system allows us to predict prognosis (course and outcome) and response to preventive and therapeutic interventions for each specific disorder.

The consequence of defining diagnostic validity in this way is that any new classification system should prove its utility for practicing clinicians; otherwise its likelihood to be adopted is very low. The necessity to prove its clinical utility is especially pressing for a classification system originating from evolutionary psychopathology. Most psychiatrists believe that evolutionary explanations are too vague and general to be useful in clinical practice and are discouraged by the fact that there are no evolutionary-based treatments for mental disorders. The reason is that clinical aspects have not been a major focus of research and discussion in evolutionary psychopathology (Troisi, 2012). When people experiencing mental distress arrive at the hospital or psychiatrist's office, what they need is a proper diagnosis and an effective treatment. And most evolutionary hypotheses and explanations, as currently presented by articles and books in the field of Darwinian or evolutionary psychiatry, do not seem to be of great help to address patients' needs.

In the rest of this commentary, building on the distinction between validity and clinical utility (Kendell & Jablensky, 2003), I review the prognostic, preventive, and therapeutic implications of a functional classification of psychiatric disorders based on a life history framework.

Prognosis

Dealing with the way individuals allocate time and energy to the various activities that compose their life cycle, life history theory should have important implications for the prediction of the longitudinal aspects of psychiatric disorders, including their course and outcome. For example, fast spectrum disorders can be expected to peak during the years that coincide with maximum mating effort and intrasexual competition. In clinical terms, this means that a psychiatric patient with a fast spectrum disorder should present the most severe symptoms during adolescence and young adulthood and improve substantially with age (Troisi, 2007). *DSM-IV* cluster B personality disorders (antisocial, borderline, narcissistic and histrionic personality disorders) meet the criteria of fast spectrum disorders and, in fact, tend to become less evident or to remit with age (van Alphen, Engelen, Kuin, & Derksen, 2006). In particular, the behavior characteristics of antisocial personality disorder (ASPD) first appear during adolescence and often disappear during the fifth decade. If ASPD is viewed as a risk-taking behavioral strategy, its improvement with age and higher

prevalence among males fit with the pattern one would predict from a life history theory perspective. Among patients with ASPD, ages 15 to 29 are those of most severe manifestation of the disordered personality traits, including impulsivity, aggressiveness, irresponsibility, and sensation seeking. Among males in the general population, these are the years of highest risk for motorcycle accidents and arrest for assault. From a life history theory perspective, the common explanation for these clinical and sociodemographic findings lies in the role of risk taking in reproductive competition, which is typically more intense for young men than for women or older men. During the teens and young adult years, competition for social and economic resources is acute, and one's fate in the mating market is being determined. For males at younger ages, the optimal strategy is to take risks to acquire resources for immediate use in mating effort, especially when environmental characteristics are uncertain and unpredictable (Hill & Chow, 2002).

In women, anorexia nervosa and bulimia nervosa first appear during adolescence and have a high rate of clinical recovery within 5 years from the onset (Keski-Rahkonen et al., 2007; Uher & Rutter, 2012). These data are in accord with their classification within the fast spectrum disorders. However, to my knowledge, there are no prognostic data confirming the distinction between subtypes with a dysregulated profile (fast spectrum) and perfectionist or overcontrolled profiles (slow spectrum).

Data are more equivocal for other disorders that Del Giudice classifies within the fast spectrum disorders. For example, according to a review of the natural course of bipolar disorder in the pre-drug era (Alvarez Ariza, Mateos Alvaerez, & Berriois, 2009), this psychiatric condition is a chronic disorder that does not remit or improve with age. In addition, in the pre-drug era, mixed states and delusional symptoms were more common in elderly patients and their episodes were longer. The same argument applies to schizophrenia spectrum disorders. Del Giudice puts a major emphasis on schizotypy and limits the focus on the subgroup of patients who present with florid positive symptoms (i.e., delusions and hallucinations). Such a description of schizophrenia spectrum disorders largely ignores those forms of schizophrenia characterized by social withdrawal, alogia, reduced motivation, anergia, and flat affect (negative or deficit schizophrenia). These forms do not meet the criteria for a classification on the fast end of the continuum and do not improve or remit with age (Tek, Kirkpatrick, & Buchanan, 2001).

Prevention

Prevention is the holy grail of medicine and psychiatry. Diagnosis and therapy are important for helping patients and fulfilling their expectations, but both take place after the disease process has already struck the individual. Prevention is much more; it holds the promise of reducing or eliminating the risk of getting sick through the application of a body of knowledge concerning the causal factors that set in motion the pathogenetic process. In particular, primary prevention aims at avoiding the development of a disease by removing modifiable risk factors involved in its etiology. Compared to other fields of medicine, psychiatry seems to be a step back in implementing successful preventive strategies, as attested by minor changes over time in the prevalence rates of mental disorders (Kessler et al., 2005).

An innovative and original contribution that the evolutionary life history framework can offer to the prevention of mental disorders is the identification of those developmental factors that result in different life history strategies. By tradition, epidemiological research in psychiatry has been concerned with the study of risk factors for mental disorders (Hyman, 2011b). More recently, studies of gene–environment interaction have searched for the links between particular disorders and particular genetic polymorphisms in the face of contextual adversity. A new way of looking at these gene–environment interactions is the evolutionary hypothesis of differential susceptibility to environmental influences, which is strictly related to the life history framework (Belsky & Hartman, 2014). This hypothesis has important implications for preventive interventions because it predicts that individuals long regarded as especially vulnerable to adversity due to their genetic makeup disproportionately benefit from environmental changes designed to foster positive functioning.

Given that early experience is a major and modifiable determinant of individual life history variation, preventive interventions targeting parenting styles and family relationships could benefit from a reclassification of psychiatric disorders based on the risk factors that channel individual development toward the fast or the slow spectrum. Unlike prevailing models of the pathogenic impact of early environment, the life history framework can refocus preventive interventions not only on childhood traumatic experiences (that are related to fast spectrum disorders) but also on parental overprotection (that is a likely antecedent of slow spectrum disorders; Otani et al., 2013).

Therapy

To be clinically useful, any new theory or hypothesis applied to medicine must have therapeutic implications. Randolph Nesse, one of the founders of the field of evolutionary psychopathology, has acknowledged that at present there are no evolutionary-based

treatments for mental disorders and has concluded that evolutionary biology's main contribution to psychiatry is more theoretical than practical (Nesse, 2005). It is unlikely that the life history framework can overcome this important limitation of the evolutionary approach to the study of psychiatric disorders.

The reason for the therapeutic impotence of evolutionary psychopathology, including the life history framework, is its distinctive focus on ultimate questions (phylogeny and adaptive function) rather than proximate questions (mechanisms). In medicine, a detailed knowledge of disease mechanisms is the prerequisite for the discovery of effective therapies. The prevailing metaphor of medicine is that of the body as a machine that the doctor is called upon to fix when it breaks. The doctor's role is that of an engineer who uses technology (i.e., therapeutic tools) to reverse the pathways leading to machine malfunctioning (i.e., the pathogenic mechanisms of disease; Childs, 1999). Thus, it is highly improbable that a reorganization of existing diagnostic categories based on functional criteria will lead to the discovery of more effective therapies, unless the new functional classification will allow a better understanding of the pathophysiological mechanisms underlying psychiatric symptoms and syndromes.

Conclusions

The theoretical merit of the life history framework is outstanding, especially when compared to the imaginative and adaptationist hypotheses invented in the past by evolutionary psychopathology to explain the origin of psychiatric disorders. It is a framework based on one of the most heuristic and validated evolutionary theories, and it allows the meaningful reinterpretation of a variety of behavioral and physiological data. However, the aim of reorganizing psychiatric nosology by classifying existing diagnoses along the fast/slow continuum is too ambitious for a number of reasons.

First, the clinical utility of the fast/slow spectrum classification is limited. Whereas prevention and, to a lesser extent, prognostic prediction of mental disorders could benefit from the new classification, its adoption is unlikely to improve therapeutic interventions. This judgment may appear too pessimistic, but it should be evaluated in the light of the new "competitors" that are entering the field of psychiatric nosology to overcome the evident limitations of the symptom-based classification systems. For example, the National Institute of Mental Health has taken the lead in developing a dimensional approach to clinical observation, codified in the new Research Domain Criteria (Insel et al., 2010). This is based on a matrix of major neural systems (specifically, negative valence systems, positive valence systems, cognitive systems, social processing systems, arousal, and regulatory systems), which are assessed across multiple units of analysis (including genes, molecules, cells, circuits, physiology, behavior, and patient report). The ultimate aim of this effort is to provide a stronger foundation for research into pathophysiology, which ultimately may inform future clinical classification schemes and, eventually, help identify new targets for treatment development (Krystal & State, 2014). With its focus on pathophysiological mechanisms (which are the foundation of nosology in all other branches of medicine), a new classification based on Research Domain Criteria is likely to attract clinicians' interest much more than a functional classification of mental disorders.

Second, it is not clear how the classification based on the life history framework deals with the issue of temporal stability of diagnostic categories. Life history strategies generally translate into stable, traitlike psychological and behavioral profiles. Thus, when applied to psychopathology, the fast/slow continuum should pertain to personality disorders or chronic disorders in which personality features play a crucial role rather than statelike disorders with a definite onset, discrete clinical manifestations, and complete resolution. To accommodate these disorders within the fast/slow classification, the model should specify the contextual conditions that allow an individual to switch off a life history strategy and the range of plasticity of life history patterns (Troisi, 2005).

Third, the use of existing *DSM* categories to rebuild a functional classification produces insurmountable difficulties. For example, the difficulty to explain the switching in the same patients from the fast to the slow end of the continuum (or vice versa; e.g., restrictive anorexic women switching to bulimia) or the possible comorbidity between fast and slow spectrum disorders (e.g., one patient presenting simultaneously with symptoms of substance abuse and social phobia). Which is the functional classification of patients with such a kind of switching or comorbidity? The concept of phenocopy (the same clinical syndrome corresponding to different functional correlates) is unlikely to explain all these nosological discrepancies. In addition, there is no reason to assume a complete correspondence between functional correlates and pathophysiological mechanisms. If the existing *DSM* categories that are phenocopies could be reclassified by either function or mechanism, clinicians are likely to choose the second option for the reason explained in the preceding Therapy section.

In conclusion, at the present time, rather than being the basis for a new psychiatric classification, the life history framework appears to be a powerful stimulus for an innovative research program that can

enrich our understanding of the etiology (early experiences, gene–environment interaction) and epidemiology (sex-related prevalence, age at onset, natural course) of mental disorders.

Note

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Psychological Inquiry, 25: 389–393, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.916193

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Strategic Choices versus Maladapative Development

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Del Giudice (this issue) provides a novel and compelling "general framework" as to how individual variations in life history strategies can inform our understanding of the structure of psychopathology. Like his adapative calibration model of individual differences in stress responses (Del Giudice, Ellis, & Shirtcliff, 2011), Del Giudice's target article draws our attention to fast versus slow life history strategies and how these are moderated by sex. From any lofty vantage point, four of the most visible, deeply etched features of the epigenetic landscape are those representing the four combinations of male/female and fast/slow phenotypes. It would be surprising if these major suites of adaptive design features were *not* related to vulnerabilities for specific forms of psychopathology. The framework represents an exploration of the relevance of life history theory (LHT) for psychopathology, and perhaps personality, more than a model for all psychopathology. In this brief comment, we suggest Del Giudice's fast–slow framework, and LHT in general, is incomplete without systematic consideration of a (mostly) independent dimension of individual differences related to neural integrity, and thus his framework illuminates some disorders better than others. Here we discuss one disorder that he did not focus on—substance abuse—and suggest that his approach has much to offer. We also discuss a group of disorders that he did focus on—neurodevelopmental disorders such as schizophrenia and autism—and suggest that here the LHT approach may provide fewer insights.

It is important to recognize that sensitivity to environmental signals that direct life history strategies represents an adaptation, facilitating reproductive fitness across the different environments humans have frequently encountered. This cue-driven adaptive tuning is partly accomplished by stress and immune regulation systems, and one thing we know about these systems is that it they are malleable. Thus, a LHT approach is perhaps on firmest ground in elucidating those forms of psychopathology that have their roots in atypical stress system functioning, that is, those types of psychopathology that are most apt to wax and wane over time and circumstance. Perhaps unsurprisingly, a perspective built on successfully implemented adaptations may work better for "disorders" that have relatively little impact on fecundity. Specifically, we suggest that Del Giudice's model has much more to say about those disorders *not* characterized by reduced neural integrity or by reduced fecundity, that is, internalizing and externalizing disorders such as depression, anxiety disorders, obsessive-compulsive disorder, substance abuse, and conduct disorders, than it does for neurodevelopmental disorders (NDDs) characterized by reduced neural integrity and reduced fecundity, such as schizophrenia and autism.

Substance Abuse

Del Giudice (this issue) addresses substance use in the context of psychopathy. There is evidence to suggest, however, that substance abuse in psychopathy may differ from substance abuse in general. For example, individuals with psychopathy may not as often report symptoms of substance dependence such as withdrawal and craving, and psychopathy has been shown to moderate the neurobiological response to drug/alcohol cues (Cope et al., 2014). These observations suggest that the neurobiological underpinnings of addiction may be different in substance-using individuals with psychopathy. We suggest the framework proposed in Del Giudice (this issue) could be readily applied to substance use disorders more generally as they occur within nonpsychopathy samples. Next we provide a brief example of how the model proposed by Del Giudice could be extended to alcohol use disorders.

Alcohol use disorders are heterogeneous in terms of course, severity, recovery, and comorbidity, prompting many efforts to identify clinically meaningful subtypes. Binary models of alcoholism are of particular interest for the LHT framework. Several classification systems have been proposed that identify two subtypes within alcohol use disorders. For the purposes of this commentary, we focus on two highly overlapping classification systems—Cloninger's Type

1 and Type 2 and and Babor's Type A and Type B (Hesselbrock & Hesselbrock, 2006). The Type 2/Type B subtype is associated with an earlier age of onset, a familial history of substance use disorders, higher rates of conduct problems and comorbid drug use, and more severe alcohol-related problems. Type 1/ Type A is associated with a later age of onset, less comorbid psychopathology, and fewer conduct problems. There is evidence to suggest that this binary typology can be applied to the general population and across cultures, making this typology an appropriate choice to consider from an LHT framework (Hesselbrock & Hesselbrock, 2006; Tam et al., 2014).

The framework proposed in Del Giudice (this issue) details specific traits and psychiatric disorders associated with the fast versus slow spectrum. Upon examination, the correlates of the Type 1/Type A subtype are consistent with slow spectrum psychopathology, whereas the Type 2/Type B subtype are consistent with fast spectrum psychopathology. More specifically, the Type 2/Type B subtype is associated with increased novelty seeking behaviors and reduced reward dependence, risk aversion, and harm avoidance (Leggio, Kenna, Fenton, Bonenfant, & Swift, 2009), all traits associated with fast spectrum psychopathology. Alternatively, the Type 1 subtype is associated with reduced novelty seeking, and with increased risk aversion, harm avoidance, and reward dependence, all traits associated with slow spectrum psychopathology. Further, Type A has been associated with comorbid psychiatric conditions identified as slow spectrum disorders, such as disorders of anxiety and depression (Hesselbrock & Hesselbrock, 2006; Leggio et al., 2009). In contrast, Type B was associated with psychiatric conditions identified as fast spectrum disorders, including drug use and conduct problems. In sum, many of the characteristics used to differentiate slow and fast spectrum psychopathology in the by Del Giudice (this issue) overlap with characteristics used to differentiate alcohol use disorder subtypes.

Last, these subtypes may emerge from different neural substrates, and hence may be helped by different types of medication. For example, Bogenschutz, Tonigan, and Pettinati (2009) reported that individuals with an Alcohol Use Disorder (AUD) classified as Type A showed improvements in drinking outcomes following a Naltrexone intervention when compared to a placebo. However, drinking outcomes in individuals classified as Type B did not differ by medication type (i.e., Naltrexone vs. placebo). In contrast, SSRIs "can worsen the prognosis and increase drinking relative to placebo in the Type B" subtype, an effect not found in the Type A subtype (Roache, Wang, Ait-Daoud, & Johnson, 2008, p. 1503). These findings suggest that the model proposed by Del Giudice (this issue) could guide treatment discovery and planning. Overall, this brief consideration of alcohol use disorders illustrates the potential scope and clinical value of the LHT approach to psychopathology.

Schizophrenia and Autism

A framework that stresses cue-driven LH strategies encounters some difficulties when applied to severe psychopathology. To understand the nature of these more serious disorders, additional causal factors must be specified, and our understanding of these may also be informed by evolutionary theory. There are two related issues here. One is straightforward and obviously recognized by Del Giudice: Some disorders are primarily characterized by disruption of adaptive processes that degrade biological functioning. The other is more theoretical: Might systematic consideration of organism quality help reveal the virtues and limits of a LHT approach?

Before we discuss our own attempts to bring evolutionary theory to bear on neurodevelopmental disorders, consider the problem posed by high rates of MZ twins discordant for psychopathology. In schizophrenia, slightly more than half of MZ twins are discordant (Gottesman, 1991), whereas in autism about 40% are discordant (Hallmayer et al., 2011). Consistent with these data, shared environment effects (c^2) tend to be small for both disorders (Plomin, DeFries, Knopik, & Neiderhiser, 2013). As monozygotic (MZ) twins reared together have nearly identical genomes and grow up in highly similar environments, these observations raise concerns about how important the "key dimensions" of "resource availability, extrinsic morbidity-mortality, and unpredictability" are for the development of schizophrenia and autism. When it comes to severe psychopathology, we believe the bulk of the causal variance lies elsewhere. The discordance data draw our attention to the importance of stochastic processes introducing "noise" into neurodevelopment pathways, and as well to organismic processes buffering development from such perturbations. These opposing processes represent the foundation of our Developmental Instability model of neurodevelopmental disorders (e.g., schizophrenia, attention deficit hyperactivity disorder, autism), a model that attempts to explain comorbidity across these disorders and the presence of shared features (e.g., lower intellectual ability, atypical lateralization, presence of more minor physical anomalies, and fluctuating asymmetry), while accounting for their persistence across generations through mutation-selection balance (Yeo, Gangestad, & Thoma, 2007). In a nutshell, our model proposes two distinct genetic influences for NDDs: a general factor that broadly increases vulnerability for atypical brain development (accounting for phenotypic similarities across

disorders), and a specific factor that increases vulnerability for a given specific form of NDD.

We have hypothesized (Yeo, Gangestad, Edgar, & Thoma, 1999), as have others (Keller & Miller, 2006), that mutation load is the single most important etiological factor for NDDs. This leads to our suggestion that NDDs are best understood as reflecting a dimension of individual differences orthogonal from the fast–slow distinction. Although fast and slow life history strategies can be viewed as adaptations, NDDs like schizophrenia and autism are simply "harmful dysfunctions" emerging from insufficiently buffered genetic and environmental disruptions. The harmful nature of these phenotypes is evident in many ways but foremost is the overall reduced fecundity of affected individuals plus that of their immediate relatives (Power et al., 2013). Table 1 summarizes many features distinguishing schizophrenia and autism from internalizing and externalizing disorders. As a whole, these features denote atypical developmental due to mutations (especially rare copy number deletions; see Yeo & Gangestad, in press, for a review), beginning early in gestation, affecting primarily cortical functioning rather than stress system functioning.

One feature of Table 1 is especially notable—the link with reduced general cognitive ability (GCA; Kahn & Keefe, 2013). Perhaps no other feature besides reduced fecundity so clearly demonstrates the harmful nature of schizophrenia and autism, because greater GCA is associated with so many beneficial life outcomes, not only in social and vocational realms but also in terms of morbidity and mortality (Deary, 2012). Among patients, uncommon copy number deletions predicts deficits in GCA and executive functioning (Yeo et al., 2013; Yeo et al., in press). In contrast to the brain's stress system, the neural networks implicated in schizophrenia and autism are not especially malleable. The cortical design characteristics conferring a "good network" are laid down early in life, accounting for such observations as the temporal stability of general cognitive ability over the lifespan (Beaver et al., 2013) and the relationship between low cognitive ability in childhood and the risk of dementia (Whalley et al., 2000). Several recent studies offer evidence of molecular genetic overlap between risk of schizophrenia and lower GCA. A large-scale study found that rare copy number variations that increase risk for schizophrenia also had a negative impact on GCA in healthy controls (Stefansson et al., 2014). Two interesting observations have emerged from studies examining in healthy controls the correlates of genetic variation in the total set of single nucleotide polymorphisms (SNPs) representing risk for schizophrenia (Purcell et al., 2009). First, controls with a heavier loading on this massively polygenic SNP risk factor show relatively lower GCA (Lencz et al., 2014). In another study, controls with a heavier loading showed relative developmental decreases in GCA over time (McIntosh et al., 2013). There is also evidence for the continuity across the schizophrenia spectrum (Nelson, Seal, Pantelis, & Phillips, 2013) of this same molecular genetic SNP factor (Bigdeli et al., 2014). Along with the recent report demonstrating the existence of an overarching psychopathology factor (labeled "p") that is negatively correlated with GCA and neural integrity (Caspi et al., 2013), these studies convincingly demonstrate the intimate association of the genetic factors underlying schizophrenia spectrum psychopathology with an important harmful dysfunction—reduced GCA. Taken together, the set of attributes captured in Table 1 suggest that the adverse effects of mutation load, moderated by available buffering capacity, may be much more important than life history strategies for schizophrenia and autism. We recognize, however, that life history strategies and attendant vulnerabilities may play a progressively greater role as one moves along the continuum from the diagnostic entities themselves through related personality disorders to normal variations in personality.

Table 1. *Comparison of Schizophrenia and Autism With Internalizing and Externalizing Disorders on Features Putatively Linked With Mutation Load.*

Feature	Schizophrenia/Autism	Internalizing	Externalizing
Reduced fecundity	yes	no	no
Developmental abnormalities			
Minor physical anomalies	yes	no	no
Dermatoglyphic anomalies	yes	no	no
Fluctuating anatomic asymmetries	yes	no	no
Atypical brain asymmetries	yes	no	no
Copy number variations	yes	no	no
General cognitive deficits	yes	no	maybe
Childhood impact	yes	no	maybe
Course	more chronic	variable	variable

Systematic consideration of the relevance of this additional dimension of individual differences can potentially sharpen applications of LHT to psychopathology, but additional research is needed. In general, LHT stresses the importance of environmental cues for life history strategies, rather than cues as to organismic integrity (though see Rickard, Frankenhuis, & Nettle, 2014). Exposed to the exact same set of environmental cues, does it make evolutionary sense that individuals of high and low neural integrity respond the same way? Would individuals with relatively lower GCA readily recognize the environmental cues? And if so, would they mount an equally robust developmental response? Taking this orthogonal dimension of "effective mutation load" into account may also help us see more clearly the covariance structure of psychopathology. For example, in evaluating statistical models of psychopathology Caspi et al. (2013) found that when they removed variance related to their "p factor," the positive correlation between internalizing and externalizing disorders turned into a negative correlation. This finding is consistent with the unidimensional nature of fast versus slow strategies and its relevance for psychopathology. Sex differences in psychopathology also became more salient, as expected if the adverse effects of mutation load are not sex specific.

Note

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Psychological Inquiry, 25: 394–413, 2014

ISSN: 1047-840X print / 1532-7965 online
DOI: 10.1080/1047840X.2014.925339

Routledge
Taylor & Francis Group

REPLY

A Tower Unto Heaven: Toward an Expanded Framework for Psychopathology

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I respond to commentaries on my target article "An Evolutionary Life History Framework for Psychopathology." I start by addressing criticism of my basic assumptions about life history strategies and their implications for individual differences in human behavior. Next, I examine the theoretical structure of the proposed framework and respond to the commentators' challenges to its generality and flexibility. I show how the framework can be expanded to include multiple levels of analysis and to integrate behavioral control with neurological functionality; I also reinterpret the recent finding of a general factor of psychopathology in the context of the expanded framework. In the last section I discuss specific psychopathological conditions, namely attention deficit/hyperactivity disorder, borderline personality disorder, substance abuse, autism spectrum disorders, schizophrenia spectrum disorders, obsessive-compulsive disorder, eating disorders, and depression. For each condition, I reply to the commentators' criticism of my life history analysis, integrate their suggestions and insights, highlight the present weaknesses of the theory, and indicate promising directions for future research.

I am grateful to the commentators for their thought-provoking responses to my target article. The breadth and depth of their comments was impressive; addressing them has greatly sharpened my thinking on life history, psychopathology, and the evolution of mental disorders. The commentators left no stone unturned in scrutinizing my proposal for a unifying framework, but their overall response was extremely constructive. In fact, some of them extended my initial analysis, suggested important additions to the theory, and applied the framework to new conditions that had not been covered in the target article.

As several commentators noted, the goal of unifying the study of mental disorders is a frighteningly ambitious one. For such a biblical task, a biblical metaphor seems appropriate. Psychopathology is a scientific Babel—a humming confusion of models, disciplines, and approaches (Abed, 2000). My framework is a plan for rebuilding the theoretical structure of the discipline from the ground up, working together in the common language of evolutionary biology. Although this imaginary tower is a long way from reaching the sky, I think its contours are beginning to emerge. In the first section of this reply, I deal with the theoretical foundations of the life history approach. Next I move to the conceptual architecture of the framework, and show how the fast–slow distinction can be expanded into the blueprint for a comprehensive taxonomy of mental disorders. In the final section I engage in the brickwork of classification by discussing the place of specific disorders within the framework.

The 18 commentaries spanned a huge range of topics; although I did my best to respond to all the major points, some interesting comments had to go unaddressed. In particular, I decided to forgo discussion of the framework's clinical applications. I sincerely thank all the commentators with a clinical background for sharing their insights. However, in my response I focus on the theoretical and empirical validity of the framework, leaving that of clinical utility as an important question for the future.

Checking the Foundations: Validity of the Life History Approach

The life history framework I presented in the target article rests on four basic assumptions: (a) at the

Color versions of one or more of the figures in the article can be found online at http://www.tandfonline.com/hpli

broadest level of analysis, life history variation can be meaningfully described by a single dimension, from "fast" to "slow"; (b) the fast–slow continuum applies not only to differences between species but also to individual and population differences within a species; (c) at the broadest level of analysis, individual differences in human motivation, personality, and self-regulation can be understood as reflecting variation on the fast–slow continuum; and (d) life history strategies are jointly determined by genetic and environmental factors and show developmental plasticity in response to cues of danger, unpredictability, and resource availability.

The commentators did a great job in pointing out potential problems and limitations of this approach. Ideas from evolutionary theory should not be adopted blindly but understood in their strengths and weaknesses, and handled with care to avoid slipping from useful generalizations to misleading simplifications. This is especially true of powerful, seductive concepts like that of the fast–slow continuum. In this section I address criticism of my starting assumptions, discuss their complexities and limitations, and try to dispel some misconceptions about the implications of a life history approach.

The Fast–Slow Continuum

The strongest criticism of my life history approach comes from Surbey (this issue), who argues that the very idea of a fast–slow continuum is outdated, theoretically unsound, and falsified by the biological evidence. According to Surbey, the fast–slow continuum is synonym with the theory of *r-K* selection (Pianka, 1970; see Jeschke, Gabriel, & Kokko, 2008), and the two share exactly the same limitations. This is incorrect. The theory of *r-K* selection was based on specific assumptions about the role of density dependence in driving the evolution of life histories; whereas those assumptions have been rejected or substantially revised (Charlesworth, 1994; Jeschke et al., 2008; Reznick, Bryant, & Bashey, 2002), the general pattern of variation identified by *r-K* theorists has been largely confirmed by later empirical studies (Jeschke & Kokko, 2009; Promislow & Harvey, 1990; Sibly & Brown, 2007; Sibly, Grady, Venditti, & Brown, 2014; Stearns, 1983). It is important to stress that the fast–slow continuum is a *descriptive* concept that makes no assumptions about the evolutionary causes of the observed covariation between traits (see Jeschke et al., 2008). Although some theorists (e.g., Roff, 2002) prefer to avoid "big picture" generalizations to focus on specific mechanisms of life history evolution, the fast–slow continuum is regarded a useful heuristic by many researchers, as testified by its increasing popularity in the scientific literature.[1]

Three commentators (Surbey; Crespi; Kennair) cite a well-known comparative study by Bielby and colleagues (2007) as an empirical challenge to the fast–slow framework. In the study, the authors factor-analyzed a number of life history variables—such as gestation length, number of offspring per litter, and age at sexual maturity—across a broad range of mammalian species. They found that life history variables clustered along two factors instead of just one; crucially, neither factor could be interpreted as a fast–slow axis of variation. Unfortunately, these results are vitiated by the authors' inappropriate use of exploratory factor rotation. In the Appendix, I reanalyze Bielby et al.'s data and find that—contrary to the authors' claims—they provide strong support for a fast–slow continuum in mammals. I also discuss a related paper by Jeschke and Kokko (2009) and show how these authors underestimated the consistency of the fast–slow continuum across methods of analysis (see the Appendix for details).

Limitations of the fast–slow continuum. While reports of the death of the fast–slow continuum have been greatly exaggerated, investigators should be aware of the boundaries and limitations of the concept. To begin, it must be stressed that the fast–slow axis is an important dimension of life history variation, but not the *only* one (Kennair); comparative data supporting the fast–slow continuum also show the existence of other meaningful axes of variation (e.g., Sibly & Brown, 2007; Stearns, 1983; see the Supplement). Employing the fast–slow continuum as a high-level descriptor should not preclude consideration of the trade-offs that form the substance of life history strategies. For example, in the target article I argued that sex differences in psychopathology are best understood at the level of specific life history trade-offs—current versus future reproduction and mating versus parenting—rather than at the level of fast versus slow strategies.

Another limitation of the fast–slow continuum is the inherent fuzziness of its definition. Although life history variables tend to cluster in similar ways in different taxonomic groups, the resulting continua are usually not identical and may differ in important ways. For example, high fecundity clusters with fast life histories in mammals but with slow life histories in fish (Jeschke & Kokko, 2009; see the Appendix). The same applies to behavioral and physiological

[1]Searching Google Scholar for publications containing both "life history theory" and "fast–slow" or "pace of life" returned a steadily increasing number of publications from 1992–1994 ($n = 6$, or 1% of all publications containing the phrase "life history theory") to 2010–2012 ($n = 153$, or 6% of all publication containing the phrase "life history theory"; search performed on April 21, 2014).

traits such as boldness, migration range, and metabolic rate, which may show different associations with life history variables in different species (see Réale et al., 2010). The take-home message is that there is no "universal" fast–slow continuum (Jeschke & Kokko, 2009); the concept needs to be adapted to the ecology of each individual species, as simple extrapolations may easily prove misleading.

Is the fast–slow continuum compatible with mixed sexual strategies? Some commentators (Holtzman & Senne; Jonason & Schmitt) note that humans can and do engage in mixed sexual strategies that involve both short- and long-term mating, and frame this observation as a challenge to the life history framework. In particular, Holtzman and Senne (this issue) argue that life history theory treats short- and long-term mating as "polar opposites." Fortunately, this is simply not the case. Life history strategies can be described at multiple hierarchical levels; the fast–slow continuum summarizes a number of specific trade-offs, each with a degree of functional independence from the others. One of these trade-offs is that between *mating effort* (which can be expended in multiple short-term sexual relations) and *parenting effort* (which, in humans, is most effectively channeled through long-term relationships). Within the limits of the trade-off, flexible allocation strategies are entirely possible, especially for individuals who are less constrained because of their superior attractiveness, wealth, or status—just as the trade-off between body growth and maintenance becomes less severe when food is abundant (see James & Ellis, 2013; see also Gangestad on condition dependence in life history traits).

Still, the trade-off cannot be completely avoided: The time and resources spent with a long-term partner and his or her children cannot be spent to pursue another sexual liaison. Thus, on average, people who invest a lot in long-term relationships will tend to invest less in short-term mating. Also, the trade-off between mating and parenting becomes more severe when people start having children; for this reason, studies of college undergraduates (e.g., Jackson & Kirkpatrick, 2007) are likely to underestimate the strength of the trade-off, and they provide a distorted picture of individual differences in this domain. That said, the contradiction between a unidimensional fast–slow continuum and mixed sexual strategies is only apparent: Life history strategies and sexual strategies exist at different levels of analysis, and both should be included in a complete theory of psychopathology (see next).

From Life Histories to Individual Differences

Extending the life history framework to include individual differences in personality and behavior is not a straightforward move (Crespi; Gangestad; Surbey). I agree with Crespi (this issue) that life history approaches to animal personality and "coping styles" provide a good role model for this task. I also thank Tops (this issue) for showing how animal and human findings can be synthesized in creative ways to yield novel insights and predictions. The field of animal personality has moved way beyond the initial focus on shyness–boldness and proactive–reactive coping styles, and the fast–slow continuum—under the rubric of "pace-of-life syndromes"—is emerging as a unifying principle for understanding multidimensional variation in behavior and physiology (see Réale et al., 2010; Wolf & Weissing, 2012). Investigators interested in life history and human behavior should acquaint themselves with the complexity of the animal literature (Réale et al., 2010), not least to avoid the pitfall of defining life history strategies in purely behavioral terms (Gangestad, this issue).

In the target article, I tried to be careful in selecting a small set of theoretically justified, empirically robust correlates of life history strategies in humans (Table 1, target article). Even so, behavioral outcomes are always overdetermined, and the associations between traits and strategies are only partial and indirect (Gangestad, this issue). The various correlates discussed in the target article should be treated as imperfect, convergent indicators of individual differences in life history strategy; as such, they should be considered together rather than in isolation and examined in light of contextual and personal factors (e.g., availability of sexual partners, attractiveness) that may moderate the relations between general strategies and specific behaviors and outcomes.

This selective approach to life-history-related traits differs in important ways from the inclusive approach followed by other researchers (e.g., Figueredo, Vásquez, Brumbach, & Schneider, 2007; Giosan, 2006; Olderbak, Gladden, Wolf, & Figueredo, 2014). I suspect that aggregating diverse measures of social functioning, personality, physical and mental health, socioeconomic status, and so forth, into ever broader superfactors (Olderbak et al., 2014) may obscure as much as it reveals about the structure of individual differences—especially when the focus is on pathological outcomes rather than normative variation (see Copping, Campbell, & Muncer, 2014, for related criticism of this approach). For this reason, I wish to caution against Glass's suggestion that simply correlating psychopathological symptoms with "K-factor" scores (see Figueredo et al., this issue; see also Figueredo et al., 2007) would be a valid and sufficient test of the framework.

Life history and human personality. As noted in the target article, I am skeptical about the General

Factor of Personality (GFP; see Rushton & Irwing, 2011) as a useful indicator of life history strategy. Setting aside the ongoing debate about the psychometric validity of the GFP, I believe that life history trade-offs are best reflected at the level of (a) the Big Five domains of Conscientiousness and Agreeableness; and (b) narrower facets of the remaining domains, such as imagination, dominance, and sensation seeking (see also Del Giudice, 2012). Intriguingly, a recent study of personality in the Tsimane (a forager-horticulturalist population of Bolivia) was unable to recover the standard dimensions of the Big Five; however, two reliable factors emerged that were largely a mixture of Conscientiousness, Agreeableness, and some aspects of Extraversion (Gurven, von Rueden, Massenkoff, Kaplan, & Lero Vie, 2013). These findings are consistent with the idea that Agreeableness and Conscientiousness reflect fundamental trade-offs in the organization of individual differences. In contrast, the Big Five domains of Extraversion, Openness, and Neuroticism are not unequivocally associated with fast life history indicators (see Holtzman & Senne, this issue, for relevant data); for this reason, deriving a GFP from the Big Five cannot be expected to yield a clean indicator of fast–slow variation in personality.

Moving beyond the Big Five, I agree with Brüne (this issue) and Jonason and Schmitt (this issue) on the theoretical and empirical relevance of "dark triad" traits (narcissism, psychopathy, and Machiavellianism). These traits should definitely be included in future elaborations of the framework. Also, the *Honesty-humility* factor in the HEXACO model (Ashton & Lee, 2007, 2008) is an excellent candidate as a slow life history correlate (see Del Giudice, 2012), as well as a strong negative predictor of the dark triad (e.g., Gaughan, Miller, & Lynam, 2012; Jonason & McCain, 2012).

Finally, I wish to address Crespi's (this issue) suggestion that fitness trade-offs associated with specific personality traits may provide a better alternative foundation for psychopathology than the broader life history trade-offs emphasized in the present framework. I see no contradiction between these two levels of analysis; in fact, all the trade-offs identified by Nettle (2011) can be easily framed in a life history perspective, as they deal with the cost and benefits of mating competition (Extraversion and Openness), vigilance to threats and dangers (Neuroticism), long-term planning (Conscientiousness), and cooperation (Agreeableness). In my view, one should consider both the specific trade-offs associated with individual personality traits *and* how correlated trade-offs give rise to broader, functional patterns of individual differences.

Genes and Environments in Life History Development

In the target article, I focused on environmental factors and dealt only cursorily with the role of genotypic differences in the development of life history strategies. This was noted by Brüne (this issue) and Schlomer and Cleveland (this issue), who argue that behavior genetics (including the study of G × E interactions and differential susceptibility) should be brought to the forefront of life-history-inspired research. Most life-history-related traits show moderate to high heritability, and (contra Jonason & Schmitt, this issue) genetic differences clearly play a large role in determining an individual's position on the fast–slow axis of psychopathology risk. Moreover, there is growing evidence that individual susceptibility to the environment is moderated by genotype (Belsky, Pluess, & Widaman, 2013; Dick, 2011; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011; but see Duncan & Keller, 2011); as noted by the commentators, these findings are highly relevant to models of life history development.

My reasons for initially concentrating on the environmental side of the coin were essentially three. First, environmental stressors play a prominent role in psychiatric epidemiology; my goal was to show how a life history approach can make sense of the stress–psychopathology link, including its exceptions (e.g., the low levels of stress associated with some subtypes of depression and eating disorders). Second, we know a lot more about the environmental variables that drive life history plasticity than we know about *specific* genetic variants associated with life history strategies in humans (some candidates are discussed by Brüne, this issue, in his commentary). Although the data on heritability are very robust (Schlomer & Cleveland, this issue), we still do not understand the architecture of life history strategies at the level of specific pathways and genes. Third, despite their remarkable empirical success, existing models of differential susceptibility have trouble explaining the evolution of *genotypic* differences in plasticity (discussed in Ellis et al., 2011).

These difficulties aside, I agree that extending the framework to include an explicit model of genotypic effects and G × E interactions is a top priority for future research. Empirically, a major challenge will be to distinguish between potentially adaptive, "strategic" genetic variation (the focus of Schlomer & Cleveland's commentary) and maladaptive variation due to mutation load (discussed by Yeo et al., this issue). For example, the available genetic data on human personality seem inconsistent with the idea of alternative adaptive strategies (e.g., Miller, 2011; Verweij et al., 2012; Verweij et al., 2010); however,

tests of balancing selection are themselves based on questionable assumptions (see Del Giudice, 2012), which greatly complicates the interpretation of empirical findings in this area.

More on the environment. Although I do not share Surbey's skepticism about the fast–slow continuum, I welcome her reminder that modern life history theory is more complex than the "executive summary" I presented in the target article. Specifically, predicting the effect of environmental factors such as mortality risk on the evolution of life history strategies is far from straightforward, and the subtleties of the relevant mathematical models literally fill volumes (e.g., Charlesworth, 1994; Roff, 2002). Applying life history theory to human development means walking the tightrope between sophisticated models with predictions that depend on detailed assumptions about mortality schedules, density dependence, and environmental stochasticity, and limited empirical data with no experimental control and myriad confounding factors. Inevitably, one has to reach a compromise between models and data, making tentative generalizations that can serve as useful heuristics for empirical research (e.g., Ellis, Figueredo, Brumbach, & Schlomer, 2009; Kuzawa & Bragg, 2012).

The point is that model predictions depend on many assumptions, some of which are extremely difficult to test with the available data. In the target article, I present a minimalist set of generalizations—about mortality, predictability, and resource availability—that I believe are reasonably robust and empirically supported. However, there is plenty of room for improvement, and I am confident that the field will continue to grow more sophisticated as new models and findings accumulate.

The commentators raise many other interesting points about the environmental determinants of life history strategies. I agree with Jonason and Schmitt's remarks about the importance of short-term mismatches between contradictory environmental contingencies in the etiology of some disorders; a related point is made by Mishra and Gonzales, who stress the importance of considering acute situational influences on behavior and their interaction with stable dispositions. From a different perspective, Gangestad notes that some effects typically attributed to ecological factors (e.g., cues of danger and unpredictability) may actually reflect individual differences in condition (e.g., genetic quality, social and material resources). Indeed, the evidence indicates that the two pathways coexist. For example, James, Ellis, Schlomer, and Garber (2012) found that earlier sexual debut was predicted by higher levels of familial/ecological stress *and* higher perceptions of mate value; moreover, earlier puberty increased self-perceived mate value and anticipated sexual debut in males (but not in females). In a recent study by Copping and colleagues (2014), high levels of attractiveness and social support predicted earlier sexual debut and more sexual partners in males (but not in females), whereas environmental security predicted later sexual debut in males and later puberty in females.

James and Ellis (2013) reviewed other relevant findings and discussed the need to integrate mate value and sex-specific effects in models of life history development. I fully subscribe to this view; reconsidering the associations between stress and psychopathology in light of condition dependence and sex-specific effects is likely to increase the power and realism of the framework. At the same time, I believe that the epidemiological patterns described in the target article will turn out to be fairly robust, even after accounting for these additional developmental processes.

Finally, Jonason and Schmitt argue that the framework should be applied to the cross-cultural study of psychopathology. I agree, and find the prospect exciting. A life history perspective may help make sense of the confusing data on the ecological and geographical distribution of mental disorders; at the same time, cross-cultural comparisons would provide an excellent test bench for the predictions of the theory.

The Construction Plan: Structure and Scope of the Framework

To succeed as a unifying approach for evolutionary psychopathology, the life history framework must prove to be (a) general enough to include a large majority of mental disorders and (b) flexible enough to accommodate an enormous range of potential etiological pathways. Naturally, these issues attracted a great deal of attention from commentators. In this section I seek to clarify the theoretical structure of the framework and address the commentators' challenges to its generality and flexibility.

An Expanding Framework

As stressed in the target article, I am not proposing the fast–slow distinction as the be-all and end-all of evolutionary psychopathology. On the contrary, I view it as the initial step toward a truly comprehensive theory of mental disorders—or more aptly, as the apex of a branching, multilevel system of analysis and classification based on functional principles. In the target article, I focused heavily on the fast–slow distinction; now it is time to be more explicit about the bigger picture and present my outlook for an

expanded version of the framework. Figure 1 shows a conceptual map of the expanded framework as I currently envision it. The left half of the figure depicts a functional hierarchy of behavioral organization; to each level in the hierarchy corresponds a level of analysis in the framework (the tower metaphor may come in handy again). The fast–slow continuum sits at the top of the hierarchy, followed by basic life history dimensions such as the current–future reproduction axis. Note that, in this diagram, higher levels are not assumed to fully explain the organization of the lower levels; also, no strong assumptions are made about the direction of causality—higher levels may either reflect the action of superordinate mechanisms or simply summarize the patterns emerging at the lower levels.

The next levels of analysis are those of self-regulation and motivation. Although their relative position in the hierarchy is somewhat arbitrary, I placed self-regulation above motivation for the following reasons: (a) basic self-regulatory parameters such as activation, inhibition, and risk sensitivity can have cascading effects on multiple motivational systems, and (b) the proactive-reactive axis of self-regulation seems to be a primary dimension of personality variation in animals; proactive coping styles can be identified across diverse taxonomic groups, from insects to mammals (Del Giudice, in press; Koolhaas, de Boer, Buwalda, & van Reenen, 2007; Koolhaas et al., 1999; Tops, this issue).

Motivational domains (see Bernard, Mills, Swenson, & Walsh, 2005; Bugental, 2000) provide a powerful criterion for the classification of mental disorders; for example, reactive obsessive-compulsive disorder (OCD) is closely linked to the domain of self-protection, whereas mating and competition are the main motivational systems involved in externalizing disorders. At this level, the analysis of mental disorders can be expected to yield overlapping classifications, as multiple motivational systems may be involved in the etiology and manifestation of a given condition.

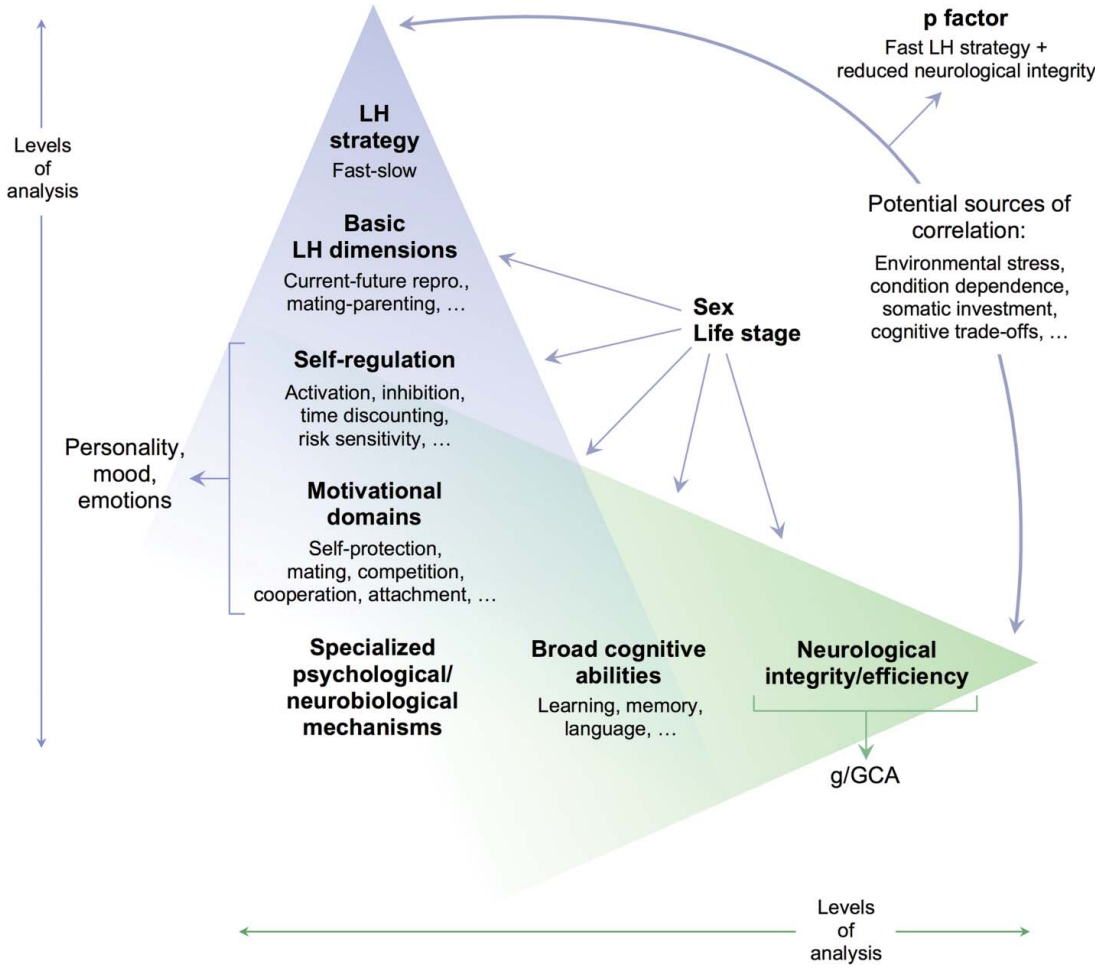

Figure 1. An expanded life history framework for evolutionary psychopathology. *Note.* LH = life history; GCA = general cognitive ability.

The hierarchy in Figure 1 ends at the level of specialized psychological/neurobiological mechanisms, such as those that mediate understanding of others' mental states, the feeling and expression of disgust, or the organism's response to stress. Of course, there are other important levels of analysis, including the cellular and molecular one (Brüne, this issue). However, those levels of analysis are unlikely to prove useful in the *classification* of mental disorders, even though they may be crucial for understanding their etiology and development. Another notable feature of Figure 1 is the "lateral" placement of personality and emotions. Personality traits and emotions emerge from the operation of self-regulatory and motivational/affective systems (Tops, this issue); although they are important pieces in the puzzle of psychopathology, I propose that they do not qualify as independent levels of analysis and should not be used as general criteria for the classification of mental disorders (see my discussion of emotions in the target article).

The hierarchy of behavioral control on the left side of Figure 1 intersects with another, conceptually independent hierarchy (shown in the lower half of the figure) that addresses the *functionality* of neural processes (Gangestad, this issue; Yeo et al., this issue). The apex of this hierarchy is a dimension of global neurological integrity/efficiency, heavily influenced by mutation load and reflected in the level of general cognitive ability, or *g* (see Deary, Penke, & Johnson, 2010; Moreno-De-Luca et al., 2013). Later I examine this aspect of the framework in more detail. Motivation, self-regulation, and broad cognitive abilities all undergo significant changes across development; as a result, different stage and transition in the human life course exhibit specific patterns of risk for psychopathology. As discussed in the target article, men and women face different constraints in their life history trade-offs, and differ in many aspects of motivation and self-regulation; likewise, consistent sex differences exist at the level of broad cognitive abilities, such as language and visuospatial skills (see Geary, 2010). Although there are little if any differences between the sexes in *average* levels of general cognitive ability, males reliably show higher *variability* in g, including a higher risk for intellectual disability (see Johnson, Carothers, & Deary, 2008).

A blueprint for evolutionary taxonomy. The diagram in Figure 1 provides the blueprint for a comprehensive evolutionary taxonomy of mental disorders. Moving from life history strategy toward the lower levels of analysis, description becomes increasingly multidimensional, to the point where specific evolved systems take center stage (Crespi, this issue; Glass, this issue); however, the framework as a whole retains the coherence and heuristic power of the fast–slow distinction (Abed, this issue; Belsky, this issue; Brüne, this issue). In other words, the expanded life history framework takes advantage of the (partially) hierarchical nature of behavioral systems to reduce the complexity of the phenotypic landscape. By comparison, the "integrated evolutionary theory" proposed by Martel suffers from a surprising lack of integration; although I appreciate the need to extend the framework beyond the fast–slow distinction, I do not think it would be productive to treat natural selection, sexual selection, and life history evolution as mutually exclusive phenomena. In fact, these evolutionary processes intersect and overlap at all levels (e.g., Geary, 2002; Höglund & Sheldon, 1998; Ryan, 1998), and the evolutionary history of the mechanisms involved in the etiology of mental disorders will often reflect a combination of all three.

In principle, the expanded framework sketched in Figure 1 is compatible with the Research Domains Criteria (RDoC) promoted by the National Institute of Mental health (Cuthbert & Insel, 2013; Troisi, this issue). The RDoC system originated in neurobiology and was developed in a bottom-up fashion starting from the level of individual mechanisms (the lower left corner of Figure 1). As a result, it lacks a clear sense of functional hierarchy in the organization of behavior. In short, I do not see RDoC criteria as an alternative to the life history framework but as a *subset* of it. My prediction is that the RDoC system will be forced to move toward a hierarchical approach, as a purely bottom-up approach to classification will prove inadequate to account for comorbidity patterns, epidemiological and developmental findings, and so forth. Also, one should remember that RDoC criteria have yet to produce anything resembling an alternative taxonomy of mental disorders; by expanding the life history framework down to the level of individual mechanisms (Brüne, this issue; Tops, this issue), evolutionary psychopathology may be able to coopt the RDoC system and assimilate it within a broader biological perspective.

How General Is the Fast–Slow Distinction?

The diagram in Figure 1 should help clarify the idea that life history strategies "set the stage" for the development of psychopathology. I am emphatically not making the assumption that mental disorders necessarily *originate* at the level of life history strategies (top left of Figure 1); the primary causal factor in the etiology of a given disorder may well be a dysfunction of a specific neurobiological mechanism, or a global reduction in neurological integrity (Gangestad, this issue; Yeo et al., this issue). However, to the extent that life history strategies organize the lower levels of the hierarchy, they can still modulate the

degree to which individual neurobiological mechanisms are vulnerable to dysfunctions, moderate the effect of reduced neural integrity on the expression of specific symptoms, and so forth. Thus, subtypes of current diagnostic categories may be either *mediated* or *moderated* by life history strategy depending on which causal factors are identified as primary, as rightly noted by Gangestad. However, this would be a problem only if the fast–slow distinction were the *only* admissible level of analysis; in a multilevel framework, the fast-slow distinction can be meaningfully applied even when the causal primacy of a disorder lies elsewhere.

As the framework develops and we learn more about the etiological processes of psychopathology, it will be useful to label some conditions as fast or slow spectrum *disorders*, and other conditions as fast or slow spectrum *variants* or *subtypes* of the same disorder. A third possibility—subtly different from the first two—is that some "conditions" presently recognized as such are best understood as nonspecific defense reactions, much like fever or cough. Both depression and generalized anxiety are potential candidates for this role. To the extent that (say) depression turns out to be similar to fever, it should be properly treated as a *symptom* cutting across taxonomic distinctions, although it may still be associated with specific fast or slow spectrum conditions. However, the fast–slow distinction should remain extremely helpful in capturing broad patterns of comorbidity and epidemiological risk, regardless of whether the relevant conditions/symptoms are mediated, moderated, or merely associated with life history variation.

Finally, I agree that some disorders may fall completely outside the fas–slow distinction because their causes and/or symptoms are effectively insulated from the rest of the behavior control hierarchy (e.g., they are independent of premorbid personality and cognition, developmental and familial risk factors, and the presence of typically comorbid disorders; see Gangestad, this issue; Polimeni & Reiss, this issue). This group is likely to include most mental disorders caused by brain injury or degeneration, acute infections, and the side effects of substances/medications. Such conditions are already treated as discrete diagnostic categories in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed. [*DSM*]; American Psychiatric Association, 2013), and should not prove especially problematic for a functional taxonomy as long as they represent the exception rather than the rule.

"State" versus "trait" disorders. Some commentators (Kennair, this issue; Troisi, this issue; Yeo et al., this issue) suggest that a life history approach may be more applicable to chronic, persistent, traitlike conditions such as personality disorders than to acute, malleable, statelike conditions such as OCD. Here I disagree; although important in its own respect, the distinction between "state" and "trait" disorders is completely irrelevant to that between fast and slow spectrum psychopathology. A stable configuration of motivational and self-regulatory traits can easily increase the risk for acute, transient disorders—for example, in response to stressful events, changes in life conditions, or hormonal changes like those of puberty and pregnancy (incidentally, the distinction between Axis I and Axis II disorders in the *DSM* was abandoned partly because of the extremely high comorbidity between the two "axes"). Conversely, the effects of brain injury may be permanent even if the condition does not fit the fast–slow distinction at all.

The equation between stable life history strategies and traitlike disorders might reflect a specific misconception about the framework—namely, that life history strategies must play a primary causal role in the etiology of each and every disorder (see earlier). In a multilevel framework, however, causal pathways can be fairly indirect, and the stability of the underlying strategy has no direct relevance to the stability (or malleability) of the associated disorders. It should also be noted that life history strategies are not cast in stone; in a long-lived species like ours, there are reasons to expect a degree of continuing plasticity, with opportunities for strategic revision in response to changes in the environment or individual condition (see Del Giudice & Belsky, 2011).

Integrating Function and Functionality

A complete framework for psychopathology must integrate two distinct but complementary aspects of the phenotypic landscape—the *function* of behavior and the *functionality* of the neural processes that control it. I thank Yeo et al. for raising the issue so clearly in their commentary. Although the two hierarchies shown in Figure 1 are conceptually independent, there are reasons to predict correlations and interactions between them. At the lower hierarchical levels, the balance between different cognitive abilities may vary along the fast–slow continuum, as postulated for example by Woodley's CD-IE hypothesis (Figueredo et al., this issue). Conversely, a scarcity of attentional and mnemonic resources is likely to constrain the effectiveness of self-regulatory processes (including executive functions; see Yeo et al., this issue).

The apical levels of the two hierarchies represent individual variation on the fast–slow continuum and in the overall level of neurological integrity/efficiency. To the extent that both respond to stressors of

various sorts, one would expect an environmentally induced correlation between fast life histories and reduced neurological integrity. This effect should be compounded by the lower levels of somatic investment associated with fast life history strategies, which may result in reduced buffering of deleterious mutations (Yeo et al., this issue), less effective maintenance and repair of brain tissue, and so forth. Condition-dependent effects in life history development (Gangestad, this issue) can also be expected to generate correlations between life history strategies and neurological integrity, although the sign might be reversed in males. In total, it is reasonable to predict a small degree of correlation between fast life history strategies and reduced neurological integrity/efficiency. Although undergraduate samples show extremely small correlations (less than .10) between g and life history measures such as the K-factor (Figueredo et al., this issue), the absence of effect may be explained by range restriction in both g and environmental stress. Also, the association between life history and neurological integrity needs not be linear across the range of possible environments, and may become especially strong at the very highest levels of environmental harshness and deprivation—that is, well outside the range sampled by most nonclinical studies.

What about the p factor? The concepts discussed in this section make it possible to reconsider the place of the "p factor" within the life history framework (Belsky, this issue; Yeo et al., this issue). In a recent study, Caspi and colleagues (2014) identified a general factor of psychopathology accounting for the comorbidity among a diverse set of disorders (dependence from alcohol, tobacco, cannabis, and hard drugs; conduct disorder; major depression; generalized anxiety disorder; phobias; OCD; mania; and schizophrenia), above and beyond the standard internalizing and externalizing factors. In the statistical model favored by the authors, the p factor was most strongly associated with mania, schizophrenia, depression, and generalized anxiety disorder. The smaller loadings of "externalizing" disorders must be interpreted with care, as this category consisted almost entirely of various types of substance abuse—that is, conditions associated with the externalizing spectrum but not strictly part of it (se Yeo et al., this issue, for evidence of heterogeneity within this category).

Higher levels of p were associated with reduced neural integrity and general cognitive ability, reduced executive functioning, higher Neuroticism, lower Agreeableness and Conscientiousness, and higher levels of developmental stress (lower socioeconomic status and higher frequency of maltreatment in childhood). In light of all these findings, the p factor can be interpreted as a combination of *fast life history strategy* and *reduced neurological integrity/efficiency* (top right of Figure 1). Note that, if neurological integrity and life history strategy were correlated at the population level (see earlier), their correlation would contribute to reinforce the p factor. Also note that Figueredo et al.'s hypothesis of higher comorbidity in fast strategies implies a stronger pattern of correlations among fast spectrum disorders, which would further "pull" a general factor toward the fast end of the continuum.

This updated account of the p factor is fully consistent with the expanded life history framework delineated in this section. Indirect support for this interpretation comes from the finding that, after controlling for p, the externalizing and internalizing factor in Caspi et al.'s dataset became negatively correlated. From a life history perspective, the internalizing spectrum is best understood as a heterogeneous mixture of fast and slow spectrum conditions (see the target article); controlling for p would have the effect of removing a considerable proportion of fast spectrum variance from internalizing disorders, leaving a negative correlation between the externalizing and internalizing factor as a statistical "shadow" of the fast-slow continuum. However, as noted by Belsky, a direct test of this interpretation of the p factor would require epidemiological data based on functional subtypes rather than standard *DSM* categories.

On the Building Site: Analysis and Classification of Specific Conditions

In the second half of the target article I applied the framework to a diverse set of mental conditions, with the goal of building a provisional taxonomy based on the fast–slow distinction. It was exciting to see commentators extend the framework to borderline personality disorder (BPD; Brüne, this issue) and alcohol abuse (Yeo et al., this issue). Some commentators built on my initial classification by offering conceptual refinements and additional evidence, whereas other criticized my analysis or voiced skepticism about the applicability of the fast–slow distinction to certain types of conditions. In this section I respond to these comments. I begin by briefly considering the place of *DSM* categories in an evolutionary taxonomy, then go on to discuss each individual category in turn.

DSM Categories: Accept, Reject, or Revise?

The first question raised by a proposal for an alternative taxonomy is, what should be done with existing diagnostic categories? The commentators have strikingly different answers to this question. At one

extreme, Martel (this issue) seems to fully accept the validity of *DSM* categories, and rejects my proposal of splitting them into functionally divergent subtypes. At the other extreme, Crespi (this issue) advocates the eventual dissolution of psychiatric nosology into a highly multidimensional, mechanism-centered, personalized approach to psychopathology. Meanwhile, Kennair (this issue) argues that a proper evolutionary taxonomy should be restricted to harmful dysfunctions (Wakefield, 1999), excluding both adaptive but undesirable strategies and the negative outcomes of properly functioning mechanisms.

My approach to these thorny issues is a pragmatic one. As I made clear in the target article, I believe that *DSM* categories are in many ways inadequate and should be heavily revised. I also believe that an alternative evolutionary taxonomy will have to develop organically over time, through a combination of top-down (e.g., the fast–slow distinction) and bottom-up approaches (e.g., the RDoC system and Crespi's proposal). In my view, subtyping existing disorders is a first effective step in this direction, with the understanding that current labels and criteria may have to be abandoned or replaced along the way.

The choice of keeping the conventional label "disorder" for all currently diagnosable conditions, regardless of whether they fit the harmful dysfunction criterion, is similarly pragmatic and provisional. However, I believe that a rigid application of the harmful dysfunction criterion would likely prove too restrictive, leaving out too much treatable suffering to be a satisfactory option (see Cosmides & Tooby, 1999; Del Giudice & Ellis, in press). Even if conditions such as BPD or OCPD were conclusively proven to reflect adaptive strategies, they would have to be included in any taxonomic system with real-world applicability.

Attention Deficit/Hyperactivity Disorder

Although I did not discuss attention deficit/hyperactivity disorder (ADHD) in the target article, a number of commentators (Abed; Brüne; Martel) make the assumption that ADHD belongs in the fast spectrum of psychopathology. This makes sense if one considers (a) the high comorbidity between ADHD and externalizing disorders (Nigg, 2013); (b) the high impulsivity, low conscientiousness, and steep time discounting found in ADHD (Demurie, Roeyers, Baeyens, & Sonuga-Barke, 2012; Martel, 2009; Nigg, 2013); (c) the predictive association between ADHD in childhood and increased risk-taking in adulthood (Ramos-Olazagasti et al., 2013); (d) the robust association with low socioeconomic status in childhood (Larsson, Sariaslan, Långström, D'Onofrio, & Lichtenstein, 2014; Russell, Ford, Rosenberg, & Kelly, 2014); and, in the context of my life history taxonomy, (e) the high comorbidity, familiarity, and predictive association between ADHD, schizophrenia, and bipolar disorder (Dalsgaard et al., 2014; Hamshere et al., 2013; Larsson et al., 2013).

Despite this pattern of convergent findings when ADHD is considered as a whole, nearly everyone agrees that the diagnostic category of ADHD is highly heterogeneous (see Fair, Bathula, Nikolas, & Nigg, 2012; Martel, Goth-Owens, Martinez-Torteya, & Nigg, 2010). Moreover, the standard *DSM* distinction between *predominantly inattentive*, *predominantly hyperactive/impulsive*, and *combined* subtypes is not very accurate (e.g., the inattentive subtypes includes many individuals with "subthreshold" hyperactive/impulsive symptoms; see Martel et al., 2010; Martel, Roberts, Gremillion, von Eye, & Nigg, 2011).

Although there is no room here for a detailed analysis of ADHD, it is worth considering two recent findings. First, when inattention and hyperactivity/impulsivity levels are measured in a bifactor model (i.e., controlling for a general ADHD factor), inattention is no longer associated with disinhibition, impulsivity, and externalizing behaviors; moreover, specific inattention is only weakly correlated with lower conscientiousness and predicts higher levels of agreeableness and withdrawal/depression (Martel et al., 2011). Second, a groundbreaking study by Martel and colleagues (2010) identified four main subtypes of ADHD based on personality profiles; although most children with ADHD (78.1%) showed "poor control" or "extraverted" profiles characterized by combined symptoms and high aggression, a minority showed profiles characterized by inattention symptoms, elevated levels of withdrawal/depression, and low aggression—an "introverted" subtype (about 10%) and a high-conscientiousness "perfectionistic" subtype (about 1%).

Taken together, these findings indicate that, although most cases of ADHD clearly belong in the fast spectrum of psychopathology, there seems to be a minority of cases (probably around 10%) whose profile of personality and symptoms is more consistent with a slow spectrum condition. This subset is primarily characterized by inattention rather than hyperactivity/impulsivity but overlaps only in part with the inattentive subtype of the *DSM* (see Martel et al., 2010). Of course, there is still a lot of work to do on ADHD subtypes, and future studies will surely improve on these initial findings. However, the available evidence should prompt evolutionarily minded researchers to look at ADHD with a fresh eye and explore the possibility of functionally distinct subtypes along the fast–slow axis of variation.

Borderline Personality Disorder

In his commentary, Brüne performs a remarkably detailed analysis of BPD from a life history perspective, moving beyond my initial emphasis on motivation to consider a range of neuropsychological, neurobiological, and genetic findings. I have only two comments on Brüne's analysis. First, "harm avoidance" in the TCI (Cloninger, Svrakic, & Przybeck, 1994) is not a measure of harm prevention in the sense of the target article, and should not be interpreted as an indicator of slow life history (as I mistakenly did while discussing OCD in the target article). The harm avoidance dimension captures a mixture of worry, pessimism, fearfulness, shyness, fatigability, and lack of energy—essentially, a combination of low extraversion and high Neuroticism (De Fruyt, Van De Wiele, & Van Heeringen, 2000). Higher levels of harm avoidance predict increased risk of both self-mutilation and suicide in BPD patients—quite the opposite of a self-protective strategy (Joyce, Light, Rowe, Clonginger, & Kennedy, 2010; Korner, Gerull, Stevenson, & Meares, 2007).

My second comment is about disgust as a life history correlate. Current research on disgust sensitivity recognizes the existence of multiple, functionally distinct domains of disgust; for example, Tybur, Lieberman, and Griskevicius (2009) distinguished between *pathogen*, *moral*, and *sexual* domains of disgust sensitivity. Both moral and sexual disgust are associated with high Conscientiousness, high Agreeableness, and low psychopathy—as expected of a slow life history correlate (see Tops, this issue, for related evidence). However, pathogen disgust shows none of these effects (Tybur et al., 2009). In short, self-disgust in BPD patients should be better understood in this framework—and, if possible, differentiated from low self-esteem—before it can be treated as a valid indicator of life history strategy.

Substance Abuse

In the target article, I briefly mentioned substance abuse as a frequent correlate of externalizing spectrum disorders. Yeo et al. (this issue) examine alcohol abuse from a life history perspective and suggest that specific subtypes of alcohol abuse can be mapped on the distinction between fast spectrum (Type 2/Type B) and slow spectrum psychopathology (Type 1/Type A). I find their analysis compelling; my only critical note is that harm avoidance should not be treated as a correlate of slow life history (see my earlier discussion here of BPD). If it can be successfully extended to substances other than alcohol, Yeo et al.'s analysis may help explain the co-occurrence of substance abuse with slow spectrum disorders, with no need to invoke the problematic concept of "switching" between opposite ends of the continuum (Troisi, this issue).

The Autism Spectrum

My classification of autism spectrum disorders (ASDs) as a (possibly heterogeneous) subset of slow spectrum psychopathology attracted a number of comments. Figueredo et al. (this issue) elaborate on my initial analysis and, on the basis of their SD-IE theory, suggest that autistic-like phenotypes represent specialized "morphs" of slow life history strategies in humans (for a similar argument, see Del Giudice, Angeleri, Brizio, & Elena, 2010). Polimeni and Reiss (this issue) state that a life history framework does not easily capture autism, but they do not explain why. Gangestad (this issue) notes that my life history analysis of autism is still conjectural, and of course I agree. Autism is a complex pathology that has proven exceedingly hard to understand. In general, I believe it is a good idea to approach ASDs from the side of autistic-like traits; when disorders are on a dimensional spectrum with normative variation, the milder variants may be more revealing of the underlying functional logic—especially if severe cases of the disorder involve compromised neurological functionality. So far, autistic-like traits have been shown to predict restricted sociosexuality, increased investment in long-term relationships, and low levels of impulsivity and sensation seeking (Del Giudice et al., 2010; Del Giudice, Klimczuk, Traficonte, & Maestripieri, in press), consistent with a slow life history strategy. Although more research is needed, the initial findings are definitely encouraging.

Martel (this issue) criticizes my inclusion of ASDs in the slow spectrum, and goes on to argue that autism should be reclassified as a fast spectrum pathology. She bases her argument on two lines of evidence: (a) the overlap between ASDs and ADHD, and (b) the findings of executive dysfunctions and reduced effortful control in individuals with ASDs. The data on executive functioning in ASDs, however, are notoriously inconsistent (e.g., Geurts, Corbett, & Solomon, 2009; Van Eylen et al., 2011). Literature reviews usually conclude that the evidence for specific inhibition deficits—that is, inhibition deficits that are not better explained by reduced flexibility or working memory—is especially inconclusive (see Geurts, de Vries, & van den Bergh, 2014). Because disinhibition is the only robust executive correlate of fast strategies (see the target article), I do not think the current evidence offers a compelling rationale for reclassifying ASDs as fast spectrum disorders.

The overlap between ASDs and ADHD is a trickier problem for my classification. Previously, I noted how ADHD is a heterogeneous category, with a subset of cases that seems consistent with a slow life

history strategy. If my classification of ASDs is correct, the overlap with the autistic spectrum should be largely restricted to the "slow" subtypes of ADHD, characterized by a strong inattention component (see earlier). This prediction is supported by a recent study by Polderman and colleagues (2013). In a population sample of adults, autistic-like traits correlated with attention problems but *not* with hyperactive traits; moreover, the correlation between attention problems and autistic-like traits was entirely explained by a shared genetic factor. The picture, however, becomes less clear in studies of children with ASD and ADHD symptoms, which typically show elevated rates of both inattention and hyperactivity (e.g., van der Meer et al., 2012).

My hypothesis is that hyperactive symptoms in children with ASD are functionally different from those observed in "pure" ADHD. This is not a far-fetched idea if one considers the vagueness of hyperactive symptoms as described in the *DSM*. Fidgeting, tapping one's hands or feet, leaving one's seat in inappropriate situations, not waiting for one's turn in conversation, talking excessively, interrupting others—all these "hyperactivity" symptoms may be easily explained as arising from repetitive behaviors and/or mindreading deficits. Although this is just a hypothesis at the moment, it is noteworthy that hyperactivity symptoms in ASDs are strongly correlated to levels of stereotypic/repetitive behavior (Martin, Hamshere, O'Donovan, Rutter, & Thapar, 2014; Rao & Landa, 2014; Stratis & Lecavalier, 2013), and—contrary to expectation—do not seem to be associated with motor disinhibition (Sanderson & Allen, 2013).

The Schizophrenia Spectrum

A number of commentators challenge my account of schizophrenia spectrum disorders. Martel expresses skepticism about the connection between schizotypy and mating but does not back up her remarks with evidence or counterarguments. Gangestad notes the conjectural nature of my hypothesis. Yeo et al. take me to task for failing to consider the role of neurological dysfunction and mutation load; I hope my discussion in the previous section has contributed to fill this gap. In this regard, it is important to stress that a central role of mutation load in schizophrenia is fully consistent with the sexual selection model (SSM) advocated in the target article (see Del Giudice, 2010). Conversely, the SSM is not *only* concerned with deleterious mutations, as wrongly assumed by Polimeni and Reiss. In the SSM, schizophrenia risk involves two distinct sources of genetic variation—deleterious mutations and schizotypy-increasing alleles (Del Giudice, 2010)—which may correspond to the two genetic factors hypothesized by Yeo et al. (this issue).

Troisi (this issue) criticizes the sexual selection model of schizophrenia for concentrating on positive symptoms while basically ignoring negative symptoms, in spite of their clinical importance. I agree that this is a weak spot in the theory, and I hope that future research will clarify the functional role of negative symptoms. In my own research, negative schizotypy in the normative range does not seem to uniquely predict any life-history-related outcome (Del Giudice et al., 2010, in press). In severe psychosis, chronic negative symptoms may partly reflect the long-term effects of neurological damage.

On a minor note, I disagree with Troisi's reading of the historical review by Alvarez Ariza, Mateos Alvarez, and Berrios (2009) as showing that unmedicated bipolar disorder does not remit or improve with age. On the contrary, the studies cited by Alvarez Ariza and colleagues reported extremely high recovery rates after one or few episodes. Chronic cases of bipolar disorder were rare and usually involved patients with late-onset forms of the disease (which are often associated with degenerative neurological conditions; see Mitchell, Hadzi-Pavlovic, & Loo, 2011).

In their commentaries, Abed (this issue) and Polimeni and Reiss (this issue) present their own hypotheses on the evolution of schizophrenia. Here I do not address the validity of these hypotheses, which to some extent may be compatible with a life history approach (Abed, this issue). I appreciate Polimeni and Reiss's point about the frequent magico-religious content of positive symptoms. This aspect of the disorder is often underplayed in the literature and may enrich existing theories about the potential reproductive benefits of schizotypy. I also thank Abed for highlighting the broader social factors involved in the epidemiology of psychosis; although I am skeptical of claims that schizophrenia did not exist before the 18th century (see Fraguas, 2009; Heinrichs, 2003), I agree that a satisfactory model of the disorder must be able to account for the observed effects of migration and urbanization. This is a very promising topic for evolutionary research in this area.

Obsessive-Compulsive Disorder

Most comments on OCD focused on the distinction between reactive and autogenous obsessions. The commentators expressed doubts about the autogenous subtype based on their clinical experience (Kennair, this issue; Polimeni & Reiss, this issue), or challenged the distinction based on the partial overlap between the two kinds of obsession (Kennair, this issue; Martel, this issue). Whereas Polimeni and Reiss correctly noted that self-reports of impulsivity have questionable validity, some studies have found the same results with laboratory-based inhibition tasks (see the target article

for references). In my opinion, the data are sufficiently robust to warrant serious consideration of the reactive–autogenous distinction. Still, there may be better ways to subtype the disorder, for example, based on personality profiles (as with eating disorders and ADHD) or neuropsychological parameters (e.g., Besiroglu et al., 2011). If—as I suspect—reactive and autogenous obsessions are merely pointing to a more fundamental distinction within OCD, their mutual overlap will cease to be problematic once a better taxonomy is developed. Clearly, more research in this area is badly needed, especially with clinical populations of patients with OCD, ASDs, and schizophrenia spectrum disorders. Tops's (this issue) hypothesis about grooming behavior in OCD as a response to novelty is another intriguing topic for future studies.

Eating Disorders

In my discussion of eating disorders, I argued that the standard distinction between anorexia nervosa (AN) and bulimia nervosa (BN) has very limited functional meaning and that personality subtypes offer a much better starting point for an evolutionary taxonomy. Abed (this issue) complements my treatment with an in-depth discussion of the sexual competition hypothesis and its main evolutionary alternatives. Martel (this issue) is skeptical about personality subtypes and argues that all eating disorders should be reclassified as slow spectrum conditions, although she offers no supporting evidence for her proposal. Other commentators agree that eating disorders cut across the fast–slow continuum but assume (incorrectly) that AN is a slow spectrum condition and BN is a fast spectrum condition (Abed, this issue; Brüne, this issue; Troisi, this issue). As I stress in the target article, this equation does not hold. Whereas fast strategies are typically associated with BN, slow strategies can be associated with both AN and BN; moreover, many patients move between AN and BN diagnoses in different phases of their disorder. Framing the analysis of eating disorders in terms of AN versus BN is likely to obfuscate the topic rather than illuminating it.

Finally, Polimeni and Reiss argued that eating disorders are an evolutionary novelty and as such cannot be explained in a life history perspective. However, as Abed notes, the idea that eating disorders emerged from a recent mismatch between human eating/mating psychology and current nutritional/social environments is in no way incompatible with a life history framework. Of course, life history strategies *alone* cannot fully account for the phenomenology of eating disorders—any satisfactory explanation will require the multiple levels of analysis shown in Figure 1.

Depression

I conclude this section with a brief note on depression. My initial analysis of depression did not yield a clear typology, and in some ways raised more questions than answers. This is not entirely surprising given the multifaceted, elusive nature of depressive disorders (Polimeni & Reiss, this issue). Abed and Kennair (this issue) specifically wonder about the clinical and explanatory value of my provisional classification. I agree with these commentators that the current state of the theory is not optimal. Of course, it is possible that depression is so lacking in specificity that it cannot be meaningfully subtyped (Kennair, this issue); alternatively, some key element may still be missing from our models. I suspect that significant insights could be gained by investigating the interplay between mood regulation and stress responsivity, and I point in that direction in the target article. At this stage in the development of the framework, the most important task is to find the right questions—if the approach is valid, the answers will come in due time.

Conclusion

At the end of this exchange, I see many reasons for excitement. Although not all the commentators see the need for a unifying framework like the one I am advancing, I am thrilled by the enthusiasm that many of them show and by their willingness to give my proposal a serious chance. I think the original idea not only survived this initial round of criticism but came out improved and more sophisticated. Whether the tower will reach the sky or crumble under its own weight, it looks like this project is going to stimulate much new thinking and research in the field. There is nothing more I can ask for.

Note

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Appendix

1. Reanalysis of Bielby et al. (2007)

1.1. Original findings. In their study, Bielby and colleagues (2007) analyzed a set of life history variables—gestation length, litter size, interbirth interval, neonatal body mass, weaning age, and age at sexual maturity—across a wide range of mammalian species. First, they log-transformed the life history variables and regressed them against adult body mass to control for scaling effects. They then proceeded to factor-analyzed them, extracted two factors, and applied an orthogonal rotation algorithm (Varimax) to the unrotated factors. Neither of the resulting factors could be easily interpreted as a fast-slow continuum. Rather, the authors interpreted the factors as two independent life history dimensions, *reproductive timing* (conceptually related to the trade-off between current and future reproduction) and *reproductive output* (conceptually related to the trade-off between offspring quality and quantity).

1.2. Problems with the original analysis. There are two main problems with this analysis. To begin with, statistically controlling for body mass does not just remove the effects of scaling constraints—it also removes part of the variance due to genuine life history trade-offs (Jesche & Kokko, 2009; Roff, 2002, p. 283). At a minimum, the results of analyses of mass-corrected data should be compared with those obtained from the uncorrected variables (see Jesche & Kokko, 2009).

Even more importantly, the authors did not seem to realize that standard exploratory algorithms such as Varimax are *designed* to break down general and bipolar factors to approximate a "simple structure", whereby each of the variables tends to load highly on some of the factors and weakly on the others (see Darton, 1980; Russell, 2002). Even if a general factor *does* exist, it usually disappears in the rotation; when there is a theoretical rationale to expect a general factor (as in this case), the unrotated solution is likely to offer a more meaningful description of the data.

1.3. Reanalysis. I reanalyzed Bielby et al.'s original dataset, which is available as an electronic supplement to their paper. As it turned out, it was impossible to exactly replicate the loadings reported by Bielby et al. Using the same statistical software employed by the authors (SPSS), I tried all the available types of factor extraction and rotation and compared the resulting loadings with those reported in

Table A1. *PCA Results in Mammalia (Whole Sample). PC = principal component. BM = body mass. IBI = interbirth interval. ASM = age at sexual maturity.*

Mammalia	Uncorrected, unrotated		Mass-corrected, unrotated		Mass-corrected, Varimax-rotated	
Variable	PC1	PC2	PC1	PC2	PC1	PC2
Gestation length	.893	.379	.469	.818	.279	.900
Neonatal BM	.791	.572	−.056	.960	−.264	.925
Litter size	−.784	.014	−.579	−.245	−.511	−.366
IBI	.866	−.255	.765	−.169	.783	.002
Weaning age	.815	−.482	.757	−.441	.835	−.266
ASM	.871	−.190	.766	−.011	.750	.156

Bielby et al. (2007). The highest similarity with the published loadings was obtained with principal component analysis (PCA) followed by Varimax rotation. Tucker's coefficient of congruence (*CC*) between the published solution and my reanalysis was $CC > .999$, both in the whole sample of mammals (Mammalia) and in placental mammals (Eutheria). The extremely high value of *CC* indicates that the two solutions are virtually identical, supporting the validity of the present reanalysis.[2]

1.3.1. Mammalia. The unrotated solution obtained from uncorrected life history variables in Mammalia is shown in Table A1 and Figure A1a. As expected, the first unrotated component (70.2% of variance explained) recovered a strong fast-slow axis of variation. All the variables reflecting a slower life history showed positive loadings on this dimension, whereas litter size (an indicator of fast life history) showed a negative component loading.

The unrotated solution obtained from mass-corrected variables is shown in Table A1 and Figure A1b. As can be seen in Figure A1b, the first component (38.4% of variance) recovered a fast-slow axis that was very similar to that obtained from the uncorrected variables. In fact, the congruence between the uncorrected and mass-corrected solution was $CC = .89$ for the first component and $CC = .91$ for the second component, indicating very high similarity in factor structure despite the correction for body mass. Unsurprisingly, the only variable that showed a sizable difference in loading after correction for adult body mass was neonatal body mass (Table A1). The component scores of individual species on the uncorrected fast-slow continuum (PC1 of the uncorrected solution) and the corrected fast-slow continuum (PC1 of the unrotated mass-corrected solution) were positively and moderately correlated ($r = .46$). In other words, individual species tended to have similar positions on the fast-slow continuum before and after correcting for body mass.

The effect of Varimax rotation on the mass-corrected solution is shown in Figure A1b. As expected, the rotation algorithm moved the first component away from the fast-slow dimension. It is important to note that Varimax rotation is *orthogonal*—that is, the components are not allowed to correlate with one another. An oblique rotation (direct Oblimin with $\delta = 0$) of the two components shown in Figure 1 resulted in a between-component correlation of .65 in the uncorrected solution and .32 in the mass-corrected solution (excluding neonatal body mass). A plausible interpretation of these results is that reproductive timing and reproductive output are partially correlated (i.e., non-independent) dimensions of life history variation, with the fast-slow continuum as a superordinate "general factor."

1.3.2. Eutheria. A reanalysis of Eutheria (placental mammals) showed the same pattern of results observed in the whole sample (Table A2 and Figure A2). The uncorrected data showed a strong fast-slow continuum (80.3% of variance; Figure A2a). Again, the first unrotated component of the mass-corrected solution (50.1% of variance) recovered a clear fast-slow axis. The uncorrected and mass-corrected solution were highly similar, with $CC = .98$ for the first component and $CC = .81$ for the second component. Furthermore, component scores on the uncorrected and mass-corrected fast-slow continuum were positively and moderately correlated ($r = .46$).

The effect of Varimax rotation in Eutheria was even more dramatic than in Mammalia. In fact, the rotation algorithm completely dissolved the fast-slow continuum, by moving the components almost 45 degrees away from the original solution (Figure A2b). Oblimin rotation resulted in a between-component correlation of .66 in the

[2]The *CC* is an index of matrix similarity, and can be employed to quantify the similarity of two factorial solutions (Abdi, 2007). A value of $CC > .80$ indicates high similarity, while $CC > .90$ indicates very high similarity (see Horn et al., 1973; Sakamoto et al., 1998).

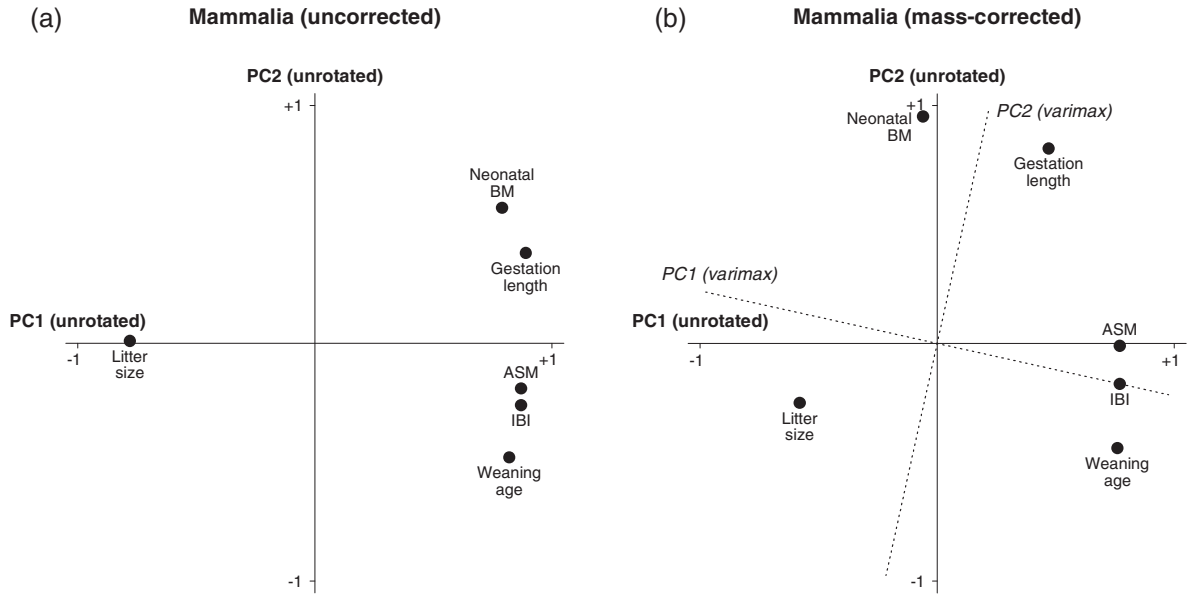

Figure A1. Graphical representation of PCA results in Mammalia (whole sample). PC = principal component. BM = body mass. IBI = interbirth interval. ASM = age at sexual maturity.

uncorrected solution and .42 in the mass-corrected solution (excluding neonatal body mass), again indicating non-independence between reproductive output and timing.

1.4. Discussion. My reanalysis showed that mammalian life histories are characterized by a strong fast-slow axis of variation; in contrast with Bielby et al.'s original findings, the fast-slow continuum could be easily recovered even after controlling for body mass. As hypothesized, Bielby et al.'s failure to identify the fast-slow continuum when analyzing mass-corrected variables was explained by their application of Varimax rotation to the dataset. Unrotated solutions clearly showed a fast-slow axis of variation, which had approximately the same structure regardless of whether uncorrected or mass-corrected variables were analyzed. Interestingly, factor scores on the uncorrected and mass-corrected fast-slow continuum were moderately correlated, contradicting the view that the two methods yield qualitatively different and/or statistically independent continua (see Jesche & Kokko, 2009; Sibly & Brown, 2007).

Of course, the fast-slow axis alone did not fully explain the observed covariation between traits, indicating the existence of more than one dimension of life history variation. The results of oblique rotations suggest that a hierarchical model may provide a better description of the data, with the fast-slow continuum as a general factor and reproductive timing and output as lower-order dimensions.

2. Reanalysis of Jeschke & Kokko (2009)

2.1. Original findings. Jeschke & Kokko (2009) employed PCA to analyze a set of life history variables—age at first reproduction, interbirth interval, lifespan, offspring mass, and fecundity—across

Table A2. *PCA Results in Eutheria (Placental Mammals). PC = principal component. BM = body mass. IBI = interbirth interval. ASM = age at sexual maturity.*

Eutheria	Uncorrected, unrotated		Mass-corrected, unrotated		Mass-corrected, Varimax-rotated	
Variable	PC1	PC2	PC1	PC2	PC1	PC2
Gestation length	.953	−.131	.849	−.229	.483	.736
Neonatal BM	.898	−.118	.402	−.843	−.259	.897
Litter size	−.816	.508	−.793	.447	−.296	−.861
IBI	.897	.340	.625	.647	.897	−.069
Weaning age	.919	.089	.791	.199	.723	.377
ASM	.888	.292	.690	.474	.830	.104

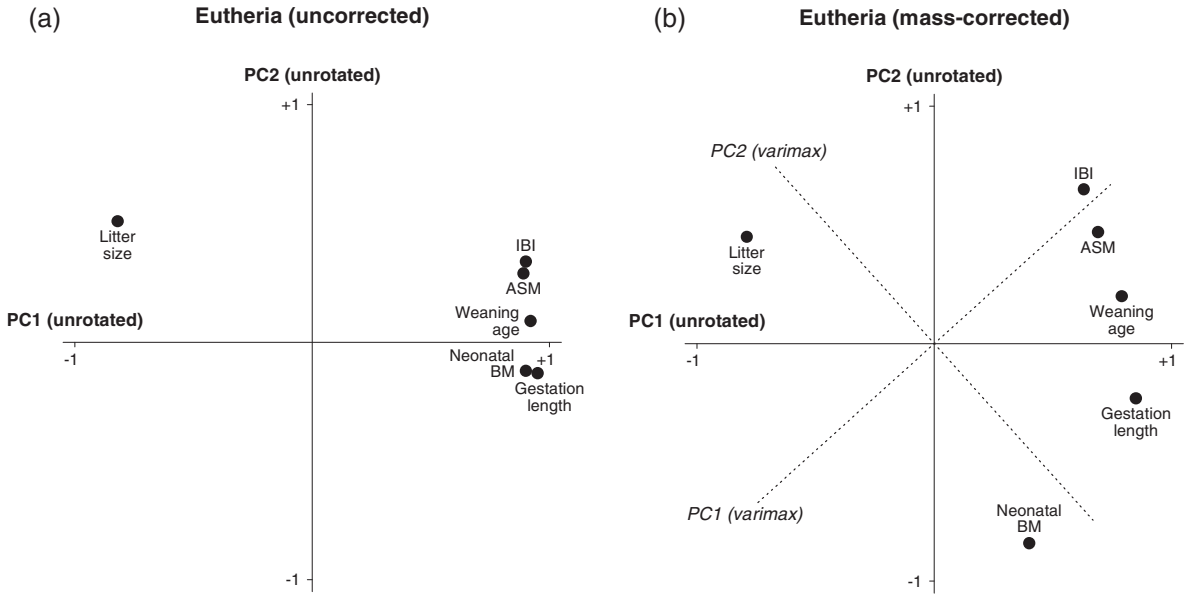

Figure A2. Graphical representation of PCA results in Eutheria (placental mammals). PC = principal component. BM = body mass. IBI = interbirth interval. ASM = age at sexual maturity.

mammals, birds, and fish. They performed PCA both on the uncorrected variables and after correcting for body mass (see above), and compared the resulting solutions to evaluate the stability of the fast-slow continuum across methods of analysis (phylogenetic corrections were also explored in the paper, but will not be discussed here). Unlike Bielby et al. (2007), these authors interpreted the unrotated solutions, and assumed that the fast-slow continuum would be captured by the first unrotated component. Their main findings were: (a) there are qualitative differences among the fast-slow continua observed in mammals, birds, and fish; and (b) analyses based on uncorrected vs. mass-corrected variables yield dramatically different results. My reanalysis is mainly concerned with point (b), i.e., the stability of the fast-slow continuum across different methods of analysis.

2.2. Problems with the original analysis. The original analysis suffers from two problems. First, the authors reported and interpreted a variable's loading on the first component only if it was the *largest* loading of that variable, regardless of its absolute value. For example, in the fish mass-corrected data, lifespan loaded .71 on the first component and .28 on the second component, while interbirth interval loaded .56 on the first component and .59 on the second component (the complete PCA results are available as an electronic supplement to Jeschke & Kokko's paper). Clearly, both of these variables had nontrivial loadings on the first component (i.e., they both contributed to define a fast-slow continuum); however, the authors interpreted these results as indicating that the fast-slow continuum in fish includes lifespan but *not* interbirth interval.

This unusual interpretation of PCA results depends on the idiosyncratic definition of "loading" adopted by the authors: "If ... traits ... are part of a continuum, they will all load on (i.e. correlate most strongly with) the first axis in a principal component analysis" (Jeschke & Kokko, 2009, p. 869). The unstated assumption is that for a trait to be part of the fast-slow continuum, the fast-slow axis must explain more of that trait's variance than any other dimension of life history variation. This assumption, however, is not part of the definition of the fast-slow continuum, and was not justified on biological grounds by Jeschke & Kokko. By relying on such an overly restrictive criterion, the authors may have severely underestimated the stability of the fast-slow continuum across methods of analysis.

The second problem concerns Jeschke & Kokko's analysis of mammalian life histories. In the mass-corrected data for birds and fish, the fast-slow continuum was represented by the first unrotated component, accounting for 43% of variance in fish and 41% in birds. However, mammals showed a slightly different pattern of results; in mammalian species, the first and second component accounted for a similar amount of variance (37% and 29%, respectively), and—in contrast with the fish and bird data—the fast-slow continuum was captured by the *second* unrotated component (see Table S6 in the supplement to Jeschke & Kokko, 2009). Apparently, the authors did not notice this, and went on to interpret the first component as a fast-slow axis of variation. As a result,

Table A3. *Loadings of Life History Variables on the Component Representing the Fast-Slow Continuum in Fish, Mammals, and Birds. UC = uncorrected. MC = mass-corrected. PC = principal component. AFR = age at first reproduction.*

	Fish		Mammals		Birds	
Variable	UC PC1	MC PC1	UC PC1	MC PC2	UC PC1	MC PC1
AFR	.90	.75	.90	.77	.90	.84
Interbirth interval	.67	.56	.73	.73	.52	−.09
Lifespan	.94	.71	.87	.10	.81	.66
Offspring mass	.49	−.49	.95	.24	.93	.42
Fecundity	.85	.73	−.86	−.50	−.40	−.85

they significantly overestimated the discrepancy between the uncorrected and mass-corrected fast-slow continuum in mammals.

2.3. Reanalysis. I recovered the complete loadings of uncorrected and mass-corrected variables from Jeschke & Kokko's supplementary material. Table A3 shows the loadings of each life history variable on the components that best represent the fast-slow continuum. As noted by the authors, the structure of the fast-slow continuum was not invariant across the three clades. However, the fast-slow continuum was almost identical in birds and mammals ($CC = .97$); only the fish data showed substantial discrepancies with mammals ($CC = .54$) and (to a lesser extent) birds ($CC = .70$).

The fast-slow continua obtained from uncorrected (UC) and mass-corrected variables (MC) were also not identical; however, the overall pattern was one of similarity rather than difference. As can be seen in Table A3, almost all the loadings were in the same direction, and most of them were of similar magnitude in the two analyses. This qualitative assessment was confirmed by quantitative indices: coefficients of congruence were $CC = .81$ in fish, $CC = .85$ in mammals, and $CC = .83$ in birds. These values indicate a high level of overall similarity between uncorrected and mass-corrected solutions. Ironically, the highest congruence was observed in mammals, in striking contrast with the original analysis.

2.4. Discussion. My reanalysis showed that the fast-slow continuum is much more stable across methods of analysis than acknowledged by Jeschke & Kokko (2009). In their paper, these authors underestimated the stability of the fast-slow continuum in two ways. First, they employed an overly restrictive criterion for interpreting component loadings. Second, a likely oversight in the analysis of mammals led them to interpret the wrong component as a fast-slow axis of variation. In the reanalysis, I assessed the congruence between uncorrected and mass-corrected solutions based on the full set of loadings, and selected the correct component for the fast-slow continuum in mammals. The results showed high levels of overall similarity in all three clades, consistent with the idea that the fast-slow continuum is reasonably robust to corrections for body mass.

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