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A tower unto Heaven: Toward an expanded framework for psychopathology

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26 April 2024

A Tower unto Heaven:
Response to Commentaries on
"An Evolutionary Life History Framework for Psychopathology"

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Abstract

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 life history 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 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 a number of specific psychopathological conditions: 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, 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. While 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 eighteen commentaries spanned a huge range of topics; while 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 broadest level of analysis, life history variation can be meaningfully described by a single dimension, from "fast" to "slow"; (b) the fast-slow continuum does not only apply 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 came from Surbey, who argued 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; while 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). While 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) cited 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 Supplement, 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 Supplement).

Limitations of the fast-slow continuum. Reports of the death of the fast-slow continuum have been greatly exaggerated. However, 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 invariably show the existence of other meaningful axes of variation (e.g., Sibly and 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-

[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).

offs—current vs. future reproduction and mating vs. parenting—rather than at the level of fast vs. slow strategies.

Another limitation of the fast-slow continuum is the inherent fuzziness of its definition. While 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 Supplement). The same applies to behavioral and physiological 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) noted that humans can and do engage in mixed sexual strategies that involve both short- and long-term mating, and framed this observation as a challenge to the life history framework. In particular, Holtzman & Senne argued 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).

However, the trade-off cannot be completely avoided: the time and resources spent with a long-term partner and his/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 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 below).

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 that life history approaches to animal personality and "coping styles" provide a good role model for this task. I also thank Tops 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" (POLS)—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).

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 in the target article). Even so, behavioral outcomes are always overdetermined, and the associations between traits and strategies are only partial and indirect (Gangestad). 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 super-factors (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' suggestion that simply correlating psychopathological symptoms with "K-factor" scores (see Figueredo et al.; 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 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 and Jonason & Schmitt 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 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 and Schlomer & Cleveland, who argued that behavior genetics (including the study of GxE 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) 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 et al., 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 were discussed by Brüne in his commentary). Although the data on heritability are very robust (Schlomer & Cleveland), 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 GxE 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.). 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., 2010, 2012); 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. While 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 whose predictions 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, and Schlomer, 2009; Kuzawa & Bragg, 2012).

However, uncertainties and discrepancies remain. For example, demographic models predict the evolution of earlier reproduction in response to increased adult mortality, but not juvenile mortality; moreover, changes in mortality that affect both juveniles and adults should have no effect of reproductive schedules (Charlesworth, 1994). In humans, mortality rates are strongly correlated across age groups, and yet higher mortality reliably predicts earlier maturation and reproduction; moreover, *juvenile* mortality seems to be an even better predictor than adult mortality (e.g., Walker et al., 2006; see Ellis et al., 2009). These puzzling findings can be accommodated in a number of ways—for example by positing density-dependent effects in juveniles (Charlesworth, 1994, p. 205 and following), or by assuming that mortality varies across life stages instead of age groups (Roff, 2002, p. 221). 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 presented 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 raised many other interesting points about the environmental determinants of life history strategies. I agree with Jonason & Schmitt's remarks about the importance of short-term mismatches between contradictory environmental contingencies in the etiology of some disorders; a related point was made by Mishra & Gonzales, who stressed the importance of considering acute situational influences on behavior and their interaction with stable dispositions. From a different perspective, Gangestad noted 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 & Schmitt argued 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

In order 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. While 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 et al., 1999; Koolhaas, de Boer, Buwalda, & van Reenen, 2007; Tops). Motivational domains (see Bernard, Mills, Swenson, & Walsh, 2005; Bugental, 2000) provide a powerful criterion for the classification of mental disorders; for example, reactive 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.

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). 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); while they are important pieces in the puzzle of psychopathology, I propose 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; Yeo et al.). 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 (GCA), or *g* (see Deary, Penke, & Johnson, 2010; Moreno-De-Luca et al., 2013). Below 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, males and females 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). While 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).

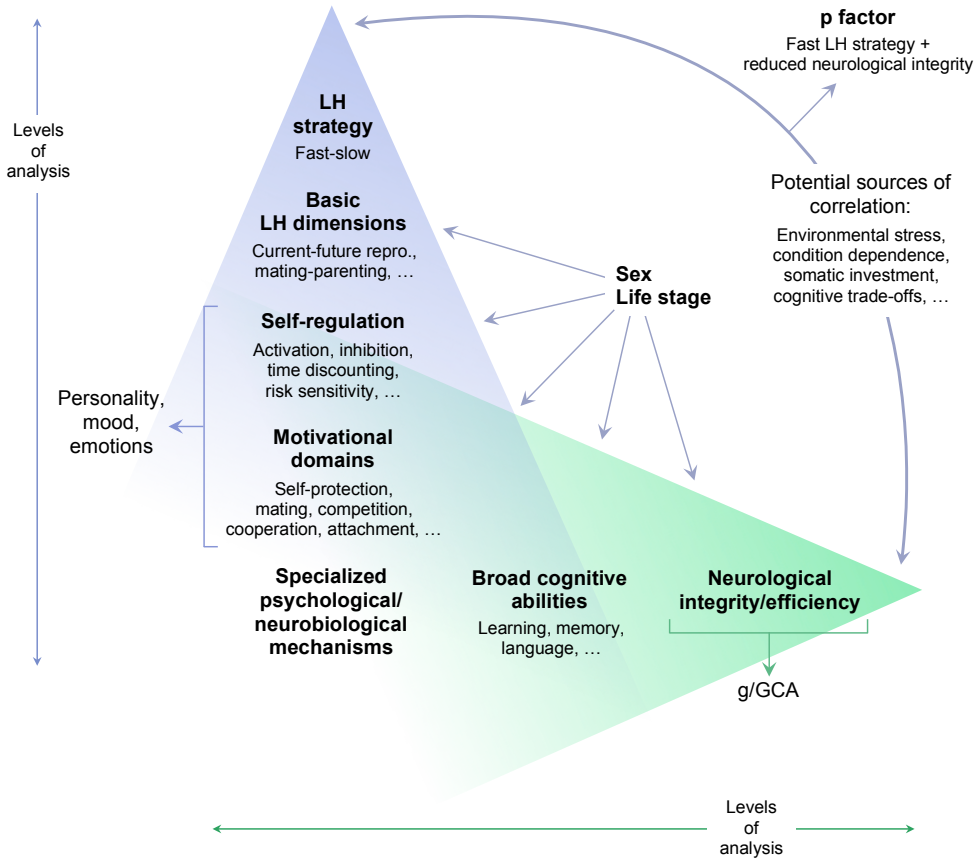

Figure 1. An expanded life history framework for evolutionary psychopathology. LH = life history. GCA = general cognitive ability.

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, Glass); however, the framework as a whole retains the coherence and heuristic power of the fast-slow distinction (Abed, Belsky, Brüne). In other words, the expanded life history framework takes advantage of the 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; while 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 shown in Figure 1 is compatible with the Research Domains Criteria (RDoC) promoted by the National Institute of Mental health (Cuthbert & Insel, 2013; Troisi). 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; Tops), evolutionary psychopathology may be able to co-opt 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 (see the target article). 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; Yeo et al). 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 mechanisms 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 like 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 fast-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; Polimeni & Reiss). 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 DSM, and should not prove especially problematic for a functional taxonomy as long as they represent the exception rather than the rule.

"State" vs. "trait" disorders. Some commentators (Kennair; Troisi; Yeo et al.) suggested that a life history approach may be more applicable to chronic, persistent, trait-like conditions such as personality disorders than to acute, malleable, state-like conditions such as obsessive-compulsive disorder (OCD). Here I disagree; while 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 trait-like 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 above). 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. While 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.). 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.).

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 kinds, 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.), less effective maintenance and

repair of brain tissue, and so forth. Condition-dependent effects in life history development (Gangestad; see above) 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. While undergraduate samples show extremely small correlations (less than .10) between g and life history measures such as the K-factor (Figueredo et al.), 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; in fact, it may become especially strong at the very highest levels of environmental harshness and deprivation—that is, well outside the range sampled by most non-clinical 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; Yeo et al.). 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 [GAD], 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 GAD. The smaller loadings of "externalizing" disorders must be interpreted with care, as this category consisted almost entirely of various types of substance abuse—i.e., conditions associated with the externalizing spectrum but not strictly part of it (se Yeo et al. 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 above), 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) and alcohol abuse (Yeo et al.). Some commentators built on my initial classification by offering conceptual refinements and additional evidence, while 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 had strikingly different answers to this question. At one extreme, Martel seemed to accept the validity of DSM categories, and rejected my proposal of splitting them into functionally divergent subtypes. At the other extreme, Crespi advocated the eventual dissolution of psychiatric nosology into a highly multidimensional, mechanism-centered, personalized approach to psychopathology. Meanwhile, Kennair argued 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 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 be adaptive strategies, they would have to be included in any taxonomic system with real-world applicability.

Attention Deficit/Hyperactivity Disorder

While I had not discussed attention deficit/hyperactivity disorder (ADHD) in the target article, a number of commentators (Abed; Brüne; Martel) assumed 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). While 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; while 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, while 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 performed 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 only have 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 et al., 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 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. examined alcohol abuse from a life history perspective, and suggested 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 discussion of BPD above). 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).

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. elaborated on my initial analysis and, on the basis of their SD-IE theory, suggested 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 & Reiss stated that a life history framework does not easily capture autism, but did not explain why. Gangestad noted 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, in press), consistent with a slow life history strategy. While more research is needed, the initial findings are definitely encouraging.

Martel criticized my inclusion of ASDs in the slow spectrum, and went on to argue that autism should be reclassified as a fast spectrum pathology. She based 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). Since 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 above). 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. While 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

My account of schizophrenia spectrum disorders (SSDs) was challenged by a number of commentators. Martel expressed skepticism about the connection between schizotypy and mating, but did not back up her remarks with evidence or counter-arguments. Gangestad noted the conjectural nature of my hypothesis. Yeo et al. took 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 paper (see Del Giudice, 2010). Conversely, the SSM is not *only* concerned with deleterious mutations, as wrongly assumed by Polimeni & 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.

Troisi criticized 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 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, under review). 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 and Polimeni & Reiss presented their own hypotheses on the evolution of schizophrenia. Here I will not address the validity of these hypotheses, which to some extent may be compatible with a life history approach (Abed). I appreciate Polimeni & Reiss' 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; while 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 mustbe 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; Polimeni & Reiss), or challenged the distinction based on the partial overlap between the two kinds of obsession (Kennair; Martel). While Polimeni & Reiss correctly noted that self-reports of impulsivity have questionable validity, many 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 SSDs. Tops' hypothesis about grooming behavior in OCD as a response to novelty is another intriguing topic for future studies.

Eating Disorders

In my treatment 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 complemented my treatment with an in-depth discussion of the sexual competition hypothesis and its main evolutionary alternatives. Martel was skeptical about personality subtypes and argued that all eating disorders should be reclassified as slow spectrum conditions, although she offered no supporting evidence for her proposal. Other commentators agreed that eating disorders cut across the fast-slow continuum, but assumed (incorrectly) that AN is a slow spectrum condition while BN is a fast spectrum condition (Abed; Brüne; Troisi). As I stressed in the target article, this equation does not hold. While 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 vs. BN is likely to obfuscate the topic rather than illuminating it.

Finally, Polimeni & Reiss argued that eating disorders are an evolutionary novelty, and as such cannot be explained in a life history perspective. However, as noted by Abed, 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 will 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). Abed and Kennair specifically wondered about the clinical and explanatory value of my provisional classification. I agree with these commentators that the current state of the theory is far from optimal. Of course, it is possible that depression is so lacking in specificity that it cannot be meaningfully subtyped (Kennair); 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 pointed 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. While not all the commentators saw the need for a unifying framework like the one I am advancing, I was thrilled by the enthusiasm that many of them showed 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.

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