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**This is the author's manuscript**

*Original Citation:*

*Availability:*

This version is available <http://hdl.handle.net/2318/1853041> since 2022-04-11T00:35:32Z

*Published version:*

DOI:10.1177/2167702615583628

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**The Life History Model of Psychopathology Explains the Structure of  
Psychiatric Disorders and the Emergence of the p Factor: A Simulation Study**

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*Clinical Psychological Science, 4, 299-311 (2016).*

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### **Abstract**

In recent years, tremendous progress has been made in mapping the structure of comorbidity between psychiatric disorders. In particular, empirical findings suggests the existence of a general “p factor” of susceptibility to psychopathology (Caspi et al., 2014). The present study employed simulation methods to test whether the observed structure of psychiatric disorders can be reproduced by the life history model of psychopathology, a recent classification model based on evolutionary theory (Del Giudice, 2014). The assumptions of the life history model were used to generate virtual epidemiological samples, which were then analyzed with the methods employed by Caspi and colleagues. Analyses of simulated data successfully replicated the key findings by Caspi and colleagues, including the emergence of the p factor and the switch from positive to negative correlation between internalizing and externalizing symptoms after including the p factor. These results offer initial support for the validity of the life history model.

*Keywords:* epidemiology; evolution; life history theory; p factor; psychopathology.

Psychiatric disorders are characterized by high rates of comorbidity; as a result, the structure of psychopathology can be described by relatively few broad dimensions of co-occurring symptoms and disorders (e.g., Lahey et al., 2011; Verona, Javdani, & Sprague, 2011; Watson, O'Hara, & Stuart, 2008). In recent years, tremendous progress has been made in mapping the structure of comorbidity between common disorders. Current models largely converge on the distinction between *externalizing disorders* marked by antisocial and rule-breaking behaviors and *internalizing disorders* characterized by anxiety, fear, and distress (Krueger, Caspi, Moffitt, & Silva, 1998; Watson et al., 2008). In a recent study, Caspi and colleagues (2014) built on previous findings by Lahey et al. (2012) to show that many common mental disorders can be mapped on a hierarchical model, with a general *p factor* reflecting a dimension of generalized susceptibility to psychopathology, above and beyond the externalizing-internalizing distinction.

While these models have considerable descriptive power, they are limited in their *explanatory* value, as they reflect empirical generalizations rather than predictions derived from theoretical principles. Linking psychopathological factors to individual differences in affective experience (positive and negative affect, fear, distress, and so forth) helps make sense of empirical taxonomies (Watson, 2005; Watson et al., 2008). However, the structure of affective experience is itself an empirical fact in need of explanation, and cannot substitute for a functional theory of comorbidity.

In a series of recent publications, I advanced an integrative framework for psychopathology based on the concepts of evolutionary life history theory (Roff, 2002; Stearns, 1992; see Del Giudice, Gangestad, & Kaplan, in press), and used it to derive a theoretically grounded model of comorbidity between psychiatric disorders (Del Giudice, 2014a, 2014b; Del Giudice et al., 2014). In those papers, I argued that the life history model explains the observed structure of psychopathology, including the emergence of the *p factor*. However, according to the model neither the externalizing-internalizing distinction nor the *p factor* accurately reflect the deep functional structure of psychopathology; instead, the large-scale structure of psychiatric disorders is best understood as reflecting a basic dimension of *fast vs. slow life history strategy* coupled with a (largely independent) dimension of *neurological integrity/efficiency* (see Del Giudice, 2014b; more on this below). If the life history model were empirically supported, it would provide a theory-driven explanation for the observed structure of psychiatric disorders, as well as constructively challenge the adequacy of current descriptive approaches.

The framework on which the life history model is based is rather complex, and the reader is directed to Del Giudice (2014a, 2014b) for an in-depth exposition. In evolutionary biology, life history theory deals with the fundamental problem of how organisms distribute their time, energy, and other resources between various components of biological fitness such as growth, survival, reproduction, mating, and parenting (Roff, 2002). The behavior, physiology, and developmental trajectories of organisms are ultimately organized by their *life history strategies*—adaptive solutions to the inevitable trade-offs that arise between competing fitness components. At the broadest level of analysis, “fast” strategies are characterized by high mortality, early maturation and reproduction, high fertility, and low investment in offspring, whereas “slow” strategies are characterized by low mortality, late maturation and reproduction,

low fertility, and high investment in offspring quality (Promislow & Harvey, 1990; see Del Giudice et al., in press; Ellis et al., 2009). Life history strategies show remarkable variation between species and individuals, and typically reflect the contribution of both genetic and environmental factors; for example, high levels of danger and unpredictability—that is, environmental stress—tend to favor the development of faster strategies (see Del Giudice et al., in press; Ellis, Figueredo, Brumbach, & Schlomer, 2009).

In humans, individual differences on the fast-slow continuum have been linked to self-regulation traits, such as impulsivity and discounting of future rewards; personality traits such as conscientiousness and agreeableness; motivational traits such as romantic attachment, risk-taking, and sociosexuality (the preference for short-term, uncommitted sexual relationships with multiple partners); physiological traits such as stress reactivity; and developmental traits such as the timing and tempo of sexual maturation (reviewed in Del Giudice, 2014a; Del Giudice, Ellis, & Shirtcliff, 2011; Del Giudice et al., in press; Ellis et al., 2009; Figueredo et al., 2005).

While life history strategies offer an organizing principle for the biological *functions* of behavior, the picture would be incomplete without considering the *functionality* of the neural processes that make behavior possible. The functionality of neural processes can also be described in hierarchical terms, from the level of specific neurological or computational mechanisms to that of broad abilities such as spatial and verbal skills, up to a dimension of generalized neurological integrity/efficiency (or, symmetrically, generalized brain dysfunction; see Del Giudice, 2014b).

The life history framework of psychopathology builds on the theory and evidence outlined above to derive a basic distinction between *fast spectrum* and *slow spectrum* disorders—that is, disorders that cluster at the fast or slow end of the life history continuum. Fast spectrum disorders are expected to show associations with social antagonism, unstable attachments, precocious and promiscuous sexuality, sensation seeking, risk-taking, and impulsivity; low levels of conscientiousness and agreeableness; early, fast maturation; and early exposure to stress and adversity. Slow spectrum disorders, on the contrary, should be associated with social compliance, stable attachments, delayed and restrained sexuality, risk aversion, and behavioral inhibition; high levels of conscientiousness and agreeableness; late, slow sexual maturation; and relatively *low* exposure to social and ecological stressors early in life (for details and a review of empirical findings see Del Giudice, 2014a). The fast-slow distinction can be employed to explain large-scale patterns of comorbidity between apparently separate disorders, but also to tease apart functionally distinct conditions that coexist within the same diagnostic category.

It is important to underscore that, in this framework, life history strategies “set the stage” for the development of psychopathology by determining individual differences in risk profiles. The causal connections between life history strategy and psychopathology are usually indirect, and may involve a variety of specific endogenous and exogenous causal factors, including stressful experiences, deleterious mutations, infections, and so forth (for extended discussion see Del Giudice, 2014a). For this reason, the life history approach does not aim to replace existing evolutionary models of specific mental disorders (for reviews, see Brüne, 2008; Del Giudice, 2014a; McGuire & Troisi, 1998); rather, the goal is to integrate existing theories within a

common conceptual framework and pave the way for a biologically plausible taxonomy of psychiatric conditions.

Based on the concepts outlined above, I advanced a provisional taxonomy of fast and slow spectrum disorders (see Del Giudice, 2014a, 2014b) by synthesizing current evolutionary models with empirical findings relevant to the fast-slow distinction. For conceptual clarity, here I refer to this taxonomy as the life history *model*, as distinct from the broader framework outlined here.

In the current version of the life history model, externalizing disorders (including conduct disorder) and borderline personality disorder (BPD) are classified as prototypical fast spectrum conditions because of their robust associations with early maturation, precocious and unrestricted sexuality, impulsivity, risk-taking, and low levels of agreeableness and conscientiousness (see also Brüne, 2014). Schizophrenia spectrum disorders (SSD), bipolar disorders, attention deficit-hyperactivity disorders (ADHD), and substance-related disorders (see also Yeo, Pommy, & Padilla, 2014) are classified as primarily fast spectrum disorders with a significant degree of heterogeneity. This means they are likely to include a smaller subset of slow spectrum conditions, even if there are still no clear-cut criteria for distinguishing between functional subtypes. Obsessive-compulsive personality disorder (OCPD) is classified as a slow spectrum condition, whereas autism spectrum disorders (ASD) are classified as primarily slow spectrum conditions with a significant degree of heterogeneity. The existing category of obsessive-compulsive disorder (OCD) can be separated into two functionally distinct subtypes: a slow spectrum subtype characterized by “reactive” obsessions and comorbidity with ASD, and a fast spectrum subtype characterized by “autogenous” obsessions and comorbidity with SSD (Lee & Kwon, 2003; Lee & Telch, 2005). Likewise, eating disorders (ED) comprise a slow spectrum “perfectionistic” subtype with primarily anorexic symptoms, and a fast spectrum “dysregulated” subtype including both anorexic and bulimic presentations (Thompson-Brenner et al., 2008a, 2008b). Finally, depressive disorders are provisionally classified as non-specific conditions that may occur anywhere on the continuum, though depression in the fast spectrum is more likely to involve high levels of somatic symptoms (sleep disturbances, eating disturbances, fatigue, agitation, and so forth). Since generalized anxiety disorder (GAD) shares most of the same liabilities of depression (e.g., Hettema, 2008; Lahey et al., 2011), the non-specific classification of depressive disorders can be extended to GAD as well (for details and discussion see Del Giudice, 2014a, 2014b).

The life history model of psychopathology has important implications for the validity of the externalizing-internalizing distinction. From a life history perspective, externalizing disorders represent a functionally coherent cluster of fast spectrum conditions, whereas the “internalizing spectrum” is a heterogeneous mixture of slow spectrum conditions, fast spectrum conditions, and non-specific conditions such as depression and GAD. The life history model predicts that if existing diagnostic categories were differentiated into functional subtypes, the fast-slow continuum would emerge as the main axis in the structure of psychopathology. The internalizing spectrum would largely dissolve, while the externalizing spectrum would be absorbed by the fast pole of the fast-slow continuum. However, current datasets are based on standard diagnostic categories, and thus predictably generate two correlated factors of internalizing and externalizing disorders (Del Giudice, 2014a).

The implications of the life history model may extend to the nature and meaning of the p factor. In the study by Caspi and colleagues (2014), the p factor was moderately associated with low agreeableness, low conscientiousness, and high levels of early stress and adversity—all correlates of fast spectrum psychopathology. Moreover, p factor scores showed smaller correlations with various indices of reduced brain integrity and cognitive impairment. Crucially, the study did not include any disorders classified as specifically or primarily slow spectrum, such as OCPD or ASD; all the investigated conditions were either fast spectrum or heterogeneous disorders. Based on these considerations, I hypothesized that the p factor reflects a combination of fast life history strategy and reduced neurological integrity/efficiency (Del Giudice, 2014b). I also argued that the model explains the surprising finding that the correlation between the externalizing and internalizing factor switches from positive to negative when the p factor is included in the analysis (Caspi et al., 2014).

In total, the life history model claims to provide a sufficient explanation of the observed large-scale structure of psychiatric disorders, including the externalizing-internalizing distinction and the emergence of the p factor. My goal in the present study was to test whether the model can reproduce the structure of psychopathology described by Caspi et al. (2014). To this end, I employed Monte Carlo methods to simulate the distribution of psychiatric symptoms in virtual samples based on the assumptions of the life history model. I then replicated Caspi and colleagues' data analysis on the simulated datasets, and compared the results obtained from the simulated data with those obtained from the real-world epidemiological data. A close match between the simulated and empirical results would indicate that the life history model can successfully reproduce the observed structure of psychopathology, and lend indirect support to the broader theoretical framework (Del Giudice, 2014a, 2014b). Note that this testing strategy is designed to work regardless of the validity and adequacy of the original analysis by Caspi and colleagues (more on this in the discussion).

## Method

Monte Carlo methods were employed to generate psychopathology scores in 1,260 virtual samples of  $N = 100,000$  individuals each. Each sample was characterized by a normal distribution of life history strategy ( $Z_{LH}$ ) and a normal distribution of brain dysfunction scores ( $Z_{BD}$ ), both with  $M = 0$  and  $SD = 1$ . Positive values of  $Z_{LH}$  correspond to fast life history strategies; negative values correspond to slow strategies (Figure 1). Positive values of  $Z_{BD}$  represent above-average values of brain dysfunction (i.e., below-average levels of neurological integrity/efficiency), while negative values represent below-average levels of dysfunction. As described in detail below, these distributions were used to derive individual symptom scores for 11 disorder categories, corresponding to those in Caspi et al. (2014): alcohol dependence, cannabis dependence, dependence on hard drugs, tobacco dependence, conduct disorder (CD), major depression (MDD), GAD, fears/phobias, OCD, mania, and schizophrenia. Since the present study focused on the cross-sectional structure of psychopathology, the longitudinal component of Caspi et al.'s (2014) dataset was not included in the simulation. Simulated datasets were analyzed following Caspi and colleagues (see below), and the results were compared with those obtained in that study. All simulations and statistical analyses were performed in R<sup>TM</sup> 2.15 (R Core Team, 2012; package *sem* 3.1-3).

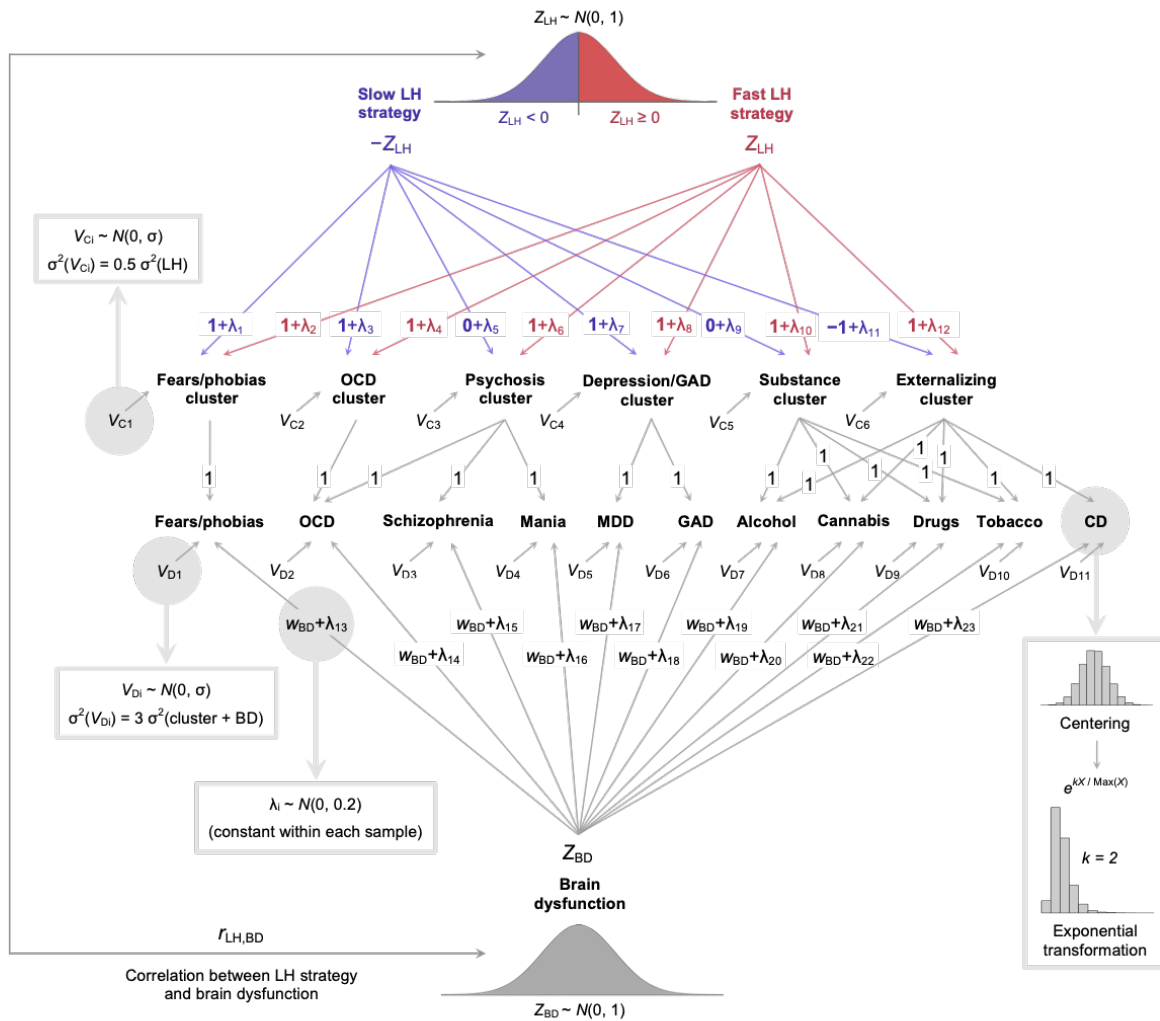


Figure 1. Structure of the simulation. See the main text for explanation. LH = life history; OCD = obsessive-compulsive disorder; MDD = major depressive disorder; GAD = generalized anxiety disorder; CD = conduct disorder.

Two parameters were varied systematically in the study: (1) the correlation between life history strategy and brain dysfunction ( $r_{LH, BD}$ ), and (2) the effect of brain dysfunction on the risk for psychopathology, relative to that of life history strategy ( $W_{BD}$ ). From a theoretical standpoint, life history strategy and neurological integrity should be largely orthogonal; however, some factors may end up affecting both—for example, exposure to early stress is expected to entrain faster life histories, but may also interfere with brain maturation and cognitive development. Conversely, high levels of deleterious mutations may contribute to brain dysfunction while triggering faster strategies in some individuals (discussed in Del Giudice, 2014b). As a result, it is reasonable to expect a modest degree of association between fast life history strategies and reduced neurological integrity. The available empirical data suggest that the correlation is probably smaller than .10 (Figueredo, Woodley, & Fernandes, 2014; Woodley, Figueredo, Brown, & Ross, 2013; discussed in Del Giudice, 2014b). The present study explored three values



of  $r_{LH,BD}$  (.00, .10, and .20) and 21 values of  $W_{BD}$  (from 0 to 1 in increments of .05; see below for details). To check the robustness of the results, 20 independent samples were simulated for each combination of  $r_{LH,BD}$  and  $W_{BD}$ , each time varying the structural parameters of the model by a random amount (see below).

### Simulation Structure and Parameters

The structure of the simulation is shown in Figure 1. Individual scores on the fast-slow continuum ( $Z_{LH}$ ; top of Figure 1) were used to generate risk scores for six clusters of disorders. A degree of clustering was introduced to account for patterns of shared liabilities between individual disorders, above and beyond those introduced by life history variation. Specifically, MDD and GAD were part of a *depression/GAD cluster* (see Hettema, 2008; Lahey et al., 2011), while schizophrenia and mania were part of a *psychosis cluster* (see Cosgrove & Suppes, 2013; Crespi, Stead, & Elliot, 2010; International Schizophrenia Consortium, 2009). In the life history model, the autogenous subtype of OCD (but not the reactive subtype) is regarded as a functional correlate of the psychosis spectrum (Del Giudice, 2014a); to reflect this assumption, OCD was included in two overlapping clusters—the psychosis cluster and a separate *OCD cluster*. The *externalizing cluster* included CD and substance-related disorders; in addition, substance disorder scores were also part of a *substance cluster*, to reflect the fact that substance-related disorders overlap only in part with the externalizing spectrum (see Yeo et al., 2014). Finally, fears and phobias constituted a *fears/phobias cluster* (see Figure 1 for a graphical representation of the clusters). The strength of the effect of clustering was determined by the amount of cluster-specific variance introduced in the model, as discussed in detail below.

**Effects of life history strategy on psychopathology risk.** The effects of life history strategy on risk scores in the six clusters were determined by a set of theory-driven weights (shown in blue and red in Figure 1). Weights were derived from the assumptions of the life history model according to the following scheme. For fast spectrum disorders (such as those in the externalizing cluster), weights were set to +1 in individuals with a fast strategy ( $Z_{LH} \geq 0$ ) and -1 in individuals with a slow strategy ( $Z_{LH} < 0$ ). With this combination of weights, psychopathology risk increases for individuals with faster life histories, and decreases for those with slower life histories. (Note that slow life history scores—i.e., negative values of  $Z_{LH}$ —were reversed in sign before weighting, as shown in Figure 1.) For fast spectrum disorders with a significant degree of heterogeneity (e.g., those in the psychosis cluster), weights were set to +1 in individuals with a fast strategy and 0 in individuals with a slow strategy. Finally, weights for non-specific disorders (e.g., those in depression/GAD cluster) or diagnostic categories comprising both fast and slow spectrum conditions (e.g., the OCD cluster) were set to +1 in both fast and slow strategy individuals. Since the current version of the model makes no strong assumptions about fears and phobias, the latter were treated as non-specific conditions, and both weights for the fears/phobias cluster were set to +1.

The weighting and clustering scheme employed in the simulation was based on a simplified representation of the relations between life history strategies and psychiatric disorders. While this approach foregoes some of the model's nuance, it also leaves no room for “tweaking” the simulation by fine-tuning the parameters to bring the results closer to expectations. If a simplified, bare-boned version of the model can reproduce the empirical data to a reasonable

degree, this indicates that the model is robust and that its predictions do not depend critically on the exact choice of parameter values. As a further check of the model's robustness, 20 independent samples were simulated for each combination of  $r_{LH,BD}$  and  $W_{BD}$ . Each time, weights were changed by a random amount through parameters  $\lambda_{1,...,12}$ . These parameters were generated from a normal distribution with  $M = 0$  and  $SD = .20$ ; this value of the  $SD$  was chosen so that even as weights changed in *magnitude*, they virtually always maintained their theoretically derived *sign*.

**Effects of brain dysfunction on psychopathology risk.** To model the contribution of reduced neurological integrity/efficiency to psychopathology, individual scores on the distribution of brain dysfunction ( $Z_{BD}$ ; bottom of Figure 1) were summed to cluster scores to generate symptom scores for the specific disorders. Brain dysfunction scores were weighted by a parameter  $W_{BD}$ . When  $W_{BD} = 0$ , brain dysfunction had an average effect of zero on psychopathology risk; when  $W_{BD} = 1$ , the average effect of brain dysfunction on disorder scores was comparable to that of life history strategy (the equivalence is not exact because of the variation introduced by clustering). In the study, values of  $W_{BD}$  ranging from 0 to 1 were systematically explored; the main goal was to test whether the fast-slow continuum could generate the expected pattern of findings even without the contribution of brain dysfunction.

To increase the robustness of the model, no further assumptions were made concerning which disorders might be more or less influenced by brain dysfunction (i.e., the same value of  $W_{BD}$  was employed for all the 11 disorders). However, weights for specific disorders were changed by a random amount in each new sample, through parameters  $\lambda_{13,...,23}$  (also generated from a normal distribution with  $M = 0$  and  $SD = .20$ ). This reflects the general assumption that the effects of brain dysfunction are unlikely to be homogeneous across disorders.

**Symptom scores.** As discussed above, symptom scores for the 11 disorders were computed as weighted combinations of an individual's life history and brain dysfunction scores. Moreover, a certain amount of stochastic variation was added to individual scores to model various sources of individual differences not systematically related to either life history or brain dysfunction. Specifically, stochastic variation was partitioned in two components—a cluster-specific component ( $V_{C1,...,C6}$ ) and a disorder-specific component ( $V_{D1,...,D11}$ ). The cluster-specific component represents those individual factors that influence an individual's risk for a certain cluster of disorders, above and beyond the effects of life history strategy and brain dysfunction. The disorder-specific component represents individual factors (both genetic and environmental) that may influence an individual's risk for a specific disorder within a cluster (e.g., a higher risk of depression vs. GAD), plus chance variation in the expression of symptoms and measurement error in symptom scores.

Since disorder-specific variance includes both chance factors and measurement error, it should be considerably larger than cluster-specific variance. Also, clustering is assumed to represent a minor source of variance compared with life history strategy. Exploratory runs showed that the exact amount of cluster-specific variance did not alter the qualitative results of the simulation, as long as cluster-specific variance was smaller than the variance associated with life history strategy (see Figures S1-S2 in the Supplemental Material available online). In all the simulations reported here, cluster-specific variance was set to one half of the variance associated with life history strategy (see Figure 1). The amount of disorder-specific variance was adjusted

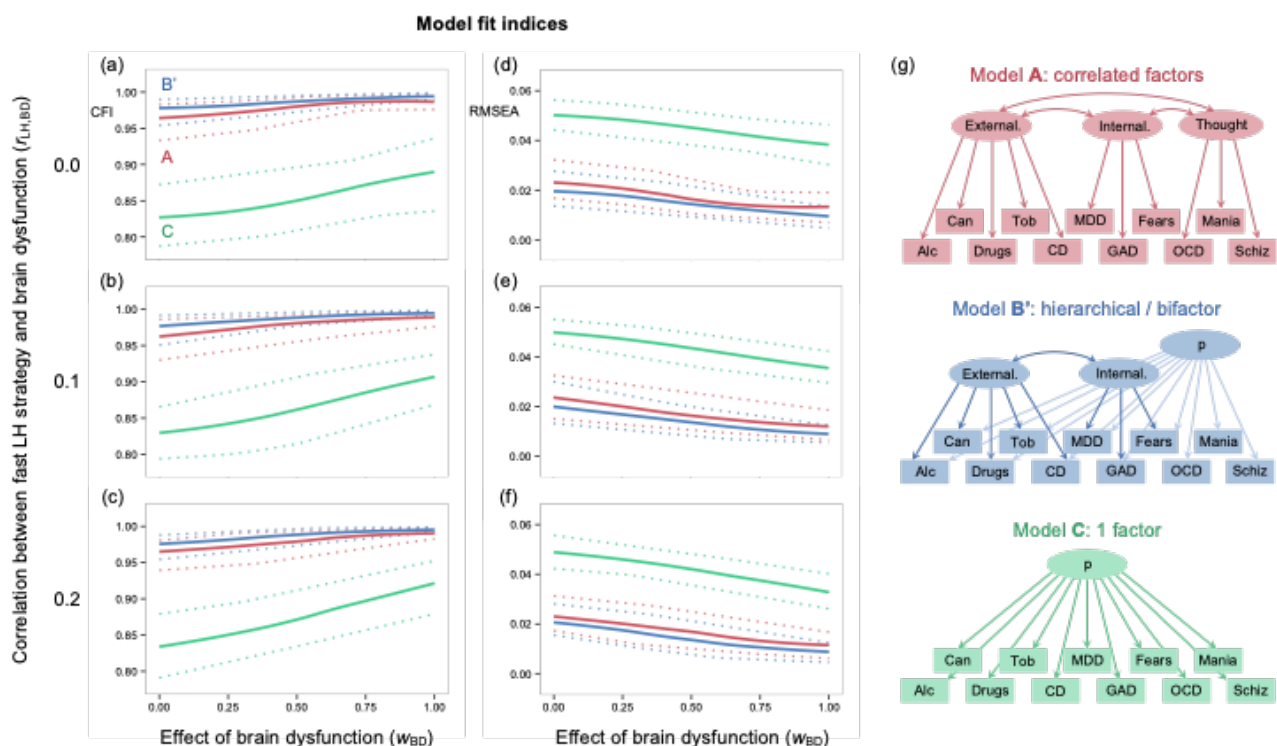
so that the average correlation between symptom scores in simulated matrices was close to .22, thus matching the average size of correlations in the study by Caspi and colleagues (calculated from supplementary tables in Caspi et al., 2014). In all the simulations reported here, disorder-specific variance amounted to three times the joint variance of cluster scores and brain dysfunction scores (see Figure 1).

As a final step, symptom scores were centered and exponentially transformed to mimic the skewed score distributions in Caspi et al.'s (2014) study (bottom right of Figure 1). Parameter  $k$  controlled the strength of the transformation. For example, transforming a normal distribution (skewness = 0) with  $k = 1$  produces a moderate level of skewness (0.7), while  $k = 3$  produces a very high level of skewness (2.7). Exploratory runs showed that simulation results were virtually identical for realistic values of  $k$  between 1 and 3 (see Figures S3-S4 in the Supplemental Material available online). In all the simulations reported here, symptom scores were transformed with  $k = 2$ , corresponding to a skewness of about 1.5 (illustrated in Figure 1).

**Summary.** In total, simulation parameters were selected as follows. The weights determining the effects of life history strategy on psychopathology were derived directly from the life history model. Both  $W_{BD}$  (the effect of brain dysfunction on psychopathology) and  $r_{LH,BD}$  (the correlation between life history strategy and brain dysfunction) were varied systematically over the range of plausible values. The cluster- and a disorder-specific components of symptoms scores ( $V_{C1,...,C6}$  and  $V_{D1,...,D11}$ ) were selected to match the average magnitude of correlations in the original dataset by Caspi and colleagues; neither the relative weight of cluster- vs. disorder-specific variance nor the amount of skewness in symptom scores ( $k$ ) had any substantive effect on the simulation results (as shown in the Supplemental Material available online). All the remaining parameters ( $\lambda_{1,...,12}$ ) were introduced to add “noise” to the simulation, and were randomly varied each time to evaluate the robustness of the results.

## Data Analysis

Each simulated dataset was analyzed by fitting three factor models to the data. These statistical models correspond to those described in Caspi et al. (2014): a *correlated factors* model including externalizing, internalizing, and thought disorder factors (model A); a *modified hierarchical/bifactor* model including the p factor, an externalizing factor, and an internalizing factor (model B’); and a *1-factor* model including only the p factor (model C). The structure of the three models is shown in Figure 2g. Models were fit to the correlation matrix via generalized least squares; the nature of the simulated datasets did not require special procedures for handling missing and/or ordinal data. For each model, the comparative fit index (CFI) and the root mean square error of approximation (RMSEA) were computed as indices of model fit. Standardized loadings and correlations between factors were also calculated. Finally, factor scores were correlated with life history ( $Z_{LH}$ ) and brain dysfunction ( $Z_{BD}$ ).



*Figure 2.* (a-f) Fit indices of the three factor models in the simulated datasets. Solid lines show average values; dotted lines show the 5<sup>th</sup> and 95<sup>th</sup> percentile. (g) Factorial structure of the models (variances and disturbances are omitted for simplicity). LH = life history; Alc = alcohol dependence; Can = cannabis dependence; Drugs = dependence on hard drugs; Tob = Tobacco dependence; CD = conduct disorder; MDD = major depressive disorder; GAD = generalized anxiety disorder; Fears = fears/phobias; OCD = obsessive-compulsive disorder; Schiz = schizophrenia.

## Results

### Model Fit in the Empirical and Simulated Data

In Caspi et al.'s (2014) study, the hierarchical/bifactor model fit the data marginally better than the correlated factors model, and considerably better than the 1-factor model. Fit indices of the three models in the simulated datasets are shown in Figure 2. Across parameter values, the hierarchical/bifactor model (B') consistently fit the data marginally better than the correlated factors model (A) and substantially better than the 1-factor model (C). Specifically, the CFI for model B' was higher than the CFI for model A in 99.7% of the simulated samples, while the RMSEA for model B' was lower than the RMSEA for model A 76.4% of the times. These results align very closely with those reported by Caspi and colleagues. As in the empirical dataset, CFI and RMSEA indicated a good fit for models A and B' and a poor fit for model C. An implication of these results is that the fast-slow continuum reliably generates a p factor in the data, even without the contribution of brain dysfunction (i.e., when  $w_{BD} = 0$ ).

As can be seen in Figures 2a-2f, the strength of the correlation between fast life history and brain dysfunction had little if any effect on the results; however, fit indices systematically improved as the relative effect of brain dysfunction increased. In Caspi et al.'s (2014) analysis, model A had CFI = .962 and RMSEA = .027; model B' had CFI = .966 and RMSEA = .025; and model C had CFI = .875 and RMSEA = .048. Fit indices computed on simulated data were closest to their empirical counterparts for values of  $W_{BD}$  in the low to middle range (about 0.25 to 0.50); in other words, when the effect of life history strategy on psychopathology risk was two to four times as large as that of brain dysfunction.

### Factor Congruence Between the Empirical and Simulated Data

As discussed in the preceding section, the hierarchical/bifactor model (B') showed the best fit in both the simulated and empirical data. To assess the similarity of factorial solutions in the simulated and empirical datasets, the respective factor loadings for model B' were compared with Tucker's coefficient of congruence ( $CC$ ). The  $CC$  is an index of matrix congruence that can be employed to quantify the similarity of two factorial solutions (Abdi, 2007). Conventionally,  $CC > .80$  indicates high similarity, while  $CC > .90$  indicates very high similarity (see Horn, Wanberg, & Appel, 1973; Sakamoto, Kijima, Tomoda, & Kambara, 1998).

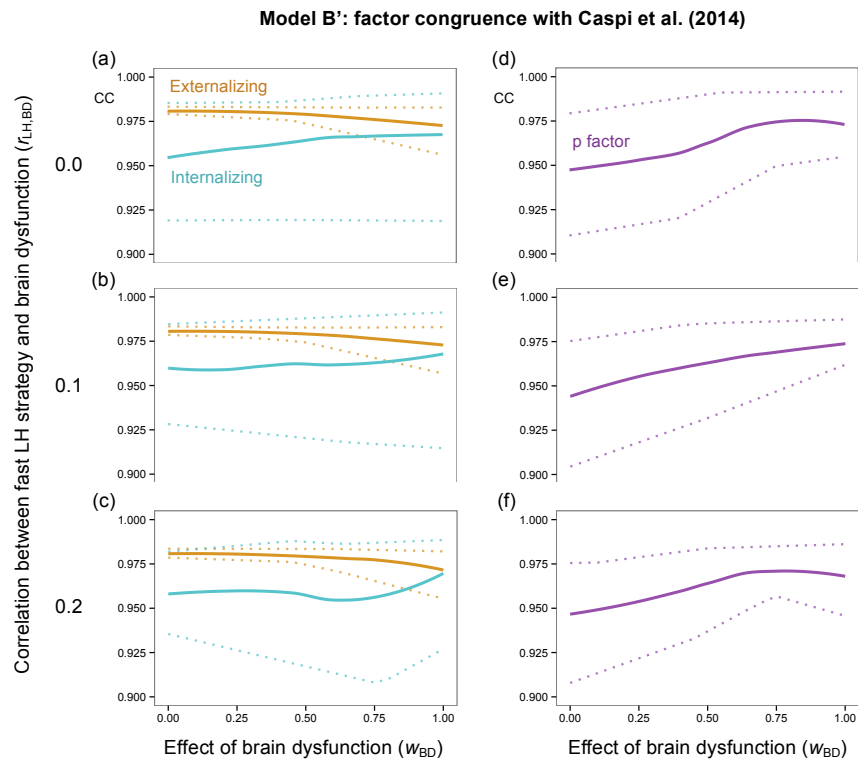


Figure 3. Factor congruence for the hierarchical/bifactor model (B') in the simulated and empirical data (Caspi et al., 2014). Solid lines show average values; dotted lines show the 5<sup>th</sup> and 95<sup>th</sup> percentile. LH = life history.

Congruence values for the externalizing and internalizing factor are shown in Figures 3a-3c, while those for the p factor are shown in Figures 3d-f. Values of  $CC$  were typically higher than .95, with the 5<sup>th</sup> percentile exceeding .90 for all parameter combinations. These values indicate very high similarity between factorial solutions in the simulated and empirical datasets. Factorial solutions were highly similar even without the contribution of brain dysfunction (i.e., when  $W_{BD} = 0$ ), further confirming that the fast-slow continuum is sufficient to reliably generate a p factor in the data. However, factor congruence for the internalizing and p factor tended to increase slightly as the relative effect of brain dysfunction increased. In contrast, the size of the correlation between fast life history and brain dysfunction had little impact on the pattern of results.

### Externalizing-Internalizing Correlations in the Empirical and Simulated Data

In Caspi et al.'s (2014) study, the correlated factors model showed a moderate positive association between the externalizing and internalizing factor, in line with the existing literature. However, when the p factor was included in the hierarchical/bifactor model, the correlation between the externalizing and internalizing factor became negative, suggesting opposite liabilities for externalizing and internalizing disorders. In Del Giudice (2014b), I argued that this surprising finding can be explained by the life history model; specifically, I noted that “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” (p. 402).

Correlations between the externalizing and internalizing factor in the simulated datasets were estimated for the correlated factors model (A) and the hierarchical/bifactor model (B'). As in the empirical study by Caspi and colleagues, the correlation was positive in model A but became negative in model B' (Figures 4a-4c). The positive correlation in model A increased in magnitude as the relative effect of brain dysfunction increased, whereas the magnitude of the correlation between fast life history and brain dysfunction had virtually no impact on the results. As hypothesized in Del Giudice (2014b), after controlling for the general effect of the p factor (model B'), externalizing scores were associated with faster strategies (average  $r = .53$ ), whereas internalizing scores were associated with *slower* strategies (average  $r = -.27$ ).

### The Nature of the p Factor

It is possible to gain further insight in the nature of the p factor by correlating p factor scores with life history scores ( $Z_{LH}$ ) and brain dysfunction scores ( $Z_{BD}$ ), as shown in Figures 4d-4f. As hypothesized, p factor scores were consistently and positively correlated with faster life history strategies ( $r \approx .50$ ). As the effect of brain dysfunction increased, its correlation with the p factor increased as well, reaching a maximum of about .70 for  $W_{BD} = 1$ . In Caspi et al.'s (2014) study, correlations between p factor scores and indices of brain dysfunction ranged from .13 to .33; again, this is consistent with values of  $W_{BD}$  in the low to middle range (see Figures 4d-4f). In total, simulation results match the empirical findings by Caspi and colleagues, and support the contention that the p factor reflects a combination of fast life history strategy and reduced neurological integrity/efficiency (Del Giudice, 2014b).

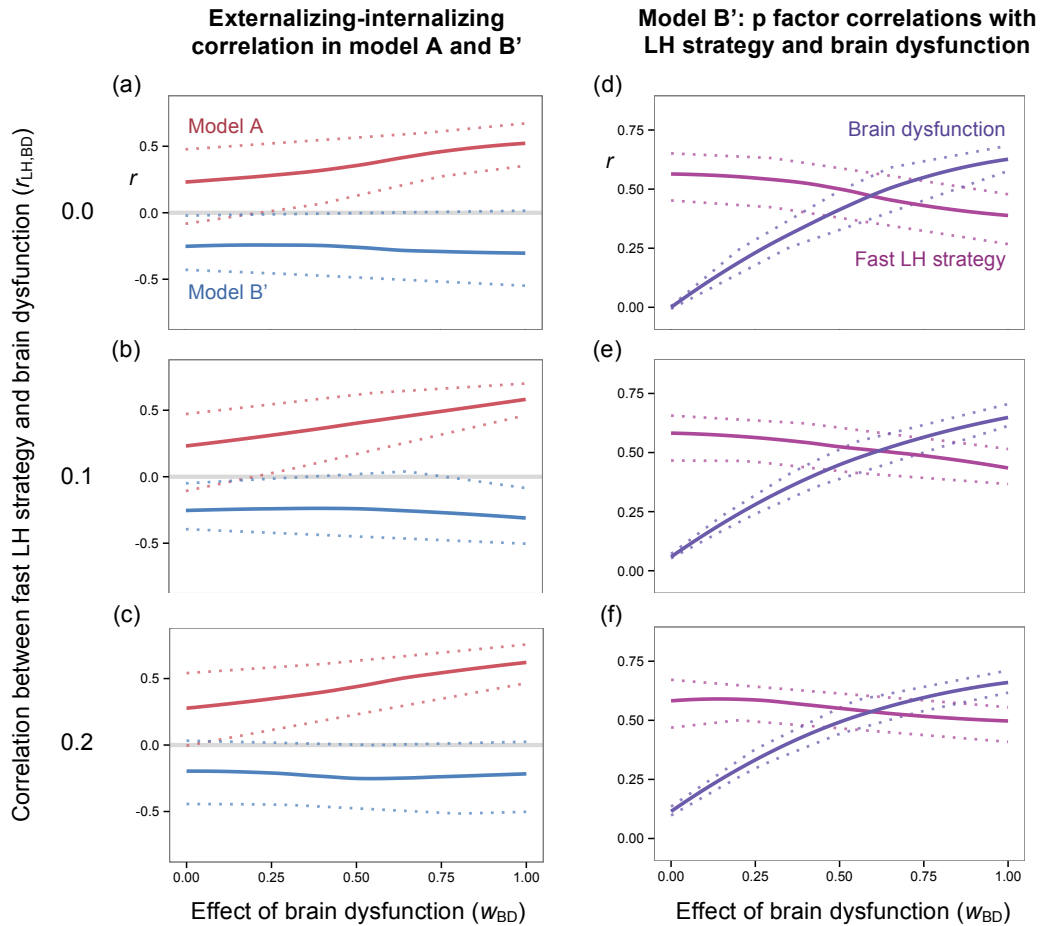


Figure 4. (a-c) Correlation between the externalizing and internalizing factor in the simulated datasets for the correlated factors model (model A) and the hierarchical/bifactor model (model B'). (d-f) Correlations between p factor scores, life history strategy, and brain dysfunction in the simulated datasets for the hierarchical/bifactor model. Solid lines show average values; dotted lines show the 5<sup>th</sup> and 95<sup>th</sup> percentile. LH = life history.

## Discussion

In this study, I employed simulation methods to test the claim that the life history model of psychopathology (Del Giudice, 2014a, 2014b) can explain the empirical structure of comorbidity between psychiatric disorders, as described by Caspi et al. (2014). The simulated datasets reproduced all the relevant findings by Caspi and colleagues; specifically, (a) the data were best accounted for by a hierarchical/bifactor model that included a general p factor, an externalizing factor, and an internalizing factor; (b) the correlation between the externalizing and internalizing factor was positive in the correlated factors model but negative in the hierarchical/bifactor model; and (c) the p factor showed a positive correlation with brain dysfunction when the effects of the latter were included in the model. Moreover, factor loadings

in the simulated data were highly consistent with those observed in the empirical dataset by Caspi et al. (2014). Taken together, these results support the validity of the life history model, and suggest that the p factor of psychopathology may be interpreted as a combination of fast life history strategy and reduced neurological integrity/efficiency (Del Giudice, 2014b).

Of course, simulations can only provide indirect support for a given theoretical model. In this study I showed that the observed structure of psychopathology can be faithfully reproduced by the statistical structure depicted in Figure 1, which in turn was derived from the life history taxonomy of mental disorders advanced in Del Giudice (2014a, 2014b). However, any alternative model described by the same structure would be equally well supported by the present results. Empirically, the interpretation of the p factor as a combination of fast life history and reduced neurological integrity/efficiency (as in Figures 4d-4f) is consistent with findings that higher levels of p are associated with low conscientiousness, low agreeableness, and high levels of early stress—all of which cluster with other fast life history traits (see Del Giudice, 2014a)—as well as multiple indicators of compromised brain integrity such as low IQ, reduced working memory, neurological abnormalities, and damage to the retinal microvasculature (Caspi et al., 2014).

Several aspects of the study are worth commenting on. First of all, the present findings do not rest on the assumption that the original analysis by Caspi et al. (2014) was fully adequate to begin with. For example, Caspi and colleagues allowed the externalizing and internalizing factors to correlate in model B', whereas in standard bifactor models all factors are orthogonal (e.g., Brown, 2015); this may raise questions about the adequacy of these authors' methodology. However, it is important to underscore that the goal of the present study was not to evaluate the validity of Caspi et al.'s (2014) original analysis, but rather to test whether the data generated by the life history model give comparable results when analyzed with the same techniques and models employed in the literature. Second, the simulation was deliberately based on a simplified representation of the life history model, and the values of all the theoretically relevant parameters were randomly varied in each simulated sample. These features of the simulation ensure that model predictions are robust, and that results cannot be explained by overfitting or parameter "tweaking". Third, the fast-slow continuum alone was sufficient to generate the observed structure of psychiatric disorders even without the contribution of a generalized factor of "brain dysfunction" (shorthand for reduced neurological integrity/efficiency), although including a small to moderate effect of brain dysfunction clearly increased the model's performance. While neurological functionality has an important role in the life history framework (see Del Giudice, 2014b), these results underscore the centrality of the fast-slow continuum. Similarly, grouping disorders into clusters provided somewhat increased realism but did not contribute substantially to reproduce the observed structure of psychopathology. In fact, exploratory simulations showed a closer match to the empirical data when the effect of clustering was comparatively *weaker* (compare Figures S1 and S2 in the Supplemental Material available online). Fourth, varying the correlation between life history and brain dysfunction within the range of plausible values (.00 to .20) had little if any effect on the results, and the p factor emerged reliably even when life history and brain dysfunction were entirely uncorrelated.

Finally, for the sake of simplicity the present simulations did not include gender as an additional determinant of psychopathology risk. In the study by Caspi and colleagues (2014), the



p factor did not correlate with gender; however, gender did show significant associations with both the externalizing factor (higher scores in males) and the internalizing factor (higher scores in females). Accordingly, the authors speculated that, net of the p factor, “the externalizing and internalizing components of the structure of psychopathology primarily represent gendered personality styles” (p. 132). The present results suggest that this interpretation is incomplete. Although gender was not included in the model, simulations reliably reproduced a negative correlation between the externalizing and internalizing factor in the hierarchical/bifactor model. Thus, gender differences are not required to explain the observed opposition between internalizing and externalizing symptoms. The life history model predicts that males should be over-represented in fast spectrum disorders characterized by high levels of risk taking (e.g., externalizing disorders); in contrast, females should be more prone to develop both fast and slow spectrum disorders that involve the up-regulation of protective defenses (e.g., anxiety; discussed in Del Giudice, 2014a). According to the life history model, then, the externalizing-internalizing distinction arises from a combination of the fast-slow distinction *and* specific gender differences in psychopathology risk (see also Martel, 2013).

The present study has several limitations that should be addressed by future research. As noted above, the simulation did not include the effect of gender on psychopathology, nor the longitudinal component of psychiatric symptoms. For this reason, the present results are best regarded as a “proof of concept” of the explanatory validity of the life history model rather than a detailed account of the structure of psychopathology. A more fundamental limitation is that simulations can only show that the empirical data are consistent with a certain model; they do not rule out the possibility that other theoretical models may explain the data just as well (or better). Ideally, it would be preferable to directly compare the predictive ability of the life history model with that of one or more competing models. At present, I am not aware of alternative models of the structure of psychopathology that are (a) derived from theoretical principles rather than empirically inducted from epidemiological data, (b) comparably broad in scope (e.g., covering ASD and SSD alongside anxiety and personality disorders), and (c) specified in enough detail to be amenable to a simulation approach like the one employed here. In absence of direct comparative tests, the evidence in favor of the life history model must be regarded as initial and provisional.

The present results have several potential implications for research in psychopathology. A counterintuitive implication is that, (a) if the p factor reflects a combination of fast life history strategy and reduced neurological integrity/efficiency, and (b) if these two dimensions are only modestly correlated with one another, then including more disorders in epidemiological studies should have the paradoxical effect of *weakening* the p factor. This is because putative slow spectrum disorders such as OCPD and ASD are predicted to be *negatively* associated with fast strategies, but *positively* associated with brain dysfunction. When only fast spectrum disorders are considered, the p factor emerges as a unitary dimension in the data (as in the present simulations). However, including slow spectrum disorders can be expected to pull apart the two components of the p factor, thus weakening its coherence as more diagnostic categories are considered.

More generally, the life history model maintains that neither the p factor nor the externalizing-internalizing distinction capture the deep functional structure of psychopathology.

Simulation results neatly illustrate this point. As shown in Figure 1, the data were generated by the interplay between the fast-slow continuum and a brain dysfunction continuum (plus a small amount of clustering between disorders). However, factor analysis of the same data supported the existence of an externalizing factor, an internalizing factor, and a general p factor. Clearly, these factors fail to accurately reflect the *actual* process responsible for generating the data. For example, while the p factor recovered from the simulated data was statistically robust, it actually reflected a mixture of two functionally and statistically distinct components, i.e., fast life history strategies and brain dysfunction.

In light of these findings, it is worth considering the possibility that current descriptions of the structure of psychiatric disorders fail to “carve nature at its joints”. The limitations of standard psychiatric taxonomies are well known; indeed, the present generation of descriptive models (e.g., Caspi et al., 2014; Lahey et al., 2011; Watson et al., 2008) stems from an attempt to overcome the artificiality of standard distinctions (e.g., that between mood and anxiety disorders) by replacing them with empirically supported ones, such as that between externalizing and internalizing disorders. However, the present results suggest that empirical studies may be missing the true functional relations that underlie comorbidity patterns. A life history approach suggests that researchers should aim directly at life history strategy and brain dysfunction as the fundamental dimensions of psychopathology structure. This would necessitate moving beyond standard diagnostic categories and starting to look for functional subtypes of common disorders. In this regard, the functional, top-down approach of the life history model may prove a useful complement to the mechanistic, bottom-up approach embodied by the Research Domain Criteria (RDoC) promoted by the National Institute of Mental Health (Insel et al., 2010; discussed in Del Giudice, 2014b).

In conclusion, it is worth underscoring that the life history framework of psychopathology is a work in progress. Despite its theoretical and heuristic value, the fast-slow distinction is only a first step toward a comprehensive evolutionary theory of mental disorders (for extended discussion see Del Giudice, 2014b). Accordingly, the life history model of psychopathology is still provisional and incomplete in many respects. At the same time, the model is already powerful enough to explain the main features of the structure of psychiatric disorders. Future extensions and revisions of the model should further improve its predictive ability, for example by including the longitudinal component of comorbidity and clarifying the functional structure of complex diagnostic categories such as ASD and SSD. I hope that the present study will encourage investigators to consider the benefits of an evolutionary approach, and integrate the concepts of the life history framework in their own research.

### **Author Contributions**

M. Del Giudice created the study concept and design, performed the simulations and data analysis, and wrote the manuscript.

### **Acknowledgments**

I warmly thank Steve Gangestad and Ron Yeo for their advice and feedback.

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